FORMAL BIOLOGY AND COMPOSITIONAL BIOLOGY AS TWO KINDS OF BIOLOGICAL THEORIZING

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This Dissertation is Dedicated to Three People:

Mine Forældre,
Grethe Grønfeldt Winther og Aage Bisgaard Winther

and the best of friends and, if I may, my bara bhai,
Amir Najmi

Les agradezco todo su apoyo. Gracias.

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There are two fundamentally distinct kinds of biological theorizing. "Formal biology" focuses on the relations, captured in formal laws, among mathematically abstracted properties of abstract objects. Population genetics and theoretical mathematical ecology, which are cases of formal biology, thus share methods and goals with theoretical physics. "Compositional biology," on the other hand, is concerned with articulating the concrete structure, mechanisms, and function, through developmental and evolutionary time, of material parts and wholes. Molecular genetics, biochemistry, developmental biology, and physiology, which are examples of compositional biology, are in serious need of philosophical attention. For example, the very concept of a "part" is understudied in both philosophy of biology and philosophy of science.

My dissertation is an attempt to clarify the distinction between formal biology and compositional biology and, in so doing, provide a clear philosophical analysis, with case studies, of compositional biology. Given the social, economic, and medical importance of compositional biology, understanding it is urgent. For my investigation, I draw on the philosophical fields of metaphysics and epistemology, as well as philosophy of biology and philosophy of science. I suggest new ways of thinking about some classic philosophy of science issues, such as modeling, laws of nature, abstraction, explanation, and confirmation. I hint at the relevance of my study of two kinds of biological theorizing to debates concerning the disunity of science.

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Introduction

The fundamental point of this dissertation is to construct, explore, and defend a fundamental difference between two kinds of theorizing in biology: formal and compositional. Briefly, formal biology focuses on finding relations, often captured in the form of laws, among mathematically abstracted properties of abstract objects. These laws both serve as the foundation for, and are extracted from, the mathematical models characteristic of formal biology (e.g., mathematical evolutionary genetics and theoretical mathematical ecology). Note that by laws here I do not mean sentences written in first-order logic (or, more generally, metamathematics), but rather claims expressed in mathematics, as is the case in actual scientific practice.

In contrast to formal biology, compositional biology includes areas of biology, such as molecular genetics, biochemistry, developmental biology, and physiology that are concerned with articulating the concrete structure, mechanisms, and function, through developmental and evolutionary time, of material parts and wholes. Compositional biology is in serious need of philosophical attention; for example, the very concept of "part" is understudied in both philosophy of biology and philosophy of science. By analyzing this form of biology, I hope to bring forward a set of new possibilities and pictures regarding abstraction, part organization, models and modeling, explanation, and confirmation.

Another way to motivate the distinction between formal and compositional biology is to note that focusing on "parts" might seem quaint,

irrelevant, and even bizarre to most philosophers of biology and philosophers of science. The concrete nature and theoretical role of parts are rarely considered topics worthy of *direct* analysis. It is remarkable that so little philosophical attention has been given to parts, considering the ubiquity of part organization in the subject matter of biology. Most research in biological science concerns relations of *compositionality* rather than of *abstraction*, whereas the inverse is the case in the investigations of philosophy of biology and philosophy of science. The central relation between the one and the many in compositional biology is one of object compositionality through the aggregation (in the broadest sense of the term) of many parts in order to produce one whole. In contrast, in formal biology, the central relation between the one and the many is one of object abstraction through the removal of many, and the concomitant focus on a few, properties (often mathematical), in order to produce one kind from many instances. The concern of this dissertation is compositionality, a topic not previously explored in a systematic way in philosophy of biology.

My distinction between two kinds of biology is meant to capture differences in theoretical or empirical *methodology* more than differences in *ontological domains of study*. That is, both kinds of biology could conceivably, and in some cases do, focus on the same domain and even ask similar kinds of questions, but bring different conceptual and material *tools* to the analysis. In some areas, such as the field(s) studying the relation between evolution and development, a process, such as the evolution of multicellularity, can be analyzed from the perspective of either kind of biology even if particular investigators often adhere to one form of biology over the other. Having said this, certainly there is a strong correlation between ontological domain *and*

methodology for many, if not most, other areas. For example, the study of evolutionary changes in gene frequencies is formal whereas the investigation of physiology is compositional. Regardless of the actual relationship between ontological domain and methodology in any given area, I make the distinction between the two kinds of biology based primarily on their respective theoretical and empirical biases and practices, rather than the biological domains that they study.

Before I turn to a detailed explication of each chapter, let me provide a brief account of each chapter followed by an explanation of the logic of the dissertation organization. In Chapter 1, I provide a philosophical framework that grounds, and allows for the diagnosis of, the two kinds of biology. This framework borrows heavily from literature in metaphysics and epistemology; it also employs theoretical physics as both a useful "sister group" to formal biology, and as a contrast to compositional biology. In Chapter 2, I develop the notion of a theoretical perspective, which I use for the remainder of the dissertation. In this chapter, I utilize that notion to understand, in detail, the varied disciplines of compositional biology, such as functional morphology, developmental biology, and biochemistry. In Chapter 3, I explore a contemporary set of debates found in the disciplines attempting to synthesize evolutionary and developmental phenomena. This is an area, with historical roots going back at least to Darwin, where the two kinds of biology meet and, potentially, clash.

In the remaining two chapters, I explore, in the context of my distinction, two classic topics in philosophy of science, models (and modeling) and explanation. In Chapter 4, I articulate four general properties of models and then show how the primarily non-mathematical models of compositional biology do

indeed meet these properties. In Chapter 5, I contrast the very different explanatory patterns that formal and compositional biology exhibit. The pattern present in formal biology is *much* more familiar to philosophers of biology and philosophers of science than the one present in compositional biology.

I have chosen this organization to try to achieve two goals. First, I want to show that distinct areas of philosophy, including metaphysics and epistemology, as well as philosophy of science, can be used to analyze and clarify biological practice, empirical and theoretical alike. It is not always clear that especially metaphysics and epistemology can be made relevant to an analysis of the practices of fields as important to contemporary society as developmental biology, biochemistry, and molecular genetics (all cases of compositional biology). I hope to show especially in Chapters 1, 4, and 5 that philosophy can be made relevant. Chapter 1 uses tools from metaphysics and epistemology. Chapters 4 and 5 employ methods and frameworks from philosophy of biology and philosophy of science, and are also a plea for using compositional biology to develop a new picture regarding abstraction, models and modeling, explanation, and confirmation in order to expand philosophy of science.

The second goal is to analyze compositional biology from many different angles in order to clearly explicate *what* it is. After clearly distinguishing it from formal biology in the first chapter, I provide, in Chapter 2, an in-depth analysis of a variety of different disciplines, and theoretical perspectives, in compositional biology. I continue this detailed analysis in Chapter 4, where I analyze different models and modeling techniques from different compositional biological sciences. In contrast, as I do in Chapter 1, Chapters 3 and 5 are balanced in terms of the attention given to both formal biology and compositional biology. The

odd-numbered chapters thus provide a comparative context in which to comprehend compositional biology. I hope that the dissertation provides a broad and robust understanding of compositional biology.

I will now turn to a summary of the content of each chapter.

In Chapter 1, I develop a framework of philosophical distinctions, regarding four theoretical commitments, that can be applied to differentiate the two kinds of biology. These are: (1) the assumed causal structure of the objects studied, (2) the way abstraction of objects into kinds is made, (3) the model types employed, and (4) the relevance and nature of part organization. The basic pattern of commitments in formal biology is to assume and employ: (1F) simple objects consisting of a few idealized causal factors, (2F) simple abstraction, which is premised on necessary and sufficient conditions (I also call this "Locke-(C.I.) Lewis" abstraction), (3F) mathematical models, and (4F) irrelevant or highly idealized part organization. The basic pattern of commitments in compositional biology is to focus on: (1C) complex objects that are themselves parts of larger wholes, which have many kinds of causal factors, (2C) complex abstraction, which is patchy and messy (I also call this "Wittgenstein-Boyd" abstraction), (3C) propositional non-mathematical models and material models, and (4C) the hierarchical and integrated organization and functioning of concrete biological systems. These are all reasonable commitments for the kind of scientific work each of the two kinds of biology have defined for themselves historically, socially, and philosophically.

In Chapter 2, I expand the notion of "theoretical perspectives" in order to provide a conceptual tool for analysis of the different ways that distinct disciplines of compositional biology *partition* a system, especially the organism. I

compare and contrast the views of Kauffman, Wimsatt, and Griesemer on theoretical perspectives, and develop my own position. In particular, I argue that a perspective could be thought of either as a collection of *all* the activity related to it, or as the set of biases and assumptions *guiding* this activity. I defend and employ the view that for purposes of philosophical analysis, the latter is the appropriate understanding. I also develop the idea of a theoretical perspective's "partitioning frame," which is the set of biases and assumptions guiding the ways in which the perspective identifies and individuates parts. I look to the canonical textbooks of different disciplines of compositional biology to explicate the partitioning frame of that particular theoretical perspective (e.g., physiology or developmental biology). Each of these fields partitions the same "object" (e.g., the organism) differently through their respective employment of a specific partitioning frame. Formal biology does not work in this way. I summarize my analysis with a table presenting "examples of parts," "criteria of the partitioning frame," and "general list of guiding biases" for each theoretical perspective of compositional biology analyzed.

In Chapter 3, I address, through a case study, the possibility of synthesis and unification between the two kinds of biology. The relationships between the phenomena of, and theories about, development and evolution remain mysterious. The current avatar of this relationship is the one between the disciplines of levels of selection theory and evolutionary developmental biology. By explicating a hierarchical view of theoretical perspectives, I show that levels of selection theory is an example of both formal biology and the "competition perspective," whereas evolutionary developmental biology is a case of both compositional biology and the "integration perspective." In both cases, these

concrete fields "inherit" the guiding biases and assumptions of the higher-level perspectives in addition to adding their own. I then elaborate, using key research papers and books as well as communications with scientists, the respective theories and experiments of these two fields for both the organism and social insect colonies, which are objects at two different levels of the biological hierarchy. It becomes clear that the two fields are methodologically different, even when studying the same objects. I also show the difference between the two fields by developing, conceptually, other aspects of a theoretical perspective, such as explanatory resources employed, and the meaning of key terms. (Providing a full conceptual anatomy of a theoretical perspective is one of my future projects.) In concluding the chapter, I elaborate on various interpretations regarding the relationships between the two fields. In doing this, I evaluate the prospects of a synthesis between the two, without arriving at a firm conclusion.

In Chapter 4, I address the issue of models and modeling in biology. I review two different analyses of models and modeling, the semantic view of theories and the mediating model view. I also consider two non-standard views, Griesemer's and Downes' respective expansive and deflationary accounts. In addition, I present two very distinct views on abstraction stemming from Cartwright and Friedman. Inspired by these varied presentations on models and modeling as well as on abstraction, I propose four general properties for models: (1) models are meaning structures of various types and at various levels of abstraction, (2) models can be "hooked up" with other models, subject to the background protocols and assumptions of the theoretical perspectives guiding such articulation of models, (3) models in biology are subject to trade-offs among pragmatic desiderata – for example a general model is rarely a realistic one, and

(4) models are used in a variety of scientific activities, including guiding further modeling and empirical activity, as well as providing explanations.

For the rest of Chapter 4, I show how different models of compositional biology meet these properties, and should therefore be considered proper models. However, this does not imply that modeling, as a theoretical activity, is as prevalent in compositional biology as it is in formal biology. In fact, I argue that while models are prevalent in compositional biology, modeling *independent* of empirical activity is rare. The picture of the activity of modeling, as well as of explanation stemming from models and the confirmation of models, is *very* different in compositional biology as compared to *all* the standard philosophy of science analyses of modeling, which are pertinent *only* to formal biology. It is to a contrast of these two pictures that I turn in Chapter 5.

In Chapter 5, I distinguish the very different ways in which the two kinds of biology produce explanations. Despite the presence of models in both kinds of biology, the forms of modeling and explanation are distinct. I present five differences. (1) The model meaning structures in formal biology are formal law-like relations between abstract mathematical properties, whereas in compositional biology they are presentations of the compositional and functional (sensu Cummins) organization of the system. (2) In formal biology, modeling activity and empirical activity are easy to differentiate, but they are difficult to distinguish in compositional biology. (3) Model articulation (theoretical explanation) in formal biology is a mathematically abstract activity independent of empirical evaluation, as described, for example, in Friedman's account of theoretical unification, whereas in compositional biology, model articulation is intimately tied to empirical activity, including the practice of model evaluation.

(4) Model application (causal explanation) works similarly in the two kinds of biology in so far as a general model is applied to a particular concrete system to explain aspects of it; however, in formal biology, formal causal relations are explained, as detailed in Cartwright's work, whereas in compositional biology, compositional and functional relations are explained, as presented in Cummins' analysis. (5) Model evaluation (confirmation) in formal biology follows protocols explicated by the semantic view (e.g., goodness-of-fit tests), whereas in compositional biology methods of "qualitative" confirmation have not yet been developed. Given the close relationship between theoretical and empirical activity in compositional biology, however, it seems clear that (dis)confirmation of a model can easily lead to straightforward changes in the meaning structures; this contrasts with formal biology, where assumptions as well as the functional mathematical relations between variables often have to be fundamentally revised in response to model disconfirmation.

This dissertation, thus, seeks to provide a framework that will allow us to begin to philosophically understand extremely important understudied biological sciences. Given the social, economic, and medical urgency of many compositional biological sciences, we could fruitfully spend significantly more effort understanding them. We lose relevance as well as insight by focusing on biological sciences rich in mathematical theoretical structure. In articulating my conceptual analysis, I hope to have shown that different areas of philosophy can be made applicable and relevant. In addition, as a feedback effect—science to philosophy, rather than philosophy to science—I believe that a study of compositional biology will lay the grounds for a clear expansion, in philosophy of science, of our picture of the development, structure, and application of both

theory and models. It is time to move beyond the picture provided by philosophers of science focusing on abstractive and formalistic science, as useful as their efforts have been.

Chapter 1: A Philosophical Framework Grounding the Distinction Between Formal and Compositional Biology

1.1 Framing and Goals

In this chapter I provide a philosophical framework, using tools from metaphysics and epistemology, that will allow us to establish and characterize a heretofore unrecognized difference between formal and compositional biology. Formal biology focuses on the relations, governed by formal laws, among the mathematically-abstracted properties of abstract objects. Particular kinds of mathematical models capture these relations, as we shall see. Formal biology, including especially mathematical evolutionary genetics and theoretical mathematical ecology, shares similarities with theoretical physics and, therefore, I will often, for convenience, employ examples from theoretical physics to help characterize the features it has in common with formal biology.

Conversely, compositional biology, which includes areas of biology, such as molecular genetics, biochemistry, developmental biology, physiology, and systematics, is concerned with articulating the concrete structure, mechanisms, and function, current as well as through developmental and evolutionary time, of material parts and wholes. Although compositional biology does seek generalizations, it shies away from laws of the abstractly formal – God is in the details, the Devil in the abstractions. Both sciences seek generalizations, but, as

we shall see, they achieve it in different ways and the extent and type of generalization is quite different in each.

The difference between these two kinds of biology is captured in my framework articulating four distinctions (pertinent to four abstract commitments made in each kind of biology)[1]: (1) simple vs. complex objects (the assumed causal structure of the objects studied), (2) simple vs. complex abstraction (the form of abstraction of objects into kinds), (3) mathematical vs. propositional non-mathematical or material models (the prevalent types of models employed), (4) irrelevance or idealized vs. relevance of part-whole organization[2] (relevance and nature of compositional organization). This chapter is arranged around an explication of each of these four distinctions. For each distinction, I first provide a *general* account of what is involved in making that commitment in the first place (e.g., assumed causal structure of the objects studied). I then present how formal and compositional biology make *distinct*, and *opposing*, *assumptions* regarding that particular commitment (e.g., simple vs. complex objects).

Note that these distinctions have their sequential arrangement for a reason. The first three concern kinds and abstraction. Although the first is also an ontological distinction, the ontology is conditioned by the theory studying those objects. The last concerns mereological[3] relations and patterns, which, as I argue below, is a distinct issue from abstraction relations and patterns. In articulating these four distinctions, note also that I am explicitly naturalizing my analysis in

[1] In Chapter 5, I will add a fifth distinction, concerning pattern of scientific explanation, to this list. In the current chapter I do not further develop this distinction since it is a very complex one requiring significant explication.
[2] "Organization" is here shorthand for structure, process, and function.
[3] "Mereology" is the philosophical study of parts and wholes. It is a little-known subfield of philosophy.

what the scientific theories actually endorse. That is, I use philosophy to explicate science, but science provides the data for this analysis.

1.2 Simple versus Complex Objects

The first distinction regards the causal structure of the concrete objects investigated by a science. First, given the naturalistic proclivities of my analysis, I want to emphasize that it is to the *scientific theory*[4] that we must turn if we want to learn what the pertinent *types of* causal factors are. In general, I consider types of causal factor as types of interactions that an object can engage in; a particular causal factor makes a difference to a particular outcome.[5]

Simplicity and complexity of the causal structure of the objects, I claim, has to do with the neatness[6] and number of causal factors[7] involved in (1)

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[4] In Chapter 2, I develop in detail the concept of "theoretical perspective," which is a coherent and integrated unit of scientific theory (at various levels of generality), which, at its lowest level of generality, is associated with a discipline, such as mathematical evolutionary genetics (formal biology) or developmental biology (compositional biology).

[5] The literature on causation is quite complex, yet there is little material on how to *individuate* a causal factor, or even a type of causal factor. Often the division of processes into types of causal factors (and types of outcomes) is taken for granted. I believe that turning to the scientific theory is of help here. Furthermore, some of the literature has defined causal factors generally as a "difference maker" and I believe this to be a workable definition of what a causal factor does in a particular case even if it does not tell us what the types of factors are appealed to by the theory (Cartwright 1989; Jennings 1913; Mackie 1974; Mill 1904; Strevens 2004 in press).

[6] By "neatness" and "neat" I mean something like clearly articulated theoretically and with little variation in causal effect within types of causal factors. For example, we know how to clearly relate, mathematically, fitness to change in gene frequency. But the mechanisms of cell signaling are incredibly diverse, and output type (i.e., what the cell does in response) varies tremendously among different cell types, different cell signaling structures, and different taxa. There is

forming an object and (2) the continued interaction of the object with other objects, of the same or of different kinds, around it. For simple objects, there are a small number of relatively neat causal factors that interact to produce *reliably equivalent* concrete objects with reliably repeated causal capacities[8]. Examples of this are electrons, chemical elements, and, I argue below, the genes of mathematical evolutionary genetics. For complex objects, there are a large number of relatively messy causal factors that form diverse concrete objects different from one another and with somewhat different causal capacities, even if such objects share aspects of their history and capacities. Examples of this are livers of different Homo sapiens organisms, or even homologous genes in different taxa.

The distinction between simple and complex objects is meant to capture the equivalence *or* diversity of objects produced from, or engaged with, causal factors of varying number and neatness. Thus simple objects are equivalent in terms of their (1) history – the same forces of oxygen and hydrogen electron shell dynamics and structure form any molecule of water, and (2) causal capacity – under given conditions, any electron will behave in the same way as any other electron. Complex objects of a type are unique in terms of their (1) historical diversity – livers in different humans develop in slightly different ways, sometimes even qualitatively so, leading to morphological or physiological "abnormalities", and (2) causal capacity diversity – livers in different humans,

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no clear theoretical relation between, for example, particular cell signaling structure and outcome.

[7] Unless otherwise noted, "causal factors" is equivalent to "types of causal factors."

[8] I shall explore the notion of "causal capacity" in detail in Chapter 5; it is a concept interchangeable with "type of causal factor."

and certainly in related species, have different levels and even kinds of functioning.

Another sense in which objects are simple or complex has to do with the *total* number of causal factors available in a theory. Biological sciences studying simple objects tend to have few total numbers of causal factors, whereas those studying complex objects, tend to have many total numbers of causal factors. In mathematical evolutionary genetics, as we shall see, there are relatively few forces theoretically ascribed to the central objects, genes. These are selection/fitness, drift, mutation, and population structure, which introduces levels of selection concerns, as we shall see in Chapter 3. In developmental biology there are many causal factors, such as signaling, movement, and death among cells, that can exist at various levels, including molecular, cellular, and organ.

Furthermore, even if a particular object in developmental biology, an example of compositional biology, is ascribed few causal factors (which is a possibility even if there are *many* total causal factors), making it somewhat simple, that object is analyzed from a variety of *other* theories that ascribe their own causal factors. For example, when physiology or functional morphology, as we shall see in Chapter 2, import their own causal structure, the object becomes much more complex from this "inter-theoretical" viewpoint. The variety of compatible theories present in compositional biology, but not in formal biology, adds another layer of complexity to the causal structure of the objects it studies.

Now that I have explicated causal complexity generally in terms of the messiness and high number of (types of) causal factors both within and across theories investigating a particular object, let me turn to an application of this

description to the two kinds of biology, using examples from chemistry and theoretical physics, when applicable to formal biology.

Formal biology focuses on simple objects. These are the objects in nature for which few and neat causal forces are involved in their origin and their subsequent interactions. Examples include most of the objects of physics: masses, electrons, photons, atoms, populations of particles in a gas. These are all objects for which a few precise essential properties can be captured. Useful theory can be produced for, say, any and every electron – they are all equivalent to one another. Furthermore, despite the fact that, for example, photons come in many frequencies, they are quantized and we can come up with very abstract and general descriptions of them that apply to all of them (e.g., Maxwell's laws). Even populations of particles in a gas are subject to relatively few and well-understood forces.[9] All these objects are excellent candidates for a neat abstraction hierarchy with necessary and sufficient conditions, as we shall see in the section 3 of this chapter. Furthermore, there is roughly only one theory applicable and relevant to each. This is not to say that these objects are perfectly simple objects. There are degrees even here – numerous gases and numerous conditions are such that the ideal gas law does not hold, but such gases are still simple objects in that they are chemical compounds of a certain kind and, at least under some interpretations of physics, highly abstract laws are powerful explanatory devices of the causal structure of objects.

Biology, in general, suggests a very different ontology from theoretical physics or the chemistry of elements and simple compounds. First of all, the

[9] And this is precisely how R. A. Fisher modeled genes and their causal forces in panmictic populations. (Michael Wade reminded me of this point.)

concrete objects of biology can often be interpreted and manipulated from a broad multiplicity of theories. Unlike many concrete simple objects in physics, the concrete object "gene" can be described from a wide variety of perspectives.[10] Clearly there are multiple causes, even multiple kinds of causes from different theories,[11] for the origin and interaction of genes. But let us focus on the genes of mathematical evolutionary genetics. Let us focus on the practice, and ontology implied, by this powerful discipline.

Unlike many other areas of biology, and even other areas of the physical sciences, mathematical evolutionary genetics, a paradigm case of formal biology, is highly driven by theoretical concerns and mathematical models.[12] In such a (mathematically) abstract discipline, it is admittedly difficult, if not paradoxical, to articulate what their ontology is. What do they believe about the "objects" they study—are these objects simple or complex objects—if confirmation of their models is relatively rare and, more important, difficult? So there is an element of *indeterminateness* in their ontological position stemming from the lack of concretization of their postulated objects.

In so far as we *can* discuss their ontological position, mathematical evolutionary genetics refers to objects that are both more complex (i.e., more

[10] In theoretical physics, there are, of course, exceptions to this point – the wave-particle duality of quantum mechanics is one such case. (Wade pers. comm.) I want to note that it is certainly beyond the scope of this dissertation to develop the ontology of theoretical physics. I rely on intuitions regarding physics in order to motivate the distinction between formal and compositional biology.
[11] The overlap and synthesis of these perspectives, as well as the different kinds of causes they refer to, is conceptually unclear and will be one of the central concerns of Chapter 3, where I discuss evolutionary developmental biology and levels of selection theory.
[12] See, e.g., Lewontin 1974. The emphasis on mathematical modeling in this field accounts, at least in part, for the relative ease with which anti-realist, or "realism neutral," interpretations can be provided for it; see, e.g., Lloyd 1988 and, for a different view on the same issue, Rosenberg 1994.

variation, less equivalent, less neat causal factors) than the simple objects of theoretical physics, yet also simpler, and more rigid, than the complex and qualitatively variable (types of) complex objects of compositional biological sciences, such as developmental biology and physiology. Put more strongly, the genes of mathematical evolutionary genetics, while not simple and not caused, or interacting, by *extremely* few and neat causal forces, do have much more in common with the point masses of Newtonian mechanics than they do with the livers of physiology or even the concrete regulating genes of developmental biology. This might seem surprising to those of us trained to view large-scaled scientific differences in terms of content-matter rather than in terms of practice or styles of analysis, both of which are included in scientific theory. Let me specify my argument more clearly.

Mathematical evolutionary genetics typically postulates a handful of causal factors: (1) selection, (2) mutation, (3) genetic drift and (4) population structure, which raises concerns regarding inbreeding and levels of selection. These factors are clearly defined mathematically and their consequences on gene frequency well understood. As long as alleles at particular loci are subject to the same forces[13] under the same idealizing assumptions, then the same consequences will occur, whether they are loci "for" an enzyme involved in the breakdown of an amino acid or a gene regulating protein. There is no variation in effect from the causal force point of view, even though, of course, the alleles, whether at the same or at different loci, vary in nucleotide sequence.

[13] On evolutionary causal factors as "forces" see Sober 1984. Note that the analogy between Newtonian mechanics and evolutionary genetics is explicit here. I discuss the presence of forces and laws in mathematical evolutionary genetics in Appendix A.

Mathematical evolutionary genetics postulates simple objects: a few clearly-defined and neat causal factors (of particular types, described by the "forces of evolution") constitute the full suite that change gene frequencies, regardless of the actual role of the genes in concrete biological processes. Simple objects are subject to equivalent consequences determined by few and well-defined causal factors.

The causal structure of the objects of fields such as developmental biology and physiology is certainly not indeterminate since the focus of these fields is on the structure, process, and function of *concrete* objects. These objects are clearly complex objects and are parts of a system. First of all, there is clear variation in the objects of the same kind, both in terms of the causal forces involved in their history, in their causal capacities, and in their very descriptive attributes (e.g., liver size, function, cell number). And there is no short list of mathematical causal forces involved. The list of causal factors for these sciences is (indefinitely) long and the factors are qualitative and not amenable to parameterization. Furthermore, there are a variety of theories from which to interpret, say, a kidney: comparative morphology, physiology, and developmental biology (as we shall see in Chapter 2). These theories have their own unique commitments to causal structure. The causal structure of the objects of compositional biology is detailed, qualitative and diverse – in short, complex.

Formal biology (e.g., evolutionary mathematical genetics) postulates concrete objects that are simple objects, subject to few well-defined causal forces leading to equivalent outcomes. Admittedly, these are not as simple and variation-free as the elements of chemistry or the fundamental particles of theoretical physics, nor are they concrete in the same way, but the model-based

practice of evolutionary genetics does suggest objects with the same sorts of properties as those of formal physical science. Compositional biology (e.g., developmental biology, physiology, biochemistry, even systematics) actively investigates concrete objects that are quite complex and are *parts* of larger systems. God (i.e., that which is desired and exalted in the field) is in the complex details here – that is the causal structure of the objects studied. The active investigation of the different and unique, of complex objects that stick out from their peers of the same kind, (e.g., the homeotic mutant to determine the effect of regulating genes or the genetic knockout to determine steps in biochemical pathways) is rewarded. These two kinds of biology display very different commitments to the causal structure of the objects that they study.

1.3 Simple versus Complex Abstraction

The second distinction concerns our categorization of concrete objects in the world into, what I will call, "theoretical kinds" at ever-increasing levels of generality. Here the important criterion of categorization is *judged similarity of objects*. When objects are judged similar in many important respects, then they are considered instances of the same theoretical kind.

In this section, I motivate my account of abstraction by first describing the tool, intensional sets with properties. I defend my tool by placing it in the context of some philosophical theories, many of which are explicitly or implicitly compatible with my account of abstraction. I then articulate *how* my account

works both in formal and compositional biology both in general and with examples.

In analytical philosophy, there is a classic distinction between intension and extension.[14] The intension of a term is the set of properties or sentences providing the "meaning" that is used to identify the concrete objects to which the term, or kind, pertains. I argue for a general account of intensional sets in which they contain the properties (or "predicates") used to define the term or kind. It is difficult to atomize and count properties and predicates, but again, following my naturalistic analysis, I place the brunt of the identification work on the scientific theory and its choice of properties (including, for example, causal factors).[15] The extension of a term consists of all the concrete objects referred to by the term. The smaller the intensional set, and thereby the larger the extension (since more objects are picked out under less constrained intensions), the more abstract the theoretical kind. Thus two theoretical kinds, one more abstract, and sometimes including, the more concrete one, have different relative sizes of intensional sets (former smaller than latter) and extensions (former larger than latter).

Intensional sets provide the means for diagnosis and recognition of theoretical kinds in this world, as the traditional theory of meaning also holds. The intensional set then determines the appropriate reference and extension of

[14] E.g., Lewis 1946, p. 39. In a very similar distinction, Frege distinguished between two senses of meaning: connotation and denotation.
[15] Furthermore, many philosophers of science also do not have good *a priori* accounts of property identification. For example, Cartwright 1989 simply assumes, as we shall see below, that abstract categories contain fewer properties or Aristotelian causes than more concrete categories. Furthermore, van Fraassen 1967, while discussing meaning relations among predicates, does not anywhere provide an account of predicate identification. Given the naturalistic and empiricist tendencies of these two philosophers, I believe that they too would turn to the scientific theory for providing an anatomy of the pertinent property structure.

the term. There are a variety of views under this traditional theory ranging from the idea of "necessary and sufficient conditions" for adequate reference of an object by a term, advocated by philosophers like John Locke and C. I. Lewis, to the idea, defended by Wittgenstein and Boyd that terms have neither necessary conditions, nor sets of sufficient conditions, to refer correctly to an object.[16]

Before I further explicate this traditional theory of meaning, I want to contrast it with what another theory of meaning, the Kripke-Putnam causal theory of reference.[17] This view holds that terms function more like proper names that rigidly designate a single individual (e.g., "Aristotle," "Ayers Rock"[18]) than like natural kind terms where degree of similarity in properties among objects is used to appropriately class the objects (the traditional theory of meaning). Under the Kripke-Putnam view, a single baptismal event ("water *is* what I have here, H_2O") determines the subsequent appropriate usage of that term. They emphasize the baptismal event and subsequent historical chain of usage of the term and leave out the epistemic events of judging particular objects as instances (or not) of particular kinds.

Since Kripke and Putnam use *proper names* as their model for terms (which are, by definition, for *one* object – the individual), rather than *kinds* (of *many* objects), their account does not handle terms well that refer to kinds of objects. For kinds, *epistemic acts of judgment* of similarity and instantiation determining adequacy of reference to an object are absolutely crucial. That is, the historical chain of term use is not sufficient. We still have to determine whether a

[16] Boyd 1991, 1999; Lewis 1946; Locke 1975; Wittgenstein 1958.
[17] See Kripke 1972; Putnam 1973; Schwartz 1977a, especially Schwartz's introduction, Schwartz 1977b. I thank Frederick Schmitt for extremely useful discussions regarding this theory.
[18] Or "Uluru," which is the aboriginal name.

particular object can be considered an instance of a kind. This we can do in a number of ways: (1) compare it to a concrete prototype, (2) abstract properties from the object and compare quantity and quality of property similarity to the intensional set of the abstract kind, or (3) judge, qualitatively, overall similarity with the kind and, if very similar, determine from that what properties the object and kind share.[19] My point is that whichever way we choose epistemically or psychologically, or champion in philosophical argument, each of these ways can be represented—rationally reconstructed—in terms of comparison of the contents of the intensional sets of the concrete object and the kind term. The traditional, rather than the Kripke-Putnam, theory of meaning should be used in evaluating the proper use, in science, of theoretical kinds.

One concern Kripke and Putnam have vis-à-vis the traditional theory of meaning is that a particular individual does not maintain its properties across possible worlds, although it is *still* the same individual. They use this as an argument in favor of their view that what provides the meaning of a term is the baptismal event of naming, and subsequent accurate reference (which, as I argued above will always rely on epistemic acts of judgment). To this, I argue that it is less important to worry about identity and reference maintenance across possible worlds, than to be concerned with accurate scientific judgment and

[19] Regarding the third option, one could further say that what is important here is to determine what *tropes* (or kinds of predicates/predicate universals, such as "redness" or "tallness") are both instantiated in the object and captured by the kind (cf. Lowe 1988). Identifying this will allow us to compare property, that is, predicate, sharing between object and kind. With respect to the first two, object identification or concept formation as occurring through either comparison with a concrete prototype or through the establishment of property similarity with an abstract kind, see Rosch and Mervis 1975; Rosch et al. 1976; Medin et al. 2000. I thank Frederick Schmitt for pointing me to this literature.

identification of theoretical kinds in this world. And here we must rely on property sharing of some sort.

Kripke and Putnam, following Quine[20], argue that ultimately the baptismal event can be shown to be "accurate" and consistent based on the microstructure of the object. That is, water *is* H_2O not only because that was the baptismal event in the past, based, partly at least, on superficial properties, but also, more importantly, because we eventually discovered the microstructure of water and were able to provide scientific criteria for differentiating water from non-water (even in cases, Kripke and Putnam argue, where non-water resembled water in all *observable*, but not *essential* properties).

Now, I do not believe that one has to adopt a Kripke-Putnam theory of meaning to endorse a view that knowledge of the microstructure is an important criterion for identifying an object as an instance of a kind. Some properties may be *necessary* for an object to be an instance of a kind (e.g., in order to be a piece of matter, the piece must have *some* mass, even if it is non-resting mass). And some of these properties are determined by the microstructure of the object. Furthermore, it is true that often properties covary in the intensional sets of particular terms (e.g., charge and acceleration, given particular conditions, covary for all electrical charges); this is precisely what laws and generalizations attempt to capture. And property correlations often have to do with the microstructure, including the causal structure, of the objects involved, as studied by that theory. None of this need involve a Kripke-Putnam style causal theory of meaning. In fact, I gladly endorse the view, agnostic with respect to realism, that

[20] See, for example, Quine 1991 (1969), pp. 169-170.

theoretical kinds and properties can be highly projectible[21] because of (a combination of) microstructure and theoretical richness.

In short, then, the Kripke-Putnam theory of meaning underestimates the role of epistemic judgments of similarity, overemphasizes the importance of possible world ontology, and has too narrow an interpretation on the importance of microstructure. I will now turn to the traditional theory of meaning and two very different presentations of it: the traditional Locke-Lewis "necessary and sufficient condition" abstraction, which I call "simple abstraction," and the Wittgenstein-Boyd "family resemblance" abstraction, which I call "complex abstraction." I claim that the former is especially pertinent to formal biology, while the latter is crucial for compositional biology. But first I must sketch my general view on abstraction.

I employ a version of Cartwright's definition of abstraction as the removal of either properties or Aristotelian causes.[22] Concerning the first, Cartwright notes that there is some difficulty in knowing exactly how to individuate properties, but that we still acquire a good intuitive idea of abstraction by thinking of it in its Aristotelian and etymological sense of "taking away," "separating," and "subtracting."[23] She writes,

> For Aristotle we begin with a concrete particular complete with all its properties. We then strip away—in our imagination—all that is

[21] See Goodman 1983 on projectibility of theoretical kinds and properties, that is, the reliability of inferences regarding the membership in a particular kind as well as ownership of particular properties, by objects thus far unobserved, made on the basis of (1) observed objects and (2) judged similarity between the unobserved and observed objects. See also Barker 1997 who discusses the general issue of "ampliative inference."
[22] Cartwright 1989, ch. 5.
[23] Cartwright 1989, pp. 197, 215; Oxford English Dictionary, 2nd Edition, online.

> irrelevant to the concerns of the moment to focus on some single property or set of properties, 'as if they were separate.'[24]

To paraphrase, we remove elements (properties) from the intensional set of the theoretical kind. A formally similar argument applies to Cartwright's more concrete account of abstraction through subtraction of Aristotelian causes. The main difference is that here the properties are collected into each of the four Aristotelian causes and then removed as a group, a cause at a time (i.e., material, efficient, formal and final). This very general account of abstraction holds for both simple and complex abstraction.

Cartwright claims that more "explanatory information" is provided when more of the Aristotelian causes are present in the "object," or what I believe can be read as "theoretical kind."[25] One advantage of the "Aristotelian causes" version of abstraction over the property version is that it provides a useful typology of the (sets of) properties that are removed. But the more *general* property account of abstraction can be implemented with my intensional set analysis.

A very similar account of abstraction was presented by the important American evolutionist James Mark Baldwin, together with an English psychologically-inclined philosopher, George Frederick Stout, in Baldwin's *Dictionary of Psychology and Philosophy* from 1903. They defined abstraction as "Concentration of attention on those parts or characters of an object which are treated as relevant to the special interest of the moment, and its consequent withdrawal from those which are irrelevant."[26] It is worth noting that Baldwin and Stout also mentioned the "parts" of an object. Below I shall explore the

[24] Cartwright 1989, p. 197.
[25] See Cartwright 1989, pp. 219-220.
[26] J. M. Baldwin and G. F. Stout in J. M Baldwin 1903, v. 1, p. 6.

problematic notion of partitioning as a form of abstraction. For now, it is best to consider abstraction merely as the removal of properties.

In Chapters 4 and 5, I will compare Cartwright and Friedman on various points, including the role they believe that abstract laws and models play, and their distinct views on explanation. Here I want to distinguish two aspects of my account of abstraction, inspired by Cartwright's notion of the *abstract-to-concrete* relation as one of *few-to-many* properties and Aristotelian causes. There is a distinction to be made between the *overall* pattern of abstraction and the *purpose* of abstraction. The overall pattern I argue for is one of property removal. As we shall see in Chapter 5, Friedman has an *incremental* theory of abstraction, which I do not endorse, although it is interesting, because I am not sure that it individuates kinds, in addition to laws and models, correctly. But an important point that Friedman's analysis reminds us of is that abstraction, in general[27], is for the purpose of *creativity* – new connections and hypotheses can be articulated.[28]

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[27] Of laws and models, and not just of terms.

[28] In a footnote discussing Whewell's notion of consilience, Friedman notes that "the whole point of *theoretical* structure is to facilitate this process of inductive 'jumping together'." (Friedman 1983, p. 242) In the literary sphere, Jorge Luis Borges makes an analogous point in an essay entitled "Kafka and His Precursors": "If I am not mistaken, the heterogeneous pieces I have enumerated resemble Kafka; if I am not mistaken, not all of them resemble each other [Wittgenstein's family resemblance!]. This second fact is the more significant. In each of these texts we find Kafka's idiosyncrasy to a greater or lesser degree, but if Kafka had never written a line, we would not perceive this quality; in other words it would not exist. … The fact is that every writer *creates* his own precursors. His work modifies our conception of the past, [just as it has to] modify the future." (Borges 1964, p. 201; cf. Borges 1970 writes "como ha de modificar el futuro") For Borges, Kafka created a new way to tie together disparate work by authors such as Kierkegård and Browning – this is, in a loose sense, analogous to creating new connections and hypotheses through the use of an abstract theoretical kind, law or model. McOuat 2001 also indirectly relates to the point of kinds providing the grounds for creative model construction, broadly construed. He also directly tackles the relationship between kind/model construction and kind/model evaluation, through measurement.

A removal view of abstraction can easily imply lack of creativity, although this is not something that I think Cartwright endorses[29]. I certainly endorse a *removal* pattern *and* a *creative* role for abstraction, certainly in formal biology. Abstract theoretical kinds (e.g., "charged particle," "gene with only fitness parameter") often serve unifying and organizing purposes in the laws and models of the theory at hand.

Let me now turn to two extreme presentations of the traditional theory of meaning: Locke-Lewis "necessary and sufficient condition" abstraction and Wittgenstein-Boyd "family resemblance" abstraction. In his *An Essay Concerning Human Understanding*, Locke adopted a nominalist position – he held that (kind) terms did not refer to essences, but were rather "the Workmanship of the Understanding."[30] What is important for my argument is his view that names, at every level of abstraction, are necessarily associated with a collection of ideas. In discussing the "Ring on [his] Finger," he makes a distinction between its real essence[31] and its nominal essence. The latter, which we can, and do, know "is its Colour, Weight, Fusibility, Fixedness, etc. which makes it to be Gold, or gives it a right to that Name."[32] Thus, for Locke a name (the nominal essence; e.g., "Gold") is captured in the properties defining it (e.g., its particular "Colour, Weight, Fusibility, Fixedness"). Lewis makes a similar point when he writes, "Traditionally any attribute required for application of a term is said to be *of the*

[29] For example, in "The Truth Doesn't Explain Much" she notes that abstract laws "unify" and "organize." This seems to grant them a creative role in generating new more concrete laws and models even if abstract laws are false and do not organize "varied and diverse" phenomena (Cartwright 1983, p. 53).
[30] Locke 1975, p. 416 (Book 3, Chapter 3).
[31] That is, its microstructure, which Locke, the empiricist, believes we will never know because it is insensible.
[32] Locke 1975, p. 419 (Book 3, Chapter 3).

essence of the thing named."[33] Lewis is very clear throughout his discussion of intension and extension that a kind term has necessary and sufficient properties – the attributes are "required." Neither Locke nor Lewis, however, have a clear dynamical account of the *process* or even *pattern* of abstraction. Furthermore, their account concerns the nature of vernacular terms and they do not discuss scientific terms. Despite these weaknesses, their account of (theoretical) kind terms as containing necessary and sufficient conditions provides a good start for my account of simple abstraction using intensional sets.

When theoretical kinds are judged, and abstracted, on simple objects, intensional sets with necessary and sufficient properties can often be found – simple abstraction can be done. Therefore, the intensional sets of theoretical kinds of simple objects have a neat overlapping structure. That is, there is only one way to abstract a more concrete theoretical kind to the immediately more abstract theoretical kind, defined by a particular intensional set of necessary and sufficient conditions *for that* more abstract theoretical kind; this way is to remove just the one right property. If we keep on doing this, we will see a neat overlapping structure of the properties of the intensional sets of progressively more abstracted theoretical kinds. We see this neat structure in simple objects because all objects of a particular simple kind are equivalent and objects of another simple kind, which can be placed together with the objects of the original ontological kind under a more abstract theoretical kind, are also equivalent.

For example, leptons, which include electrons and muons, are fundamental particles with no strong force interactions. All electrons have a

[33] Lewis 1946, p. 41. See also Schwartz 1977b.

particular mass (.000511 GeV/c^2) and charge (-1e); all muons also have a particular mass (.106 GeV/c^2) and charge (-1e).[34] Each of these simple kinds can be judged similar theoretically using just the mass, which is a necessary property; however, it may not be sufficient (some particle in the universe may just happen to have that same mass and not be an electron). However, if we add "charge" and "fundamental particle with no strong force interactions" to our intensional set, then we have a robust intensional set with necessary and sufficient properties for the theoretical kind, whether it be an "electron" or a "muon." Now, we can produce an even more abstract theoretical (simple) kind by ignoring mass and charge and simply saying "a fundamental particle with no strong force interactions" – this is the definition of the theoretical kind "lepton" (it is not important for purposes of this argument that "fundamental particle" and "strong force interaction" are themselves theoretical kinds and simple objects, respectively – these kinds are defined independently). Note that the extension of the "electron" theoretical kind is identical to all the simple kind electrons out there; the extension of the lepton theoretical kind is the sum of the electron, muon, and four other fundamental particle simple kinds in the world.

After giving this scientific example of abstraction into theoretical kinds, let me turn briefly to mathematical abstraction, which I consider a special case of abstraction using intensional sets. To the best of my current knowledge, there is not much literature on the act of abstracting an object (e.g., "evolutionary gene") or equation (e.g., "gene frequency change based on selection and drift")

[34] http://www2.slac.stanford.edu/vvc/theory/leptons.html

mathematically.[35] Various forces, under particular background conditions (to be further discussed below under the name "*ceteris paribus* intensional set") are assigned to objects in the form of properties defined by those forces.

Formal biology, best represented in mathematical evolutionary genetics, appeals and employs simple mathematical abstraction. Although there is a diversity of mathematical models in evolutionary genetics, the genetic parameters and variables, and formulas followed, are basically agreed upon and neat hierarchies of mathematical kinds and formulas representing various numbers of genetic parameters and variables are developed in the model hierarchies for a particular problem areas under a particular set of assumptions.[36]

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[35] Steiner 1989 does discuss related matters, but he does not focus on the actual process of abstracting mathematical properties from object properties, or from producing an abstraction hierarchy of such mathematical properties, both of which are my concern here.

[36] Lloyd argues that the view of evolutionary (genetic) theory as hierarchical applies primarily to the very *abstract high-level theory* of evolution, but not to the much more concrete "theoretical models" "which are formed from specific theories through the adoption of additional empirical assumptions." (Lloyd 1988, p. 13) For example, consider the point that both Fisherian and Wrightian mathematical modeling schools agree on the basic (abstract!) formulas and forces of evolution, formally considered (Michael Wade, pers. comm.). Lloyd thus suggests that neat hierarchy exists at very abstract high levels of evolutionary theory, but not at the low-level (e.g., theoretical models) and the middle-level (e.g., model-types). In fact, Lloyd's philosophical analysis of the units of selection controversy (e.g., Lloyd 1988, 2000a) shows, convincingly, the mismatches in empirical assumptions and theoretical goals between different theoretical models and model types of "group selection" and, thus, the absence of a consensus neat model hierarchy. But, underlying this diversity, there does seem to be a mathematical ideal imperative toward—and, to an extent, an actual situation of—theoretical unification as has been shown in recent work showing the mathematical translatability of different parameterizations of group and individual selection (Dugatkin and Reeve 1994; Kerr and Godfrey-Smith 2002a, 2002b) as well as earlier work on the unificatory power of the Price covariance approach (Wade 1985; Frank 1997), to be further discussed in Chapter 5. It is unclear to how low a level of model abstraction these unification attempts pertain. Furthermore, however, it is clear, and Lloyd *does* show this, that within a set (family) of assumptions and modeling techniques, there is a fairly neat abstraction hierarchy of models.

In fact, the simple theoretical kinds employed in these mathematical models have necessary and sufficient properties by *definition*.[37] Such simple abstraction of these theoretical kinds (genes) and formulas is an aspect of the clear and definite logic of mathematical abstraction (by removal of parameters and variables). That is, mathematical properties for a gene represented by parameters and variables, postulate, that is, theoretically define properties for the model-based gene that are then measured in the empirical gene (e.g., fitness effect and mutation rate of a gene is defined, represented in models, and then, sometimes, measured).[38]

I will now turn to complex abstraction as advocated by Wittgenstein and Boyd, which I argue is prevalent in compositional biology. In his *Philosophical Investigations*, Wittgenstein argued that terms could not be presented in terms of intensional sets of necessary and sufficient conditions, but should rather be thought of as having (something like) disjunct sets of properties. That is, the objects (extension) referred to by a particular term do not all share the same properties; an object may share property X with another object, while sharing property Z with a third object. In discussing how one determines "kinship" between different images, he writes,

> In such a difficulty always ask yourself: How did we *learn* the meaning of the word ("good" for instance) From what sort of examples? in what language-games? Then it will be easier for you to see that the word must have a family of meanings.[39]

Terms have a "family of meanings" that are learned by ostension and by the appropriate use of language. I interpret this as Wittgenstein arguing that the connotation of terms is constituted by a multiplicity of intensional sets.

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[37] This is certainly not the case in compositional biology.
[38] I will return to mathematical abstraction in the next section, on models, of this chapter.
[39] Wittgenstein 1958, 36e, ¶77.

Boyd accounts for the meaning of theoretical kinds by appealing to what he calls "homeostatic property clusters."[40] Instances of theoretical kinds, Boyd argues, tend to have some, but not all, properties (and values of those properties), to a large extent, in common. Furthermore, Boyd claims, they have this homeostatic cluster of properties in common because these properties are projectible to unobserved instances of the kind, under the particular theory under discussion. Boyd is also a realist. He claims that the properties provide an accurate classification when they *accommodate* the causal structures postulated by the theory.[41] It is under these conditions that the kinds can serve in our inductive and explanatory practices. Boyd claims that the correlation between the properties need not be causal (either through mutual interaction or common cause), but given his realist commitments to kinds, entities, and causes it is difficult to see under what conditions the cluster properties would *not* be causally interwoven. For example, his paradigm case of homeostatic property clusters is species and the members/instances of species have similar properties *precisely* because of (1) a common cause, genealogy, and, (2) maintenance of character cohesion through gene exchange of members of that species.[42]

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[40] See, e.g., Boyd 1991, 1999.
[41] Boyd 1999, especially pp. 146-148, 165-167, 176-179.
[42] Note that Boyd's, as well as Wittgenstein's, account of natural kinds, does not fit well with the received view of kinds and Laws, which includes the "necessary and sufficient view" of abstraction as well as the Law-based view favored by many of the logical positivists, to be further explored in Chapters 4 and 5, and also in Appendix A. First of all, predictions and explanations are fallible because the projectibility of any given property is not perfect across instances (there are no necessary conditions for all instances of a particular kind), nor is it perfect into the future because Boyd allows for the possibility of change in the causal explanations pertinent to particular kinds (for example, the way a liver works in an ancestor could be different from the way it works in a descendant). Boyd thus allows for predictive imperfections as well as Laws (or, more aptly put, loose correlations or mechanisms) that change over time.

Furthermore, he does not believe in the reduction of properties to some sort of intrinsic microstructure or essence, which can ultimately be represented in powerful (formal) laws. Homeostatic property clusters are complex and cannot be reduced further. Thus, the received view of necessary and sufficient properties for natural kinds seems at philosophical odds with Wittgenstein's family resemblance and Boyd's homeostatic property cluster views. I will now place my articulation of theoretical kinds and abstraction in the context of this debate.

When theoretical kinds are judged, and abstracted, on complex objects, necessary and sufficient properties cannot often be found. We can still talk about the intensional sets of such theoretical kinds, but now the intensional sets for the *same* theoretical kind will be malleable – that is, multiple intensional sets will correspond to the same theoretical kind.[43] Properties in these intensional sets of a theoretical kind will sometimes not be causally correlated with one another, and they will certainly not be neatly related to a more abstract theoretical kind by subtraction of the same properties from the different intensional sets that give the same more concrete theoretical kind.[44] That is, there will be more than one way to get to the same more abstract theoretical kind. Put differently, for complex objects, especially the parts of a system investigated by compositional

[43] As a general example consider the fact that there are multiple types of pathologies for almost every organ of the human body. And in many cases, humans still manage to live satisfactory extended lives with these pathologies. The pathological organ of a human organism does not have the same set of properties as that for the "ideal" or "normal" organ. And yet we would refer to it as an instance of the same theoretical kind – we would call it, for example, a human liver or, more abstractly, a mammalian liver. Furthermore, there are also many non-pathological variations in function type and magnitude.

[44] Eco 1984 made this point in criticizing the single-branching structure in the Porphyric Tree of ever more concrete concepts.

biology, there are often properties that are important to the definition of particular theoretical kinds and adequate reference to objects, but few, if any, properties are genuinely necessary and few sets of properties are genuinely sufficient for all the objects of a particular theoretical kind. For example, "heart" can be defined in many ways, depending on the theory used, the taxa studied, and the level of generality desired. Even for a particular clade (e.g., mammalian) there are not really any necessary and sufficient conditions for defining "heart" – exceptions can almost always be found, and "hearts" are not equivalent to one another.

A complex object can often be analyzed from very different theories, as I will explore in Chapter 2 where I also develop the idea of "theoretical perspectives." Thus, there are multiple possible *kinds* of abstraction hierarchies, since there are *very* different ways of formulating theoretical kinds. In organismal biology, for example, comparative anatomy, developmental biology and physiology provide distinct ways of defining, recognizing, and explaining theoretical kinds. Each of these ways involves distinct theoretical kinds many of which are *not translatable* across perspectives, in the members of their intensional sets, since the properties are so qualitatively different. Of course, a variety of perspectives exist in theoretical physics as well, but there phenomena tend to be of one kind or another (e.g., gravitational or electromagnetic) and there is often a clearly best way to analyze any object or process (i.e., simple object), thus theories do not tend to overlap and provide distinct relevant explanations for the same object. Furthermore, in theoretical physics, theories, many of which can be related to basic forces, apply either to very distinct levels of nature (e.g., classical

mechanics vs. quantum mechanics) or, more importantly, can often be unified[45] to a single theoretical perspective (e.g., quantum electrodynamics and, now, superstring theory).

In theoretical physics, and, on analogy, in formal biology, a few basic properties are considered important and can often be translated across theories; furthermore, there are clear ways of integrating perspectives either through abstraction (theoretical unification of forces) or through theoretical interrelation (addition of forces of different types in a "vectorial" fashion in a particular situation). Theoretical physics and compositional biology (but not formal biology!) differ in that the former tends to deal with simple objects and the latter with complex objects. But they also differ in that the variety of theories in compositional biology has real potency and can be interpreted to lead to its disunity, as I will explore in Chapters 2 and 3, whereas the variety in physics can be mostly overcome through abstraction and/or interrelation.

Now that we have discussed some of the differences between simple abstraction, often on simple objects, and complex abstraction, often on complex objects, let us develop a formalism for the concept of necessary and sufficient properties as well as the denial of this concept.[46] This will allow us to clarify the above discussion, and present it in a different manner. Note that a particular theoretical kind, TK, (supported by a theory) is defined by an intensional set consisting of a variety of properties, P. Objects are partly determined by a theory (e.g., the additive or interactive effects of genes are interpreted differently in Fisherian or Wrightian population genetics; the developmental liver is not

[45] I will explore unification in theoretical physics and formal biology, under Friedman's view, in Chapter 5.
[46] I thank Steve Crowley for assistance with this formalism.

equivalent in all respects to the physiological liver). This, however, does not affect the formalism. It only implies that the list of objects changes as theories are changed.

The following implies that we accept the existence of at least one property necessary for instantiation of a theoretical kind:

$\forall SO \forall TK(\exists P_I)[(SO \in TK) \rightarrow SO(P_I)]$ [1.1]

[Note: SO = "simple object"; "$SO \in TK$" should be read as, "SO is an instance of TK"; " $SO(P_I)$" should be read as, "SO has property P_I." The variables are intended to hold for all simple objects, all theoretical kinds, and all properties, under particular theories (which, themselves determine the theoretical kind).]

An example of this would be SO: electrons, P: electron charge

The following implies that we accept the existence of at least one property sufficient for instantiation of a theoretical kind:

$\forall SO \forall TK(\exists P_I)[SO(P_I) \rightarrow (SO \in TK)]$ [1.2]

It may be difficult to find examples for sufficiency of just one property, but we can extend the formalism by introducing sufficient *sets* of properties:

$\forall SO \forall TK(\exists P_I)[SO(P_I \wedge P_L \wedge \ldots) \rightarrow (SO \in TK)]$ [1.3]

An example of this might be SO: electrons, P: electron charge, electron mass, fundamental particle with no strong force interaction.

Note that for complex objects, in general, the above sentences would all be *false*. That is, we would deny necessity and sufficiency of properties (or property sets) for instantiation (and determination of the extensional set) of complex objects of particular theoretical kinds. This would be represented formally by adding a negation (¬) in front of each existential quantifier and also replacing all the SOs with COs (i.e., "complex objects").

Introducing sets of properties brings up the possibility of introducing *disjunctions* of properties and sets of properties. For example, a Wittgensteinian would concede the necessity of a sufficiently long property disjunctive list in [1] above [i.e., replace " $SO(P_I)$" with "$CO(P_I V P_L V \ldots)$"]. With the disjunction of P_I's sufficiently long, the Wittgensteinian would have to agree that the disjunction is necessary. Of course, she could claim that it would have to be infinitely long, or at least as long as there are members in the *extensional* set, and that such a "save" misses her point.

Interesting cases arise in the epistemic area between simple objects and complex objects. As we move from complex objects to simple objects, the disjunction list gets shorter and shorter (the limit being one property). Consider elements of the same chemical group. They share many properties, yet they vary in some as well. There a disjunction list longer than one, but not *much* longer, would capture the necessary set of properties to be an object of that group. Such a case would be *intermediary* between, say, hadrons (all of which share the crucial property of being affected by the strong force, which leads to many other similar properties) and kidneys of humans (which share some properties, especially high-level functional ones, but are extremely variable and have all kinds of

mal/misfunctions; these mal/misfunctions can be represented as distinct properties).

The general point here is that we can almost always abstract using necessary and sufficient properties—simple abstraction—in the case of simple objects, whereas complex abstraction is almost always required for complex objects. However, there is a middle ground with short disjunctions of necessary sets of properties.

These arguments regarding theoretical kinds can be summarized in the following table, which captures the different characteristics of the intensional set defining and abstracting the two extreme form of objects given or postulated by theory. Note that the middle ground of objects is not depicted in this table, but its characteristics would be between the answers in either column.

	Objects which are "given" /postulated by the theory[47]	
Theoretical Kinds: Characteristics of their Intensional Set	**Simple Objects**	**Complex Objects**
(1) What is the "minimal definition" at any level of abstractness of theoretical kind?	Short	Long(er)
(2) Are there any necessary and sufficient conditions for belonging to a theoretical kind?	Yes	No (maybe rarely)
(3) Are the intensional sets of ever more abstract theoretical kinds compositionally set-related?	Yes	No
(4) Can multiple distinct intensional sets define the same theoretical kind? [By distinct I mean also that the different properties are NOT correlated (including causal correlation).]	No[48]	Yes[49]
(5) Can multiple different (but not distinct) intensional sets, with potentially correlated properties, define the same theoretical kind? (e.g., Boyd and perhaps Eco)	Yes, but not necessary given (2) and (3)	Yes
(6) Can there be variation for particular properties among instances of a theoretical kind?	No	Yes
(7) Are there any properties for which there can be NO variation?	Yes	Yes

Table 1. (1.1) Characteristics of the intensional sets of the theoretical kinds of simple and complex objects.

[47] Note that I want to remain agnostic about the realism issue with respect to the objects of the theory. The two distinctions I have thus far presented—the causal structure of the objects and the abstraction of objects into kinds—are highly theory-dependent, but the first concerns the putative *causal structure of the entities* prescribed by our theories while the latter focuses on the *epistemic categorization* of those entities into hierarchical groups captured by similarity.

[48] Two different (but not distinct!) intensional sets to pick out, say Calcium, would be equivalent because the different properties they employ (e.g., ionization energy and atomic number) are causally correlated.

[49] Developmental homology ≠ structural homology. That is, developmental criteria do not pick out the same spatio-temporal regions as structural criteria, yet both can be used to pick out, say, mammalian hearts. Another example is Wittgenstein's famous example of games.

Thus far, I have spoken about the nature of the intensional set that determines the theoretical kind, at a particular level of generality, under study. What I want to introduce now is a concept that I will call the "background" or "*ceteris paribus* properties" of this intensional set. The basic idea here is that the utility—appropriateness of reference and projectibility—of the theoretical kind *depends* on what the *other* properties ascribed to the theoretical kind are. That is, what other assumptions are made about the theoretical kind and how reliable are the inferences licensed by these assumptions about properties?

One argument is that while abstraction can proceed mostly problem-free in theoretical physics and formal biology, and is, in fact, one of the goals and generalizing principles of those kinds of sciences, abstraction is highly limited in use in compositional biology. There generalizations about the causal role of parts are much more context-dependent and subject to exceptions – even when the "foreground" properties of the intensional set are met in complex abstraction, the background properties can fail to be met in many different ways.

For example, consider ideal gases and the simple and formal relationships captured by the ideal gas law. We can define a very abstract theoretical kind—an ideal gas—with a very broad extension. Of course, the realism of this abstraction is not always very high. In fact, and this is a general point, the realism of this theoretical kind only applies under a particular *ceteris paribus* clause, which can be captured in the intensional set (e.g., the gas should have the properties of not being subject to any kinds of intermolecular forces, of not being subject to magnetic fields present for gas molecules with the property of having a magnetic moment, etc.). This clause can thus be thought of as part of the intensional set defining the kind, but it is the *background* of this set. It is the background in two

senses: (1) it is usually covert and (2) it provides the context in which the relevant properties of intensional set accurately capture the right extension (it "fills in" the properties not mentioned in the definition). When this clause holds, the theoretical kind (e.g., ideal gas) accurately captures all the relevant properties of the objects under its extension. When the clause does not hold, and it often does not, to varying degrees, then the theoretical kind misses some of the (causally) relevant properties and is, therefore, not realistic. But the intensional set of the theoretical kind still captures the important properties of an abstract and explanatorily potent theoretical kind, which, as we shall see in Chapter 5 when discussing Friedman's view, can allow us to derive more concrete laws, models, and theoretical kinds.

For theoretical physics and formal biology the background part of the intensional set is much more flexible and significantly smaller than the part for compositional biology. That is, often even when one or more of the elements of the background does not hold (i.e., does not describe the situation at hand – the theoretical kind does not apply, *strictly speaking*, to the particular object), the theoretical kind still captures many relevant and causally important properties of the object. To a significant first approximation, the kind is still applicable. This is the "flexibility" of the clause. Furthermore, there are fewer meaningful ways in which the *ceteris paribus* clause can fail to be met when applied to the simple objects of theoretical physics and formal biology. Since these kinds are causally simple, there are fewer, *different in type*, ways for the causal context to be different, and, thus, for defining the theoretical kind differently. For example, the orbits of the planets of the solar system can, with great accuracy, be predicted from the set of pairwise interactions between each planet and the Sun. One can

think of these pairwise interactions between point masses, captured by the Universal Law of Gravitation, as a theoretical kind. Of course, the gravitational force of the other planets and even of close stars should be taken into account (i.e., the *ceteris paribus* clause of a pairwise interaction does not, strictly speaking, hold), but the orbit can still be calculated with great accuracy from just pairwise interactions. Furthermore, given that the primary force acting on the earth is gravity, there are few ways different in kind in which the clause can be inapplicable (e.g., electromagnetic and other forces are not relevant).

This picture of theoretical kinds with the background members of their intensional sets helping to specify the context in which they apply, also pertains to compositional biology. But here the background components are often falsified, both in number and kind of properties and conditions. Due to the causal complexity of parts, there are so many conditions that need to be met for a particular part—complex object—to "count as" an instance of a theoretical kind. If any of these are missing (e.g., a liver not engaging in the right kinds of mechanisms), then that complex individual would not, strictly speaking or even *usefully* speaking, count as an object of that complex theoretical kind (e.g., it would be a *pathological* liver). And there are many ways, both within and across types of ways, for a complex object not to match the prescriptions of the *ceteris paribus* clause of its putative abstract theoretical kind. For example, consider the lack of predictive power that inferences about gene function often have across different taxa. We can establish similarity of gene sequence and thereby define a particular gene type. But, the concretization of this gene in particular systems[50] is such that whatever properties could be ignored in the source system from which

[50] Note the explicit consideration of part-whole relationships here.

the gene, and its causal roles, were abstracted, cannot now be ignored in the target systems. Different properties and kinds of properties come to the fore, and the *ceteris paribus* clause in the intensional set does not hold. The complexity of the causal networks in which genes are involved are such that inferences made on the basis of abstract theoretical gene types are extremely problematic and fallible.[51]

A further difference between the two kinds of biology becomes clear when one considers reactions to failures of the *ceteris paribus* clause and the subsequent accuracy of abstract theoretical kinds. Two sorts of responses are available, one employing theoretical activity, the other using empirical manipulation.[52] (1) With respect to theoretical activity, the appropriate response to a situation with a new force or a new complication is to add formula variables (which can be thought of properties as in the intensional set pertinent to that object) that take that into account. For example, in the ideal gas laws we can add van der Waal forces when they become pertinent. There are precise and well-defined ways to add forces to specify the properties of the more concrete theoretical kinds of theoretical physics and formal biology.[53] In compositional biology, there are not well-

[51] Again, note that such inferences are much less problematic in the case of kind-based sciences in which subsumption of abstract theoretical kinds is much more straightforward, certain and useful.
[52] For more detail on the distinction between theoretical and empirical activity, which is not the same as the distinction between theoretical and empirical content (!), see Chapter 4.
[53] In a chapter entitled "Physical Law," Duhem 1977 explicitly considers laws under increasingly complex and realistic conditions. Under such conditions, he explains how variables can be incrementally added to the symbolic relations that constitute laws in his opinion. A similar argument can be made for theoretical kinds considered as mathematical kinds. Consider a point mass in a problem involving only gravity. Now imagine that a collision with another massive object occurs. Then this is a particular kind of point mass whose properties (acceleration) can be precisely derived from combining gravitational and impact

defined ways for further specification of theoretical kinds. If a *ceteris paribus* clause should not be met, it is unclear how to theoretically model, or explain or predict concrete situations that fail to meet the background properties. At least one reason for this is because causes often interact in causal, non-quantitative, and non-predictable ways in compositional biology. So removing or adding a causal factor from the intensional set describing a theoretical kind cannot be interpreted in the additive "vectorial" fashion that it often can in formal biology.

(2) With respect to empirical manipulation, failures of the *ceteris paribus* clause in theoretical physics can be remedied by controlling the factor and, thereby, reinstating the *ceteris paribus* clause. That is, if for example a magnetic field causes the deviation of some magnetically inducible gas from the ideal gas law, then simply turning off or neutralizing the magnetic field can control the situation. Of course this is not always possible, but often it is. This is more difficult, however, to do in formal biology. However, there are methods of statistical randomizing effects in the populations pertinent to ecological or evolutionary genetic studies, for example. In compositional biology, however, it is very difficult to control for factors in part because the system is so intimately *integrated* that trying to control it might cause the whole system to malfunction and even if it still functions, the outcome of a control might not be the same outcome as the outcome that actually exists in the system naturally.

The utility of very abstract theoretical kinds (in part determined by the realistic applicability of that kind) is high in theoretical physics and formal biology, but rather low in compositional biology.

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forces in a vector fashion. We have precise mathematical ways of defining acceleration ("a") of this kind ("m").

In this section on abstraction, I hope to have motivated and sketched a general account of abstraction. I am inspired by Cartwright's version of abstraction as removal of properties; but, as Friedman (and Borges), among others, reminds us, abstraction is a *creative* process, so we must remember that removal of properties can also be creative. My general account of abstraction relies on the notion of an intensional set with properties pertinent to a theoretical kind as members. Abstraction, then, is removal of properties from this set. This can happen in a neat and hierarchical fashion, as is the case with simple abstraction on simple objects in formal biology. Or it can happen in a much messier and non-overlapping fashion, as is the case with complex abstraction on complex objects in compositional biology. The intensional set can be construed as being very large, including all kinds of properties determined by the theory, including properties that allow for the idealization of the theoretical kind. The properties that are commonly used in the explicit *definition* of the theoretical kind I consider the foreground properties. The ones that allow for this theoretical kind to be useful and stable, I call the background properties in the *ceteris paribus* clause. The background properties are much more easily controlled and understood in formal biology than in compositional biology. In short, abstraction is much clearer and more powerful in formal biology, although it is certainly present (although more fallible) in compositional biology.

1.4 Mathematical Models versus Propositional Non-Mathematical or Material Models

The third distinction concerns modeling, which is a crucial part of scientific work. In Chapter 4, I will analyze the concept of models and modeling in detail. Here I want to contrast, in a basic manner, mathematical to propositional non-mathematical or material models. I argue that formal biology tends to employ mathematical models, whereas compositional biology generally uses the other two general model forms. In this chapter, I will not analyze the semantic view, or expansive/deflationary views of it, or even orthogonal approaches – I leave that for Chapter 4.[54] In that chapter, I argue that modeling is a theoretical *activity*, although it certainly has significant empirical *content*. There I also develop a framework describing four general properties of models possessed by *all* the model forms employed in biology. Thus I postpone an important argument for the idea that non-propositional models (e.g., narrative and diagrammatic models) and material models (e.g., scale models and Griesemer's "remnant models") *are* indeed models. In this current section, I motivate how modeling, and the model forms employed, in each kind of biology are related to other methods and assumptions (captured by my other three distinctions). I am interested in *how*, rather than *what*, models represent.

[54] Proponents of the semantic view of theories include Lloyd, Suppe, Suppes, Thompson, and van Fraassen. Both Griesemer and Downes can be thought of as adopting some aspects of the view, while remaining skeptical about other features of it. Advocates of orthogonal approaches on models and modeling include Cartwright, Friedman, Morgan, Morrison, and Suárez.

I will first explore mathematical models, commonly found in formal biology, and will then analyze the models prevalent to compositional models. In analyzing each model form, I will show how it relates to my other three distinctions between the two kinds of biology. I will, for example, show how simple mathematical abstraction pertains to the models of formal biology and how complex abstraction relates to the models of compositional biology. I will also draw some general conclusions regarding where the main *locus* of manipulation and intervention work occurs in the two kinds of biology: does most of the activity occur in modeling or in experimental work?

Mathematical models, prevalent in formal biology, can be interpreted in the language of first-order logic[55] or, as is the case much more often in science, written directly in mathematics. The philosophy surrounding this form of models is extremely extensive and cannot possibly be reviewed here, but in Chapter 4 I will provide a little more detail.

Developing a mathematical model involves a large amount of (mathematical) abstraction and idealization: (1) properties of objects and processes need to be quantified, thereby ignoring many different kinds of qualities that they have, (2) only some of the objects and processes under study can be quantified, thereby ignoring a host of other objects and processes, (3) measurement errors in the eventual confirmation of the model are often *ignored* as a possible limitation, or problem, that needs to be addressed during the development of the models, and (4) multiple causal interactions need to be ignored for purposes of computational expediency. The list is very long and has

[55] There is, of course, active debate regarding the generality and even utility of this claim. Elisabeth Lloyd holds that the claim is not generally true – e.g., consider models using natural numbers.

been discussed elsewhere.[56] The point I want to make here is that mathematical abstraction, while powerful, involves significant loss in "qualitative" data. It is no accident that mathematical models apply well to simple objects since, to an extent, it actively *makes* simple objects out of some objects that are, perhaps, *inherently* complex (e.g., genes).

As we saw above, one aspect of abstraction on which there seems to be relatively little written concerns the process of parameterization, or quantification in general. That is, how are the properties of objects and processes mathematically abstracted? What aspects are *ignored* when doing this and what does the abstraction *capture* about those objects and processes? This process interests me because formal models consist of some very special sorts of theoretical kinds, what one might call "mathematical kinds."

In order to relate formal models to the first two distinctions, I will explore some the arguments in favor of the view that mathematical abstraction of model development can be understood in terms of the intensional set account of abstraction presented in the previous section. First, let us assume that the parameters and variables of a model are its "properties." I am not here concerned with determining the exact analogical relationship between property *and* parameter or variable, I am merely asking the reader to grant that these can be thought of, at least quantitatively and, in a loose sense, qualitatively, as properties. Now, a model with more parameters and variables is a model with more *properties*, that is, a more concrete model. This matches the mathematical intuition well. A model with more (appropriately articulated) parameters and variables is one that can be made to match the concrete system more closely – it

[56] E.g., Duhem 1977; Levins 1966; Wimsatt 1987; Cartwright 1989.

can be (dis)confirmed more easily and with greater precision. It is also, as the literature on trade-offs in model building points out, a model with less generality (applies to a narrow range of concrete and exact cases) and computationally more expensive. So thinking of parameters and variables as properties of *models* is consistent and useful, even if, as the semantic view has argued, we should be wary of the appropriateness of linguistic analyses of mathematical modeling (and abstraction).

Mathematical models are not well suited to capture the complex, varied, and concrete nature of part-organization (part-organization is the fourth distinction). It is difficult to represent the multiple kinds of processes and functions in which a single kind of part, as well as many kinds of parts, engage. A very large number of variables and parameters would have to be introduced into the models, and these variables and parameters would not be able to capture the qualitative and "articulated"[57] nature of the parts and their interaction.

Theoretical physics and formal biology can easily mathematically abstract and idealize concrete objects (primarily simple objects). The objects have clear quantitative properties and causal relations among these properties, belonging to objects of the same and of different kinds, can be expressed in terms of functional mathematical relations. Physical science is replete with mathematical functions relating variables. Ever since at least the development of mathematical natural philosophy in the 17th century, such functions have been interpreted as formalizations, that is, mathematical representations, of natural laws[58] and the

[57] "Articulation of parts explanations," as described by Kauffman, will be explored in Chapter 2.
[58] Cf. Appendix A. Giere 1995 provides a historical and philosophical analysis of "laws of nature."

variables and parameters have been understood as stand-ins for particular properties of the objects and processes under study.

Consider Newton's second law of mechanics: F = ma.[59] "F" can be interpreted as the total force applied on an object, "m" as the inertial or "resistance" mass of the object, located at the center of gravity of the object, and "a" as the acceleration, or rate of change of velocity, of the object. The function, expressed with an equality and a multiplication represents a law. This law is supposed to have all the desirable properties a universal law of nature should have. Numerous other laws, represented mathematically, are familiar to the reader. The point here is that in theoretical physics and formal biology there are well-articulated and straightforward mathematical relationships[60] among simple objects, abstracted as theoretical mathematical kinds.

We see this kind of mathematical abstraction and idealization in mathematical evolutionary genetics as well. There is a difference, however, in that confirmation of this theory is significantly more difficult due to the complexities of biological systems.[61] Roughly put, formal biology is more top-down, or "theory-first," than mathematical physical sciences. However, the commitments about and to abstraction are similar: develop mathematical variables and parameters representing various properties of objects, processes and systems, and articulate the relationships between such properties using mathematical functions.

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[59] I am using the iconic formula. Newton expressed force in terms of rate of change of momentum.
[60] The "input laws" of mathematical models. On this point, as well as many other points concerning formal and mathematical laws and models discussed, or alluded to, in this section, see Appendix A.
[61] See, e.g., Lewontin 1974 and Lloyd 1988.

One of the basic variables in evolutionary genetics is frequency of gene types. A basic explanandum in this field is to give a dynamically sufficient causal account, at least in principle, of changes in these frequencies over evolutionary time (across generations). This is done by employing an exhaustive set of distinct evolutionary causes; these causes, or "forces" as they are sometimes called, are represented, even *defined*, in mathematical terms. Put strongly, *the forces are nothing more than their mathematical representations in the form of models*.[62] This is, in fact, why mathematical evolutionary genetics was so amenable to semantic view analysis, as indicated by the work of, especially, Beatty, Lloyd, and Thompson. Even if one is a realist believing in the truth of such forces, rather than an ontologically agnostic modeler emphasizing only the empirical adequacy of such representations, the scientifically pertinent content of the models *is* just the mathematical representations. The causal processes underlying the representations are unimportant for purposes of theoretical articulation. In formal biology, theory (in the form of particular models) is developed through the manipulation of formal symbols.

Now consider the models prevalent in compositional biology, propositional non-mathematical models and material models. The former are model forms that have propositional content, that is, some sort of direct semantic meaning, not represented mathematically. The latter are made out of matter, but are still more like representations of a concrete system than of the concrete system *itself*. In this part of the section on models, I will show how these model forms are related to the other three distinctions. I will focus more on material

[62] I am intentionally alluding to Hertz's statement "Maxwell's Theory is Maxwell's Equations."

models, especially scale models, as compared to the propositional models, because I explore the latter in detail in Chapter 4.

Diagrammatic and narrative models can be thought of as propositional non-mathematical models.[63] Diagrams refer immediately to physical objects. There are, for example, relatively fewer kinds of manipulations that can be done in diagrammatic models, as compared to mathematical models. Diagrammatic models, to speak metaphorically, do not exist in a theoretical world of their own the way mathematical models do. The biologist simply represents, say, the steps of a gene network in terms of a flow-chart. Although such diagrams, as we will see in Chapter 4, can guide research, useful manipulation tends to be done empirically, in laboratory experiments. This is because there are next to no "degrees of freedom" in the model itself and there is little internal logic to these models, independently of the concrete system that they directly represent. The models represent, but cannot provide very much independent insight as a consequence of manipulation. This is different from saying that very limited generalizations can be formed from them. A model that cannot, in general, be manipulated to achieve new results, or learn new things about nature, could still be a model with a fair amount of generalizability – it might represent, to an extent, and explain, many objects in nature, even objects of different kinds.

Consider a diagrammatic representation, such as a flow chart, of a hierarchical gene regulatory network. This representation indicates the genes and their temporal pattern of activation and inhibition, often showing early genes affecting, in a cascading fashion, downstream genes. First, note that this

[63] On diagrammatic models, see, for example, Goodman 1976, 1978; Lopes 1996; on narrative models, see, for example, Danto 1985; Griesemer 1996; Hull 1975, 1981, 1992; Richards 1981, 1992.

model cannot be manipulated. It is the result of many experiments, often using genetic knock-out techniques, employed to establish patterns of activation and inhibition of genes on other genes (patterns include one-to-one, many-to-one, or one-to-many activation and/or inhibition). For example, if knocking out a gene has particular phenotypic effects, then effort is employed to work out the mechanisms by means of which that gene achieves those effects (e.g.: What other genes does it affect? What are the biochemical pathways by means of which the genes involved aid in producing the phenotypic effect?) The manipulation exists not in the model itself—in the representation—but exists instead in the *practice* of the laboratory, which aims to figure out the causal processes at work among component parts of the system through experimental manipulations and controls. *The manipulation or intervention*[64] *work, which is essential for all science, occurs, for formal biology, often in the model itself whereas, for compositional biology, it happens in the experimental work of which a model is then made to summarize (or guide further) results*.[65] I am not denying either, or both, that empirical work is important for formal biology or that theoretical is crucial for compositional biology, but I am arguing that the locus of manipulation and resource employment is quite different in the two kinds of biological science.

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[64] Hacking 1983.

[65] I realize that this is a contentious claim that focuses on one particular understanding of models as *just* a representation device for theoretical purposes. Some might argue that the laboratory experimental work is itself guided by protocol models or what one might call "models of method or practice." Practice oriented sociologists and philosophers of science such as Bruno Latour, Elihu Gerson and James Griesemer seem to advocate a broader notion of models (e.g., Latour and Woolgar 1986; Gerson and Griesemer 1997). While I fully invite a broader notion of models, for my limited purposes in this dissertation I focus on models as a theoretical activity. So when I use the term "model" it does refer to an inscribed representation with theoretical import.

As an example of the extent and utility of generalization in compositional biology, let us explore a flow chart of, say, *Hox*-gene action. The chart might be applicable to many organisms. This is precisely because such gene regulatory networks are highly "conserved" across taxa, as work over the last 20 years on this high-level regulatory gene type has shown. Again, this can only be known through repeated inductive efforts, and the diagram must be repeatedly checked with experimentation in diverse new taxa. Although predictions for *Hox*-gene action in a previously untested target system are possible from known source systems (often "model systems"), such predictions are fallible and require constant experimental verification (and the background properties in the intensional set can repeatedly fail to apply!).[66] Flow charts of downstream and more taxon-specific genetic regulatory networks are even less generalizable.

Similar arguments could be made for narrative models, which are also descriptions, in idealized form, of the action occurring in concrete systems. Such models describe, in language, the important features of a concrete set of events; they analyze and represent such events into stages, objects and processes.

I will now turn to material models and how they relate to the other three distinctions in this chapter. Consider a scale model of the jaw of a snake. This model could show the shape, structure, and way in which the jaw disarticulates when the snake eats a large prey item. But note that it is highly abstracted – there are numerous properties of a real snake jaw that it does not show. Even a real snake jaw, showing just the bone and teeth, could not show the blood vessels, the muscles, the nerves, and could certainly not show the *active* jaw. Of course one

[66] In Chapter 5 I will discuss how model development and confirmation are difficult to separate in compositional biology.

could observe a live snake, but then one could not observe the jaw itself. Models of physical parts[67] involve abstraction in order to be useful.

A specimen removed from its habitat in nature, prepared appropriately, and stored in a museum is a *generalization* of the species in nature. The properties of the specimen represent the wider range of properties of its conspecifics in nature. Even if we have multiple specimens of the same population, these specimens are representations of the complete population in nature. They do not capture the full range of variation in anatomy, physiology and behavior of all individuals of that population. Notoriously, specimens also cannot show breeding potential between populations, which is a criterion for defining species, or general behavior of individuals. Many properties of the individuals and populations in nature are left out in the construction of what Griesemer calls "remnant models," that is, museum specimens that generalize from the species in nature.[68] Note that these models are themselves *parts* of a species, and the species that they represent can also be thought of as parts of an ecosystem.

Strictly speaking, there are two forms of abstraction going on in these cases of material modeling, both of which can be described by eliminating properties (from an intensional set). First, there is abstraction in the strict sense discussed above: properties of objects are removed. For example, the properties of the bone are lost if one makes a plastic model or the full range of variation of the population (a property) is lost due to sampling. Second, there is abstraction defined in terms of *omitting certain objects that, often, are parts of a system*. This is

[67] These models thus refer directly to part organization (distinction 4 below).
[68] Griesemer 1990; I shall return to remnant models in Chapter 4.

what Gerson calls "buffering."[69] In the jaw case, leaving out nerves or blood vessel is buffering; this can also be reinterpreted as a case of abstraction in that certain properties of the system (some of its parts, and interactions among parts, which can be thought of as relational properties[70]) are left out of the representation.[71] Note that these are two very different forms of abstraction. In the case of buffering an idealization occurs because *concrete objects* are removed from consideration in actuality – in the case of abstraction proper (removal of properties), all the concrete objects remain in actuality, but any number and type of their properties are subtracted in imagination. Buffering can be described as a removal of system properties, but the *concrete* nature of this removal—through the removal of concrete parts—must be noted.

These cases match the intensional set account of abstraction quite well, as can be seen in how straightforwardly discussion about properties applies to these model forms. What about the *components* of the material model, its parts, can they be considered theoretical kinds? I believe that they can. The poison-fangs of a snake jaw-model, for example, capture some of the important features of many concrete snake jaws in a patchy way. Naturally, they do not represent the full variation of fangs, or even the functioning of fangs, but they do represent the approximate structure and placement of these weapons and they also hint at the functioning of all such fangs in nature. Thus, the fangs represent a complex

[69] Gerson 1998 (in prep).
[70] I am grateful to Elisabeth Lloyd for this suggestion; Gerson seeks to keep abstraction and buffering as two separate and unrelated concepts (Gerson pers. comm.).
[71] Computer imaging techniques could be interpreted as providing another way of doing this. Computer modeling, in general, is an important activity that falls outside of the scope of my dissertation. Undoubtedly, a full account of modeling, and a full story of the two kinds of biology that I am here presenting, requires an analysis of computer techniques.

theoretical kind, subject to complex abstraction. Similar arguments could be made for other parts of the material model.

In conclusion, I want to explore some general considerations concerning the model forms pertinent to formal and compositional biology. Simple objects can often be usefully represented in terms of mathematical models. For example, the stability and reliability of formation of NaCl can be explained in terms of differences in a quantity, electronegativity, and the mathematically-expressed dynamics of the electron shell structure of each of the two constituent elements. The simple object "point mass" is an abstract assumption of gravitational theory; it serves a specific role in the mathematics involved in this theory. However, simple objects can also sometimes be explained, with significant loss of realism, by material models.[72] NaCl can also be represented in material model form. However, such a model does not adequately represent the notion of charge nor probability distribution of electron location. The latter quantum phenomenon is one of many phenomena that cannot be represented in macrophysical models, but can be represented in mathematical models.

Conversely, complex objects can, with significant loss of realism, be represented in mathematical models. For example, when computationally-tractable mathematical models of gene networks are presented they often do not capture much of the real nature of the action of real genes.[73] They may capture the dynamical patterns of "interaction," but they do not capture the actual biochemical mechanisms underlying the dynamical patterns. Complex objects are often best represented by propositional non-mathematical or material

[72] Hence, my first and third distinction do not completely covary.
[73] For example, Kauffman 1993; von Dassow et al. 2000.

models, whether scale, remnant, diagrammatic, or narrative. Complex objects or processes that differ from each other (of even the same kind!) in important qualitative ways not captured well by quantitative methods, even in a space of sufficient dimensionality, are better represented in terms of propositional non-mathematical and material models.[74] For example, it is theoretically easier and more accurate to describe different cell or tissue types with qualitative properties rather than mathematical theoretical kinds. It is more pertinent to articulate the reactions occurring in the Krebs cycle of biochemical respiration using qualitative aspects of the types of chemicals involved (e.g., how one enzyme type removes a certain part of a molecule to form another molecule). Explaining the mechanisms among parts is a science of qualities, not a science of quantities. Furthermore, articulating the *functions* of parts can also not be done usefully in mathematical expressions but requires, rather, an explanatory narrative. These are themes to which I shall return in Chapter 4. Suffice it to say here that the models of compositional biology rely on complex abstraction on parts, which are complex objects, of a system.

[74] This is not to say that mathematical models never serve a purpose in compositional biology. For example, mathematical models developed by systems theory in the 60s and 70s were explicitly meant to analyze compositional systems. Furthermore, recent work developing formal models in, for example, genetic regulatory networks (e.g., Kauffman 1993; von Dassow et al 2000) and communication and task allocation in ant colonies (e.g., Gordon et al 1992.; Franks and Tofts 1994) also shows the presence and occasional utility of mathematical models in compositional biology. However, it is clear that such models are rare and are often received with skepticism.

1.5 Idealization or Irrelevance versus Relevance of Part-Whole Organization

The fourth distinction concerns the importance, and conceptualization, of part-whole organization in the two kinds of biology. My argument is that formal biology either ignores or has a very idealized notion of parts (e.g., demes of a population), which is defined by a few mathematical properties and does not capture the mechanistic details of these parts. Compositional biology, on the other hand, is specifically concerned with working out the details of the compositional system, including the structure, process, and function of its hierarchically-organized and integrated parts.

In philosophy, wholes and parts, despite their importance, have received significantly less attention than either the kinds and instances, or objects, properties, and relations. Philosophy of science has also not concerned itself very much with them, despite their significance in, for example, the biological sciences and in articulated complex systems.[75] In order to motivate an analysis of parts, let me first define exactly how I will use "parts" and "wholes." There are two ways in which I will *not* use these terms. First, there is a tradition concerning what one might call "parts of ideas" in which mental constructs such as ideas, classes, (intensional!) sets, etc. are thought of as being decomposable into sub-ideas, sub-classes, or even aggregations of extensional objects.[76] Here, "part" is used in the sense of "part of a concept/idea." I am specifically not using part in this way as

[75] Although, see Kaufmann 1971; Levins and Lewontin 1985; Lewontin and Levins 1988; Nagel 1961; Wimsatt 1974, 1986.
[76] For example, Lewis 1990.

this is not, and could not be, the object of study of any scientist, biologist, physicist or chemist.

Second, there is an intellectual discourse interested in what I will call "idealized parts" in which assumed concrete objects of any sort are idealized as individuals and then a logic of aggregation is developed that is supposed to capture concepts such as identity, existence, uniqueness, etc. This analysis of wholes and parts is found particularly in abstract metaphysics and logic. Here parts have been defined by very abstract spatio-temporal logical criteria. The modern investigations in mereology (the study of parts) by, among others, Lésniewski, Carnap, and Leonard and Goodman were concerned with the abstract formal properties of individuals and their spatio-temporally defined parts.[77] Contemporary discussions continue in this vein.[78] The relevance of abstract mereology to dynamic and functionally-based biological systems seems very tenuous, if at all existent. This is because, in the metaphysical and logical tradition of mereology, the material nature, interaction, and theory-dependence of the objects are all ignored – objects and processes *qua* parts are idealized to the extent that they can no longer be scientifically studied. Often, only topological relations among parts are considered interesting.

In this dissertation, I shall *not* be concerned with these views on parts (and wholes). I consider the "part of a concept/idea" a branch of set-theory or, more broadly, logic. This does pertain to my analysis of abstraction using intensional sets; it is irrelevant, however, for an analysis of parts as studied by scientists. I view the "idealized part" concept as an extremely impoverished notion of parts

[77] Leonard and Goodman 1940; Simons 1987; Frederick Schmitt pers. com.
[78] E.g., Bahm 1972; Burge 1977; Lee 1977; Ruben 1983; Simons 1987; Lewis 1990; Sanford 1993; Scaltsas 1990; Simons 1987; Van Inwagen 1990, 1993.

and have not found it useful at all in an analysis of how parts are actually investigated in biology, whether it be formal or compositional. Even in formal biology, the parts are idealized using mathematical, not metaphysical, *tools*. Furthermore, the *purposes* of part idealization are also very different in mereology and in formal biology.

The parts that I am concerned with are the ones pertinent to scientific investigation. Many systems in nature are composed of parts. The paradigmatic system for such an analysis, currently as well as in the history of philosophy and of science, is, of course, the organism.[79] But other kinds of concrete systems also have parts, in some sense: planetary systems with a central star; atoms with protons, neutrons, and electrons; (perhaps) light beams with "photons"; artificial mechanical systems such as bridges and houses; artificial electronic systems including computers; and, in biology, genes in the genome, organisms in populations, populations in species, and species in clades. This list is not exhaustive by any means.

In this section of the chapter I argue that although *some* of the systems investigated by theoretical physics and formal biology can be thought of as compositional, the theoretical focus in analyzing these systems is to abstract and

[79] Two particularly captivating accounts, which discuss the relationships between mechanism and teleology in their analysis of the integrated and compositionally hierarchically-organized organism, are Aristotle's *Generation of Animals* (1990) and *Parts of Animals* (1993) and Kant's *Critique of Judgment* [the Third Critique, 1952 (1790)]. The conceptual confusion that the organism has caused vis-à-vis especially teleology is captured in Kant's statement that "it is absurd for men… to hope that maybe another Newton may some day arise, to make intelligible to us even the genesis of but a blade of grass from natural laws that no design has ordered." (Kant 1953 (1790). This confusion is evidence for the significant differences between an analysis of the compositionally-organized organism and abstracted physical "mechanistic" systems. In a sense, it is precisely this difference that I am trying to capture in my dissertation.

idealize mathematically, rather than provide representations and explanations of the compositional organization. Put differently, formal biology does study, for example, genes and populations as components of systems (i.e., the genome and metapopulations, respectively), but the emphasis is undoubtedly on abstraction through finding a few simple mathematically-expressed relations between the few properties of the objects investigated. Compositional biology, on the other hand, seeks to describe and explain, through a variety of model forms and a particular kind of compositional explanatory pattern to be explored in detail in Chapter 5, the compositional structure, process, and function of a system. It does not necessarily seek to capture the few essential properties, precisely because parts, which are non-equivalent within a kind, and are complex objects, rarely have such properties.

Much of theoretical physics is interested in characterizing the properties and relations of highly abstract objects (e.g., electrons, fundamental particles, photons, charges, point masses, etc.). Although these can be sometimes be thought of as components of a system, articulating their general properties and relations between such properties captured in mathematical laws are the focus of investigation. Expressing such laws does not require considering them as parts of the concrete system in which they are (sometimes) found. Furthermore, even when characterization of a "system" is at issue (e.g., the Schrödinger equation for some element, such as Helium) the parts of the system are not considered interesting qua material parts. Abstractions, not partitioning, are of interest here. In these cases the following are not considered of interest, or even coherent: (1) feedback processes among parts, (2) the function of parts (and what has, loosely,

been called, "teleology"), (3) the uniqueness of particular parts (i.e., non-equivalence/non-interchangeability of parts).

Furthermore, certainly it is true that aspects of the mathematical laws and principles were discovered through material or thought experimentation on compositional systems, loosely speaking. For example, the solar system, mechanical systems with levers and pulleys, electric circuits, atomic structures, multiple wave sources, etc. were used to determine abstract laws, but, again, the theoretical goal was not to describe these particular "compositional" systems, but rather to explain the mathematical properties, as well as the laws and principles employing those properties, of the objects and processes under the purview of the particular area of physical theory. Compositionality in much of theoretical physics is not considered important – it is almost irrelevant.

There is, however, an area of theoretical physics, in which consideration of, and explicit reference to, compositional systems seems particularly important – thermodynamics. Here the behavior of *populations* of molecules is explicitly explained in terms of the behavior of the component/part molecules (e.g., heat content explained in terms of average speed of the molecules). I still think that this is quite different from the compositional focus that exists in compositional biology. Compositionality in thermodynamics is much less rich, refers to significantly fewer kinds of relations, is completely "additive" in both the mathematical and metaphorical sense, explicitly assumes the equivalence of component "parts," has no functional or "teleological" component, and is completely captured in precise mathematical formulations.

Furthermore, the goal of (statistical) thermodynamics is, again as in mechanics, to articulate general principles among variables that do not refer to

the compositional structure (e.g., heat content, efficiency, entropy). In short, while many areas of theoretical physics rely on compositional systems for discovery of theoretical principles, and while a few areas contain compositional systems, in a loose way, in their theoretical principles, the goal, as discussed in the preceding three distinctions, is to describe through mathematical laws and models the essential simple properties of the simple objects through simple abstraction; as we shall further explore in chapter 5, these laws and models can often be unified. It is in these senses that parts are irrelevant to most of theoretical physics, or highly idealized in an area that can be construed as compositional, thermodynamics.

The goal of mathematical evolutionary genetics and theoretical mathematical ecology, key cases of formal biology, is, methodologically speaking, the same as the goal of theoretical physics, as I have been arguing throughout this chapter: seek general and idealized mathematical laws and models. Although the genome is clearly a compositional system with multiple interacting genes, the theoretical framework of mathematical evolutionary genetics abstracts away from this in a number of ways that I will detail. Furthermore, the compositional and hierarchical structure of populations is also abstracted in a number of ways. In what follows, I will show how compositional organization is made irrelevant or highly idealized by the mathematical evolutionary genetic framework.

Typically, the models of evolutionary genetic focus on the properties of individual genetic loci – an "additive" bias is quite powerful in this field.[80]

[80] See, for example, Lloyd 1988, chapters 5 and 7; Wade and Goodnight 1998; Wimsatt 1984.

Evolutionary forces are often represented by causal factors acting on single loci. Consider the Hardy-Weinberg principle (in its simplest form), the infinite-allele model of mutation, Fisher's fundamental theorem of natural selection, and definitions of genetic additive effects (on genetic variance) in quantitative genetics. A powerful claim for the legitimacy of such an additive bias stems from George Williams's argument for the universal validity of calculating additive genetic effects by averaging the effect of a gene across all genetic backgrounds in a population; this is justified, according to Williams, "No matter how functionally dependent a gene may be, and no matter how complicated its interactions with other genes and environmental factors."[81] Under this view, genetic context is considered unimportant because it can be averaged out of causal existence. This is a prevalent view in population and quantitative genetics, and is part of the Fisherian school of thought, to be further discussed in Chapter 3.

Of further interest for the relationship between theoretical physics and formal biology is the fact that Fisher even employed an analogy between natural selection and thermodynamics:

> It will be noticed that the fundamental theorem [of natural selection]… bears some remarkable resemblances to the second law of thermodynamics. Both are properties of populations, or aggregates, true irrespective of the nature of the units which compose them; both are statistical laws; each requires the constant increase of a measurable quantity, in the one case the entropy of a physical system and in the other the fitness… of a biological population.[82]

Fisher did qualify this analogy by stating that, for example, that thermodynamic systems are permanent whereas biological systems are not and that entropy leads to disorganization whereas higher fitness leads to "progressively higher

[81] Williams 1966, p. 57.
[82] Fisher 1958, p. 39.

organization in the organic world."[83] Despite these qualifications, it is clear that Fisher had a methodological orientation compatible with that of theoretical physics.

Let us return to the notion of genetic average effect. With the presence of population structure and subdivision, averaging across all individuals in the *total* population is no longer empirically adequate or justified because individuals in different subpopulations are *isolated*, to an extent, from other individuals and "experience" a different genetic background. That is, the gene frequencies of particular loci are different in different demes. This means that particular gene alleles that are positively correlated in one deme could be negatively correlated in another deme, hence the additive genetic effect of a particular locus will *differ* in the two demes.[84] With population structure we can no longer average across total (meta)population background. *Compositional* organization has become crucial from a theoretical point of view. This point is made and explored in some of the key textbooks, for example Hartl and Clark 1989 on population genetics,

[83] Fisher 1958, p. 40.

[84] This is a separate point from the importance placed on interactions between pairs, triples, etc. of loci using the additive (and dominance) effects of *each* locus. This latter point concerns the relative weight placed, theoretically and empirically, on interaction effects built up, using, for example, analysis of variance, from additive effects, for calculation of total genetic variances in the population. (See, e.g. Falconer and Mackay, pp. 129-130; Wade 1992, pp. 39-42.) Here too there is an additive bias in the field, in the sense of being biased against the importance of interaction. Falconer and Mackay, for example, write: "[Statistical] Interactions involving larger [than three] numbers of loci contribute so little variance that they can be ignored, and we shall confine our attention to two-factor interactions since these suffice to illustrate the principles involved." (p. 129) Wade 2000 argues that given enough loci (and, in reality, there are *many* loci) the number of three-way and higher order interaction terms between as many loci (e.g., three-way interactions between three loci) become, in aggregate, "orders of magnitude greater than the number of additive and dominance terms typically used to describe evolutionary processes." (p. 215) For Wade, then, we are unjustified in ruling out *a priori* the effect of these interaction terms on genetic variance, irrespective of the existence and nature of population structure.

and Falconer and Mackay 1996 on quantitative genetics, even if it is underplayed particularly in the latter and in a fair amount of the theoretical work presented in population genetics and quantitative genetics.

Yet, even when gene interaction becomes important due to population structure, and both are investigated and modeled[85], the amount and kind of compositionality is highly limited and idealized. Few mathematical properties of the highly abstracted parts are measured or postulated [e.g., additivity, dominance, and interaction phenotypic and fitness effects; gene frequency in, and sizes of, hierarchically-structured populations; inbreeding effect/F-statistic (indicating extent of population subdivision)]. From very few variables, a fair amount of the theoretical structure of mathematical evolutionary genetics concerned with population structure has been developed.

In these cases, kinds of idealizations concerning compositional structure similar to those in theoretical physics get made. Consider, for example, that genetic effects are measured in terms of effects on the phenotype or on fitness rather than in terms of an understanding of the *content* of the effect (i.e., what is the mechanistic cause? What is the qualitative nature of the interactions?) The different loci are still considered qualitatively equivalent in that each of them has the *same* kinds of variables and parameters assigned to them, even if they have different quantitative values for the given variables and parameters. The detailed nature of the myriad qualitative processes occurring is not considered. Furthermore, in so far as population structure is concerned, the parts there are also considered equivalent and defined by a few variables and, therefore, few

[85] See Wolf et al. 2000 as well as Wade et al. 2001, and references therein. See also Christiansen 1999; Otto and Feldman 1997; Wagner et al. 1998.

relations exist between them, which can indeed be expressed mathematically. Furthermore, both for gene interaction and population structure, in so far as the mathematical theory is concerned, the parts do not, in any sense, exist "for the sake of the whole" – they are not mutually-determining and necessary as they are, for example in organismic development or in the appropriate functioning of biochemical networks in molecular biology (e.g., Krebs cycle).[86] It is to these "incarnate" parts, the components investigated by compositional biological, that I now very briefly turn.

Concrete parts and wholes are studied primarily in complex, integrated, and hierarchically-organized systems with division of labor. In compositional biological sciences, such as comparative anatomy, developmental biology, physiology, biochemistry, cellular biology, and even systematics[87] integrated, hierarchical and functionally-distinct parts are the central objects of investigation. System partitioning and articulation[88], in empirical and theoretical activity, are the primary concern.

The parts and wholes of compositional biology are the topic of much of this dissertation. Chapters 2 and 4 are dedicated, respectively, to how different

[86] Although we no longer believe in "teleology" in scientific biology, there is a sense in which the issues of mutual part-whole determination and necessity still remains an important theme, captured particularly well by both Aristotle and Kant. See, for example, McLaughlin 2001.
[87] Although systematics does not study the functioning of organic systems, it does study parts in order to establish hierarchical similarities across systems to infer genealogy. The detailed particularities of parts of different species are compared to establish similarities and differences across taxa. This is the comparative method. Systematics does use computer algorithms and quantitative methods of analysis to find most likely trees, but I interpret these methods as methods and not as theory. Theory in systematics is genealogy inferred from similarity of the parts of the species. It is, unfortunately, beyond the scope of this dissertation to analyze systematics.
[88] See Winther 2003 (in press).

theoretical perspectives analyze different kinds of parts via their different "frames of partitioning" and to how models concerning these parts are articulated. Half of chapters 3 and 5 also concern how compositional biology, respectively, studies parts through the discipline of evolutionary developmental biology and how various aspects of parts are explained. Here I will thus not repeat my downstream analysis of parts.

Whereas parts are the central object of study in compositional biology, in theoretical physics and formal biology, parts are considered irrelevant or are highly idealized.

1.6 Coda: Summarizing the Difference Between Formal and Compositional Biology

The analysis I have just provided for the two kinds of science is summarized in the following table.

	Formal Theoretical Physics	**Formal Biology** (e.g., Mathematical Evolutionary Genetics)	**Compositional Biology** (e.g., Developmental Biology, Physiology, Biochemisty)
1. Causal Structure of Objects Studied	Simple Objects	Indeterminate; Simple Objects	Complex Objects
2. Abstraction of Objects	Simple Abstraction	Simple Abstraction	Complex Abstraction
3. Models Used	Mathematical	Mathematical	Propositional Non-Mathematical or Material
4. Relevance of Part-Whole Relations	Irrelevant or Idealized (for Thermo-dynamics)	Irrelevant or Idealized	Crucial

Table 2. **(1.2)** A comparison of the two kinds of biology and theoretical physics with respect to the four distinctions.

Note that, when comparing methodological commitments and goals, formal biology is much more closely related to theoretical physics than to compositional biology. A cladogram, a sort of "branching resemblance tree"

indicating similarity, would show the two formal sciences as much more similar than either is to compositional biology. This indicates, I think that the deep commitments of a broad scientific perspective play a larger role in organizing the work and theory of science than the area of study (e.g., simple physical vs. complex animate objects). The commitments of formal versus compositional biology (and science) are different with respect to every distinction of my framework, as can be seen in Table 2. (1.2). Are these differences merely descriptive ones that are a consequence of particular historical developments or are they "in principle" differences that stem from deep in the logic and methods of the two kinds of science? Answering this question gets to the heart of the issue of whether a synthesis between these two kinds of science is possible and, more fundamentally, what a synthesis even means (e.g., compatibility, but independence, of distinct theories *or* deep integration of different theories). I will leave an exploration of this question to Chapter 3, but I hope that in this chapter I have made it clear that we need philosophical tools, particularly from the field of metaphysics and epistemology, to tease apart differences in methodology and goals between formal and compositional biology.

Chapter 2: An Empirical Analysis of Theoretical Perspectives in Compositional Biology

2.1 Framing and Goals

In Chapter 1, I made a distinction between "formal biology" and "compositional biology" that I developed using four distinctions: (1) attention to simple versus complex objects, (2) employment of simple versus complex abstraction, (3) usage of mathematical versus material or non-mathematical propositional models[89], and (4) consideration of part-whole organization as irrelevant or idealized versus relevant.[90] There, I provided the grounds for a clear distinction between the two kinds of biology *primarily* through their different practices of abstraction, which includes their practices of modeling.[91] [Although I did mention parts (e.g., as (1) complex objects, as well as (4) being relevant for

[89] Note, as I argued, that compositional biology does, on rare occasions, use mathematical models that refer explicitly to the material part structure and organization (e.g., topological models for developmental biology) – these mathematical models could in principle be mapped in a state space and thus be subject to a semantic view analysis, but they rarely are and, furthermore, they are not subject to as much independent mathematical manipulation as models of theoretically (in the mathematical sense) rich mathematical evolutionary genetics. Furthermore, mathematical evolutionary genetics and theoretical ecology do, on occasion, use material and even, for example, diagrammatic models (see, e.g., Griesemer 1991a; Plutynski 2001). But the contrast I am making here is based on a strong correlation between form of model used and kind of biology.

[90] In Chapter 5, I develop a fifth distinction having to do with the patterns of explanation common to both kinds of biology. For the sake of clarity and linear argument development, I will not be concerned with this distinction until then.

[91] I will return to this theme in Chapter 4.

compositional biology, but either irrelevant or highly idealized for formal biology).] The attention given to the first three distinctions was disproportionately large compared to the last, because both types of science rely heavily on abstractions (including models) whereas only compositional science focuses on parts.[92] In this chapter, I will complement the analysis of abstraction given in the first chapter with an explicit analysis of parts.

The goal of this chapter will be, first, to discuss theoretical perspectives, as consisting of guiding biases and assumptions. I will employ the work of Kauffman, Wimsatt, and Griesemer, but will develop my own views on theoretical perspectives and their relationship to disciplines. I will also show that textbooks can be used as a source from which to glean the guiding biases and assumptions of the theoretical perspective of a discipline. Furthermore, such perspectives exist in both formal biology and compositional biology, but they play different roles in these two kinds of biology.

After this general discussion, I will provide an *empirical* analysis (*sensu* conceptual data gleaned from textbooks, for a philosophy of science analysis) of theoretical perspectives in compositional biology. I will examine a set of disciplines in compositional biology—comparative anatomy, functional morphology, physiology, developmental biology, cell biology, and biochemistry—and explicate their respective guiding biases and assumptions. I

[92] In distinguishing between the two kinds of science (or even two things in general), one can discuss either those aspects that they have in common, but might differ somewhat on, or those aspects that are completely different. Of course there is a continuum between these (i.e., if common aspects differ too much, then such aspects become different aspects), but discussing abstraction in the two types of science falls in the former category (i.e., both sciences abstract, but do so differently), whereas discussing parts falls in the latter category since parts are the sole purview of compositional biology.

will extract the assumptions and biases guiding each field by analyzing the organization and content of canonical advanced textbooks in each of these six fields in compositional biology. One result of my analysis is that each of these disciplines partitions a system very differently. Compositional biology is a non-unified (and, potentially, "non-unifiable") endeavor consisting of multiple perspectives, an important reason for the complexity of its objects and its processes of abstraction.[93] I will summarize my investigation in a table indicating, for each theoretical perspective (discipline), (1) examples of parts, (2) criteria of partitioning, and (3) general lists of guiding biases.

After presenting my study of textbooks, I will relate the results of my analysis to the framework developed in Chapter 1. The series of six case studies investigated here teach an interesting lesson. My general framework paints the picture of two distinct "ideal types" of kinds of biology, formal and compositional. But, although each of the six case studies is a clear and recognizable case of compositional biology, some of them fail to meet exactly *all* the four distinctions of Chapter 1. In particular, the existence of complex abstraction and the use of material or non-mathematical propositional models are sometimes not met completely in the theoretical perspectives of compositional biology explored below. For example, functional morphology has

[93] To what extent formal biology, and formal science, such as theoretical physics, is unified and unifiable is an open question. For arguments in favor of its unification see Friedman 1974, 1981, 1983; Kitcher 1981, 1985; Smocovitis 1996; for arguments against its unification see Galison and Stump 1995; Cartwright 1999. As I will discuss in Chapter 5, I ally myself with the view that unification is possible, desirable, and even actual in some of the theoretical work of formal science and formal biology. For instance, Michael Wade noted that he considers the Price-Hamilton formulation of kin selection and group selection an important piece of unification with a hierarchy of abstract models articulated in, for instance, Wade 1985; see also Frank 1997, 1998.

a mathematical component that assumes (to an extent) simple kinds and employs formal models. And the molecules of biochemistry can be thought of as simple objects, to an extent. Although some of the theoretical perspectives I discuss sometimes have formal aspects, they are still a compositional biology, as I show below. As a result of both such intra-category variation, and complex abstraction in *delineating a kind of biology*, defining them and determining their extension cannot be done through the dogmatic exercise of applying necessary and sufficient conditions.

2.2 On Theoretical Perspectives

In what follows, I will first turn to an analysis of Wimsatt's, Kauffman's, and Griesemer's notion of theoretical perspective. Although their views differ, their opinions provide some common ground for defining this concept. After providing a robust view of theoretical perspective, I provide a map for how I will employ this concept in my analysis of the different methods of partitioning adopted by the different disciplines of compositional biology. The sources of data for my philosophical (not scientific!) empirical analysis are the canonical textbooks of each discipline, a method for which I provide justification.

2.2.1 Kauffman, Wimsatt, and Griesemer on Theoretical Perspectives

In philosophy of science, there is a long tradition of describing and emphasizing the importance of the theoretical units involved in scientific activity. In the 20th century, philosophers such as Kuhn, Lakatos, and Quine

emphasized the theory-ladenness of both observation and experiment in their respective notions of paradigm, research program, and web of belief. In philosophy of biology, the importance of theoretical units in guiding scientific activity has been developed in the work of Kauffman, Wimsatt, and Griesemer.[94] Their work has numerous similarities, including an emphasis on the guiding biases and assumptions of the theoretical units and a concern with, primarily, the static aspects of scientific theory as opposed to a focus on change and its causes, which are two themes that Kuhn, Lakatos, and Quine stress. I am particularly drawn to their work because it focuses on biology and it takes the compositional organization of biological objects, in nature and in theory, as central. This is *not* a topic of concern in Kuhn, Lakatos, and Quine, who develop a more general account of scientific theory and its processes of accommodation of, and change in response to, novel phenomena, theories, and methods.

I will now turn to an analysis of theoretical perspectives, with a particular focus on biases concerning partitioning, as it appears in Kauffman's, Wimsatt's, and Griesemer's work. After this, I will articulate my own views on these issues.

In an important paper that has practically been forgotten, the biologist Stuart Kauffman develops a particular kind of explanation that he calls an "articulation of parts explanation." He justifiably claims that these explanations are "prevalent in biological sciences."[95] Such an explanation emphasizes the processes by means of which the parts of a system interact to cause a certain outcome.[96] The explanation *is* the account of how such parts and processes

[94] Kauffman 1971; Wimsatt 1974, 1994; Griesemer 2000. Also Gerson pers. com.
[95] Kauffman 1971, p. 257.
[96] As we will see in much more detail in Chapter 5, this explanation is an instance of a Cummins' style analytical explanation that appeals to the capacities of parts

articulate to cause an outcome. Presumably, the description of the activity of a transmembrane protein sensitive to some extracellular chemical (e.g., a hormone) which then releases an intracellular molecule involved in some metabolic cascade would count as an articulation of parts explanation. Kauffman remains vague both in his account of what an explanation is and in what the "articulation" relation is (i.e.: is it the way that parts relate to one another via processes as the "glue"? is it the relation between parts and processes? is it the relation of the part-process system to the outcome?).

Despite the weaknesses of his analysis of explanation and process, Kauffman's concept of "adequate description" is a useful one similar to Wimsatt's and Griesemer's concept of "theoretical perspective." He writes, "[1] Given an adequate description of an organism as doing any particular thing, we will use that description to help us decompose the organism into particular parts and processes which articulate together to cause it to behave as described. [2] For different descriptions of what the organism is doing, we may decompose it into parts in different ways."[97] An "adequate description" thus provides grounds for partitioning an organism (in different ways for different descriptions); "theoretical perspectives," as we shall see, play the same role.

In an effort to provide further theoretical underpinnings for his notion of an adequate description, Kauffman develops the idea of a "conjugate, coherent decomposition." For his argument to be internally consistent, the system

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of a system to explain a capacity, from a particular theoretical point of view, of the system. Part capacities are different in kind from one another and are simpler than system capacities.

[97] Kauffman 1971, p. 258.

decomposition must *be determined* by the adequate description. Kauffman explains:

> Parts and processes are accepted more or less jointly, and with them, the adequacy of a particular articulation of parts explanation in which just those parts and processes are seen as fitting together to yield the behavior in question. Other causal consequences of these parts are then considered irrelevant. I will call such a decomposition of a system a conjugate, coherent decomposition, for it is conjugate to a particular view of what the system is doing, and coherent in that it provides an articulation of parts explanation of how the system does whatever is specified in that particular view of it.[98]

An adequate description thus sets the framework for a decomposition of the system, which ultimately provides a description-specific articulation of parts explanation. This is exactly the same inferential process as in Wimsatt 1974 where a theoretical perspective sets the framework for a part-decomposition of the system, which, in turn, suggests an explanation of how the system performs its perspective-specific behavior.

Consistent with, and inspired by, the themes in the science of the 60s and 70s, Kauffman suggested that a purely symbolic "cybernetic model" first be developed when attempting to develop an articulation of parts explanation. This model would only subsequently be filled in by actual causal mechanisms. Thus, the adequate description was supposed to produce a decomposition that then specified an abstract and symbolic cybernetic model that then, together with "a specific set of causal mechanisms," became a "hypothesis requiring verification."[99] Kauffman fails to follow up this inferential train with a theory, or even an account, of hypothesis (or model) *confirmation*. Kauffman also does not present the form that such a cybernetic model would take, but presumably it would

[98] Kauffman 1971, p. 260.
[99] Kauffman 1971, p. 263.

consist of (feedback) relations, of various strengths of correlations, between variables – these variables would fundamentally refer to the part-whole organization of the system, thus while the model is mathematical (my 3rd distinction), it refers intimately to the part-whole organization (my 4th distinction).

Whereas Kauffman focuses on a particular form of explanation, Wimsatt is concerned with the overall theoretical structure of different biological sciences, including their guiding biases and assumptions, particularly with respect to the activity of partitioning. In a relatively recent article, Wimsatt displays his broad concerns in that he discusses the role of problems and "explanatory closure" (see footnote 109) in theoretical perspectives, "there is a reasonably well-defined class of problems which can be solved without bringing in information from outside the perspective. …Thus there are paradigmatic anatomical, physiological, and genetic problems, though no one believes that these approaches individually exhaust what may be said about the organism."[100]

In an almost classic article 20 years earlier, Wimsatt notes that each theoretical perspective "implies or suggests criteria for the identification and individuation of parts, and thus generates a 'decomposition' of the system into parts."[101] Wimsatt then argues that such decompositions could be simple (for the objects of physics) or complex (for biological systems). A very informative figure, presented by Wimsatt, representing the partitioning of a rock and a fruit fly under distinct theoretical perspectives captures this distinction between simple and complex partitioning (see Appendix Figure 2.1). The spatial partitioning of a

[100] Wimsatt 1994, p. 260.
[101] Wimsatt 1974, p. 70.

rock remains fairly robust under different theoretical perspectives, but the spatio-temporal partitioning of a fly certainly does not.

Wimsatt contrasts this *compositional* activity of partitioning with the *abstractive* activity of representing a hierarchical and complex part-whole system in terms of highly abstract state variables.[102] Note that as was the case with Kauffman, Wimsatt's suggestions regarding mathematical abstraction specifically concern part-whole systems rather than, for example, the properties of electrons or monadically-considered genes. Wimsatt's abstract representation is very difficult to follow (Appendix Figure 2.2). As I understand it[103], the properties of a system are quantified, under a particular theoretical perspective, as state variables (to eventually make a state space) and then the strengths of causal interactions (determined from statistical associations?) between these variables are assessed. Sets of state variables with strong mutual causal interactions are then partitioned as clusters or parts (i.e., S_1, S_2, and S_3 in 2a). By reducing state space dimensionality, such parts would seem to make modeling as well as numerical analysis simpler.

It is unclear to what extent these state-variable mathematical parts map onto the concrete parts of an organism (under any theoretical perspective). Although numerous concrete parts would seem to have greater intra-part as compared to inter-part causal interaction, thereby providing the grounds for a tight mapping, state-variable parts are not an important, common, or even

[102] Wimsatt 1974, pp. 72-74.
[103] I thank Elisabeth Lloyd for extensive discussions on this part of Wimsatt's paper.

necessarily useful tool for compositional biology[104]. Wimsatt further claims that when there are strong causal interactions between state-variable parts *across* theoretical perspectives, interactional complexity exists, whereas when there is no such cross-perspective interaction, interactional simplicity exists. Wimsatt does not clarify what allows for the *measurement* of cross-perspective interactions. Presumably some meta-perspective measurement or mapping methods are necessary. Wimsatt's account of interactional complexity is opaque.

Let us turn to comparing one aspect of Kauffman's and Wimsatt's respective arguments. An interesting difference in their accounts of part decomposition and mathematization of system descriptions is that for Kauffman the two are related. A symbolic cybernetic model provides the ground for formulating hypotheses about the appropriate causal interactions between concrete parts. For Wimsatt the two are unrelated and are distinct ways of representing the system. Given the history of the compositional biological sciences examined here, I would say that Wimsatt's understanding is more accurate. Few, if any, practitioners in compositional biology have actually employed the "top-down" hypothesis-generation methodology that Kauffman suggests. This is not to say that such practice is impossible, but the burden of proof is on Kauffman, and those following his ideas, to show that such praxis can be useful and effective in compositional biology.

Similarly to Wimsatt, Griesemer is also concerned with the overall structure of scientific disciplines but, in addition, he is also concerned with the

[104] Although this by itself is certainly not an argument against the utility of mathematizing certain aspects of compositional biology. Consider the work of Kauffman 1993, von Dassow et al. 2000, Gordon et al. 1992 even if such work has been largely ignored.

material practice of the science and the way that theoretical perspectives "coordinate" models, protocols, and phenomena.[105] In a figure (Appendix Figure 2.3) expanded from Ronald Giere's model of scientific reasoning, Griesemer suggests that physical *images* are the "concrete expression of theoretical perspectives."[106] It is unclear how images can *always* be the expression of all theoretical perspectives, at different levels, for all biological (or physical) sciences, formal or compositional. Griesemer seems to be overextending his claim, but the idea that there are *mediating* representations (and/or activities) between a perspective and its models, protocols, and phenomena is a very provocative one. Griesemer is not concerned with either the role perspectives play in partitioning systems or in the way that systems could be represented mathematically. His concern is to describe perspectives in a very broad and, possibly, complete sense – a laudable goal indeed. I will now turn to my more modest interest and comprehension of theoretical perspectives, which borrows much from Kauffman's, Wimsatt's, and Griesemer's accounts, but also differs from them at times.

2.2.2 My Account of Theoretical Perspectives

Phenomena, methodologies, and theories are all aspects of scientific activity coordinated by theoretical perspectives. These activities provide answers to the research questions deemed of interest by that perspective. This immediately raises an interpretative problem: is a perspective the *collection* of all scientific activities (including, for example, methodologies and models of various

[105] See, e.g., Griesemer 2000; Griesemer and Gerson on protocols.
[106] Griesemer 2000, p. S349.

types, as well as the activities—mental, physical, and social—linking models and methodologies necessary to confirm the models) or is it the broad biases[107] and assumptions (some implicit and hidden) *guiding* all this activity? That is, is the perspective an aggregate of the multi-level and multi-type scientific activity or does it consist only of the biases coordinating and *prescribing* such activity? So, for example, does the theoretical perspective of developmental biology include all the details of (i) lab discussions and meetings, both to discuss progress and to troubleshoot, (ii) implementation of specific molecular biology protocols for particular purposes, (iii) the physical reagents and instruments, and (iv) the general biases directing research, all four used for a particular purpose (e.g., to determine the size of a DNA clone) or does a perspective consist, primarily, only of the general biases directing the researcher in all these activities (e.g., "set up a PCR/gel electrophoresis experiment to determine the size of your DNA clone")?

This is a well-known question with analogies in other fields such as developmental biology or philosophy of mind: when attempting to explain how a system changes over time, or even how a system works, should we focus on the *dynamics of the system as a whole*, such as the developing organism or the mind-body-action complex *or* should we be concerned with *capacities of a few (kinds of) parts* in the system that seem to have special power either causally or representationally (or both), such as genes or intentional states?

[107] Note that by "biases" I do not intend to imply that the assumption is devious or reprehensible, as vernacular usage suggests. Rather, I intend to use the word to encompass explicit and implicit, "objective" and "subjective," examined and unexamined, high-level and low-level, deep and superficial, etc. assumptions guiding scientific activity.

I do not think that this problem has been satisfactorily addressed in either Wimsatt's or Griesemer's work. Griesemer's image of scientific reasoning[108] suggests the interpretation that perspectives are guiding assumptions. I believe that there is an ambiguity in the term "theoretical perspective" and that, depending on the purpose of the philosopher or sociologist of science, the term can be used as either a descriptor of scientific activity or the biases guiding such activity. In what follows, I will employ the term in the sense of guiding biases as well as assumptions. For any particular perspective, such biases and assumptions are more compact and different in kind than the sum total of scientific activities.[109]

The guiding assumptions (constituting theoretical perspectives) are distinct in kind between formal biology and compositional biology. In formal biology, for example, assumptions govern the abstraction of simple kinds (in particular mathematical kinds) from simple objects. Abstraction of a simple kind, as discussed in Chapter 1, is based on very few, easily quantifiable, properties that can be captured in relatively simple laws. In population genetics, for example, quantities such as gene frequencies, fitnesses, and population sizes are used in models and empirically measured. Additive or multiplicative genetic models are based on, and provide, different assumptions guiding the theoretical work employing such quantities.[110] This example also brings forth the hierarchical structure of perspectives, because both additive and multiplicative

[108] Griesemer 2000, p. S349, Appendix Figure 2.3.
[109] These biases and assumptions also guide an "explanatory closure" between the theoretical goals of a discipline (e.g., produce models of a certain sort, explain or describe certain kinds of phenomena) and the methodologies for achieving such goals. (I owe this point to Frederick Schmitt.) The guiding biases direct scientific activity, in a discipline, in a consistent manner.
[110] Wade et al. 2001.

genetic models are aspects of the single theoretical perspective of mathematical evolutionary genetics, but they themselves constitute two different perspectives at a more detailed level of analysis.

Furthermore, in formal biology, and in formal science such as theoretical physics, there are relatively few *distinct* theoretical perspectives (e.g., electromagnetism and mechanics), and they often apply to *different* concrete objects (e.g., charge vs. mass, and electrical vs. mechanical forces).

In compositional biology, on the other hand, the guiding assumptions of a perspective are those that prescribe the *decomposition of a system into parts*. These assumptions determine the individuation and identification of parts – that is, they select the essential properties for partitioning the system. For example, anatomy is guided by different assumptions than physiology in its partitioning of a biological system. More specifically, anatomy is guided by considerations regarding structure, whereas physiology is coordinated by assumptions concerning process.

Furthermore, in compositional biology, there are myriad theoretical perspectives that overlap in application to the *same* concrete objects (e.g., organisms). These perspectives, even when they pertain to the same concrete object guide the partitioning of a system in different ways. The important guiding assumptions in formal biology concern abstraction whereas the crucial ones in compositional biology concern partitioning.

Now that I have delineated my use of the concept of theoretical perspective and contrasted some of their important biases in formal biology as compared to compositional biology, I will turn to the relationship between a theoretical perspective and a discipline.

Is a theoretical perspective[111] (1) a position or a school of thought within a discipline (e.g., Wright's Shifting Balance Theory vs. the Fisherian "Large Population Size Theory" in mathematical evolutionary genetics[112]), (2) a discipline (e.g., mathematical evolutionary genetics or comparative anatomy), or (3) a set of disciplines grouped by some larger scale set of similarities (such as the competition vs. integration "super-perspectives" to be discussed in Chapter 3 – see, e.g., Diagram 3.1; and fleshed out in Winther 2003 in press)? Wimsatt further generalizes this question when he writes "Is there a paranoid schizophrenic's perspective? One or many? Is there a female perspective? is it cultural or biological? is there a feminist one? …Does each new interest group or reference group individuate a perspective or a component of a perspective? Does every person? Does every life stage?"[113] These are broad and difficult questions better left to the psychologist and sociologist who, unlike me, has tools to address them. But I do have some tentative answers pertinent to my analysis of compositional biology.

First, I suggest that we can identify disciplines robustly through a variety of means.[114] There are sociological facts about how university departments and programs are structured and often fairly robust organizations are found when

[111] In what follows, I will consistently use Wimsatt's term, although it should be noted that Kauffman's view is basically isomorphic to Wimsatt's, as argued above.
[112] E.g., Wade and Goodnight 1998; Goodnight and Wade 2000; Coyne, Barton, and Turelli 1997, 2000.
[113] Wimsatt 1994, p. 263.
[114] Sander Gliboff has pointed out some inadequacies of my account. It is notoriously difficult to articulate identification and individuation criteria for "disciplines." Perhaps, Gliboff suggests, it is better to refer to the theoretical perspectives as "approaches" than to identify them with disciplines. For the purposes of this dissertation and in the interest of receiving *further* insightful critiques of these ideas, I will continue to argue that the perspectives can be (loosely) identified, under some conditions, with disciplines.

comparing universities. Furthermore, particular canonical upper-level undergraduate and introductory graduate textbooks, in their attention to detail and in the conceptual framework they provide, serve to identify, and further entrench, disciplines. Third, our shared intuitions as students of science often provide us with robust disciplinary identifications. Thus, university structure, textbooks, and our intuitions as science scholars all provide ways for individuating disciplines.

The six disciplines in compositional biology that I will focus on are (1) comparative anatomy, (2) functional morphology, (3) physiology, (4) developmental biology, (5) biochemistry, and (6) cellular biology. The first four can be thought of as instances of "organismal biology," while the latter two can be considered cases of "molecular biology." I choose these six disciplines because they all relate to evolutionary developmental biology, a new discipline[115] that I will explore in more detail in the subsequent chapter. I do not therefore consider compositional biological sciences such as immunology or neurobiology, or even medicine, which while not necessarily a biological science, is certainly compositional.

A single large-scale theoretical perspective, I argue, guides such disciplines. Certainly more fine-grained theoretical perspectives, sometimes at

[115] Addressing theory change and theory novelty is beyond the scope of this dissertation; my notion of theoretical perspective describes, primarily, a static and stable biological science. Evolutionary developmental biology (evo-devo) is, however, a *new hybrid* field and there is a historical story and a philosophical account to be told here. I will, however, side-step this and will merely claim that evo-devo has a set of guiding biases and assumptions that overlap with a variety of the compositional biological sciences here described. However, the total set of such biases in evo-devo has still to be carefully articulated, both in actual scientific activity and in science studies. Chapter 3 is, in part, an attempt to do so *without* telling a historical narrative or developing a philosophy of hybrid theoretical perspectives.

odds, exist, but these include the guiding assumptions of the disciplinary theoretical perspective. This is why major practitioners of a discipline may appear to disagree on almost everything, although they actually agree on many aspects of their science.[116] So here I take a stronger position that Wimsatt when he writes, "[p]erspectives may sometimes correspond loosely to disciplines, but need not."[117] Although perspectives are hierarchically-structured, one level at which they exist is the disciplinary one. And it is *this* perspective that is captured in the canonical advanced textbook(s) of the discipline; textbooks show the relative unity and commonality of methods, theories, and data of a discipline. In conjunction with the idea, explored above, that theoretical perspectives determine the mode or frame of partitioning of a system, it then follows that textbooks capturing the disciplinary perspective provide a locus from which we can glean the modes of partitioning endorsed by the perspective. And this allows us to satisfy the goal of this chapter, which is to analyze the very different modes

[116] Although if two people have the same vocabulary to be able to disagree so vehemently, this would seem to imply that they actually *share* many assumptions. For example, Wade and Coyne, though differing on many issues, certainly share, for example, a commitment to the power of mathematical formalization as a suitable and useful representation of the evolutionary process. They also agree on many of the basic evolutionary forces. Consider how they *both* differ from a biologist, such as Goodwin (1989, 1994) or Gould (1989, 2002), who would emphasize the importance of constraints, developmental and phylogenetic, in the evolutionary process and who, in the case of Goodwin, would be skeptical of the power of the sort of mathematical abstractions presented by evolutionary genetics. Although Gould accepted the explanatory power of population genetics for microevolution, he did believe that microevolution alone could not explain macroevolution; and although he even (co-)engaged in mathematical abstractions regarding macroevolution (e.g., Lloyd and Gould 1993), his work tended to be non-formal and compositional. (I thank Lloyd for clarifications here.) As in systematics, similarity is a matter of degree. Disciplines, like species, can also be classified as "groups under groups" [Darwin 1859, p. 433] (although, in the case of disciplines, there is not a single "natural" classification system).

[117] Wimsatt 1994, p. 264.

of partitioning of the six different compositional biological sciences indicated above. These differences are especially notable for the disciplines that focus on the *same* system (i.e. the first four focus on the organismic level and the last two investigate the molecular level).

For the purposes of my general analysis of different compositional biological sciences and their corresponding frames of partitioning, textbooks are a satisfactory source of data. I certainly accept that textbooks are *simplifications* of disciplinary activity and that they engage heavily in rhetoric and self-proclaimed authority.[118] A *full* analysis of the theoretical perspective of any specific discipline[119], including its detailed guiding biases and assumptions, would require both attention to other sources of data (such as research papers, laboratory practice, communications with practitioners of the field) and more nuanced philosophical and sociological methods.[120] Furthermore, I also understand that active practitioners of a field rarely refer to textbooks and have certainly moved beyond them in terms of detail. But even if textbooks are the ladders that researchers subsequently kick away[121], the importance of textbooks as a summarized collection of the scientific activity of a discipline[122] and as the

[118] See, e.g., Kuhn's discussion of the role of textbooks in science in Kuhn 1970, pp. 136-138. He claims that they are loci of authority and ignore the richness of the history of the discipline.
[119] In Chapter 3, I will investigate evolutionary developmental biology and levels of selection theory in explicit detail using both research papers and communications with scientists.
[120] See, e.g., Latour and Woolgar 1986; Lloyd 1988.
[121] I thank Melinda Fagan for this Wittgenstein-inspired way of putting the point as she described to me her own development as a molecular biologist who received a PhD from Stanford University.
[122] Michael Wade informed me that when a potential candidate for tenure is up for review a criterion often used in the sciences is whether that candidate's work has made it into the advanced textbooks of the field. Thus, since textbooks provide credibility, this suggests that they are considered important.

resource with which the next generation of practitioners learn the tools of the trade cannot be underestimated nor should it be forgotten. It is precisely because textbooks indicate (1) "the generally-accepted" and (2) "that which is learned early" that I use them.

In short then, I have argued that three criteria allow us to identify disciplines independently of knowing anything about their theoretical perspective. Each discipline is guided by an overall theoretical perspective, which can be gleaned from the textbooks of the discipline. One aspect of a theoretical perspective from compositional biology is its mode of partitioning. Different disciplines partition differently and also have other distinct guiding assumptions as biases, as summarized in the table at the end of this chapter [Table 3. (2.1)].

2.3 An Empirical Analysis of Theoretical Perspectives

I will now turn to an analysis of each of the six compositional biological sciences in turn as motivated above. I call my analysis "empirical" from the point of view of a philosopher of science who is using "data" of scientific practice in the form of textbooks. That is, textbooks are one instance of scientific practice, and they are also excellent summaries of such practice. The first four compositional sciences belong to "organismal biology," whereas the latter two (biochemistry and cell biology) are cases of "molecular biology." The level of investigation is clearly different between these two clusters of theoretical perspectives.

2.3.1 Comparative Anatomy (Organismal Biology)

Comparative anatomy textbooks, such as Libbie Hyman's famous *Comparative Vertebrate Anatomy*[123] are organized according to structural systems in a taxonomic context. In her first chapter she describes "general considerations of animal form" such as those of "planes and axes," "symmetry," "metamerism or segmentation," and "cephalization."[124] Note that these are all *anatomical* considerations. She also has a section in that chapter on "homology and analogy" where she does discuss function. She even partially defines homology in terms of "similarity or identity of specific physiological …mechanism." It is unusual to define homology in terms of mechanism and "function,"[125] but given that Hyman claims that mechanisms indicating homology have to be *extremely* similar, her assertion is not so odd. She writes, "thus an insect leg and a cat leg serve the same broad general function, that of walking, but the mechanism of walking is quite different in the two cases."[126] So this aspect of her definition of homology is *still* tied to anatomy (and common descent) in that homologous parts have to be extremely similar to one another anatomically; they are of the same anatomical kind. After her introductory chapter on animal form, she has a chapter on (1) the classification of the chordates, (2) the "essential features of the lower types

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[123] Hyman 1942, 2nd edition.
[124] Hyman 1942, pp. 1-4. Cephalization is the "pronounced tendency for the anterior end of the body to become more and more distinctly separated and differentiated from the rest of the body as a *head*." p. 3.
[125] In this chapter, I use "function" in the sense associated with Cummins' "causal role functions," to be described in Chapter 5. These are the current contributions to a system behavior that structural or processual parts have. However, *Hyman's* use of "function" falls fairly close to Wright's (1973) notion of a function as what an object (or, I would add, a process) *did* in the past (or today) such that it persisted under a regimen of natural selection (see also Godfrey-Smith 1993, 1996).
[126] Hyman 1942, p. 4.

[chordates]," (3) the "external anatomy and adaptive radiation in gnathostomes [jawed-vertebrates]," and (4) chordate development. These chapters employ primarily anatomical data for their arguments. This is especially clear in (2) and (3).

All of the remaining nine chapters concern the comparative anatomy of distinct structural systems with their constituent parts (e.g., skeletal in four chapters, muscular, digestive and respiratory, circulatory, urogenital, and nervous). That is, Hyman describes each of these systems across different chordate groups [e.g., sharks, bony fish, mudpuppy (which is an amphibian), turtle, pigeon, cat and rabbit]. And while these nine chapters do have brief descriptions of physiology and evolution (literally in fine print!) the focus of each is comparative anatomy. *Anatomical dissections, not measurement of dynamic mechanisms in living organisms, are the order of the day*. In fact, the book was originally written as a laboratory manual for comparative anatomy classes, but in her second edition she also decided "that the book shall now serve as a text as well as a laboratory manual. It is believed that students will not need to purchase any accompanying textbook…"[127] Detailed textual descriptions of part shape, topological relation to other parts, and part structural identity, were, in the second edition, meant to aid the direct viewing of the dissected organism. As a telling case of scientific bias, and of theoretical "cephalization," Hyman ends her book with a 6-page discussion of the structure of the mammalian brain; she also adds (in fine print) a 1-page description of the "functions of the *parts* of the brain."[128] That organ of the putatively greatest majesty, the mammalian brain, is

[127] Hyman 1942, p. ix.
[128] Hyman 1942, pp. 511-517, emphasis mine.

described in terms of the central theme of the book, anatomy. This book served as a central textbook for many years.[129]

But, as Marvalee Wake points out in a third edition of this book, the discipline of comparative anatomy, or "morphology" has changed. Wake writes, "Until the early 1950s, comparative morphology emphasized the evolution of structure alone. Since that time evolutionary morphologists have emphasized the history of change of function (i.e., evolution) as well as of structure, for the concept of adaptation stresses that structures change to provide functional advantage. This book [3rd edition], then, presents, system by system, the evolution of structure and function of vertebrates."[130] For each chapter, a different expert evolutionary morphologist was assigned to supplement and, if necessary, change, Hyman's basic text in a manner consistent with Wake's comment.

Such a change of commitments within comparative anatomy is not, however, universal. Goodwin certainly did not make this change.[131] Baron, a less radical comparative morphologist, also does not endorse such a move. Baron argues against the causal efficacy of selection to explain the appearance and developmental dynamics of variant morphologies of organisms.[132] While theoretical debate over the precise relation between selection ("function") and morphology is ongoing, a significant amount of work in (evolutionary) comparative anatomy today is still practiced as it was represented in Hyman's

[129] This can be gleaned from the first sentence of Marvalee Wake's "Preface to the Third Edition," "Several generations of zoology students have used Libbie Hyman's *Comparative Vertebrate Anatomy* as their guide to the study of vertebrate structure and evolution." (Wake 1979, p. ix)
[130] Wake, M. (ed.) 1979, p. 1.
[131] Goodwin 1989, 1994.
[132] Baron 1991. See also Wake, M 1992 who is sympathetic to Baron's concerns while not fully endorsing them.

1942 text. This is especially true at universities with natural history museums and at museums such as Berkeley's Museum of Vertebrate Zoology, the Field Museum in Chicago, Harvard's Museum of Comparative Zoology, and the California Academy of Sciences in San Francisco. Comparative anatomy is an independent theoretical perspective.

2.3.2 Functional Morphology and Physiology (Organismal Biology)

Let us now explore two very different, but related, disciplines that partition the system, typically an organism, according to processual criteria: functional morphology and animal physiology. As we will see, the former in particular has aspects pertinent to formal science although I categorize it as a compositional science. My main argument here is that these two disciplines are concerned with processually-defined parts and interpret them in a way distinct from comparative anatomy, even if functional morphology, similarly to comparative anatomy, focuses on the level of gross-scale organismic structure whereas physiology investigates a variety of organismic levels.

2.3.2.1 Functional Morphology: Basic Content

A key text in functional morphology, *Functional Vertebrate Morphology*, was edited by, among others, Milton Hildebrand and David Wake. Even a cursory glance at the table of contents reveals a completely different organization as compared to Hyman's book. The book chapters, each written by a different expert, are primarily *activities*: "Walking and Running," "Jumping and Leaping,"

"Swimming," "Ventilation," and "Feeding Mechanisms of Lower Tetrapods."[133] This is very different from Hyman's organization around structural systems of the organism.

Let us examine some of the salient characteristics of the theoretical perspective of functional morphology, as represented in this book. First, many of the chapters provide a *classification* of the different types of activities under their purview. For example, in a chapter on swimming, the authors differentiate between axial undulatory propulsors (e.g., fish that use their body or hind fin to undulate through the water), and appendage propulsors. This last category is further subdivided into undulatory (such as mantarays, which undulate their huge fins in a direction orthogonal to the direction of motion) and oscillatory (such as all amphibians, reptiles, birds and mammals that jerk their appendages through the water).[134] Classifying the numerous relevant processes is an important first step in the complex and detailed compositional science of functional morphology.

In functional morphology, parts are identified in two different ways. First, *activities are subdivided into distinct activity-parts.* For example, Bramble and Wake, in their chapter entitled "Feeding Mechanisms of Lower Tetrapods," partition the *process of feeding* into body movement, head thrusting, tongue projection, mastication (if present), and swallowing.[135] The first three can be either (activity-)parts, in "generalized predators [that] frequently combine all three tactics in a

[133] Hildebrand et al., 1985. One telling exception is a chapter on "The Vertebrate Eye"; I think this is yet another case of theoretical cephalization or what some call "ocular discourse" – the brain and the eye are considered unique and crucial parts of the organism, and, as such, are theoretically privileged.
[134] Webb, P. W. and Blake, R. W 1985, p. 111.
[135] Bramble, D M and Wake, D M 1985, pp. 252-260.

single predatory act"[136] or kinds, in cases where specialized predators rely, for example, primarily on tongue projections (in salamanders, anurans, and chameleons). In such a case a concrete activity-part can become a stand-in, a *name* (e.g., HLP – hyolingual projection[137]), for a *type* of behavior when that activity-part constitutes a significant portion of the entire behavior. This is sort of nominalization is based on a part of a process. In many of the other chapters of the Hildebrand et al. book, activities are also divided into their processual components.

The second way parts are individuated is *by using behavioral activity criteria* instead of the anatomical criteria used in Hyman's text. Processually-individuated structural parts, and groupings of parts, are identified according to what they do. For example, Hildebrand depicts the *leg* as a unit that undergoes movement and Bramble and Wake unite a variety of muscles and bones into particular groups in their representation of feeding behaviors.[138] In comparative anatomy (e.g., Hyman 1942), muscles and bones are *not* grouped together. In functional morphology, however, activity determines (structural) part individuation and identification.

Functional morphology, as represented in this textbook, uses (1) parts of processes as well as (2) structural parts identified by their integrated role in processes. The former is a partitioning of an activity, the latter is a structural

[136] Ibid, p. 252.
[137] Ibid, p. 252.
[138] This grouping of structural subparts to make a unit, which is then part of some larger unit, can be seen especially in Hildebrand's diagrams of animal cartoons in motion and an idealized diagram of a leg in motion (Hildebrand 1985a, pp. 39, 41, 46); it can also be seen in a diagram entitled "the mechanism of intraoral transport in the model generalized tetrapod" in Bramble and Wake's chapter (p. 238), Appendix Figures 2.4-2.7.

partitioning according to processual considerations. This is very different from the purely structural partitioning found in comparative anatomy, represented by Hyman's textbook.

Before we move to physiology, it is necessary to discuss the role of mathematics in functional morphology, especially in view of my claim that this biological science is a compositional science. In so far as it explicitly models a biological system using mathematical models imported from physics or engineering, does it also become a formal science?

2.3.2.2 Functional Morphology: Model-Types

This is not an easy question to answer. In order to attempt to answer it, I will turn to a brief description of three model-types frequently used in functional morphology: diagrammatic and scale models, narrative models, and mathematical models.[139]

Diagrammatic and scale mechanical models are ubiquitous in functional morphology. A paper in a 1960 symposium volume on "Models and Analogues in Biology" describes a variety of mechanical models including a depiction of the vertebrate hind limb as a contraption involving a carriage on a rail and various rods, springs, and axels (Appendix Figure 2.8). This same paper notes that "all mechanical devices can be broken down into two elementary systems – levers and wheels," and thus many functional morphological systems can be represented, physically or diagrammatically, in this way.[140] Papers in the

[139] A more complete explication of model-types will be found in Chapter 4.
[140] Brown, R H J 1960.

Hildebrand et al. text also describe concrete[141] mechanical scale models. Such models contain parts as explicit components. Since they are idealized, these models are incomplete for a variety of reasons: (1) there are scaling non-isomorphisms, (e.g., length and volume scale in different powers), (2) the model materials differ from the actual biological materials and may not be representative of the actual physical properties such as elasticity, tensile strength, and density, (3) not *every* bone and muscle is represented through rods and springs – some are omitted from the model[142], and (4) heterogeneities, within and between individuals, in muscle and bone structure, energy demands and supplies, etc. are not represented. Thus, these concrete mechanical models are impoverished idealizations. They do, however, refer directly to the *compositional* nature of the system being represented.

Narrative models in functional morphology are linguistic representations of the stage-like nature of activity. For example, Bramble and Wake provide a narrative model of chewing. They divide chewing into a series of opening and closing phases at two different speeds, fast and slow. They *define* each of these phases with (1) the movement of different parts of the body (e.g., head and neck, mandible, and skull) and (2) the activation of different muscle groups.[143] Other series-of-stages narrative models are presented in the chapter on ventilation and, to a lesser extent, in the chapter on digging of quadrupeds.[144] Narrative models

[141] As opposed to abstract.
[142] Recall the general discussion in Chapter 1 of buffering as, under one interpretation (*not* Gerson's), one *form* of abstraction.
[143] Bramble and Wake, pp. 235-240. I will explore this specific model in much more detail in Chapter 4.
[144] Liem, K F 1985; Hildebrand 1985b.

are especially common in the recounting of history, biological or human.[145] In human history it is difficult to empirically evaluate narrative models due to their intrinsic "multi-perspectivality," the unknowable intentions of the human agents making history, and the complexity of conditions. In functional morphology, in contrast, there are concrete objects to which we can and do turn to verify the models. Thus, narrative models in functional morphology can be empirically evaluated[146] more readily than such models in history. These models, of course, are also highly idealized in that they are an abstraction and do not exactly represent the system and its parts. Through language they do, however, explicitly refer to concrete parts, and changes in them; they are thus models pertinent to compositional biology. Ideally, the representation of an organism through functional morphology would most readily occur through a combination of models such as narrative models and scale models.

Mathematical biological models, as discussed in Chapter 1, are also an undeniably important model-type in functional morphology. Mathematical models are used to model dynamic activities such as swimming or the properties of body support,[147] and are an integral aspect of that discipline. In the case of swimming, a large number of diverse formulas and parameters (Appendix Figures 2.9, 2.10) are used to represent different forms of swimming such as undulatory and oscillatory propulsion; the fundamental distinction between these two is that in the former case the moving entity (body or fin) moves smoothly and in the same direction as the whole body whereas, for the latter,

[145] Danto 1985; Griesemer 1996; Hull 1975, 1981, 1992; Richards 1981, 1992.
[146] All within the framework of having chosen one's position vis-à-vis the inevitable trade-offs between, for example, generality, precision, accuracy, and simplicity, as we shall see in Chapters 4 and 5.
[147] E.g., Alexander 1985; Webb and Blake 1985; Vogel 1988.

motion causes drag and is discontinuous with motion of the body. The equations used for the two cases are remarkably different, the equations of the former case involve primarily parameters describing smooth reactive forces on the body by the water whereas those of the latter one entail parameters describing both (discontinuous) drag of the appendage on the water and thrust that provides a torque force on the body.[148] Different mechanisms of movement entail very different mathematical representations. In the case of body support, multiple parameters represent the forces involved, angles of bones and tendons, tensile strength of the biological materials, cross-sectional area of bone, elastic deformations, etc.[149]

Complex engineering formulas stemming from fluid dynamics and structural engineering are thus ubiquitous in functional morphology. Sometimes situations, however, are *too* complex for models to be formulated: "Some detailed hydromechanical models for oscillating lifting surfaces have been developed, but none has as yet been applied to swimming animals, partly because of their complexity."[150] Furthermore, the compositional organization of the system is intimately appealed to in these models, even if mathematical abstraction idealizes away from within-organism and among-organism (of the same species) variation. Thus, these mathematical methods serve a different role than those ubiquitous in, and even defining of, formal biology. In this latter kind of biology,

[148] Webb and Blake 1985, pp. 113-126.
[149] Alexander 1985, pp. 27-31.
[150] Webb and Blake 1985, p. 126.

the mathematical parameters are highly abstract, referring to a few simple properties, many of which are statistical aggregate measures (e.g., fitness).[151]

2.3.2.3 Physiology

Physiology provides a more processual and activity-oriented outlook than functional morphology. In the latter, behaviors are still intimately connected to morphological, gross-scale structures such as organs, whereas in the former, activities are related to a variety of levels. Furthermore, the environment external to the organism is considered more explicitly in physiology, at least in part because the environment is perceived as a disturbing force against which physiology confers organismic homeostasis.

One set of physiology textbooks, Eckert et al.'s *Animal Physiology*[152] and Hoar's *General and Comparative Physiology*[153], revolve around activities, as can be seen from their chapter titles: "Enzymes and Energetics," "Propagation and Transmission of Signals," "Exchange of Gases," and "Feeding, Digestion, and Absorption" in the former, and "Homeostatic Mechanisms," "Receptor Mechanisms," "Nutrition and Digestion," and "Excretion" in the latter. Note that these activities span a variety of levels and parts seen as distinct in Hyman 1942 as well as Hildebrand et al. 1985. Here I want to provide descriptions of the presentations of molecular, nervous, and gas exchange activity.

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[151] Furthermore, as we shall see in Chapters 4 and 5, the equations of formal biology, which produce "transition theories" (sensu Cummins 1983), are easily amenable to semantic view analysis.
[152] Eckert et al. 1988.
[153] Hoar 1983.

Molecules are described from the point of view of their role in dynamic metabolic pathways, many of which are circular never-ending closed loops such as the Krebs cycle. Structural diagrams of molecules are, of course, provided, but the *flow* of the cycle, which produces ATP, is emphasized using arrows between intermediary products and tallying numbers of ATP molecules produced. Furthermore, a small section on "oxygen debt" emphasizes both the multi-level impact, and description thereof, of molecular activity on animal tissue (Appendix Figure 2.11).[154]

Nervous signals are also described as central agents in depictions of nervous system activity. Diagrams of structures that produce them are only one aspect of the diagrammatic and narrative models that represent the activity of these signals. Simple circuit diagrams, flow charts of sequential activity, and electrical intensity graphed as a function of time all capture activity of nervous signals (Appendix Figures 2.12, 2.13).[155]

In considering the process of gas exchange, a variety of models are employed to describe it. Schematic diagrams represent the exchange of different gases between different organs, tissues, cells, or even molecules (e.g., lung and circulatory system at different levels, or hemoglobin) as well as schematic diagrams of particular properties (e.g., pressure) at particular locations (i.e., parts) of the process. Graphs showing lung volume as a function of time indicate the different component phases of breathing. A mathematical graph indicates the roughly linear relationship between lung area and body weight in different species. Formulas relate rates of gas transfer to such variables as diffusion

[154] Eckert et al. 1988, p. 63. See also Hoar 1983, Chapter 2.
[155] Eckert et al. 1988, Chapter 8. See also Hoar 1983, Chapters 6 and 8.

distance and concentration difference across the membrane. Again the activity and process of gas exchange is emphasized in all these representations (Appendix Figures 2.14-2.18).[156]

Many kinds of parts are present in the processually-oriented image of nature presented by physiology. First of all, activities are broken up into activity-parts[157] such as the component activities of cell respiration (including glycolysis and the Krebs cycle), or the various components of nerve activity (including stimulation and refractory period), or even the various stages of breathing through expansion and contraction of the lung. The activity is divided into temporal segments according to individuation criteria that state that the different segments are *different in kind from each other* (e.g., glycolysis is different from the Krebs cycle).

There are also the parts that could be thought of as the *agents* of the activity, rather than as segments of the activity. These are the structures, such as molecules or organs, which engage in the process. Note, though that these parts are not necessarily defined structurally. That is, processual considerations such as the *capacity* of a structure to engage in certain activities are used to identify the relevant parts of a system. For example, the heart would be considered a blood pump in the circulatory system in which the right atrium and ventricle are involved in moving deoxygenated blood, whereas the left atrium and ventricle move oxygenated blood (Appendix Figure 2.17). From a structural point of view, all four heart chambers are integrated in a single organ, but from a processual point of view, the right side is engaged in a different, though related, activity –

[156] Eckert et al. 1988, Chapter 14. See also Hoar 1983, Chapter 13.
[157] This same classificatory partitioning process is done in functional morphology, as described above with the example of hyolingual projection.

the two sides are, if one were to draw a physiological flow chart *separated* in activity, with the lungs in between.

Another set of physiology textbooks, Willmer et al.'s *Environmental Physiology of Animals*[158] as well as Schmidt-Nielsen's *Animal Physiology*[159] also organize their analysis in terms of activities. But, they add another crucial aspect, the environment. Wilmer et al.'s book, for example, includes a whole section entitled "Coping with the Environment" in which they discuss the problems posed by environments, such as marine, fresh water, and terrestrial habitats. They also have a section on parasitic habitats. For each of these chapters, they discuss how physiological processes (e.g., maintenance of water balance) address the particular problem posed by that environment. Similarly, Schmidt-Nielsen's text is explicitly environmentally-oriented. Instead of focusing on habitat-types, he presents a *problem-orientation* reflected in the division of his text into five parts that addresses how animals acquire and maintain: (1) oxygen, (2) food and energy, (3) temperature, (4) water, and (5) movement, information, and integration. He organizes different physiological activities under each of these categories of problem. Both Schmidt-Nielsen's and Willmer et al.'s organization provides an explicit way to tie function to process (rather than focus exclusively on process). That is, particular processes are here understood as solving environmental problems – processes have specific functions. As I will show in Chapter 4, process is not the same as function, even if the two are related.

Let us summarize how parts are pertinent to functional morphology and physiology. What sorts of parts, as represented in their textbooks, do these fields

[158] Willmer et al. 2000.
[159] Schmidt-Nielsen 1997.

employ? How do these theoretical perspectives partition a system? Parts of a process, and processually-defined structural parts (i.e., the agents of the process) are the two important (very abstract) kinds of processually-defined parts. Although I do not claim that biologists explicitly differentiate between these parts or even self-consciously employ one or both in their work, I argue that these parts are implicitly employed in most of the classificatory, representational, and explanatory practices of processually-oriented compositional biology such as physiology and functional morphology. Almost any representation or explanation stemming from these fields can be shown to consist of process partitioning. Differently put, the theoretical perspectives of physiology and functional morphology partition a system processually.

2.3.3 Developmental Biology (Organismal Biology)

The parts pertinent to developmental biology are of a different type altogether. The concern here is with the construction, over ontogenetic history, of the organism[160]. The parts appealed to in explanations of the ontogenetic process are *molecular genes*[161] acting over ontogenetic time, *regions* of developmental activity such as endoderm, mesoderm and ectoderm, as well as *dynamic signaling processes* such as cell signaling activities and morphogenetic gradients. I will discuss this discipline in more detail in Chapter 3. As a theoretical perspective, the parts appealed to in its explanations and descriptions are parts that both (1)

[160] Or, as we shall see in Chapter 3, on higher-level integrated systems such as social insect colonies.
[161] These molecular genes are exactly what Moss 2002 calls "genes-D."

change over developmental time[162] and (2) are involved in causing such change[163].

Again, a brief exploration of the standard contemporary textbook on this topic, Gilbert's *Developmental Biology*[164], will serve my purposes. Three important aspects of development can be inferred from this text's detailed table of contents. The organization mixes ontogenetic history (early to late) and organizational levels (molecular to organismic), two crucial aspects of development. Although there is a clear developmental temporal narrative component to the text's structure, there is also a clear switching among different levels (e.g., molecules,

[162] This is one way in which the parts are *complex*.

[163] I sidestep the issue of distributed cause here. I am extremely sympathetic to the view articulated in the work of Lewontin, Oyama, and others on the topic of extended and distributed developmental cause (and multiple developmental causal "agents", although the investigators mentioned would probably not be satisfied with the reification of distributed causes into agents). But while my philosophical proclivities lie in that direction, my scientific pragmatism suggests to me a "naturalizing" move that resists such proclivities. That is, if I am to naturalize my analysis in scientific practice, I will probably have to abandon many of the points adumbrated by the distributed-cause-during-development advocates. Developmental biology practice does tend to be "reductionist" in general even if there are ways to be simultaneously a "holist" and a "reductionist," as exemplified by the work of Raff and Wagner (e.g., Raff 1996; Wagner 2000; Wagner et al. 2000). For now I would prefer to set aside conflicting philosophical and scientific pulls and simply provide an analysis that is independent of commitments to holism or reductionism.

On a related note, Frederick Schmitt pointed out to me that there is a clear tension between the *philosophical* views emphasizing distributed cause and compositional organization, respectively. The more distributed cause we have, the less compositional organization there can be, and vice-versa. Thus, there is some tension in my philosophical views where I would like to emphasize both. But Lewontin and Levins also have this tension in their dialectical view, in which they also focus on both! By sidestepping the whole issue of distributed cause I will leave the resolution of this tension for future work. [However, in Chapter 5, I will also briefly note that the explanatory pattern advocated by Cummins, while accurate for compositional biology, is a "reductionist" way to flesh out explanation in this form of biology – other alternatives could perhaps exist.]

[164] Gilbert 1997.

cells, and organs). Third, the effect of the environment is minimized in the text, i.e., normal development is context-independent.

Let us turn to the first two points. The book is divided into five sections. After an introductory section addressing basic concepts, techniques, and levels, there are sections on "Patterns of [early] Development," "Mechanisms of Cellular Differentiation," "Specification of Cell Fate and the Embryonic Axes," and "Cellular Interactions During Organ Formation." Note that there is a clear historical direction through these sections, from the early stages of development (with chapters on cleavage, gastrulation, and early axonal specificity[165]) through the middle stages (with chapters on cell fate specification through cell-cell interactions, and the establishment of body axes in mammals and birds[166]) and culminating[167] in the formation of organs, which are the key parts of adult morphology (with chapters on secondary induction, the tetrapod limb, and

[165] Chapters 5, 6, and 8 respectively.
[166] Chapters 15 and 16 respectively.
[167] It is of some interest to note that developmental processes, which can be captured in narrative models, do not develop the narrative tensions present in human narratives, such as plays or novels. That is, there are no dramatic elements that build up and, subsequently, require resolution. There is no easing of dramatic tension. That is why I use terms like "culminating" ironically, to note important differences between biological processes and human creations. Despite efforts by some philosophers and scientists to note continuities of all kinds between the biological and social worlds, these putative near-isomorphic analogies fail repeatedly and we should constantly be alert to the crucial discontinuities between biology and sociology. We should not fall prey to the traps set by the explanations of the sociobiologists, evolutionary epistemologists, or, even, those set by the more benign analogies, advocated even here in my dissertation, of commonalities of representational and explanatory resources in the biological and social sciences, such as narrative models (e.g., the work of Hull, Richards and Griesemer). We must always be vigilant about suggested similarities, in representations of the biological and the social, which can be used both for misrepresenting nature (although, who determines what a "misrepresentation" is?) and for oppressive social purposes.

hormones as long-distance mediators of development[168]). This is a developmental historical narrative.[169] The parts *instantiating* structure and process *at a particular time* as well as those (same or different kinds of) parts *causing* the structure and process of *subsequent stages* are the parts involved in this narrative.

This is an important distinction that does not exist in the other two processual organismic compositional biological sciences, functional morphology and physiology. Developmental biology strongly emphasizes the distinction between those parts that are the sources, causes, and agents of change (especially "genes-D," as described in Moss's work) *and* those parts that instantiate or are involved in the change, such as developmental regions (e.g., endoderm, mesoderm, and ectoderm). There is a clear hierarchical *causal* divide in this discipline; it represents and explains how change from one level (molecular, especially genetic) to another (especially adult morphology) happens. This causal divide has been the subject of many metaphors (e.g., the gene as a homunculus containing the "representation" or "program" of the whole organism) and much philosophical critique.[170] Due to the absence of this clear and drastic hierarchical *causal* divide in the other two theoretical perspectives of functional morphology and physiology, this distinction is not important for them; in these cases the agents (that is, causes) of change are the same units as those involved in the change.

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[168] Chapters 17, 18, and 19 respectively.
[169] Note that the processual narratives of development are directional ("time's arrow"), whereas the processual narratives of physiology are circular in the sense both of homeostatic cycles and metabolic cycles ("time's cycle").
[170] Again, while I am sympathetic to the philosophical literature, it is also clear to me that most biologists believe in this causal divide. And since my articulation of compositional biology is naturalized in the theories and methodologies of biologists, I will not address the philosophical literature here.

The narrative models of developmental biology, and the parts pertaining to the models, exist at *various levels* here. For example, the section on mechanisms of cellular differentiation concerns transcriptional and translational regulation of gene expression through, in the former case, activation of promoters (Chapter 10) or chromatin (Chapter 11) or, in the latter case, differential RNA processing and translation (Chapter 12). In other words, there is a temporal sequence of molecular events during which regulation can occur by virtue of changing reaction timing and intensity. This, in turn, causes cellular differentiation (that is, *producing difference from sameness*). But cellular (e.g., cell signaling, Chapters 15 and 17) and histological (e.g., primary and secondary induction, Chapters 17 and 18) levels are also examined as regions and agents of cellular differentiation. That is, developmental changes and developmental causes at higher levels are considered valid and potent.[171] Thus, the parts that figure in developmental explanations exist at a variety of levels, from regulating molecules to inductive tissues.

Let us now turn to the third point, the point that environmental influences are downplayed. Developmental biology is typically considered an "internalist" field in which internal processes, guided or self-determined, are described.[172] The work of Brian Goodwin or Humberto Maturana and Francisco Varela are two extreme versions of this view of developmental biology.[173] The "self-

[171] E.g., the effects of the apical ectodermal ridge on limb bud formation, and effects on cell movement and nervous system formation by the organizer, discovered by Spemann.
[172] On internalism and externalism see Godfrey-Smith 1996; Winther 2001a, 2001b. For discussions of how developmental biology typically ignores environmental influences, see van der Weele 1995. For examples of recent attempts at remedying this omission, see Tollrian and Harvell 1999; Gilbert 2001.
[173] Goodwin 1989, 1994; Maturana and Varela 1980.

determination" of the genetic program is also potentially part of this argument, though for the three authors just mentioned it certainly is not since they believe, in different ways, that morphogenetic fields or homeostatic processes provide internal regulation and use genes as mere resources. The views of these three authors also make clear an important point: *an understanding of organisms as compositionally-structured (as opposed to an extreme "distributed cause" view) does not require a gene-centric position*. Others, however, consider development an internally-driven process *precisely because* the genetic sequence is internal to the organism and potent in its development.[174] Gilbert's book also endorses this assumption by having to set aside a chapter entitled "Environmental regulation of animal development" rather than keeping that discussion integrated with the rest of the book. Developmental biology as a field relies on the idea of development as a self-organizing or, more commonly, as an organized-by-genes, process of part construction.

[174] See, for example, Lewis Wolpert's work in which he discusses whether the egg is computable (Wolpert 1994). A passage from a book chapter entitled "Ex DNA Omnia" will make the point: "The power of DNA lies in its containing both the instructions for making all the proteins in the cell, and the programme which controls their synthesis." (Wolpert 1991, p. 77) Others have since promulgated, even putatively "proven," Wolpert's view to be "correct." (See, e.g., Portin et al. 1998). Mayr's distinction between proximal and ultimate causation also relies on this notion.

Again, there is cogent criticism of this position by Keller, Lewontin, Moss, Oyama, among others, who emphasize the distributed cause view. Again, I appeal to biological practice, which promulgates internal drive in development, although I certainly wish to distance myself from the extreme gene-centrism of someone like Wolpert.

2.3.4 Intermezzo: On Organismal Biology and Molecular Biology as Clusters of Disciplines

Now that we have explored the guiding biases, in particular with respect to criteria of partitioning, of the theoretical perspectives of organismal biology, I would like to sketch how molecular biology can be considered a compositional biology. The various theoretical perspectives of molecular biology have not received *sufficient* philosophical attention[175], particularly considering their economic, medical, and social relevance. Here I only *begin* to do justice to molecular biology as a, primarily, compositional biology consisting of theoretical perspectives such as biochemistry and cell biology.

A way in which molecular biology has received some philosophical attention is in its relationship with organismal biology, which is considered a relationship of theoretical reduction.[176] I side with those that argue against the actuality, or even possibility, of a reduction relation from organismal biology to molecular biology. The diversity and richness of scientific practice, including that of the independent theoretical perspectives of organismal biology, indicates that no such theoretical reduction exists and is not likely to be achieved. The spread of molecular techniques to and in organismal biology is not *theoretical* reduction; it is not even *methodological* reduction in that even those molecular methods are employed for theories (problem and question framings as well as models) *intrinsic* to that higher-level organismal biological science and some of the methods, such as dissection and transplantation still pertain strictly to

[175] Although see, for example Culp and Kitcher 1989; Moss 2002; Schaffner 1980, 1993.
[176] See the work of Nagel and Schaffner. For criticisms on these points see, e.g., Culp and Kitcher 1989; Kitcher 1984; Sarkar 1998; Fagan 2002.

organismal biology.[177] I want to avoid further discussion of theoretical, or other kinds of, reduction. What I want to address is whether, and if so how, the two bona fide theoretical perspectives of molecular biology that I will explore, biochemistry and cell biology, partition their system of study.

My argument concerning different guiding biases (including criteria of partitioning) works best in the compositional science cluster of organismal biology where there is a *complex* and *hierarchical* system, *the organism*, that is decomposed in many different kinds of ways. In discussing organismal biology, I did not explicitly describe how these theoretical perspectives investigated complex objects, employed complex abstraction, although I did indicate how they used either a variety of model types and, of course, referred explicitly to the concrete part-whole organization of the organism. It seems evident that these sciences do focus on complex objects in that the parts of organisms have many dispositional properties and have complex histories, developmental and evolutionary alike, that are relevant to determining those properties (e.g., organs of the same kind in related clades have different kinds of properties). Pertinent to complex abstraction, (1) consistent hierarchically-organized intensional sets of these numerous dispositional properties can be captured by particular theoretical perspectives, of which there are many, (2) once the relevant intensional sets has been captured by the perspective, there are many kinds (at numerous hierarchical levels) of objects in the theoretical perspective, and (3) there are

[177] Presenting the different definitions of "reductionism" is beyond the scope of this dissertation. Sarkar presents five different meanings of the term, premised on issues surrounding approximations and spatial and abstraction hierarchies (Sarkar 1998, 43 ff.). Zucker distinguishes eight senses, including "ontological," "mathematical," "mechanical," and "methodological" reductionism (Zucker 1996, pp. 23-26).

many ways that different complex objects can belong to the same complex kind (i.e., particular properties can be present in some objects and absent in other objects of that kind).[178] So, for example, eyes and livers can be described under many perspectives, as we have seen, and a fair amount of intra-species and inter-species variation can be accommodated in theoretically-useful intensional sets (constituting the complex kind) of these kinds.

The story of part decomposition is not so complex in molecular biology. There is not a single integrated "molecular system" to decompose in various ways there. Part of the reason for this is that, in a trivial sense, the organism is indeed composed of "nothing but" molecules and diverse kinds of reactions, which are the "elemental parts" of biological systems. Put differently, there are no radically different ways of partitioning the same kind of molecule, or molecular pathway. In molecular biology, structure, process, and function are closely related and radically different spatio-temporal decompositions of molecules and molecular reactions do not happen when we turn from, say, a structural to a processual perspective. That is why, as I will show, biochemistry includes *all* these perspectives. There is no radical relativism of perspective-dependent part decomposition in molecular biology as there is in organismal biology. Rather, the variety of perspectives in molecular biology has to do with *level* of analysis (such as molecular vs. cellular).

[178] As we saw in Chapter 1, in formal biology, there are few theoretical perspectives, few kinds of objects within a perspective, and rigorous ways for objects to be described by a particular simple kind.

2.3.5 Biochemistry (Molecular Biology)

Biochemistry is a compositional biology at a particular *level* rather than of a particular set of structures or processes or functions.[179] In this way, it differs in an interesting way from the other four compositional *organismal* biological sciences thus far considered. Stryer's book is divided into 6 overarching sections, all but the last, pertaining to phenomena at the molecular level. The last one concerns the "interaction of [molecular] information, conformation, and metabolism in physiological processes."[180] Even in this final section, molecular phenomena are the operative representations and explanations. Biochemistry is a science that focuses on a particular level rather than on a particular, broadly construed, cause, such as structure (comparative anatomy) or process (temporal – developmental biology; atemporal "homeostasis" – physiology), as the other compositional sciences explored above are. This is confirmed by looking at the second section on proteins. This section of the text explores protein "conformation, dynamics, and function"[181] – these correspond, respectively, to structure, process, and function.

In biochemistry there are many different kinds of structural parts acting in the processes it describes [see, e.g., Section I ("Molecular Design of Life") and Section IV ("Biosynthesis of Macromolecular Precursors") of Stryer's text]. At the most general level, there are four basic kinds of molecules: nucleotides, proteins, carbohydrates, and lipids. But these kinds are not simple and categorical as is

[179] I do not here consider the relation of biochemistry to organic (or even inorganic) chemistry. This is, of course, an important question and a full analysis of biochemistry as a compositional biology "under" the disciplinary cluster of molecular biology is required.
[180] Stryer 1988, p. viii.
[181] Stryer 1988, p. vii.

evidenced by the existence of glycolipids (carbohydrate and lipid molecules) and glycoproteins (carbohydrate and protein molecules), both of which are key components of cell membranes, as well as by transfer RNA, which is a nucleotide sometimes activated with a signature amino acid. Thus even at the most abstract level, the categories of structural parts are fluid and complex, consistent with complex abstraction being characteristic of compositional biology.

As the level of analysis becomes increasingly concrete, the types of parts increase dramatically in number. Furthermore, there is much flexibility in the intensional set of properties pertinent to a molecule or molecule-kind when considered as an instance of a certain more general kind. For example, as long as a molecule performs a certain function, e.g., decarboxylation, it can have a broad variety of structures (i.e., the intensional set covering that function is flexible with respect to structural properties). Conversely, a specific molecule can also serve a variety of functions (e.g., acetyl CoA is a precursor to the Krebs cycle *and* to the production of cholesterol; see Appendix Figure 4.11), and thus the intensional set of the structural properties of that molecule would also include a variety of functional properties. Note also that my discussion points to the fact that the *objects* of biochemistry are complex (my first distinction between the two kinds of biology) since they involve many causes (i.e., multiple functions and processes, as well as a complex structure).

Stryer's text is replete with diagrams capturing the molecular agents at different stages of reaction. Thus, molecules serve as parts (structural and processual) in multiple reactions. They can be interpreted as the *agents* involved in, and causing, change. Their function is explained by their biochemical

properties.[182] Furthermore, biochemical processes can be partitioned into *stages*, such as the three stages of cellular respiration or the stages of gene expression (i.e., most generally, transcription and translation).

Biochemistry is a detail-oriented science with many kinds of parts. Abstract principles and general statements about processes or functions of structures are difficult to come by in that field. All of the following have exceptions: (1) Crick's central dogma (information flows from DNA to RNA to proteins), (2) the universality of the genetic code and (3) the universality of the processes of cellular respiration and photosynthesis. Such exceptions include: (1) reverse transcription and, perhaps some proposed mechanisms for adaptive mutations in bacteria, (2) the genetic code of mitochondria, (3) the existence of different alternative respiratory pathways, such as aerobic and anaerobic (including fermentation and lactic acid production), and different pathways of carbon fixation during photosynthesis, such as C_3, C_4, and CAM plants. There are multiple exceptions to the crucial generalizations in molecular biology, but we would still, for example, call all the different pathways of carbon fixation (parts of) "photosynthesis." Thus, biochemistry is aptly characterized as possessing complex ("Wittgenstein-Boyd") abstraction. The kinds of parts, whether they be structures, processes, or functions of molecular biology are complex kinds despite clear regularities in biochemical properties at very basic part-levels, such as functional groups.

[182] As I will specifically show in Chapter 5, when I discuss Cummins' notion of causal capacities.

2.3.6 Cellular Biology (Molecular Biology)

I now briefly turn to cellular biology, another key area in molecular biology.[183] Similar to biochemistry, cellular biology is a compositional biology focusing on a specific *level*, the cellular one. Again, it includes structural, processual, and functional considerations. Alberts et al.'s book is divided into four general sections: "Introduction to the Cell," "Molecular Genetics," "Internal Organization of the Cell," and "Cells in Their Social Context."[184] The *cell* is the lens through which the book is structured. For example, the structure of chromosomes and the role of the cell nucleus are considered in one chapter as a way to conceptually frame DNA replication as well as RNA synthesis and processing, while discussion of proteins as "machines" that do work necessary to the cell is taken up in another chapter.[185] Furthermore, enzymes are not considered in a separate chapter on the biochemistry of respiration, say, but are rather discussed in a chapter focusing primarily on the *cellular* loci of energy conversion, mitochondria and chloroplasts.[186] Gene expression and development are also considered in terms of cellular differentiation.[187] It is clear that the cell, rather than molecules, is the focal point of this book.

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[183] There is some legitimate concern about whether cellular biology should be characterized as a molecular biology (Melinda Fagan, pers. comm.). In part, I appeal to historical and sociological facts regarding the structure of this discipline as compared to, say developmental biology, which had its origin as an organismal biological science. I also appeal to the intuition that cellular biology is much more closely tied to molecular structure, process, and function compared to developmental biology.
[184] Alberts et al. 1994, p. xiii.
[185] Alberts et al. 1994, Chapters 8 and 5 respectively.
[186] Alberts et al. 1994, Chapter 14, entitled "Energy Conversion: Mitochondria and Chloroplasts."
[187] Alberts et al. 1994, Chapters 9, and 21, 22 respectively.

The book explores the structure, processes, and functions of cells. For example, it explores both the structure of the cell membrane and the processual transport of small molecules and macromolecules across it.[188] It explores one important structure maintaining the integrity of the cell, the cytoskeleton, as well as the mechanisms and timing involved in making *more* cells.[189] Structures and processes are both emphasized in the models and explanations of the book. Functions, of molecular activity, pertinent to the larger system—cells—are constantly emphasized.[190]

[188] Alberts et al. 1994, Chapters 10, and 11, 13 respectively.
[189] Alberts et al. 1994, Chapters 16, and 17, 18 respectively.
[190] As mentioned in footnote 96, this notion of function can be analyzed using Cummins' framework of compositional and functional explanation to be explicated in Chapter 5.

	Comparative Anatomy	**Functional Morphology**	**Physiology**	**Developmental Biology**	**Molecular Biology (Biochemistry & Cellular Biology)**
Examples of Parts (Concrete and General)	• Bones, organs • Structural parts (structural modules)	• Leg, jaw (usually musculo-skeletal system) • Morphological units that have a Cummins' function • Parts involved in/causing the activity • Activity parts (processual → functional morphological modules)	• Molecules, hormones, pancreatic tissues (parts involved in activities at various levels) • Parts involved in/causing the activity • Activity parts (processual → physiological modules)	• Molecular genes ("genes-D"), morphogenetic gradients, inductive regions • Parts involved in the activity (e.g., endoderm) • Parts causing the activity (e.g., genes-D) • Activity parts (processual → developmental modules)	• Molecules – especially genes and enzymes, cells • Structure of molecules, cells • Active parts of molecules (e.g., enzyme active site) • Parts involved in/causing the activity • Activity parts (processual and structural molecular modules)
Criteria of the Partitioning Frame	• Shape • Connectivity to other parts • Topological relations with other parts • Position in system • Structural identity	• Activity that is tied closely to morphology (rare to study, e.g., respiratory system) • Cummins' function	• Activity that is tied to dynamic metabolic and reproductive processes • Cummins' function	• Activity that is intense inside a unit over ontogenetic time • Activity that changes surrounding regions over ontogenetic time • Cummins' function	• Structural identity at molecular and cellular level • Molecular and cellular activity • Cummins' function

	Comparative Anatomy	**Functional Morphology**	**Physiology**	**Developmental Biology**	**Molecular Biology (Biochemistry & Cellular Biology)**
General List of Guiding Biases	(1) Study the static adult; ignore development and physiology (2) Dissect, label, organize parts by type (3) Use diagrammatic and scale models (4) Compare *same* parts across taxa; use this to infer *phylogenetic history*	(1) Study the functionally dynamic adult; ignore development and physiology (2) Observe behavior of animals; "experiment" with scale models (3) Use diagrammatic, narrative, and idealized formal models, tying them to part-structure (4) Infer Wright function	(1) Study, primarily, the functionally dynamic adult (small-scale temporal change); physiology of embryos is also studied (2) Experiment with live organisms; measure levels of "index" molecules (3) Use diagrammatic and narrative models (4) Infer Wright function	(1) Study the developing organism; include the study of ontogenetically changing morphology and physiology (historical change) (2) Experiment with live organisms (e.g., gene knock-outs, stains, or other "index" methods for changing parts) (3) Use diagrammatic and narrative models (4) Note how "difference arises from sameness"	(1) Study structures and processes at the molecular and cellular level (2) Experiment with molecular and cellular systems in vitro or in situ (3) Use diagrammatic, narrative, and scale models (4) Characterize link between molecular (and cellular) structure and the processes and Cummins' function determined by that structure

Table 3. (2.1) A table, divided in two due to space constraints, indicating different aspects of parts for different theoretical perspectives. The first four columns are cases of organismal biology.

In this table, I have tried to capture examples and assumptions about parts for the theoretical perspective constituting each discipline. The theoretical perspectives are the columns of the table. The first row provides examples of parts studied in each discipline. Concrete examples are given (e.g., bones or hormones) as well as general kinds of examples (e.g., activity parts, of which one example, under molecular biology, is glycolysis, the Krebs cycle, and oxidative

phosphorylation – the three parts of cellular respiration). Note that the distinction between (1) parts *involved in* an activity and (2) parts *causing* an activity exists only in developmental biology, as discussed above in section 3.C.

Note that in the last entry of each cell for the first row, I have provided names to the kinds of "modules" (parts) that I think are important for that theoretical perspective.[191] The names are provisional and even ambiguous. For example, in the case of purely process-oriented perspectives, such as functional morphology, physiology, and developmental biology, there is an ambiguity about what "processual" part—named, more specifically, a "module" for each perspective—means. The *parts involved in*, and *causing the*, activities studied by each perspective are identified using processual criteria (i.e., the intensional sets defining kinds of parts in each theoretical perspective contain criteria pertinent to process, as characterized by that perspective). For example, developmental biology defines regions such as endoderm, mesoderm, and ectoderm in terms of what they *do*, whereas physiology defines systems and parts thereof in terms of their role, for example, in maintaining homeostasis. But the *process itself* (e.g., gastrulation rather than the cells and regions involved in gastrulation) is also divided into stages (e.g., invagination at various phases) – there are *activity parts*. Thus, "processual" part includes both (1) processually-defined parts involved in, and causing the, activity and (2) activity parts. A better nomenclature could be provided.

Whereas the first row provides examples of parts, the second row states the criteria *constituting the* theoretical perspective's "partitioning frame." A partitioning frame is the set of biases and assumptions that describe the way that

[191] See also Winther 2001.

a discipline carves up the system under study into parts. In compositional biology, such frames are extremely important. They, of course, also rely on abstraction – kinds of parts and models concerning parts need to be delineated and elaborated, even if not explicitly, in order to provide content to the frame. But the *referent*s of these frames, are fundamentally, concrete parts.

The entries in each cell of the second row under each perspective are intended to be general. The functions—capacities requiring explanation—of functional morphology and physiology are different since the explananda are different in those two fields. In functional morphology, the capacity concerns gross-scale external behaviors such as locomotion and feeding, whereas in physiology, they are pertinent to dynamic internal states (often involving molecular activity) such as homeostasis.

In the last row I mention some of the general biases pertinent to each of the theoretical perspectives: (1) general object of study, (2) experimentation methods, (3) model-types employed, and (4) general inferences that are desired and justified. Partitioning criteria are also biases, but these are given in the row above. There are many other guiding biases, but the four kinds I delineate help to capture much of the structure, method, and purpose of a theoretical perspective.

Table 3. (2.1), then, helps to clarify how different theoretical perspectives pertinent to each discipline of compositional biology investigate parts.

2.4 Conclusions: The Complexity and Relativity of Organismal Biology

In this chapter, I have shown that a variety of compositional biological sciences investigate (1) complex objects using (2) complex abstraction and (3) material and propositional non-mathematical models. In addition, they are clearly concerned with (4) the concrete compositional structure, process, and function of the integrated and hierarchical systems that they study. Thus, my framework distinguishing formal from compositional biology using four distinctions, is confirmed by, and helps us understand, the theoretical and empirical structure of biological science.

Furthermore, I have developed the notion of a "theoretical perspective" in order to provide a conceptual unit that allows us to individuate the practices (including theoretical practices) of particular communities of scientists concerned with specific types of methods and goals. A theoretical perspective is a set of guiding biases and assumptions that coordinates and leads scientific activity, particularly the activity of a given *discipline*.

I have provided an "empirical" analysis of six disciplines of compositional biology using textbooks endemic to each as the source of data for their commitments to guiding biases and assumptions concerning particularly *partitioning*. I have sought to describe the "partitioning frame" peculiar to each theoretical perspective/discipline. I believe, and hope to have shown, that the partitioning frame of each discipline I have investigated is presented in its textbooks – as long as we look carefully enough and focus on, for example, the

structure of the textbooks. I have summarized the guiding biases and assumptions of each perspective in a table at the end of the chapter.

Let me end here on a tentative note. Focusing on the complexity and variety of compositional biology, particularly organismal biology, points to an interesting set of issues – relativism and the disunity of science. What do we make of the phenomenon, which I hope to have shown here, that completely different theoretical structures can analyze the *same* entity—the organism—so differently, without being in conflict and providing *competing* hypotheses for the structures, processes, and functions of that entity? Usually we have an image of different scientific theories concerning the same structure as being in conflict, with the difference between them being resolvable by experiments or even "non-rational" criteria (e.g., the Ptolemaic and Copernican interpretation of the universe). Perhaps this is the case in much of physics and even formal biology, where objects are simple and well-defined (recall Appendix Figure 2.1). Furthermore, in those cases, objects often fall under the purview of one perspective or another. In compositional biology, genuinely different and *incommensurate* perspectives exist blithely side-by-side. In fact, this is a strength for a robust analysis of systems from the point of view of compositional biology. Relativism implies robustness here. Disunity leads to strength.

Chapter 3: On the Prospects of Synthesis Between Levels of Selection Theory and Evolutionary Developmental Biology

3.1 Framing and Goals

In Chapter 1, I differentiated abstractly between two kinds of biology, formal and compositional. Using textbooks as data, I elaborated on compositional biology and its diverse theoretical perspectives, or disciplines, in Chapter 2. In this chapter, I will compare the two kinds of biology using both levels of selection theory as representative of formal biology, and evolutionary developmental biology as an instance of compositional biology. As case studies in this contrast, I will explore how each kind of biological science investigates, partitions, and interprets two biological levels: organisms and social insect colonies.

I develop the two respective hierarchies of theoretical perspectives, as presented in Figure 1. (3.1), to show how formal and compositional biology are instantiated at different levels of specificity of guiding assumptions and biases[192]. This contrast emphasizes that although levels of selection theory and evolutionary developmental biology study similar units (e.g., multicellular organisms), they address them differently. For example, as I will show, they employ different forms of abstraction, use different model-types, differ in their

[192] As explained in Chapter 2, I do not intend to imply that the assumption is devious or reprehensible, as vernacular usage suggests. I use "bias" to describe a "commitment" guiding empirical and theoretical investigation.

views of the relevance of compositional organization, and employ different explanatory resources. Any attempt at synthesizing the more general theoretical perspectives that they represent, which is the aim of current attempts at providing a unified account of evolution, development, and genetics, will be extremely arduous.[193] It is even unclear what such a synthesis would *mean*. Thus, we should not glibly proclaim that a synthesis between evolution (*sensu* levels of selection theory) and development (*sensu* evolutionary developmental biology) is forthcoming. The wide chasm between formal biology and compositional biology has yet to be bridged and few researchers in either field can articulate what essential or useful concepts might lie across such a bridge. This makes it unclear what the nature of such a synthesis will look like, a question to which I shall turn toward the end of this chapter when I discuss four distinct interpretations of the relationship between the two hierarchies of theoretical perspectives.

Let me briefly summarize the organization of the chapter. In section (2), I provide some general considerations of the two "super"-theoretical perspectives that levels of selection theory and evolutionary developmental biology fall under, the *competition* and the *integration* theoretical perspectives, respectively. In section (3), I show in great detail how these two instantiations of the super-theoretical perspectives investigate both multicellular organisms and social insect colonies. In section (4), I discuss different possible interpretations of (failures of) synthesis. In section (5), I conclude with a literary analogy.

[193] An early attempt at doing this can be found in the collaboration between Sewall Wright and Theodosius Dobzhansky (Frederick Churchill and Michael Wade, pers. comm.'s).

3.2 Two Super-Perspectives: General Considerations

In this section, I want to distinguish between two perspectives that coordinate work in each of the two kinds of biology. I will explore various kinds of differences between these two "super-perspectives," including their employment of similar words to mean very different kinds of things. I also present their differences in a hierarchical diagram.

3.2.1 The Basic Distinction

In previous work, I have distinguished between the *Competition Perspective* and the *Integration Perspective* as two theoretical perspectives guiding work in a variety of disciplines.[194] Here I want to (1) discuss the characteristics of these "super-perspectives" and (2) relate these, through the use of a diagram, to the two kinds of science and the two particular cases that I will investigate, levels of selection theory and evolutionary developmental biology.

First, let me provide a description of each of these two super-perspectives. Research under the *competition* perspective explores the selective processes acting among units at different levels of the genealogical hierarchy (e.g., gene, organelle, cell, organism, superorganism, species, and clade). It employs the replicator/interactor distinction. Replicators cause, or are statistically correlated with, interactors and copies are made of them. Interactors interact as a whole with their environment and cause the differential reproductive success of

[194] Winther 2001a, 2003 in press.

replicators.[195] The competition perspective is fundamentally interested in the patterns and processes of changes in replicator (e.g. gene) frequencies, across generations, in populations of a mathematically idealized hierarchy of interactors. For example, in social insect colonies, selection occurs at both the level of the social insect *organism* and the social insect *colony*. However, some instances of the competition perspective, such as gene selectionism, are not particularly interested in the hierarchical structure of the interactors – they focus on the competitive dynamics between the replicators. In general, the competition perspective explores, for example, the conditions necessary for the fixation of genes for cooperation at the expense of genes for defection, rather than focusing on the integrative mechanisms, such as the physiological and behavioral relations that are involved in organizing the colony.

Investigations in the *integration* perspective are concerned with the interactive mechanisms among parts and with the patterns of evolutionary change of processual and structural parts. Mechanisms of interest to this perspective can be roughly divided into two categories: (1) developmental, i.e., those processes involved in causing the dynamical path taken during the production of an individual (diachronic), and (2) physiological, that is, those processes occurring at each step of this path (synchronic). For example, in multicellular organisms, this perspective investigates both the different embryonic regions interacting during development and the specialized physiological processes that ensure organismal cohesiveness and functionality.

[195] Dawkins 1976; Hull 1980; Brandon 1982; for a philosophical review see Lloyd 2000a; for a more sociologically-oriented review see Lloyd 2000b.

3.2.2 When Similar Words Mean Very Different Things

Words like "competition," "defection," "selfishness," "manipulation," and "independence" are often contrasted with words like "cooperation," "integration," "altruism," and "sociality." Although the words do not clearly fall into neat categories, and although the relations between the terms are many, there is a contrast between "inward-independence" and "outward-interaction." I realize that this is very general and vague, but I do think that these two extremes can be found, in very different ways, in each of the two perspectives – particularly with respect to how parts (*mathematically idealized* parts in the case of the competition perspective[196]) behave. I will now present how the contrast plays out in each perspective.

For the *competition* perspective:

Modular (part) cooperation ["outward-interaction" sensu competition perspective] is explained as a strategy, on the part of a mathematically idealized interactor module, to maximize its inclusive fitness or its reciprocal fitness benefits[197], or both, in a group context. Alternatively, modular cooperation can be enforced by various higher-level control methods subject to higher-level selection.

Modular (part) defection ["inward-independence" sensu competition perspective] is accepted. It happens when a mathematically idealized interactor module gains short-term fitness benefits at the expense of

[196] Recall the discussion in Chapter 1. The idealized parts (e.g., mathematically defined loci) are equivalent in kind and are structurally, processually, and functionally depaupered. They lack the concrete richness of the parts investigated by the integration perspective.

[197] Due to space constraints I do not, in this chapter, specifically discuss this type of fitness structure, common to game theoretic models. But see Winther 2003 in press.

another module or the whole collection of modules, that is, at a cost to other modules or to the whole individual consisting of modules. It is believed that modular defection occurs in situations inimical to cooperation, i.e., when genetic relatedness among modules is "too low," when reciprocal fitness benefits in mutualistic relationships are "too low," or when the higher-level controls on lower-level defector variant modules fail, or a combination of all three.

For the *integration* perspective:

Modular (part) integration ["outward-interaction" sensu integration perspective] is interpreted as diverse kinds of developmental and physiological processes that establish functional integration of the whole. The parts engaged in the processes ("agent parts," as explained in Chapter 2) have capacities that explain their integrative behavior[198]. These capacities interact to cause higher-level system behavior. Furthermore, the processes are divided into phases, which I called "activity parts" in Chapter 2. Integration is the typical result of development. Note that interaction in this perspective is understood not as a cooperative act, on the part of strategizing modules bent on increasing their fitness.

Modular (part) independence ["inward-independence" sensu integration perspective] is interpreted as mechanistic dysfunction. This occurs when the module does not engage in its appropriate capacity. This is not

[198] Which I will explore in detail in Chapter 5 in discussing Cummins' views, and comparing them to Kauffman's and Wimsatt's views on part decompositions and mechanism ascriptions to provide what Kauffman calls an "articulation of parts" explanation.

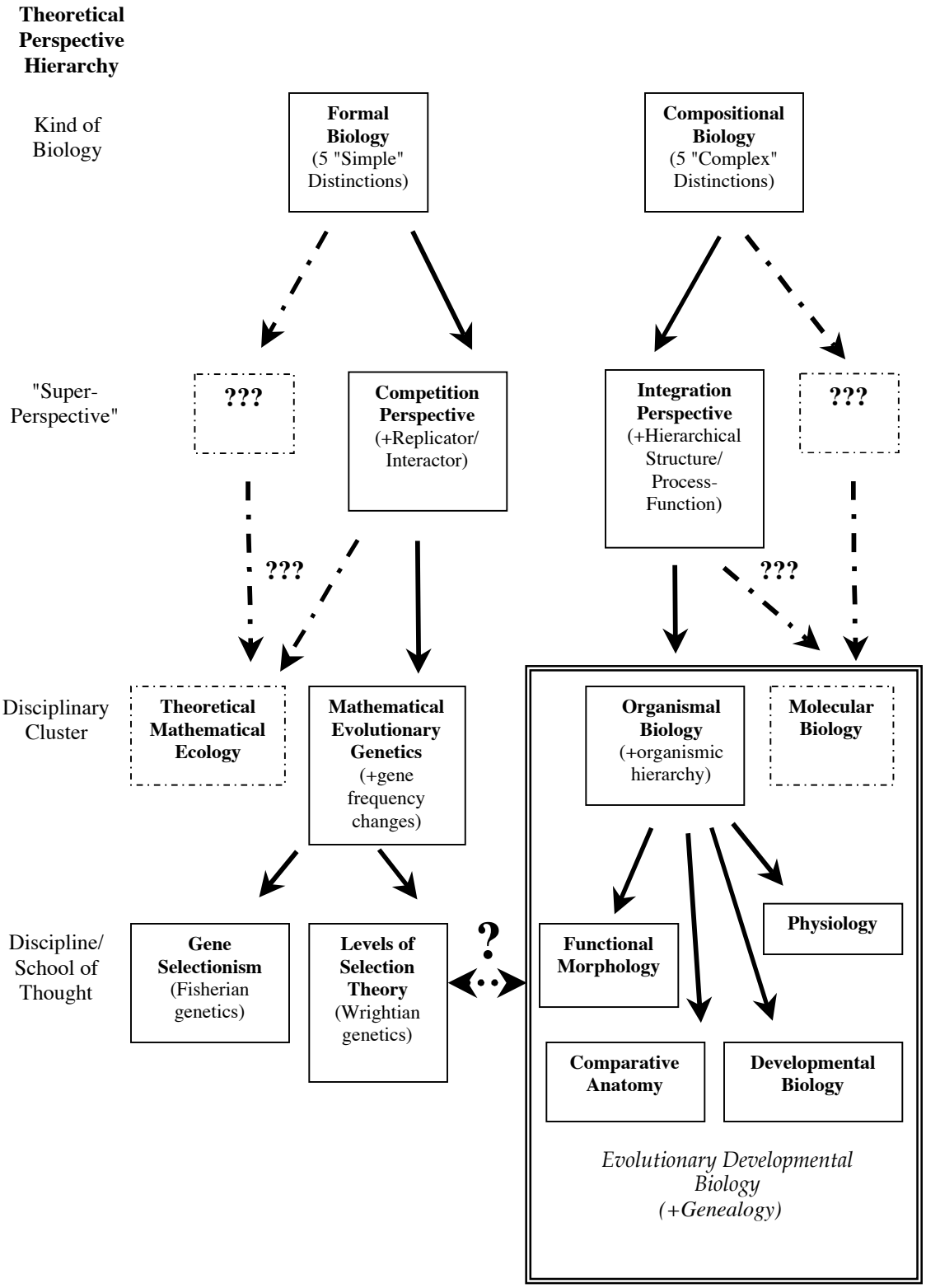
interpreted as being of any advantage, selective or otherwise, for the module.

3.2.3 A Diagram: Two Hierarchies of Theoretical Perspectives

As described in Chapter 2, a theoretical perspective is a set of guiding biases and assumptions pertinent to scientific activity. I mentioned how theoretical perspectives can exist at multiple levels in an abstraction hierarchy. My goal is to develop a full hierarchy of theoretical perspectives, with increasingly fewer guiding biases and assumptions as we move up the hierarchy. However, the fact that there are *fewer* biases does not imply that the high-level biases are narrow or uncreative.[199] I depict this hierarchical organization in Figure 1. (3.1) on the next page.

[199] In Chapter 5 I will distinguish between two alternative dimensions regarding abstraction: removal-incremental and creative-mundane. My endorsement of Cartwright's removal view of abstraction, does not imply that I adopt the view that more abstract statements, models, or (sets of) biases tie fewer things together or are less powerful. In fact, biases common to very abstract theoretical perspectives should be interpreted as extremely powerful. They creatively guide much work. In this sense, only counting number of biases to evaluate level of abstraction, while necessary, is not going to fully capture the qualitative aspect of the biases.

Figure 1. (3.1) Diagram of two hierarchies of theoretical perspectives.



3.2.3.1 General Structure of Diagram

The hierarchy of theoretical perspectives can be imagined to go all the way up to the two kinds of biology. That is, the five distinctions[200] explicated in Chapters 1 and 5 can be *thought of* as guiding biases and assumptions (e.g., for formal biology assume and investigate simple objects). Further specification of the theoretical perspectives occurs through the addition of (more concrete) biases. The first level of specification is to implement the central biases pertinent to the two theoretical perspectives here considered, *competition* and *integration* (called the level of *super-perspectives* in the diagram). These biases are, respectively, the replicator/interactor distinction (mirrored, particularly, in abstract mathematical models), and the view of a complex hierarchical organization of material structure and process-function parts (represented especially in representational non-mathematical and material models). There may be other biases that further specify super-perspectives out of the kinds of biology – these would then produce a different set of super-perspectives suggested by the dashed boxes and arrows. I will leave these additional guiding biases unspecified at this point.

These super-perspectives are then further concretized to form *disciplinary clusters*. Mathematical evolutionary genetics has a particular commitment to the replicator unit – genes (or groups of genes, if they are in linkage disequilibrium) are replicators. Other disciplinary clusters, such as theoretical mathematical

[200] I.e., (1) attention to simple versus complex objects, (2) employment of simple versus complex abstraction, (3) use of mathematical versus propositional non-mathematical or material models, (4) irrelevance or idealized vs. relevance of part-whole organization, and (5) causal and theoretical explanation in formal and compositional contexts (the distinction will be explained in Chapter 5).

ecology might have different commitments to the replicator unit – such units could be groups, avatars or species[201] or they might have altogether different sorts of commitments (guiding biases). Describing which super-perspective motivates the disciplinary cluster of theoretical mathematical ecology, as indicated by the dashed boxes and arrows, is beyond the scope of this dissertation.

In the compositional biology hierarchy, the disciplinary cluster "organismal biology" singles out the organism as the central unit in the hierarchies of structure and process-function (see Chapter 4). The organism can be further partitioned[202] into various levels, and kinds, of parts. These different decompositions are done by the various disciplines within organismal biology (e.g., functional morphology and developmental biology) as represented in the diagram and as explained in Chapter 2. In this way organismal biology differs from molecular biology both in focusing on a different level and, more importantly, in being intrinsically hierarchical. Molecular biology (e.g., biochemistry, cellular biology, and molecular genetics) seems to be *a level of* the hierarchy *within* organismal biology, and is therefore most likely "foundational" ("mono-hierarchical") in the sense developed, tentatively, in Chapter 2. Thus, as portrayed by the dashed boxes and arrows, it is unclear which super-perspective coordinates work in molecular biology, but this too lies outside of the scope of this dissertation.

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[201] See the table in Brandon 1990, p. 97, Appendix Figure (Table) 3.1.
[202] Or aggregated. But aggregation of organisms does not usually take place in the work of organismal biology; this scientific activity is left to, for example, the ecologists (into populations and communities) and systematists (into genealogies and clades).

The last and most concrete level in the hierarchy of theoretical perspectives here considered is that of *disciplines* and *schools of thought*. Conceptually, both have many detailed guiding biases coordinating their work. In mathematical evolutionary genetics, differences in such biases between gene selectionists and levels of selection theorists sometimes lead to acrimonious debate.[203] I use the descriptors "Fisherian genetics" and "Wrightian genetics" as short-hand summary indices for the distinct assumptions regarding population size, population structure, genetic architecture, relevance of development, etc. pertinent to each of these two schools of thought. Basically, Fisherian genetics is committed to large population sizes with little structure, genes are assumed to act additively, and the map between genotype and phenotype is assumed to be fairly simple and not, per se, worth exploring in so far as evolutionary questions are concerned. In contrast, Wrightian genetics believes in the existence of small structured populations in which interactions among different sets of genes existing in each group are crucial; development is considered important too in that developmental processes, mediated by different genes in different groups, can express the *same* gene differently.

In organismal biology, differences in guiding biases (e.g., biases regarding partitioning) are more fundamental but tend not to lead to debate, but rather to peaceful co-existence or even blissful mutual ignoring. Perhaps this is because *theoretically-motivated* disputes are not as clearly framed and motivated in compositional biology as they are in formal biology, where a clear mathematical theoretical structure exists that provides grounds for disagreement. Sociologically, some universities have different departments of the organismal

[203] E.g., Lloyd 2000a, 2000b.

biological sciences, such as Pharmacology and Physiology vis-à-vis Organismal Biology and Diversity at the University of Chicago[204] but none have different departments of evolutionary genetical schools of thought (e.g., "Fisherian genetics" or "Wrightian genetics") even if a number of researchers in a department may embrace one of the schools (e.g., at Indiana University, consistently ranked very highly in evolutionary biology, much research occurs in the Wright tradition – consider the work of Edmund Brodie, Curtis Lively, and Michael Wade). The differences at this lowest level of theoretical perspective are of different magnitudes in the two trees of theoretical perspectives here depicted – they are greater for the disciplines of compositional biology.

3.2.3.2 The Concept Collider: Evolutionary Developmental Biology

The various disciplines of organismal biology, together with the concepts and protocols of molecular biology, as well as assumptions and tools from systematics regarding genealogy[205], have been subject to a process of synthesis over the last 20 years. This synthesis is called evolutionary developmental biology, or "evo-devo."[206] Because it is a "hybrid zone" of research, evolutionary developmental biology does not include *all* the conceptual and methodological biases of its various "constituent" disciplines. Furthermore, it has its own guiding biases. It has its own problem structure and employs a variety of disparate methods, acquired from the parent disciplines contained within the double-lined box of Figure 1. (3.1), to address those problems. In particular, it is concerned

[204] I thank Michael Wade for this example.
[205] E.g., the now-triumphant school of Cladism. See Hull 1988; Wiley 1991.
[206] See Hall 1998; Raff 2000; Wagner et al. 2000; Robert et al. 2001.

with the roles of genes and higher-level (organismal) modules, or parts[207], as both constraints and causes, in the evolutionary process. Among other specific issues, it is concerned with homology assessment, the constraining and causative influence of genetic architecture[208] and developmental patterns in evolution, evolutionary "somatic" innovations (e.g., fins, jaws, and wings), and the relationship between micro and macroevolution.[209] Note that these are concerns involving the structural, processual, and functional integration of parts on both developmental and evolutionary time scales. The focus tends to be on the relationships among the phenomena of *genetics*, *development*, *anatomy*, and *phylogeny*.

As can be seen from a variety of sources, such as Brian Hall's evo-devo "textbook" and various programmatic articles by Raff, Wagner and co-workers, Kirschner and Gerhart, and Robert and co-workers, the processually and functionally oriented disciplines of functional morphology and physiology are underplayed in evolutionary developmental biology.[210] This lacuna is not absolute, Kirschner and Gerhart do discuss physiology in their article and book, and Wagner does, implicitly at least, allude to functional morphology in his work on homology, to be discussed below. Evolutionary developmental biology, even though it is a hybrid field with many interests, still has relatively little research on fitness and evolutionary function. In part this is because concerns

[207] As discussed in Chapter 2.
[208] By "genetic architecture" I mean mechanistic interaction among genes. For a description of such interaction see Davidson 2001.
[209] See the summary, and programmatic, statements of evo-devo in Raff 2000, Wagner et al. 2000, and Robert et al. 2001.
[210] Gerhart and Kirschner 1997, Kirschner and Gerhart 1998; Hall 1998; Raff 2000; Wagner et al. 2000; Robert et al 2001. See also Love 2003 on the role of functional morphology.

with natural selection are distinct, even anathema, to a number of the parent disciplines of evolutionary developmental biology.

3.2.3.3 Four Distinctions, Some Limitations, and the Goal

The diagram of hierarchical theoretical perspectives, albeit incomplete, helps to fix thoughts on the hierarchical organization of the guiding biases and assumptions of scientific work in a (large) subset of the biological sciences. This hierarchical structure adheres formally to the general account of abstraction that I articulated in Chapter 1 where I took abstraction, of *scientific kinds of objects,* to be the removal of properties (of various kinds) from intensional sets referring to objects. We can think of theoretical perspectives as hierarchical kinds, or at least *being describable* in this way. Here the "objects" are the sum total of scientific activity abstracted (for purposes of definition) into key guiding biases and assumptions, as discussed in Chapter 2. Now, by removing single biases[211] at each subsequent generalized level, it seems that I am advocating simple abstraction as the way that theoretical perspectives themselves can be abstracted from a philosophy of science vantage point. I want, however, to remain agnostic about this. While addition of single central biases is, perhaps, necessary to theoretical perspective specification, it is not sufficient. A number of other biases, particularly regarding laboratory and field practice, are correlated, and are specified concomitantly, with these biases. Furthermore, the qualitative nature of the biases added may be different for lower-level biases. Due to constraints on

[211] Or, going in the concretizing direction indicated in the diagram, by *adding* single biases.

conceptual tractability of my analysis, I do not address these issues, although in Table 3. (2.1), I included some of the laboratory and field practice biases in the row entitled "General List of Guiding Biases."[212]

I want to mention two caveats regarding the diagram. First, the diagram does not represent, or even purport to represent, *all* biological activity. For example, there is a significant amount of work in empirical ecology and even systematics that cannot obviously be subsumed under the two kinds of biology I have here developed, although perhaps they *could be*. Although empirical ecology is certainly not formal, it is also not necessarily compositional in that it does not necessarily nor explicitly address species or populations as parts of an integrated system. Further work is required to explore which kinds of biology (*possibly* formal or compositional) this and other disciplines of biology would instantiate.

Second, the diagram is idealized and does not represent the intra-perspective variation and inter-perspective similarity in actual cases that exists in the two distinct hierarchies. For example, an actual investigation may employ guiding biases from developmental biology and comparative anatomy, or, more pertinently to my analysis, another project may employ the very distinct biases from both levels of selection theory and developmental biology.[213] This is a problem that I addressed in Chapter 2, where I considered variation within compositional biology vis-à-vis the four distinctions. My position is that the lines

[212] For analysis of such biases in laboratory practice, see, for example Gerson 1983; Latour and Woolgar 1986; Latour 1987; Griesemer and Wade 1988. A self-reflexive piece on such matters by a group of scientists and statisticians is Neyman, Park, and Scott 1956.
[213] Although below I classify Buss' seminal 1987 book as a work within the levels of selection theory perspective, it does use biases belonging to developmental biology.

of work coordinated by the two kinds of hierarchy of theoretical perspectives here analyzed are obviously distinct in kind even if, on rare occasions, (putative) synthetic work appears. It is to such work that we must turn our attention if we are, as I am, motivated to find synthesis between theoretical perspectives in distinct kinds of biology (represented by the large question mark).

Let me now turn to the larger goals of this chapter. What is the relationship between formal biology and compositional biology? Is it one of different empirical domains (i.e., they study different objects and processes)? Is it one of different methodological resources and epistemological biases applied to similar or different domains? Is it both? To ask a different set of questions, are they incompatible and in tension, do they ignore each other, or can they strengthen each other through some sort of synthesis and unification? Clearly these are not easy questions, but in this chapter I will provide a preliminary analysis of the questions through the *particular case studies* of levels of selection theory and evolutionary developmental biology, as instances, respectively, of formal and compositional biology.[214] I will show how the two disciplines, and, therefore, the two kinds of biology, overlap partly in domain of study, but because of their very different methodological and epistemological stances, synthesis between them is difficult and even unclear in principle. I present four interpretations regarding the relationships between the two disciplines, only one being a "synthesis." I will not attempt an actual synthesis, which would amount

[214] Levels of selection theory employs mathematical models and idealizations in describing the simple objects under its investigative purview. Evolutionary developmental biology explores various facets of organismal biology, with its concrete hierarchical part structure and function.

to an answer to the large question mark at the bottom of the diagram – such a synthesis would require the work of *many* biologists and philosophers.

I will now turn to a detailed exposition of the two disciplines.

3.3 Two Super-Theoretical Perspectives: Case Studies

I will here explore how the respective instances of the two super-theoretical perspectives, levels of selection theory and evolutionary developmental biology, both investigate multicellular organisms and social insect colonies. Doing this in detail will show how each discipline/school of thought is distinct in terms of the (epistemic and methodological) guiding biases and assumptions it employs.

3.3.1 Levels of Selection Theory on Social Insect Colonies

In discussing levels of selection theory, I will start with the social insect colonies because it is with these systems that levels of selection theory in the competition perspective originated. Historically, this started with Darwin's worries about the evolution of sterility in hymenoptera, where the drastic reduction of immediate organism (or gene) fitness (i.e., sterility) in favor of a higher-level group of related organisms is most apparent. Only in the last two decades has a levels of selection research program been applied to multicellular organisms, starting with Buss' 1987 book. As discussed in Chapter 1, a commitment to different levels in nature, and selection among units at those different levels, does not imply a commitment to the relevance of concrete

compositionality. In fact, parts are *highly* mathematically idealized in levels of selection theory. In my analysis to follow I sometimes contrast the ontological and modeling (epistemic) commitments of investigators following levels of selection theory with those of researchers in the gene selectionist tradition. I do this in order to clarify and emphasize the guiding biases of levels of selection theory and to show diversity of biases within the competition perspective.

3.3.1.1 The Origins of Levels of Selection Theory, 1964-1975: The work of W. D. Hamilton and G. R. Price

Why are some organisms, such as hymenopteran workers, sterile? This seems to amount to having a fitness of zero. The origin and maintenance of cooperation, which is often called altruism, was a problem that Darwin wrestled with in his famous book.[215] His prescient answer appealed to family-level selection of "fertile parents which produced most neuters with… profitable modification[s]."[216] Both Sewall Wright and G. C. Williams started to extend Darwin's logic, but the first full mathematical exposition of this group selection argument was developed by Hamilton more than 100 years after Darwin's comment.[217] Initially, however, Hamilton argued against group selection and felt that inclusive fitness made kin selection an extension of individual selection.[218] Hamilton was interested in why organisms would reduce their fitness, for the benefit of other organisms, through behaviors such as defending the other

[215] Darwin 1964 (1859), chapter 7.
[216] Darwin 1964 (1859), p. 239.
[217] Wright 1945; Williams and Williams 1957. See Sober and Wilson 1998, pp. 58-64.
[218] Hamilton 1963, 1964a, 1964b.

organism or helping it to reproduce. Qualitatively, he argued that an allele that caused a behavior detrimental to a particular individual would increase in frequency when the recipients of the behavior were close kin who, with a high probability, carried the same allele. Quantitatively, he noted the conditions under which alleles for cooperation could increase in frequency: $rB - C > 0$, or $r > (C/B)$. In this equation, r is the coefficient of relatedness (e.g. in diplo-diploids (e.g. mammals) organism-to-sibling $r = .5$; organism-to-first cousin $r = .125$), B is the fitness benefit the given behavior provides to the recipient, and C is the fitness cost, to the benefactor (focal individual), of performing the behavior. This equation is known as "Hamilton's rule" and it represents the conditions under which kin selection occurs.

What Hamilton noted was that in hymenoptera, females are more closely related to their sisters ($r = .75$) than to their offspring of either sex ($r = .5$), provided that the females have the same father.[219] This high relatedness[220] occurs because hymenoptera are haplo-diploid: males have only one chromosome of each pair of chromosomes, females have both chromosomes of each pair of chromosomes. Thus, on relatedness grounds alone, a female should choose to help her mother rear offspring, which are her sisters: "Our principle tells us that even if this new adult had a nest ready constructed and vacant for her use she would prefer, other things being equal, returning to her mother's and

[219] Michael Wade noted that "many 'modern behaviorists' are less enamored with the Haplo-Diploids than with the within-colony conflict between queen and worker over the sex ratio as the best supporting evidence for Hamilton's theory." (Michael Wade, pers. comm., July 3, 2003)
[220] Historically, this became generalized later to high gene *identity*. I thank Elisabeth Lloyd for reminding me of this historical shift.

provisioning a cell for the rearing of an extra sister to provisioning a cell for a daughter of her own."[221]

There are complications to this simple theory, however. Hamilton noted, as others subsequently have, that multiply-mated queens produce female offspring with a relatedness coefficient smaller than .75. If the queen has mated with two males, and assuming equal contribution from the two males and no sperm competition, among-sibling relatedness is .5. If the queen has mated with more than two males, the relatedness coefficient is smaller than .5 and converges to .25 as the number of males gets very large, given the assumptions stated above.[222] (In honey bees, a queen will typically mate with more than 20 males![223]) Hamilton noted that, despite this, cooperation would still be favored given appropriate *B* and *C* parameters. Furthermore, in some genera, for example, worker fire ants of the genus *Solenopsis*, which lack ovaries, reproduction is not even a possibility. Thus, other parameters and conditions besides *r*, *B*, and *C* need to be considered. Another complication that Hamilton discussed, but did not suggest an explanation for in his early articles, is that a worker is only related by .25 to her brothers, whereas she would be related by .5 to a son. Trivers and Hare subsequently suggested that workers would still prefer to raise sisters rather than offspring if they could skew the sex ratio of sibling reproductives toward a 3:1 gyne[224]:male ratio.[225] Complications such as these have convinced

[221] Hamilton 1996 (1964b), p. 58.
[222] Hamilton 1996 (1964b), p. 62; Hamilton did not explicitly mention sperm competition.
[223] Michael Wade, pers. comm.
[224] A gyne is a reproductive female.
[225] Trivers and Hare 1976; see also Crozier and Pamilo 1996. Lloyd critiques the Trivers and Hare finding in Chapter 8 of Lloyd 1988.

investigators that Hamilton's rule is shorthand for more elaborate quantitative and population genetic models.

Investigators in the competition perspective employ Hamilton's rule. Their focus is on the reproductive dynamics of social insect colonies leading to gene frequency change. They have furthermore tended to only estimate *r*.[226] This is in part because it remains conceptually unclear how to estimate *B* and *C*, which both depend on ecological conditions. For example, which metric could we use to compare alternative worker strategies of egg-laying and foraging in estimating *B* and *C*?[227]

Despite complications regarding Hamilton's rule, it has turned out to be powerful, given assumptions of genetic variance additivity. A number of population geneticists were skeptical of Hamilton's rule until it could be shown in the 1980s by Feldman, Uyenoyama, Wade, and others that it describes, under additivity assumptions, changes in gene frequency and not just an inevitable fitness maximization. The rule and mathematical models developed from it are a good example of formal biology as can be seen with how the four differentiae are met in this case. (1) The rule applies to *any* (additively acting) gene that somehow increases altruism – the rule and models pertinent to it represent genes as *idealized* simple objects (that are equivalent vis-à-vis one another and can therefore be represented with the same formula). Furthermore, details of the behaving individuals who constitute the social group are ignored. (2) It involves simple abstraction in that a broad variety of different genes, behavioral mechanisms (including those involved in both defense and reproduction!), and

[226] Cf. Gadagkar 1991; Bourke and Franks 1995; Queller and Strassman 1998.
[227] Deborah Gordon pers. comm.

ecological contexts are ignored, or at least averaged out, and the *same* few necessary and sufficient properties determine each of the mathematical kinds of the rule (r[228], B, and C). Note that r is a property of the replicator, whereas B and C are properties of the interactors (and interactor-environment averaged context). Furthermore, elaborations of Hamilton's rule in more concrete models follow precise additions of variable types with necessary and sufficient definitional properties. (3) Hamilton's rule and the models developed from it are formalized mathematical models. They are based on principled assumptions regarding genetic relatedness and cost-benefit relations among simple objects with precise mathematically-defined behaviors. Furthermore Hamilton's rule is law-like. (4) Compositional organization is not considered relevant and is idealized by abstraction. The few precise salient properties of the idealized units/agents of the social system are captured in the different mathematical kinds, for purposes of the principled mathematical model.

Hamilton initially emphasized that kin selection was an extension of individual selection; he used the term "inclusive fitness."[229] Price's covariance approach to selection radically changed Hamilton's view on kin selection.[230]

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[228] Even in recent derivations of the rule, employing different definitions of relatedness, for example, there are still precise definitions about how to abstract these different variables (i.e., there are clear necessary and sufficient properties accruing to each variable type); see Frank 1997 who distinguishes two types of similarity correlations ("r") that serve different purposes (intra-generational vs. inter-generational dynamics).

[229] E.g. Hamilton 1964a.

[230] In his autobiographical introductory sketch to the reprint of the article, Hamilton writes: "…I am proud to have included the first presentation of Price's natural selection formalism as applied to group-level processes. I wish George Price had been alive to see it published." (Hamilton 1996, p. 318) In the actual article, Hamilton writes: "A recent reformulation of natural selection can be adapted to show how two successive levels of the subdivision of a population contribute separately to the overall natural selection. The approach is not limited

(Covariance is a probabilistic and statistical measure of the *correlation* between two variables.) Price sought to develop a "general selection theory."[231] He realized that selection could be thought of as a covariance between the fitness of the hierarchical units under study and their properties. These properties could be genotypic or phenotypic. Price, and others, have shown mathematically that this covariance can be decomposed into two components, each of which describes selection at one of two levels – (1) *within* the interactor unit (that is, *among lower-level* interactor units) and (2) *among* interactor units.[232]

A hierarchical selection process thus causes gene (replicator) frequency change. In social insects, a non-zero first component representing within-colony selection could be caused by workers altering the colony sex-ratio and thereby altering the normal Mendelian ratios (i.e. under-representing maternal genes by destroying males, over-representing paternal genes by not destroying gynes). This is analogous to meiotic drive in organisms.[233] In social insects, a non-zero second component indicating among-colony selection would occur whenever some colonies left more offspring colonies than other colonies. Such selection is also part of some sex-ratio evolution models.[234] Among-colony selection is analogous to organismal selection in organisms. Price's multi-level selection equation, which decomposes the causes of gene frequency change, can be further expanded to any number of levels so that we can have, for example, among-

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to Mendelian inheritance but its usefulness in other directions (e.g., cultural evolution) has not yet been explored." (Hamilton 1996, p. 332)

[231] Price 1995, p. 389; Price 1970, 1972.

[232] Price 1970, 1972, 1995; Wade 1980, 1985; Frank 1995. See especially the clear derivations in Wade 1985, pp. 62-64, 68; Frank 1995, pp. 375-377. There are potentially some problems with this interpretation of Price's equation as has been recently shown in Kerr and Godfrey-Smith 2002a, 2002b.

[233] Cf. Werren et al. 1988; Hurst et al. 1996.

[234] Michael Wade pers. comm.

colony, among-ant (e.g., sex ratio conflict within colonies), and within-ant (e.g., meiotic drive in queen ants) selection in social insects. The Price Equation is one of the central, as well as theoretically unifying, mathematical models of levels of selection theory.[235]

Hamilton employed Price's equation in an article in which he argued that kin selection was, indeed, a multi-level selection process.[236] Cooperation could evolve (i.e. alleles for cooperation increase in frequency) if among-colony selection for such alleles was stronger than within-colony selection against such alleles. One way to increase among-colony additive genetic fitness variance was precisely to have colonies with only one or a few queens mated with only one or a few males. This is analogous to unicellular bottleneck reproduction of sexual organisms.[237] In these cases most additive genetic fitness variance would be among colonies, rather then within them.

Kin selection, whether conceptualized as a single-level or multi-level process, describes the selective dynamics among interactors at multiple levels of selection, such as ant-organisms and social insect colonies. Replicators, such as alleles for cooperative behavior, can increase in frequency as a consequence of these dynamics.

[235] Cf. especially Wade 1985; Frank 1997, 1998. But see Kerr and Godfrey-Smith 2002a, 2002b.
[236] Hamilton 1975.
[237] Michod 1999a.

3.3.1.2 The Expansion of Levels of Selection Theory, 1976-2003

Subsequent to the development of Price's equation, levels of selection theory was expanded by a number of investigators.[238] Broadly stated, these investigators found that most cases of selection can be understood as hierarchical selection processes with hierarchical selective components. All cases of selection in populations with social interactions can be decomposed into at least two components: selection among groups and selection among individuals. Importantly, all cases of kin selection are hierarchical and Hamilton's rule can be shown to be a necessary condition for selection among groups to override opposing selection among individuals.[239] Not all cases of hierarchical selection need involve kin, however: consider selection on symbiotic relations such as lichens. Here, selection of the two-species group caused the symbiotic relation to evolve.

There are indeed few cases in nature to which a hierarchical approach could not be applied. Those cases that approach the Fisherian idealization of extremely large, randomly-mating, unstructured populations are candidates. The empirical adequacy of the Fisherian idealization is an ongoing topic of debate. Even if the idealization is false, however, its utility in making mathematical models simple and tractable still remains a powerful argument, in its favor, for gene selectionists.[240] The levels of selection approach has been further applied

[238] Uyenoyama and Feldman 1980; Wade 1980, 1985, 1996; Wilson 1980; Wilson and Colwell 1981; Queller 1992a, 1992b; for historical and philosophical reviews see Lloyd 1988, 2000 and Sober and Wilson 1998.
[239] Wade 1980.
[240] See, for example, Coyne et al. 1997, 2000.

with great fervor to social insects in recent years.[241] It can be used to understand the selective dynamics occurring among and within social insect colonies, considered as abstract units.

But the hierarchical approach has met resistance from investigators adopting a gene selectionist viewpoint regarding kin selection. A number of behavioral ecologists still interpret inclusive fitness and kin selection as an organismal or genic level process or property, as Hamilton originally did in his articles from the 1960s.[242] According to these investigators, complete models can be built using inclusive fitness defined only at the single level of the organism or gene.

But perhaps the difference between gene selectionists and levels of selection theorists is not significant. A number of investigators have cogently shown that single-level inclusive fitness is *mathematically equivalent* to hierarchical selection if the former is defined, modeled, and estimated correctly.[243] Two of these researchers argue that:

> a good understanding of altruism, and the evolution of higher levels of organization in nature, is fostered by the ability to make repeated 'gestalt-switches' between the different perspectives. That is, we should cultivate the ability to switch between seeing groups as fitness-bearing in their own right [levels of selection theory] and seeing groups as part of the milieu that determines the fitnesses of individuals [gene selectionism]. Each perspective makes some facts vivid and obscures others.[244]

Kerr and Godfrey-Smith's pluralism includes an argument against Sober and Wilson's critique of averaging. In their book, Sober and Wilson had claimed that

[241] E.g. Bourke and Franks 1995; Frank 1998.
[242] E.g. Dawkins 1976; Grafen 1984; Krebs and Davies 1993.
[243] Queller 1992b; Dugatkin and Reeve 1994; Bourke and Franks 1995; Kerr and Godfrey-Smith 2002a, 2002b.
[244] Kerr and Godfrey-Smith 2002a, p. 479.

a fundamental mistake in gene selectionist models is that such models *define any* allele-type that increases in frequency in the population at large as selfish, even for "altruistic" allele-types that are *only* increasing in frequency *because* of the existence of population structure (i.e., they would actually *decrease* in frequency in a panmictic group, and do so within groups anyway). Sober and Wilson argued that we must avoid this "averaging fallacy" of only looking at average population-at-large effects because it obscures the causal structure[245] of natural selection. Furthermore, gene selectionists "help themselves," to use a term I got from Lloyd in conversation, to the hierarchical fitness structures of the models. That is, they derive the single-level fitness parameters from previous hierarchical fitness structures. Kerr and Godfrey-Smith, however, argue that averaging occurs *in all models, albeit in different ways* – for example in the Price equation, the covariance between relative fitness of individual types and the properties of individual types is averaged *over all groups*, giving the expectation term of the Price equation. In this case, there is an implicit "group" averaging in the "individual" component of the Price equation.[246]

Kerr and Godfrey-Smith may be correct in their claims that (1) averaging occurs in different ways in the two schools of thought, and hence is a sin common to both, and (2) the models of the two schools of thought can be shown to be equivalent when parameters are defined carefully (i.e., precise equalities are articulated) for each school of thought model-type. Like Sober, Wilson, and Lloyd, however, I believe that causal structure will be obscured in gene selectionist models and hierarchical fitness parameters are crucial elements for

[245] And correctly articulating causal structure is a desideratum of theory.
[246] Waters made a similar argument in Waters 1991.

research programs that *honestly* try to capture the hierarchical selective dynamics. However, I also believe that this debate will continue and *will not* be resolved on rational grounds alone.[247]

I want to add a psychological and sociological dimension to this debate. Even if we grant that (1) the mathematics of Kerr and Godfrey-Smith is completely accurate, (2) the averaging is done by both schools of thought, and (3) "group" and "individual " level parameters and operations are conflated in the Price equation and other formulations of group selection, the two schools of thought and respective model-types are *not* equivalent – each one carries with it psychological biases (in the broad sense of Chapter 2) regarding which causal structures and processes should be looked for, and assumed, in the world. It is no accident that, sociologically, it is mainly advocates of levels of selection theory that have been interested in, for example, evolutionary transitions: Leo Buss[248], Steve Frank, William Hamilton, Richard Michod, and Michael Wade.[249] (Of course, there are exceptions to this correlation, such as Maynard Smith[250], but by and large it holds.) I argue that it is precisely because of their multi-level mathematical modeling bias that they are interested in multi-level processes in nature, and vice-versa. If our research program interest is the evolution of hierarchical organization, then we should employ levels of selection theory, for *pragmatic* psychological and sociological reasons. The *biases* concerning the model

[247] See also Okasha 2001.
[248] In a provocative passage, Buss writes, "Economic and political reality favors the language of genic selection. In these essays, I have, nonetheless, adopted the opposite position: that of a hierarchical perspective on the units of selection problem." Buss 1987, p. 179.
[249] Buss 1987; Frank 1997, 1998; Hamilton 1975; Michod 1999a, 1999b; Michod and Roze 1997; Wade 1992; Wade and Goodnight 1998; see also Lloyd 1988 and Sober and Wilson 1998.
[250] Maynard Smith 1988; Maynard Smith and Száthmary 1995.

set-up of parameter and variable definitions in order to represent selective and genetic causal dynamics in levels of selection theory are the *right* ones to capture the complexity and hierarchical structure of evolutionary transitions.[251] Further discussion of pragmatic reasons for adopting levels of selection theory should complement current "theoretical" (*sensu* mathematical) discussion.[252] Furthermore, one set of theoretical divergences and arguments within mathematical evolutionary genetics has now been clarified.

3.3.2 Levels of Selection Theory on Multicellular Organisms

Now that we have explored levels of selection theory on social insects, which is where the key mathematical models of kin selection, and multi-level selection, were developed, let us turn to the organism level which has also recently received attention as a case of hierarchical selection, both currently and in its origin.[253] The key question for the competition perspective with respect to the organism is: Why did, and do, cell lineages in organisms cooperate rather than defect? Defection appears to be favored at the cell level; cell lineages leaving more cell offspring have a higher fitness, at that level, than those that do not.

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[251] I am not advocating that we restrict the use of levels of selection theory to evolutionary transitions. It can also be used for more micro-evolutionary questions, such as in Sewall Wright's Shifting Balance Theory. Furthermore, I here leave open the question of the use of levels of selection theory by more compositionally-minded biologists such as S. J. Gould, as represented in Gould 2002 and in Gould and Lloyd 1999. Clearly there are interesting synthetically-minded biologists (including also primarily "formalists" such as Lewontin and Wade, and mainly "compositionalists" such as Günter Wagner).

[252] See Wimsatt 1984 for an important early attempt at doing this.

[253] Hamilton (like Maynard Smith and Szathmáry following him), in contrast, believed that "the bodies of multicellular organisms" consisted of clones and, "…our theory predicts for clones a complete absence of any form of competition which is not to the overall advantage and also the highest degree of mutual altruism." (Hamilton 1996 (1964b), p. 55)

Cooperation seems to be disadvantageous at that level; somatic specialization and the attendant curtailed reproduction lowers the immediate fitness of a particular cell. A number of solutions, particularly kin selection (selection among organisms as collections of genetically related cell lineages) and organismal control (policing and punishing of "rogue" cell lineages by the immune or other systems), have been proposed. I divide my discussion into investigators who claim that cell-lineage defection is mainly absent in well-integrated organisms and those who claim that it is prevalent.

3.3.2.1 Cell-Lineage Defectors are Rare in Well-Integrated Organisms

The *locus classicus* for discussion of the evolution of multicellularity, which is an evolutionary transition, is Buss' *The Evolution of Individuality*.[254] He notes that an organism is actually not a "genetically homogenous unit"[255] although it does consist of "clonal lineages"[256]. An organism is an environment "populated by normal and variant cells."[257] Cell lineages, which are interactors, compete within this "somatic ecology"[258]. Variant, that is, defector, cell lineages divide in an uncontrolled fashion and contribute little to *somatic* cell function. They are often detrimental to the whole organism. There is also strong cell-lineage selection for variant cell lineages to enter into the areas, or be part of the lineages, that fulfill

[254] Buss 1987; see also Buss 1983, 1985, 1999.
[255] Buss 1987, p. 19 (contra Hamilton, footnote 253 above).
[256] Buss 1987, p. 77.
[257] Buss 1987, p. 76.
[258] Buss 1987, p. 139.

the *reproductive* functions. Defector cell lineages can therefore disrupt both somatic and reproductive organismal functions, according to Buss.[259]

Since organismal stability, early in the evolution of multicellularity, was threatened by defector cell lineages, methods of controlling defection were strongly favored at the organismal level. For example, (1) the evolution of a sequestered germ-line eliminated the possibility that a variant cell lineage could be heritable *across* organismal generations. Furthermore, since germ cells undergo significantly fewer cell divisions than somatic cells, there is a smaller likelihood of mutation occurring in them, given an approximately constant mutation rate per cell division. Buss also considers (2) maternal control of early development and (3) inductive interactions as organismal control methods. If maternally-derived egg cytoplasmic mRNA and proteins determine division patterns and cell fate, then a cell-lineage variant, with its own genotypic and phentoypic properties, cannot arise until maternal control stops.[260] Similarly, inductive interactions between cell lineages "restrain[ ] or direct[ ] the activities of neighboring cells, …[thereby] enhanc[ing] their own replication and the survivorship of the individual harboring them."[261] This last control method is the only one in which the direction of selection is the *same* at both the cellular and the individual levels (i.e., an appropriately inducing cell lineage increases its own, as

[259] Michael Wade noted to me the interesting reciprocal case: cell lineages undergo apoptosis, or "suicidal" destruction through the activation of internal genetic mechanisms. This suicide is also for the good of the organism. Cell suicide, for example, accounts for normal development of fingers, where the cells in the "web" between the fingers die. This is an interesting phenomenon deserving more attention in the competition perspective. Due to its, up to now, inadequate coverage in the research of the competition perspective, I will not be further concerned with it.
[260] Buss 1987, p. 54 ff.
[261] Buss 1987, p. 78.

well as the organism's, replication rate). Note that all these methods of control are considered higher-level adaptations at the organismal level. Note also that for Buss, the cooperation of cell lineages is the crucial central explanandum.

Sometimes these methods of control fail as when mammalian cancers occur.[262] Less dramatic somatic mutations can also occur. Thus, Buss implies that defection by somatic cell lineages does occur in well-integrated organisms, despite numerous control methods. Germ-line mutational variants are, however, rare since there are "overlapping periods of maternal direction and germ-line sequestration."[263] The generation of germ-line variation is mainly a consequence of meiosis and recombination.

In his book's last chapter, aptly titled "The Evolution of Hierarchical Organization," Buss argues that "The history of life is a history of transitions between different units of selection."[264] This is because "any given unit of selection, once established, can come to follow the same progression of elaboration of a yet higher organization, followed by stabilization of the novel organization."[265] Thus, stabilization of individuals through control methods occurs after transitions to *that* level of individuality. Buss's book is an exploration of the transition to, and stabilization of, the multicellular level. In his conclusion he notes that there are many other levels requiring investigation, such as "the association of individuals into kin groups"[266], which is the case in social insects.

Buss work was groundbreaking in that it was the first clear and cohesive case of the competition theoretical perspective applied to the organism. It is of

[262] Buss 1987, p. 51.
[263] Buss 1987, p. 116.
[264] Buss 1987, p. 171.
[265] Buss 1987, p. 172.
[266] Buss 1987, p. 171.

interest that most of Buss' models are narrative and diagrammatic, that is, representational non-mathematical models. His rich conceptual discussion refers constantly to detailed developmental, molecular, and genetic mechanisms. There is not a single mathematical biological model in his book. Such representational strategies tend to be the purview of the integration perspective. So this is a case where the *concepts* fit under the competition perspective, but the *models* and *reference to compositional organization* fit under the integration perspective. Today, judging by who pays attention to the book, Buss' work is seen as a quintessential example of the competition perspective. Many practitioners of evolutionary developmental biology, a case of the *integration* perspective, have since judged his work mistaken and irrelevant.[267] Investigators of levels of selection theory hail it as a landmark and have ignored the detailed mechanistic arguments and representational non-mathematical models Buss gave and have, instead, provided mathematical models congruent with Buss' work.

Buss explored one explanation for the evolution of cellular cooperation: higher-level control methods. Another crucial explanation investigated subsequently to Buss is kin selection, which Hamilton first developed in a mathematically rigorous fashion, as described above. In their book on transitions, Maynard Smith and Szathmáry argue that control methods are not required for the stabilization, during evolution, of higher-level organisms.[268] Genetic similarity among cells and cell lineages of an organism is sufficient to arrest any

[267] See reviews of the book by Raff 1988 and Wolpert 1990; Günter Wagner has referred to Buss' research program as a "failed one" in conversation. See Winther 2001a, pp. 125-126. But see Gilbert 1992, who discusses Buss favorably. I will return to Gilbert's paper at the end of this chapter.
[268] Maynard Smith and Szathmáry 1995; see also Szathmáry and Maynard Smith 1995.

potential conflicts.[269] Alleles for cooperation can reach fixation given such high degrees of relatedness ($r \approx 1$) between mathematically idealized parts, that is cell lineages. With this argument, these authors ignore the potentiality, and reality, of mutation and they do not consider the full range of selective parameters (e.g. when defection benefit is high). If mutation occurs, and the selective differential is sufficiently high, cooperation will not reach fixation despite generally high relatedness.[270]

Regarding kin selection, Maynard Smith has argued that it should be understood as happening at the level of the individual – the organism.[271] But when he and Szathmáry discuss cell (*sensu* individual) versus organism (*sensu* group) selection, it is unclear whether they argue that kin selection has only a cell-level component or whether it also has an organism-level component (see, for example, their analogy between the "stochastic corrector model" and kin selection[272]). They do, however, state their allegiance to the "gene-centered approach" of Williams (1966) and Dawkins (1976)[273] and they do not cite any of the literature on multi-level kin selection theory. Both of these actions imply that Maynard Smith and Szathmáry believe that cellular kin selection occurs only at the cell-level and are, in effect gene selectionists. Hence Maynard Smith is an (rare!) example of a gene selectionist interested in evolutionary transitions.

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[269] Maynard Smith and Szathmáry 1995, pp. 8, 244. This is also what Hamilton argued in footnote 253.
[270] Michod 1999a.
[271] Maynard Smith 1976, 1982.
[272] Száthmary and Maynard Smith 1995, pp. 227-229.
[273] It is interesting to note that Dawkins 1989 does endorse a form of species selection, as Lloyd pointed out to me. However, this pales in contrast to his *general* advocacy of genic selection.

Michod and co-workers provide detailed mathematical models that employ both multi-level kin selection and higher-level control methods to explain the origin and maintenance of multicellularity.[274] Consistent with levels of selection theory, Michod has both cell-level and organism-level fitness parameters in his models (e.g. replication rate of defector versus cooperator cells; organism-level fitness as a function of cooperator cell frequency). If higher-level, that is, organismal, selection is sufficiently strong, alleles for cooperation will increase in frequency in the population. However, a number of parameters need to be considered to determine whether cooperation can reach fixation[275]: total number of cell divisions in an individual, mutation rate, and relative benefit to a defector cell (which can be less than one; in this case mutations are deleterious at both the cell and organism level). In most of the parameter space Michod explores, alleles for cooperation do *not* reach fixation even though idealized interactor cell lineages are related by common descent.[276] Kin selection is not sufficient for the origin of organismal individuality, higher-level organismal control methods such as germ-line sequestration and defection-policing mechanisms (e.g. immune systems) are necessary.[277] Michod's models are thus a mathematical articulation of Buss' emphasis on organismal control methods.

[274] Michod and Roze 1997; Michod 1999a, 1999b.
[275] As any of these three parameters increases, the equilibrium frequency of cooperation diminishes (Richard Michod, pers. comm.). Note that by "fixation" I mean an extremely high frequency of alleles for cooperation: recurrent mutation ensures that no allele is every fixed, strictly speaking.
[276] Michod 1999a, chapter 5.
[277] Michod 1999a, chapter 6.

3.3.2.2 Cell-Lineage Defectors are Common in Well-Integrated Organisms

A number of authors who endorse the competition perspective claim that cell-lineage selection occurs with substantial frequency in well-integrated organisms. Otto and co-workers have investigated germ-line cell-lineage selection in contemporary organisms.[278] They argue that the number of cell-divisions from zygote to zygote is sufficiently large to consider their mutations and mutation rate evolutionarily important (e.g. 50 in Maize, 25 for *Drosophila*, 25 for female mice, and 23 for human females, per generation[279]). The mathematical models of Otto and co-workers indicate that depending on the hierarchical (i.e. cell-level and individual-level) costs and benefits of mutations, intraorganismal selection can increase or reduce the mutation rate. Furthermore, selection between germ-line cell lineages can also decrease the mutation load in a population since deleterious mutations in such lineages will tend to be eliminated as they compete.

These selective scenarios differ crucially from Buss', Michod's, and Maynard Smith and Szathmáry's in that selection is interpreted as often acting in the *same* direction at both levels. Most loss-of-function mutations deleterious at the individual level are also deleterious at the cellular level. Furthermore, "mutations that improve the efficiency of metabolic pathways may often be beneficial at both levels."[280] Insofar as selection operates in the same direction at both levels, control methods are not necessary. But if a fraction of mutations have

[278] Otto and Orive 1995; Otto and Hastings 1998.
[279] Otto and Hastings 1998, p. 510.
[280] Otto and Hastings, 1998, p. 520.

beneficial cell-level effects, but deleterious individual-level effects, control methods will be necessary. An interesting research project investigating the relative frequency of this case of opposing directions of selection at the two levels, and the evolution of control methods as a function of its increased frequency awaits exploration.[281]

Nunney also emphasizes the reality of modular competition, in the form of cancer, in well-integrated organisms.[282] Growth-controlling genes that suppress the defector consequences of mutations in other growth-controlling genes would be selectively advantageous at the organism level. Despite such controls, cancers are almost inevitable in idealized modules with high replication and turnover rates (e.g. epithelial cells in the skin, hemopoietic cells in bone marrow and lymphatic tissue). Similarly to Michod and Buss, Nunney emphasizes the case of multi-level selection operating in opposite directions. However, he differs from them in believing that modular defection is ubiquitous.[283]

Levels of selection theory has been applied consistently to both social insect colonies and multicellular organisms. The existence of hierarchical competition is clearer in the former than in the latter, yet theory has been applied successfully to the latter as well. In particular, levels of selection theory has provided explanations for the origin of germ-line sequestration.

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[281] Sarah Otto, pers. comm.
[282] Nunney 1999a, 1999b.
[283] One problem with these views in general is that defection (especially cancer) often occurs after effective selection among organisms, that is, after organism reproduction (Michael Wade, pers. comm.). This has, to my knowledge, yet been clearly addressed in the literature.

3.3.3 Evolutionary Developmental Biology on Multicellular Organisms

In discussing evolutionary developmental biology, I will start by discussing multi-cellular organisms because these were the units first studied by this theoretical perspective. In fact, an embryonic version of evo-devo began with Darwin's, Haeckel's, and Weismann's nineteenth-century syntheses of evolution, development, and heredity.[284] Investigators studying organisms have also recently explicitly employed the concept of "modularity," that is, parthood. Only in the last 15 years or so has the possibility of an evolutionary developmental biology research program of social insect colonies become real. However, as we shall see, this research program is flourishing.

As a set of examples of evolutionary developmental biology at the organism level, each employing a different module type, let us explore the work of four contemporary practitioners in this discipline, Gerhart (biochemist) and Kirschner (cellular biologist), Raff (developmental biologist), and Wagner (comparative anatomist, developmental biologist, and population geneticist). Although their work is interdisciplinary, each investigator (the first two considered as a pair) tends to focus on one *kind of module*, as described in Chapter 2, which tend to be correlated with their *main* discipline of interest. They share the integration perspective, because the latter two focus on the complex integration of hierarchical systems at the organismal level and although Gerhart and Kirschner emphasize molecular biology, they illustrate the complex integration of systems such as metabolic cycles and gene regulatory networks.

[284] E.g., Churchill 1987; Gerson 1998; Winther 2000, 2001b.

3.3.3.1 Molecular Modules

In their book, Gerhart and Kirschner explore two important themes: (1) the conservation of basic molecular modular processes within and across taxa and (2) the intensity of interaction among molecular modules.[285] They explain the evolutionary conservation of protein function and structure in terms of processes of connectivity among multiple intra-cellular metabolic and regulatory systems. This connectivity is "contingent" in that molecular and cellular networks require multiple inputs for proper functioning. For example, protein kinases, which change the conformation of other proteins by adding a phosphate group from ATP to them, quickly activate or inhibit the other proteins.[286] This is an example of contingent connectivity in that catalytic proteins *depend* on other catalytic proteins for their state of activation or inhibition. Contingent connectivity also indicates the importance of complex networks of processes, networks of molecular modules, in Gerhart and Kirschner's approach. In their approach, molecular modules are crucial in explaining development and evolution.[287]

3.3.3.2 Developmental Modules

Raff and Wagner both focus on the organismal level but Raff concentrates on developmental modules, whereas Wagner emphasizes structural modules. I shall explore aspects of their respective theoretical and empirical research

[285] Gerhart and Kirschner 1997.
[286] Gerhart and Kirschner 1997, p. 80 ff.
[287] See also Kirschner and Gerhart 1998; Moss 2002 discusses built-in redundancy in molecular networks.

programs in some detail since I am concerned with the organismal level in my account of evolutionary developmental biology.

Raff and co-workers study the genetic and developmental differences between two sister species of sea urchins, *Heliocidaris erythrogramma* and *H. tuberculata*.[288] The former species develops directly from an egg to an adult, whereas the latter is an indirect developer, that is, it has a pluteus larval stage.[289] In the direct developer, in contrast to the indirect developer, all of the early cells of the morula are the same size, "thus, the unequal cleavages typical of indirect-developing sea urchins have been eliminated." In direct developers "cell types homologous [i.e., similar in structure] to those of indirect developers have different precursors."[290] There is thus a mismatch of early developmental modules even though the adult structural modules are congruent. This case is of interest because, to rearrange the words of the title of Chapter 7 of Raff's 1996 book, similar animals are built in different ways. What is also remarkable is that the splitting of these lineages occurred only 4-5 million years ago[291], which means that the evolution of their radical developmental differences occurred quickly.

Raff studies this system because he is interested in a variety of issues concerning developmental modules, such as: (1) the radical reorganization of early development in related species (to produce the same adult structure), (2) assessment of which developmental patterns and processes are evolutionarily ancestral and which are derived, (3) the co-option of genetic systems for new processes, such as the reorganized developmental modules. The sea urchin

[288] E.g. Raff 1996; Raff and Sly 2000.
[289] Raff 1996, 2000.
[290] Raff 1996, p. 231.
[291] Personal communication to Rudolf Raff by Kirk Zigler who used Lessios et al. 1999 calibration data.

system provides a window into these issues because Raff can study the radical differences in development between the two sister species through techniques such as cell fate mapping with dyes, transplantation, egg/sperm hybridization, in situ hybridization, and gene knockouts.[292]

A number of questions pertinent to evolutionary developmental biology can be addressed with this kind of data: (1) is indirect or direct development ancestral (put differently, is the pattern of evolution recapitulatory (adding on new terminal stages to development over evolutionary time) or can there be insertions of new types of stages in earlier parts of development over evolutionary time?), (2) (related to (1)) what was the Cambrian echinoderm (and other invertebrate) fauna similar to: modern larval or adult forms?, (3) what is the architecture of the genetic regulatory systems underlying these changes?[293] This research, as well as his theoretical discussion of morphogenetic fields during mid and late development, and the "hourglass model of development," in which mid development is highly constrained, whereas early and late

[292] Raff 1996, Raff and Sly 2000; E. Raff et al. 1999 and Nielsen et al. 2000 both present recent successes with hybridization experiments.

[293] These are unresolved issues. Raff argues that direct development is ancestral and that, therefore, insertion of new stages is the correct evolutionary pattern and that the body plan of Cambrian fauna was small modern-adult-like echinoderms (Raff 1996, 2000). Davidson and co-workers, on the other hand, argue that indirect development is ancestral and that recapitulation is correct in this case – the "set-aside cells" in larvae that give rise to the adult structures are therefore a derived character of an ancestral character, the larva (rather than, under the Raff hypothesis, a derived character of a derived character). [For a competition perspective on the evolution of set-aside cells, see Blackstone and Ellison 2000.] Davidson argues that the Cambrian fauna consisted of small modern-larva-like echinoderms (Davidson et al. 1995; Peterson et al. 2000). Each side provides genetic regulatory data supporting their view. Note that while Raff's sea urchin studies certainly provide data regarding contemporary gene regulation in two recently branched species, significantly more data, gathered from a significantly broader taxonomic viewpoint, is necessary to aid in resolving conundrums regarding genetic and developmental patterns and processes in the deep phylogenetic past.

development shows flexibility (Appendix Figures 3.2 and 3.3), all indicate Raff's emphasis on developmental modules.[294] He is concerned with what causes such modules, including the genetic regulatory network, and how the modules get reorganized, and are processual blocks, in the reorganization of body plans, during evolution.

3.3.3.3 Structural Modules

Wagner studies morphology, developmental biology, systematics, and population genetics. Wagner takes structural modules (parts) as his central object of investigation. For him, these are deeply connected to homologues: all modules of a particular kind are homologues and vice-versa.[295] Unlike Raff and many others, Wagner prefers a structural rather than a phylogenetic definition of homology,

> Structural identity is more fundamental than common descent. In fact the phylogenetic definition of homology implicitly requires the notion of structural identity… What is gained by Darwin's concept [of homology as the derivation of the "same" character/part, in two descendants, from a common ancestor] is not a deeper insight into the phenomenon of structural identity, but an explanation why structurally identical characters are distributed among recent species in nested sets.[296]

Thus, Wagner rejects the phylogenetic definition of homology both as a *criterion* and as a *cause* of homology.[297] He holds that homologues are the basic structural

[294] Gilbert et al. 1996; Raff 1996, chapter 10.
[295] Wagner 1994, 1995, 1996. See also my distinction between module-kinds and module-variants-of-a-kind in Winther 2001a, p. 120.
[296] Wagner 1995, p. 281 and personal communication; see also Wagner 1996.
[297] On this distinction, see Bolker and Raff 1996, particularly Appendix Figure (Table) 3.4. The debate over the homology concept is *extremely* complex and it is

"building blocks" of biological structure[298] and must be recognized by *criteria* that establish the structural identity of different concrete parts of the same kind. He appeals to structural and developmental *causes* of homology stabilization and constraint during development and evolution.[299] Thus, his criteria of homology identification are structural and the causes are structural and developmental. Although he has a hierarchical approach to causes of homology maintenance, he appeals strongly to relatively unique sets of expressed genes as causative agents.[300] For example, for the case of fin development explored immediately below, he presents an "epigenetic trap model" of growth in which the fin is a "semiautonomous character complex."[301] Structurally defined modules are conserved across taxa (i.e., are caused) because of developmental constraints mediated by gene action ("generative" and "morphostatic" constraints).[302]

In addition to his concerns with structure, Wagner is also clearly interested in development. For example, his studies on fin structure in bottom-dwelling blenny fish have, as one of their objectives, the goal of

> demonstrat[ing] that the developmental basis of evolutionary novelties need not be studied in phylogenetically old and highly complex organs, such as the vertebrate eye, where little interspecific variation is available. The fin hooks are anatomically simple and have all the attributes of a new morphological character.[303]

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beyond the scope of this dissertation to enter it. However, this debate serves as an excellent example of how different partitioning (and abstraction) criteria are employed in organismal biology, the paradigmatic case of compositional biology. I am currently working on a project exploring distinct notions of homology stemming from distinct theoretical perspectives of compositional biology.

[298] Wagner 1995.
[299] Wagner 1994, 1995; see also Roth 1991, 1994.
[300] Wagner 1996; Wagner and Altenberg 1996; Mezey et al. 2000.
[301] Wagner 1989, p. 1168.
[302] Wagner 1994.
[303] Wagner 1989, p. 1166; see also 1994.

Note the use of structural terminology in the second sentence. Wagner develops a narrative model of inhibitory and inductive interactions between different regions of the fin[304] from careful observation of development. His model is a positive feedback model for fin hook[305] development, in that the development of each part of the fin assures (even through inhibition of the other parts) the production of fin *hooks*.[306] Although he does not make the connection explicit, his discussion of the hypothetical role of mutations in this "epigenetic trap model" indicates his view of (semiautonomous) *genetic* networks as causal of these character complexes.[307] Unfortunately, Wagner has not had further opportunities to test this model.[308] I consider his approach to modularity and homology as primarily structural, because although he considers developmental mechanisms and constraints explanatory of homology, what he seeks *to* explain is the structural identity and individualization of morphological sets of characters (caused by gene networks[309]) during development and evolution.[310]

Wagner's research on fins and fin hooks in blenniids provides a good example of compositional biology as can be seen by inspecting how the four

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[304] These are: fin web (the transparent material between "fingers" of the fin), mesodermal ridge (the middle part of a cross-section of each "finger" of the fin), and cuticula (the outer circumference layer of each "finger").
[305] The "end" of each "finger."
[306] Wagner 1989, p. 1168, see Appendix Figure 3.5.
[307] See also the explicit representation of underlying gene networks in Wagner and Altenberg 1996, p. 971, see Appendix Figure 3.6.
[308] Wagner pers. comm.
[309] In this dissertation I have not discussed *Hox* genes and genetic networks due to space constraints. I do think that research on genetic architecture and regulation is crucial to evolutionary developmental biology and I take these investigations as central cases of the integration perspective under compositional biology – genes are here interpreted as multi-functional mechanistic components of real material systems. The interested reader can consult Raff 1996; Gerhart and Kirschner 1997; Hall 1998.
[310] Wagner 1994, 1995.

differentiae are met in this case.[311] (1) The fins and fin hooks are, in fact, complex objects with a developmental (and evolutionary) history, an adult structure and physiology, and a function in the behavioral ecology of the fish – the hooks have a history and multiple dispositional causes or capacities. (2) Even though some properties of the fins and fin hooks seem to be necessary (i.e., the fin as consisting of three components), much intra-, and inter-, specific variation is allowed – this is why these component types are described very generally. Furthermore, in inferring homologies even more variation and complex abstraction will need to be allowed. (3) The models provided are both diagrammatic and narrative. These are propositional non-mathematical models. (4) There is a clear reference to the material compositional organization in Wagner's research.

The work of these four key evolutionary developmental biologists provides a good overview of the breadth of topics in this field. Although their work combines guiding biases from various organismal biological sciences (especially comparative anatomy and developmental biology), they each tend to focus on, and employ, the biases, explored in Chapter 2, from the theoretical perspective they were primarily trained in: molecular biology for Gerhart and Kirschner, developmental biology for Raff, and comparative anatomy for Wagner.[312] Fitness and functional considerations are mostly absent. Genetic,

[311] Compare this to my discussion above of how Hamilton's rule met the four differentiae for formal biology.
[312] Rupert Riedl, an Austrian comparative anatomist with many interests, was Wagner's Doktorvater. I will explore Riedl's structural hierarchy of the biological world in Chapter 4. Wagner's work, in particular, is of significant interest precisely because he tries to tie comparative anatomy, developmental biology, and population genetics together so intimately. It is beyond the scope of this dissertation to analyze his contributions.

structural, developmental, and phylogenetic analyses of parts (modules) are ubiquitous.

3.3.4 Evolutionary Developmental Biology on Social Insect Colonies

I now want to make a seemingly paradoxical claim: the study of social insect colonies can be interpreted as organismal biology. There is a long tradition of thinking of social insect colonies as "superorganisms" dating at least to Wheeler's famous article "The Ant-Colony as an Organism."[313] Such colonies present division of labor both between germ and soma as well as within the soma, have a variety of well-defined structures, develop over time as a unit, and are highly integrated with multiple kinds of communication processes (e.g., chemical and touch) as well as homeostatic mechanisms (colony physiology). Here I will argue that because of these organismal features of social insect colonies, an evolutionary developmental biology with them as the object of study is possible and even desirable.

An evolutionary biologist once claimed that "evolution is the control of development by ecology" (van Valen, 1973). A significant amount of work has been done on the behavioral ecology of social insects.[314] (For a detailed discussion of the differences between Deborah Gordon's and E. O. Wilson's research projects on the behavioral ecology of ant colonies, see Winther 2001c and 2003 in press.) Thus, we have significant data on the ecological context in which social insect evolution occurs. The competition perspective has also provided

[313] Wheeler 1911. See also Emerson 1939, 1956; Wilson and Sober 1989, Sober and Wilson 1998; Winther 2001a, 2001c, 2003 in press.
[314] Wilson 1967, 1971, 1985, 1994; Oster and Wilson 1978; Wilson and Hölldobler 1988; Hölldobler and Wilson 1988; West-Eberhard 1987; Franks 1989; Gordon 1989, 1996, 1999; Gordon et al. 1992; Bourke and Franks 1995.

voluminous information on evolutionary genetic aspects of insect societies, as we began to see above. However, there is much less work on the development of insect societies considered as physiologically-integrated wholes in an ecological context. In other words, we know relatively little about the patterns and processes of differential gene expression, and developmental pathways, of social insect colony modules and colonies. An evolutionary developmental biology of social insect colonies requires that we investigate this.

3.3.4.1 Colony Development and Physiology

Schneirla performed an early set of investigations into army ant colony-development.[315] Army ant colonies have two discrete alternating stages: nomadic and statary. The 15-day nomadic stage of *Eciton burchelli* starts when a cohort of adults have just eclosed from their pupal case and a distinct cohort of eggs have just hatched into numerous hungry larvae (on the order of 100,000s) requiring large amounts of food. The 20-day statary phase commences when these larvae pupate. Ten days into the statary phase, the queen starts laying eggs again (Appendix Figure 3.7). The timing of this 35-day cycle, with the synchronized development of the two generations, is the result of multiple reciprocal chemical, tactile, and nutritive interactions among queens, workers, and brood. Schneirla, following Wheeler, called these interactions "trophallaxis."[316]

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[315] Schneirla 1971.
[316] Schneirla 1971; see Hölldobler and Wilson 1990, pp. 577-579 for a note of skepticism regarding the validity of these multiple interactions as an explanation for this cycle; consistent with their mainly context-independent interpretation of behavior (see Winther 2001c, and 2003 in press), they focus on the role of the queen in controlling this cycle.

More detailed research on developmental integration of social insect colonies has appeared subsequently.[317] In her review of the different mechanisms involved in reproductive-somatic caste determination, Diana Wheeler discusses queen effects (parental manipulation) on (1) worker behavior, (2) larval development, and (3) egg production or quality, or both (Appendix Figures 3.8 and 3.9).[318] In bees and wasps, a queen pheromone affects the building of gyne wax cells; the pheromone usually suppresses its construction. Furthermore, larvae in gyne wax cells receive more food from workers. As a consequence of this, they have higher levels of juvenile hormone (JH), which is necessary to develop into a reproductive female. The production of JH contingent on nutrition quantity is called a "nutritional switch." Further elaboration of organism and colony physiology leading to reproductive caste differentiation in honeybees (*Apis mellifera*) can be found in Hartfelder and Engels (1998). Similar mechanisms involving pheromones, nutrition, and JH are found in ants despite the absence of brood cells. A queen pheromone acts during a critical period of ant larval development to induce the loss of the capacity of larvae to develop as gynes. This inhibition occurs before the nutritional switch. Regarding egg production and quality, the ant queen can control how many eggs she lays, which is, of course, a function of how much nutrition she ingests, the temperature to which she is exposed, and other factors. More importantly, the queen can allocate different amounts of nutrition, mRNA, or hormones, or a combination of all three, to different eggs. In *Formica polycenta*, for example, large eggs with relatively large

[317] E.g. Wilson 1985; Wheeler 1986, 1991; Hölldobler and Wilson 1990; Robinson et al. 1997; Hartfelder and Engels 1998; Evans and Wheeler 1999, 2001; Robinson 1999.
[318] Wheeler 1986.

amounts of maternal mRNA develop into gynes.[319] There are multiple strategies available to hymenopteran queens and workers for reproductive caste determination of the developing brood, as well as for worker caste polymorphism[320], not discussed here.

The hymenopteran colony can be interpreted as an individual with mechanisms of developmental differentiation. When evolutionary developmental biology investigators study module differentiation in organisms, they study patterns and processes of differential gene expression as, for example, Raff and Davidson, with their respective co-workers, do in sea urchin development. An evolutionary developmental biology of social insect superorganisms requires a search for such patterns and processes in hymenoptera. Recently, Evans and Wheeler have found reliable differences in patterns of gene expression between honeybee workers and queens.[321] They also found that "several genes with caste-biased expression in honey bees show sequence similarity to genes whose expression is affected by hormones in *Drosophila*."[322] Thus, hormones such as JH may be involved in differentially activating genes correlated with morphological and physiological differences between workers and queens.

Let me turn to an analogy with the developmental framework presented in Chapter 2, and as summarized in Table 3. (2.1). The same kinds of developmental modules found for organisms can be seen in social insect colonies. Recall the distinction between parts *involved in* and parts *causing* the

[319] Wheeler 1986.
[320] Wheeler 1991; the developmental basis of worker caste polymorphism is not known as well as the developmental basis of reproductive caste determination.
[321] Evans and Wheeler 1999, 2001.
[322] Evans and Wheeler 2001, p. 64.

process. The queens and workers constitute the "parts involved in (colony) developmental activity." They are the processual "parts" or "agents" that are *involved in* the activity. The genes that get differentially expressed (genes-D) are, on the other hand, "parts *causing* the activity." On the other hand, the different behaviors employed and performed by the queen and workers (e.g., pheromone secretion and nutritional control) are "activity parts"; the cycle of the colony can be partitioned into these different processual segments, with queens, workers, larvae, and eggs all playing different roles. The parts causing, and the parts involved in, activity, as well as the activity parts are all developmental modules.

Behavior is a crucial factor in colony development and physiology. In fact, since behavior is usually attributed to an organism, and since ant-colonies are *composed* of organisms, organisms and organism behavior become, as units and mechanisms, respectively, parts of colony development and physiology. Behavioral ecology of social insect colonies is thus a study of *colony physiology*.[323] The same kinds of guiding biases pertinent to developmental biology and physiology in organisms apply to those areas of study in social insects even if they are not as well characterized. That is, the ontogeny of the colony is a phenomenon separable from colony physiology, even if organismal behavior is involved in both. The former has a temporal dimension and involves characterizing the causes of differential gene expression, many of which occur inside the nest; the latter tends to be studied at a time slice, particularly in the mature colony and often involves the activity of ants outside of the nest. This

[323] Again, see the work of both Gordon and Wilson. Due to space constraints, I do not review their work here. See in depth reviews in Winther 2001c and 2003 in press. Some of Thomas Park's work on flour beetles was also explicitly considered a study in population physiology.

corresponds accurately to the temporal and spatial biases of organismal developmental biology and physiology, respectively. Gene Robinson and co-workers have explicitly called for the study of "the molecular genetics of social behaviour in ecologically relevant contexts."[324] Their research program seeks to synthesize processes involving gene expression, hormones, pheromones, neurophysiology, behaviors, and ecology. They want to do this by "Focusing on genes [which] provides a common language and convergent research themes."[325] Whether a synthetic theory of colony integration (developmentally and physiologically) requires a genetic focus merits further discussion.

3.3.4.2 Is an Evolutionary Developmental Biology of Social Insect Colonies Possible?

Thus, in the last 15 years, several biologists have investigated the developmental and physiological mechanisms of social insect colonies. This application of the integration perspective has similarities to the evolutionary developmental biology synthesis that has been occurring at the organism level. Is an evolutionary developmental biology of social insect colonies possible? Clearly differential gene expression of colony-modules has been found. But this does not necessarily imply that we can consider social insect colonies individuals when it comes to assessing module homologies, establishing ancestral and derived colony-level developmental patterns, describing the origin of colony-level innovations, and linking organismic change in characters with large-scale colony

[324] Robinson 1999, p. 204; see also Robinson et al. 1997.
[325] Robinson et al. 1997, p. 1099.

evolution.[326] Progress, however, has been made on some of these aspects of the evolutionary developmental biology of social insect colonies. Colony-level properties such as nest morphology have been used in determining robust phylogenetic trees in wasps[327]; some superorganismal modules can therefore be used for establishing homologies. Furthermore, Anderson and McShea argue that organs, or "intermediate-scale structures", such as teams (workers adopting different sub-tasks in order to perform a task – e.g. carrying a prey item) and nests, exist in social insect colonies.[328] These and the other results discussed in this section on social insect colonies indicate that an evolutionary developmental biology of social insect colonies is possible. We should embark on such a project, which would also involve a conceptual investigation of the individuality of social insect colonies. Conclusions from this research would provide data, at a new hierarchical level, that would facilitate further conceptual developments in evolutionary developmental biology in general.

Thus, we have seen that these two theoretical perspectives at the disciplinary/school of thought level can be applied to the same biological systems, organisms and social insects, even if there is more work on the levels of selection in social insects and the evolutionary developmental biology of organisms. The domains of units studied by these theoretical perspectives thus overlap. But levels of selection theory and evolutionary developmental biology abstract very differently. The two theoretical perspectives abstract the same objects in fundamentally different ways (e.g., materially complex homologues vs. formally simple replicator genes), have radically different methodologies (e.g.,

[326] Raff 2000, p. 75; Wagner et al. 2000, p. 820.
[327] Wenzel 1993.
[328] Anderson and McShea 2001.

knocking out genes and vs. constructing mathematical models), and provide different model-types for these biological systems (e.g., mathematical models, such as Hamilton's rule, vs. propositional non-mathematical models, such as Wagner's model of fin hook development in blenniids). Furthermore, the levels of selection perspective has a highly ideal frame of partitioning[329] that provides the replicator as well as the gene-molecule-cell-organism-group-species-clade interactor hierarchy.[330] Thus, the two perspectives differ in how they abstract and how they partition.

Let us now turn to an elaborate discussion of the many deep ways in which they differ and what we should think about the possibility of synthesizing them.

3.4 On the Prospects of a Synthesis

In exploring details of the theory, practices, and data of levels of selection theory and evolutionary developmental biology, I hope to have made two general points: (1) these two disciplines /schools of thought differ in their epistemic and methodological resources in ways to which I will presently turn and (2) they partly overlap in the units and general processes that they study and interpret. As we shall see, there is a subtle interplay of similarity and difference between these two theoretical perspectives and there is, therefore, a variety of

[329] Recall from Chapter 2 that the partitioning frame of a perspective contains criteria allowing for the conceptual and actual decomposition of the system under study.
[330] Again, see Appendix Figure (Table) 3.1.

interpretations about *how* these perspectives *actually* relate. After some preliminary discussion, I will turn to four such interpretations.

These theoretical perspectives differ in the epistemic and methodological resources that they apply in their explanations and descriptions. First, they differ with regard to my four distinctions, which I will explore one at a time.[331]

> (1) Even when they study the same objects (e.g., genes) they interpret and model them, respectively, as simple (e.g., mathematically-defined "genes") or complex (e.g., phylogenetically-conditioned regulatory genes). Furthermore, evo-devo studies the "somatic" or "phenotypic" complex parts of organisms and social insect colonies—with a structure, development, and physiology—in a way not focused on by levels of selection theory (although see Buss).
>
> (2) They make distinct idealizing assumptions about their respective objects of study and they abstract them differently. Levels of selection theory employs necessary and sufficient conditions in defining the precise variables and parameters employed by its principled biological mathematical models, whereas evolutionary developmental biology employs complex "family resemblance" abstraction in picking out the types that it represents in its qualitative immediate models. Even though typology is crucial to evo-devo[332], recognition and bottom-up abstraction

[331] I.e., (1) attention to simple versus complex objects, (2) employment of simple versus complex abstraction, (3) usage of mathematical models versus material and propositional non-mathematical models, and (4) consideration of part-whole organization as irrelevant and highly idealized or relevant.

[332] See Amundson 1998, 2001 on the importance of essentialist thinking in developmental biology. Topics like homology and developmental constraint revolve around such concerns. See both Amundson's work and Winther 2001 for

of such ideal types is a highly contingent process based on qualitative assessments of similarities among the (overlapping) intentional sets of properties ascribed to different concrete parts eventually judged as "of the same kind."[333] Such contingency does not exist in the precisely defined mathematical variables and parameters of formal biology.

(3) Each perspective employs model-types that are fundamentally distinct. The former employs principled mathematical biology models often based on axioms and clear simple assumptions. Much of the model-building here is based on abstract mathematical principles (including "laws") and logical operations. Compositional biology tends to use mainly material and propositional non-mathematical models that refer directly to the material compositional structure of the system at hand. It does on occasion employ mathematical models, but these, as discussed in Chapters 1 and 2, are often highly complex and refer intimately to part-organization.

(4) Thus, while formal biology is concerned primarily with (mathematical) abstraction, compositional biology emphasizes the articulated part organization of complex biological systems. Levels of selection theory either makes parts irrelevant (as in its lack of interest in somatic or phenotypic parts) or highly idealizes them, as in the case of epistatic genes. Evolutionary developmental biology is, however, clearly about concrete parts.

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discussion of the importance of variation and commonality, respectively, for levels of selection theory and evo-devo.

[333] Recall complex Wittgenstein-Boyd "family resemblance" abstraction as articulated in Chapter 1.

The perspectives also differ in the *explanatory resources* they employ.[334] This term encompasses a variety of things, including the model-types employed (third distinction) by the perspective, cases of which I have discussed above.[335] But explanatory resources also include (1) the contrast classes considered relevant in an explanation[336], (2) the meanings of terms employed, (3) the research questions asked, and (4) the broad aims of the explanations (e.g., theoretical unification in the case of theoretical explanation, or, in the case of

[334] I found Amundson 2001 particularly thought-provoking regarding these matters. In comparing the "lack of common ground" between adaptationist functionalists and developmental structuralists, he "offer[s] a sketch of the contrast between these two kinds of evolutionary explanation. Adaptationist explanations depict evolution as changes to what I will call the *current genetic state* of a population, with selective forces fully accounted for but the current genetic state itself assumed as an unexplained background condition… Structuralist explanations depict evolution as changes to the *processes that give rise to phenotypes*, with those developmental processes fully accounted for at each step but selective forces assumed as unexplained background conditions. There are shortcomings to each approach. The structuralist approach ignores selection, and so it contains no account of the motor of evolutionary change… On the other hand, by treating the current genetic state as mere background the adaptationist account ignores the biases in available variation that are created by the existing developmental system." (Amundson 2001, 323) Thus, the explanans and the explandanda, as well as the explanatory background varies drastically between these two fields, which correspond, roughly to formal and compositional biology as represented, for example, in levels of selection theory and evolutionary developmental biology. The main disanalogy, however, lies in what I take to be the lack of explicit focus on *adaptation*, or selective function (sensu Wright 1973), in population genetics. This does sound rather odd, but population genetics is not, per se, focused on the function of particular parts. It is focused on the forces, one of which is selection, that change gene frequency. Perhaps we can think of particular genes as adaptations for particular purposes, but usually we think of characters (components of the interactor) as carrying function. I will here postpone a full discussion of the relation between adaptation/function and formal biology.

[335] E.g., Hamilton's rule and Wagner's model of fin hook development in blenniids.

[336] See van Fraassen 1989 on the pragmatics of explanation, as well as Dretske 1972; Garfinkel 1981; Sober 1986; Mitchell 1992; van der Weele 1995; Amundson 2001.

causal explanation, part capacity ascription or causal inference; these will be discussed in Chapter 5). The set of explanatory resources that a perspective employs is a complex battery of theoretical and methodological guiding biases and assumptions. Amundson has described how the contrast classes and meanings of a few key terms[337] differ in population genetics and developmental biology. I have not and shall not be concerned with the contrast class issue, although I think that it is important. I have shown that meanings of (general) terms, included under the "outward-interaction" and "inward-independence" distinction, do differ drastically between the two perspectives. Under the competition perspective, cooperation is described in terms of ultimate fitness benefits (i.e., gene "copy" maximization) accrued from population structure (i.e., kin group) whereas in the integration perspective, integration is defined in terms of mechanistic interaction that ensures the functional integration of the whole. I shall address the issue of *questions asked* separately below.

With respect to the last aspect of explanatory resources here discussed, to be elaborated upon in Chapter 5, it is clear that the general aims of explanation differ in the two kinds of biology. In the case of levels of selection theory as a formal biology, one aim is theoretical unification through mathematical means.[338] The equivalent of a "grand unified theory" for the forces and dynamics of gene frequency change is sometimes sought.[339] Evolutionary developmental biology, as an instance of compositional biology, intends to describe the capacities of the

[337] E.g., "constraint" and "evolution." See also Amundson and Lauder 1994 on different notions of function.
[338] On theoretical unification, see Friedman 1974, 1981, 1983; Kitcher 1991 (1981), 1985.
[339] Some consider the Price Equation one way of achieving the goal of theoretical unification; see Wade 1985; Frank 1997, 1998.

parts it investigates. The goal here is to disaggregate the different kinds of behaviors of a system (the whole) in terms of component capacities of the parts.[340] Theoretical unification and part capacity ascription are distinct explanatory aims to be discussed in Chapter 5.

But if these perspectives were *completely* different, there would be little motivation to address both of them at the same time or even worry about their relationship. We could merely ascribe them to different ontological *domains*, such as was done, and entrenched, by the formulation and highly complex interpretation of a variety of distinctions such as Mendels' factors and characters, Weismann's germ and soma, Johannsen's genotype and phenotype, Morgan's transmissional and developmental genetics, Mayr's proximate and ultimate, and Hull's and Dawkins' distinction between the replicator and interactor. Although this is by no means an actually universal interpretation, nor could it even *be* a monolithic interpretation, the former term in each pair refers to the processes of variation-heredity-development-reproduction (*production* of units!) whereas the latter term refers to, or is pertinent to, selection. There are feedback effects between the two classes of terms, but this is the basic *general* distinction. For example, Amundson has shown how these distinctions played a *historical* role in entrenching differences between fields concerned, respectively, with evolutionary and developmental phenomena.[341] According to these classic distinctions, compositional biology (concrete production of units) and formal biology (selection in populations of idealized units) belong to distinct ontological domains.

[340] On part capacity ascription as a form of explanation see Cummins 1975, 1983.
[341] Amundson 2000, 2001.

What is of supreme interest now is that despite the difference in representational and explanatory resources, the two (hierarchies of) theoretical perspectives now partly overlap in ontological domain. That is, tools used to analyze populations are now applied to, for example, cell lineages (developmental phenomena) – thereby interpreting them as populations. Conversely, tools used to examine the production of units are now implemented in, for example, social insects and constraints on genetic variation (usually considered the domain of evolutionary studies). Furthermore, each of the respective disciplines (that is, levels of selection theory and evolutionary developmental biology) now understand that they must take the preferred domains of the other fields seriously – formal evolutionary studies cannot ignore development, and compositional developmental studies cannot ignore the force of selection[342]. The fields can continue to provide their own definitions of function and structure [as in Table 4. (3.1) below], but a complex overlap of domain is occurring and it is this hybrid zone of formal and compositional theory applied to developmental and evolutionary phenomena, respectively, that a synthesis may or may not happen.

[342] Although compositional developmental studies have endorsed *phylogeny* as an evolutionary aspect, they have yet to come to peace with the relevance of the force of *selection*.

	Mathematical Evolutionary Genetics (E.g., Levels of Selection Theory)	**Evolutionary Developmental Biology (Including Organismal Biology)**
Function	- Mathematically-formulated *selection* on genes - Optimization models	- Capacity (function) ascription to parts of a system (Cummins)
Structure	- Mathematically-formulated constraint equations - Gene variance/covariance matrix in quantitative genetics	- Morphology - *Developmental*, mechanistically-formulated, constraints

Table 4. (3.1) Function and structure as defined by each of the two fields here examined [see also Figure 1. (3.1)]. Note that the bulk of research occurs in the upper left-hand and the lower right-hand cell.

But as it stands, I argue, the two perspectives are ill-equipped to deal with this partial overlap in domain. That is, their extreme differences have not yet been clearly articulated, let alone overcome. As a community of biologists and philosophers of biology, we currently desire to develop an integrated account of the relationship between levels of selection theory and evolutionary developmental biology, but in order to do this, it is not enough that those employing each of the two different hierarchies of perspective investigate the similar phenomena. We must also consider whether and how the serious differences in representational and explanatory resources can be accommodated or synthesized under one meta-perspective, through theoretical and

methodological (1) integration, (2) translation, and/or (3) complementation.[343] A full account of this is beyond the scope of my dissertation. I can only provide some suggestions for how this might be done, and speculate about whether it can even be done.

In what follows, I elaborate on four different interpretations of how levels of selection theory *relates* to evolutionary developmental biology, the key relationship indicated by the big question mark toward the bottom of Figure 1. (3.1). The four interpretations are named according to how they foresee a "synthesis" between the two hierarchies of theoretical perspectives happening (or not). I will turn to each interpretation in turn, but here I first want to indicate the properties of each vis-à-vis the above discussion.

The four interpretations I present are: (1) the explanatory exclusivity interpretation, (2) the two domains interpretation, (3) the two kinds of biology interpretation, and (4) the eventual convergence interpretation [see Table 5. (3.2)]. The first three interpretations all argue that the two hierarchies of perspectives rely on different *explanatory resources*; the last argues that some sort

[343] There is some philosophical literature on "scientific controversies," in addition to the more extensive sociology of science literature on this topic, which I will not touch here. Kitcher 2000 in Machamer et al. discusses the beginnings, middles, and ends of scientific controversies under "rationalist" (evidence determines outcome) and "anti-rationalist" (epistemologies determine outcomes) models. Kitcher does not capture the richly pragmatic and relativistic nature of scientific debates. I do not think that the debate I am analyzing here can be couched in rationalist or anti-rationalist terms or that a single account stemming from one of the hierarchies will triumph and be shown to be "true." Perhaps a meta-theoretical perspective will be found between the two hierarchies of theoretical perspectives that I describe, but the resolution to this grand controversy will not be based on "data" favoring one perspective over another. The situation is more complex. Sociological models of scientific controversy and their resolutions, such as Gerson's notion of theory realignment (Gerson 1998), are better able to describe the theoretical richness of the debates and differences between the two hierarchies that I am describing in this chapter.

of integration, translation, and/or complementation of explanatory resources is possible between them. The second interpretation argues that *different processes* are studied by formal and compositional biology; the third and fourth interpretation argues the opposite – the same or overlapping units and processes are studied in the two kinds of biology. The first interpretation is indeterminate on this point, being either agnostic or agreeing with the second interpretation.

Two further "character-states" can be looked at to differentiate the interpretations that argue that the explanatory resources are different (1-3). First, do they hold that the difference in resources leads to *tension* or to *mutual ignoring* between perspectives? The first interpretation argues the former, the second and third interpretation argues for the latter. Second, what do they consider the *source* of the explanatory differences to be? In the second interpretation, but also, I suspect, in the first, the source is interpreted as being the classic distinctions of Mendel, Weismann, etc. mentioned above, which are commitments about the ontology of the biological world. In the third interpretation, and this is one way that I think my analysis aids understanding, the source is taken to be differences in *kinds of science*, i.e., differences not so much in commitments about ontology, but rather, differences in primarily epistemic commitments about abstraction, model-building, relevance of part-whole organization, and explanatory resources.[344] In a sense, this chapter, and dissertation more generally, has been a defense of this third interpretation. I certainly do not want to argue either that the classic distinctions are free of epistemic commitments or that, even if mainly

[344] Let me underscore the ontology/epistemology point: Mendel, Weismann, etc. studied, of course, biology and so they formulated ontological (domain) distinctions. As a philosopher of biology, I study theories of biology and so I develop (primarily) epistemological and methodological distinctions.

ontological, they play no role in the differences between levels of selection theory or evolutionary developmental biology. But I do hold that my analysis of formal and compositional biology provides a new framework that allows us to see the epistemic differences between these two disciplines/schools of thought in a new and cogent light.

	Explanatory Exclusivity	**Two Domains**	**Two Kinds of Biology**	**Eventual Convergence**
Explanatory Resources	Different	Different	Different	Synthesized
Units/ Processes Studied	Indeterminate or Different	Different	Same/ Overlapping	Same/ Overlapping
Relationship Between Two Hierarchies of TPs	In tension	Mutual Ignoring	Mutual Ignoring	Synthesized
Source of Explanatory Resources	Classic Distinctions	Classic Distinctions	Epistemic Distinctions Regarding Kinds of Science	None – Differences Overcome

Table 5. (3.2) The four interpretations on the relationship between levels of selection theory and evolutionary developmental biology compared with respect to a variety of issues as discussed in the text. "TPs" means "theoretical perspectives."

Let us now turn to the differing interpretations of the relationships between levels of selection theory and evolutionary developmental biology.

(1) The *explanatory exclusivity* interpretation. Some researchers believe that the kinds of processes adopted by biologists in the two theoretical perspectives contradict, or are in conceptual conflict, with one another. For example, some key investigators of evolutionary developmental biology hold that the idea of multi-

level selection, particularly in developmental processes, is incoherent and not explanatory, *in any sense*, of development (e.g., Raff and Wolpert; Wagner, to a much lesser extent, also holds this view[345]). Some competition perspective workers believe that development can be black-boxed as long as the path from genotype to phenotype is reliable (and a change in genotype leads to a change in phenotype) – in fact the statistical and probabilistic edifice of mathematical evolutionary genetics is premised on attempts to *eliminate* developmental considerations and decompose the phenotype directly into the genotype (statistical quantitative genetics) or assign fitness values directly to genotypes (probabilistic population genetics). (Proponents of levels of selection theory do often emphasize gene interactions, as Wright did – this is not exactly the same, however, as focusing on developmental dynamics per se.) The explanatory exclusivity interpretation thus claims that: (1) development is not in need of any evolutionary (selective) explanation, or (2) development need not be taken into account in explaining evolution, or both. This interpretation thus describes a case where each theoretical perspective rejects the explanatory strategies *and* even the ontological domains of the other. The perspectives are *explanatorily exclusive* (what I have previously called the "irreconcilable" view[346]). This is not, and cannot lead to, a synthesis in any way. It is a welcome change that increasingly more levels of selection investigators are taking developmental (and epigenetic) effects more seriously. Unfortunately, the converse cannot be said for evolutionary developmental biology workers with respect to multi-level

[345] See Raff 1988; Wolpert 1990; Wagner pers. comm.
[346] See Winther 2001, pp. 125-126.

selection processes. However, as we shall see, it is incorrect to claim that the views are *in actuality* or *in principle* in tension.

(2) The *two domains* interpretation. Although an explanatory tension can sometimes be explicitly read from the work of some investigators such as Raff or Wolpert, the more common interpretation of the relation between levels of selection theory and evolutionary developmental biology is that the two concern *different* sets of biological processes. This interpretation has a strong historical lineage. Many of the orthodox distinctions "fundamental" to a conceptual account of biology, with roots in Mendel's and Weismann's work, such as (1) reproduction-selection, (2) proximate-ultimate, (3) replicator-interactor, and (4) integration/cooperation-competition imply a "separate but equal" view of each contrasting member of each distinction. The general picture here seems to be that there are two distinct explanatory projects in biology: (1) explaining the similarity relations, development, and reproduction of units (the former member of each distinction) and (2) explaining the selection and inter-generational change of these units, in populations of (mathematically idealized) units, over time (the latter member of each distinction). The Modern Synthesis consecrated these distinctions. However, in the last decade or two, there has been a move to undo the distinctions both through biological work in levels of selection theory and evolutionary developmental biology as well as through various philosophical arguments. But we have not gotten very far in undoing these entrenched distinctions (and it still remains unclear whether we *should* undo them). If levels of selection theory and evolutionary developmental theory are really two different domains then it is unclear what a synthesis between them would even

be. Presumably a synthesis breaks down distinctions. But in the two domains interpretation, a synthesis would be, at best, a plea for *both* processes to be considered theoretically, economically, and socially important. It is hard to see how this is an actual synthesis.

This interpretation too has problems. Above, I have shown how each perspective investigates two kinds of biological units, organisms and social insects, so they concern similar overlapping domains. However, perhaps someone could claim that the two perspectives concern different aspects of the same units. But, Buss, for example, interpreted developmental mechanisms from a levels of selection point of view, and some of these interpretations have been kept. Furthermore, as argued above, selective and ultimate phenomena can certainly also be interpreted from an evolutionary developmental biology point of view – functional morphology, for example, is used to ascribe *adaptation* to parts of organisms. So, although it has been enshrined almost as dogma, the two domains scenario is not a robust interpretation either.

(3) The *two kinds of biology* interpretation. In the two interpretations already discussed, there was an implicit assumption that the two perspectives were different kinds of biology. In this third interpretation, this fact is emphasized along with the potential synthesis [contra (1)] and domain overlap [contra (2)] of the two perspectives. This interpretation claims that the biases regarding abstraction, model-building, relevance of part-whole organization, and explanatory resources are so different in each of the two theoretical perspectives

that no synthesis in terms of more abstract framework can (ever) be had.[347] The point of this interpretation is that evolutionary developmental biology and levels of selection are different kinds of science. The two sciences will always remain apart unless a concerted and explicit attempt is made by conceptually-oriented biologists and philosophers of biology to articulate ways to bring compositional biology and formal biology together. My dissertation provides descriptions of these deep differences. I believe that this interpretation describes the *current situation* in which we find ourselves.

(4) The *eventual convergence* interpretation. The future is, of course, very difficult to predict. I am convinced, however, and hope to have shown, that there is as of yet no actual synthesis, even remotely, between levels of selection theory and evolutionary developmental biology. I see one of my goals as convincing biologists and philosophers that if we declare the existence of such a synthesis this is equivalent in truth-value to the declarations of the emperor in Hans Christian Andersen's fairy tale. A deep divide exists between the competition and integration perspectives. Now, either I am wrong (and such a synthesis exists) or I am right and I hope to motivate others to think about whether such a synthesis is possible, and, if so, how. Here I will sketch three areas of investigation potentially pertinent for achieving such a synthesis. These are

[347] I am aware of the notoriety of statements of the form "X can never happen in science" – this was said about electromagnetism and quantum mechanics, and Kant erroneously said 69 years before the *Origin of Species* that "it is absurd for men… to hope that maybe another Newton may some day arise, to make intelligible to us even the genesis of but a blade of grass from natural laws that no design has ordered." (Kant 1953 (1790). There is an irony, though: Darwin did not actually provide laws for the *production* of units; he provided the grounds for laws for the *selection* of units. Until the advent of developmental biology and molecular genetics in the 20th century, Kant was right.

extremely preliminary suggestions – one person's imagination, however, should not limit the work of a whole field of superb philosophers and biologists.

First, the concept of *genetic information* has received an increasing amount of attention in recent years.[348] As an important part of their argument concerning evolutionary transitions, Maynard Smith and Száthmary argue that the important evolutionary transitions are the ones for which there were new ways of transmitting (genetic) information.[349] Griesemer rightly points out that this is a distinct focus from the focus on structural (compositional) hierarchy.[350] He argues that translations among these two forms of hierarchy, and the *process hierarchy*, which he favors with his "reproducer" concept, should be articulated. A significant amount of research is required to relate these hierarchy concepts.

The idea of information transfer and the idea of hierarchy are related in discussions regarding transitions in evolution. The concept of information is a particularly suggestive place to explore relationships between the two kinds of biology articulated in this chapter since "genetic" and other forms of information could be thought of as either (1) statistical measurements of the relationships among genotype, phenotype, and fitness (for levels of selection theory) or (2) summaries of mechanisms or causal capacities (for evolutionary developmental biology). Theoretical integration of these concepts, particularly for the period *during* which evolutionary transitions occur, can and should be explored particularly since causal capacities do have a direct effect on fitness (and are

[348] Maynard Smith and Száthmary 1995; Godfrey-Smith 2000; Maynard Smith 2000; Sarkar 2000; Sterelny 2000.
[349] E.g., origin of template replication, origin of genetic code, origin of epigenetic inheritance in cell lineages of multicellular organisms, emergence of language. Maynard Smith and Száthmary 1995, pp. 13-14.
[350] Griesemer 1999, p. 127.

summarized that way) and fitness can be fleshed out, if a mathematical evolutionary geneticist should care to do so, in terms of, for example, genetic, developmental, and ecological mechanisms. As Griesemer puts it: "because transition is an evolutionary process, the hierarchy that results is a *product* of evolution, not an assumption in need of independent justification. Evolution generates transitions and transitions create hierarchy."[351] Studying the hierarchy itself, and the different ways that information is transmitted among units *at a particular level in the hierarchy*, gives us a glimpse into how the generative mechanisms of the units, and selective dynamics of populations of units, relate. It is important to keep in mind criticisms regarding the whole idea of information[352], but this still seems like a provocative place to start thinking about the relationship between formal and compositional biology.

Second, *higher levels of selection*, such as the species and clade selection[353], might also be a good place to consider the potential interrelationships between levels of selection theory and evolutionary developmental biology. This is a topic that I have sidestepped here because of space constraints. There are *mechanisms* that are positively correlated with speciation, such as the existence of a high degree of population structure in marine invertebrates that brood their young rather than disperse their eggs and sperm – these species will tend to have more inter-population variation and will therefore speciate faster than species that have large panmictic populations.[354] These mechanisms, Lloyd and Gould (1993)

[351] Griesemer 1999, p. 128.
[352] E.g., Oyama 2000a.
[353] E.g., Damuth and Heisler 1988; Lloyd and Gould 1993, Gould and Lloyd 1999; Grantham 1995.
[354] This example is from Jablonski 1986; discussed in detail in Lloyd and Gould 1993, who provide the argument regarding variation.

argue, are traits whose variation is correlated with variation in "emergent fitnesses" of the species. Alternatively, Damuth and Heisler have an entirely different view of species selection in which evolutionary change is driven by selection between guilds of competing species.[355] This can be interpreted as a community ecology approach. Both Lloyd and Gould's, as well as Damuth and Heisler's, views could be of use in articulating a synthesis involving mechanisms and selection at levels higher than the group.

Third, *selection and integration* are particularly clearly interrelated in *multicellular organisms*. A thought-provoking paper that is suggestive of issues requiring consideration in synthesizing levels of selection theory and evolutionary developmental biology is Gilbert's piece on the relevance of Buss' argument to developmental biology. Gilbert provides a historical account of critiques of Weismannism.[356] He also discusses at length "the notion that the cell lineage can be a unit of selection," which is, he claims, "a particularly robust idea that I do not want to dismiss."[357] His analysis of assumptions regarding concepts and metaphors underlying the notion of cell lineage competition focuses on what he considers the "western" notions of unit independence, self-reliance, and economic competition between units. Gilbert interprets, and finds fault with, Buss' view as one of independent cell lineages attempting to assert their economic-theory-like selfish interests by cooperating with other types of cell lineages.[358] Gilbert puts forth his view that "the units of selection [are]

[355] Damuth and Heisler 1988.
[356] See also Griesemer and Wimsatt 1989; Winther 2001b.
[357] Gilbert 1992, p. 481.
[358] Gilbert thus seems to interpret Buss as a Dawkins for which the cell is the replicator. Gilbert seems to imply that the following quote by Dawkins could have been written by Buss: "The integrated multicellular organism is a

sociocentric, relational, contextualist, and holistic as opposed to being egocentric, individualist, selfish, and autonomous."[359] He certainly does not want to deny cell lineages the status of units of selection, but he does take issue with their representation, in his opinion, as selfish and independent agents (replicators or interactors?) in the evolutionary process, both during and after the transition to multicellularity.

Biochemical evidence for the similarity of structure, process, and function of *cell-surface proteins* used in fertilization (both of isogametic protists, which presumably resemble our single-celled ancestors, and heterogametic sperm-egg) and embryonic inductive events serve as evidence for his claim that units of selection, cell lineages, are interdependent (i.e., have cell membrane structures and processes essential for communication). It is in these cases that

> we see that those cells which gave rise to multicellular organisms, rather than being solely selfish replicators, are also outer-directed cooperators. As much as they 'want' autonomy and dominance, they also 'want' to interact. As much as they have the mitotic apparatus driving them to proliferate, they have a cell membrane that drives them to communicate. The same genome that instructs cell division instructs cell interactions.[360]

I think that Gilbert's analysis, although suggestive and of extreme interest for prospects of a synthesis, is problematic. It conflates three separate distinctions: (1) what I have called the competition and integration theoretical perspectives, (2) "outward-interaction" and "inward-independence," which are

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phenomenon which has emerged as a result of natural selection on primitively independent selfish replicators. It has paid replicators to behave gregariously. … In practice the organism has arisen as a partially bounded local concentration, a shared knot of replicator power" (Dawkins 1982, p. 264). I think that this is a misreading of Buss, particularly if one examines Buss' arguments against genic selectionism as represented, for example, in footnote 248.

[359] Gilbert 1992, p. 482.

[360] Gilbert 1992, p. 483.

defined *differently*, but exist as concepts, in each of the two perspectives, and (3) reductionism and holism. In previous work, I have explicated in great detail why, in particular, (1) and (3) are not the same distinction.[361] There are holists in the competition perspective (e.g., Buss and Wade) and there are reductionists in the integration perspective (e.g., proponents of the Human Genome Project, such as Walter Gilbert). Furthermore, Gilbert does not clearly distinguish the explanatory resources in the two perspectives that he is trying to synthesize. A proponent of the competition perspective could easily say that rates of cell division *summarize* the effect on fitness of all the mechanisms, described as independent or interdependent. Such an investigator would simply not care about the mechanisms. They want to measure *fitness*, even if it is hierarchically or interactively, or both, determined.[362] In his focus on integrative mechanisms, rather than dynamics of gene frequency changes, Gilbert, it could be argued, simply misses the crucial explanatory project of the competition perspective. However, his views certainly deserve more consideration – his analysis of the use of *metaphor* in theory and practice construction in the two perspectives described in my chapter is important.

The difficulty of bridging the two (hierarchies of) perspectives can also be seen in a recent article Gilbert wrote, entitled "Evo-Devo, Devo-Evo, and Devgen-Popgen." In this article he notes, without evidence, that,

> …not all parts of developmental biology and not all parts of evolutionary biology are involved in these new unions. What is happening, I believe, is a series of interactions occurring between

[361] Winther 2003 in press. See in particular tables 1 and 2 in that paper.
[362] There are also clear ways to model and measure fitness provisioning versus fitness reception among interacting units. See Queller 1992b; Frank 1997, 1998; Wolf and Wade 2001.

> *population genetic* models of evolution and *developmental genetic* models of evolution. Both of these models emphasize genes. But in one case, evolution depends upon the frequency of gene variants within a population. In the other case, evolution depends on variations of gene expression between populations. As Ron Amundson has noted, the new union might be characterized as "devgen-popgen"; the genes are not going away when these models are synthesized.[363]

This is a provocative passage. Gilbert and Amundson are the two commentators on evo-devo who have done some work on relating the two disciplines/schools of thought that I discuss. But Gilbert does not present evidence for the interaction of these models or even how such interaction would occur. The remainder of his article discusses either which disciplines of compositional biology, to import my term, belong in evolutionary developmental biology or how genes are an important causal agent. He does not discuss the synthesis that would answer the question mark in my diagram. I do not blame him. The task is difficult and before attempting a synthesis, we have to clarify the severe differences between the two hierarchies of theoretical perspectives; this chapter is an attempt to do so.

3.5 A Literary Analogy

In this chapter, I have shown how the two theoretical perspectives described differ in very deep ways. Although ontology is now, to an extent, shared, methodological and epistemic commitments, as described

[363] Gilbert 2003, p. 348 (his emphasis).

in my four distinctions from Chapter 1, differ radically. Furthermore, even in studying the same or overlapping units and processes, both (hierarchies of) theoretical perspectives employ distinct explanatory resources, such as the pertinent contrast classes, the vocabulary, the research questions posed, and the general goals of explanation.

I am of two minds about the prospects of synthesis. Sometimes I adopt the *two kinds of biology* interpretation and think that the ways practitioners in each respective hierarchy *think about* and *do* science is so fundamentally different that finding translation principles, as well as common (1) vocabulary, (2) abstraction tools, and (3) explanatory resources is well-nigh impossible. For example, consider Gilbert's efforts at synthesis. Despite his laudable efforts he is still thoroughly situated in one hierarchy—one way—of understanding biology. How can the two sides ever understand one another?

At other times I adopt the *eventual convergence* interpretation and believe that a real synthesis is possible. But it will not be easy. We must first, like Dante in the *Divine Comedy*, realize that we first have to tread through a philosophical morass—a hell—before we can achieve this synthesis. The work on (genetic) information and levels of selection above the group provide hints of synthesis. But I remain unsure of how this will unfold. In this chapter, and in the dissertation in general, I hope to provide some guiding principles for moving through the philosophical hell. If I can just be one commentator who plays the role of Virgil, I will be satisfied. I certainly leave the role of Beatrice to others, even if there is a heaven—a synthesis—to be reached.

Chapter 4: Models and Modeling in Formal and Compositional Biology

4.1 Framing and Goals

The concept of a "model" is a rich one. It seems to be unproblematic to say that scientists, and biologists in particular, seek to represent the dynamics (i.e., causes as well as objects and processes) of systems in terms of simplified, generalized, and idealized models. Science seeks both to represent and intervene, and, as we shall see, it does both of these through models. But (1) how to articulate the relation between a model and a theory and (2) how to define all the pertinent properties that a "theoretical unit" must have in order to be considered a proper model are two difficult tasks. There are no simple, or even consensus, views on these issues. Here what I will do is to present the basic version of a model as portrayed by the semantic view of theories. By comparing this version with the account of models from the "mediating model view," and complementing the comparison with concepts from both Cartwright's and Friedman's work on physical laws in addition to Griesemer's and Downes' work on non-standard models, I develop a list of the general properties any model must have.

Some of these properties, in particular those concerned with the *explanatory power* of a model will be interpreted as anathema to the views of at least some advocates of the semantic view who claim that science does not do

any "explaining" – i.e., that explanation is not a proper, or actual, part of scientific work.[364] I describe this view in more detail in Chapter 5. I believe that models serve important roles in scientific explanation, and, as I will argue in Chapter 5, the two different kinds of biology use their respectively different models and model-types to serve different patterns, or what I shall call "pictures," of explanation.

My presentation of models is but a sketch. But if I am successful at my goal, I will be able to present a notion of model sufficiently general to account for the different models and model-types used in *both* formal biology and compositional biology. Formal biology tends to employ mathematical models, which specify transitions through a mathematically-defined state space; compositional biology tends to use non-mathematical models that refer to the compositional structure and capacity of concrete systems. The advantage to my "generalizing" methodology is that I will be able to develop a notion of model sufficiently robust to account for an incredibly diverse range of types of model-types. Subsequently to developing this framework, I will flesh out some of the model-types ubiquitous in compositional biology (e.g., scale, remnant, diagrammatic, and narrative).

[364] For example van Fraassen 1980, 1989; Lloyd 1988.

4.2 General Properties of Models

In this section, I want to present the general properties of a model. In order to achieve such a generalization, I explore two different views on models, the semantic view of theories and the mediating model view, and also contrast these positions with Griesemer's and Downes' unorthodox accounts of models. This analysis, together with some considerations stemming from Cartwright's and Friedman's respective ideas on modeling trade-offs, allow me to articulate the general properties, which I will then use, in the subsequent section, to evaluate particular models from compositional biology.

4.2.1 Models According to the Semantic View of Theories

One powerful and widespread view on models is the semantic view of theories. This view holds that a theory is a family (or, more generally, a hierarchy of families) of formal mathematical models. These models are *presentations* and *interpretations* of various axioms and assumptions (including assumptions about the state-space type[365]). But, "in a semantic definition, the set of sentences that are theorems of the theory is *not* defined by interpreting a set of axioms, but through directly defining the class of structures."[366] That is, although models (at various levels of abstraction) require axioms and assumptions, these source aspects of the models are not the central aspects of theory. Theory is fundamentally about the

[365] van Fraassen 1989, p. 223.
[366] Lloyd 1988, p. 19.

actual class of theoretical mathematical structures—models—represented as possible trajectories in a chosen state space pertinent to the purposes and subject matter ("intended scope"[367]) of that theory.

The semantic view was designed to explicitly contrast with the axiomatic and logic-driven focus on sentences and Laws of the so-called syntactic view of the logical positivists and logical empiricists.[368] The syntactic biases of this school were their biases to formalize, and unify, the propositions of (all of) science in first-order logic. But this syntactic view falls prey to various problems: the very large, in fact, infinite, number of models consistent with any set of logically-formulated laws, the inability to deal with unpleasant consequences of the arbitrary conjunction of laws as well as with the problem of relevance, the apparent explanatory symmetry of laws, the contentious distinction between observational and theoretical terms and entities, and the lack of fit between logical formalizations of theory and how scientists actually represent theoretical knowledge in their day-to-day theoretical activity.[369] Because of its completely different focus (on models of, with, and in, mathematical state spaces), the semantic view does not run into any of these problems.

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[367] Suppe 1977, p. 223.

[368] Here is not the place to do history. I remain agnostic about many points in the history of this topic. And I certainly do not want to claim that logical positivists and logical empiricism cannot provide us with any useful philosophical insights. See for example the recent work by, among others, Creath and Friedman showing how the positivists and empiricists, particularly people like Carnap, got some things "right." In this dissertation, however, I do adopt the biases of the semantic view insofar as they are focused on models. As we shall see, however, there are other ways to cash out the notion of models and there are even legitimate formulations of laws.

[369] These, and other, problems with the syntactic view cannot be further discussed here. Again, the reader is referred to other literature on this topic. See, for example, Friedman 1974 Kitcher 1976, 1991 (1981); Kuhn 1970; Putnam 1962; Suppes 1967; Suppe 1977, 1989; van Fraassen 1989.

This, then, allows us to make the point about the theoretical content of the semantic view slightly differently. There certainly are laws (axioms) in the models of the semantic view, but these are construed as constraints on the allowable trajectories through the state space. I will return to this point in Appendix A. Such laws are: (1) laws of coexistence (i.e., rules specifying which states can exist simultaneously in a model; think of the constraints placed on state-variable instantiations by the Boyle-Charles ideal gas law "PV = nRT" in a 4-dimensional state space), (2) laws of succession (i.e., rules for determining how a state will evolve from a previous state; think of formulas for free fall determining changes in states, at different rates, of time, position, velocity, and acceleration, or of transition probability matrices of Markov chains), and (3) laws of interaction (i.e., "aggregative" ("additive") composites of laws, for cases where the interacting systems and objects are of the same kind, such as an n-body gravitational system, or "interactive" ("non-additive") composites of laws, for cases where the interacting systems and objects are of different kinds – van Fraassen here refers to the uses of perturbation theory and scattering theory; in formal biology, interaction of different forces can be captured in a more specific model with more variables representing the forces[370]).[371] Certainly the laws are derivable, and sometimes even are, part of the theory, in a deep sense. But what is of interest is classifying and defining the set of structures, the *models*, that are *possibly* or, when we compare the theoretical model with data models, *actually*,

[370] See van Fraassen 1970, p. 332 for a brief discussion of laws of interaction, which are generally not considered in formal biology and are absent from Lloyd's 1988 discussion on pp. 19-20.
[371] Suppe 1977, pp. 226-227; Giere 1988b, p. 42; Lloyd 1988, pp. 19-20; van Fraassen 1989, pp. 222-225.

instantiated.[372] Characterization of the models, not of the laws, is the fundamental concern of the semantic view. As I will show in Appendix A, however, I believe that a particular kind of *law-based* view of theories is compatible, even necessarily related, to the semantic view.

Patrick Suppes' slogan summarizes the semantic view: "the correct tool for philosophy of science is mathematics, *not* metamathematics" (i.e., axiomatized sentences in first-order logic in some language *L*).[373] Or, to state the strength of the semantic view with respect to science rather than the *philosophy of* science, van Fraassen notes regarding relativity theory (and, through the argument presented in his book, also with respect to quantum theory) that "I want to insist that the point of view which I have been outlining—the *semantic view* as opposed to the received view [syntactic view]—is much closer to practice…."[374] The strengths of the semantic view are many. While I do not want to argue for the absolute superiority of this view—in fact, I think that it inappropriately biases *all* construals of scientific theorizing in a manner consistent with formal theoretical

[372] On model hierarchies, including the important concept of data models and "models of the experiment," see Suppes 1962. Giere 1988a, 1988b, 1997 presents the view that theoretical models are linked to data models through "theoretical hypotheses." That is, theoretical models are self-consistent and are "more like definitions than sets of empirical statements." (Giere 1988b, p. 40) Their *applicability* to empirical bits of the "intended scope" of the theory is a separate concern from their self-consistency. The latter certainly does not imply the former. When the theoretical model fails, the question, of course, is why. Is the failure in the assumptions about what causes and objects are involved in the empirical system? Is the failure in assumptions about the appropriate laws, mathematically represented? I will return to these questions, and the status of the distinction between theoretical model applicability and theoretical model truth, in Appendix A.
[373] Cited in van Fraassen 1989, p. 221.
[374] van Fraassen 1989, p. 224.

physics[375] and I do not believe that theory can be *defined exclusively* in terms of models, though they are central aspects of theory—I think that their focus on models is extremely helpful.

4.2.2 Models According to the Mediating Model View

There is another interpretation of models and model*ing* that has become increasingly popular over the last few years, the notion of "mediating models."[376] At the heart of this interpretation is the view that (1) theoretical models are not just a subset of theory and (2) data models are not just an organized collation of observations:

> It is common to think that models can be derived entirely from theory or from data. However, if we look closely at the way models are constructed we can begin to see the sources of their independence. It is because they are neither one thing nor the other, neither just theory nor data, but typically involve some of both (and often additional 'outside' elements), that they can mediate between theory and the world.[377]

Models are independent entities, "technologies for investigation" that are "outside" of the hierarchy[378] of data-to-theory.[379] They are neither (completely)

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[375] For example, in 1970, in discussing the part of natural language that was "adapted for a technical role in the language of science," van Fraassen notes that "this meaning structure has a representation in terms of a *model* (always a mathematical structure, and must usually some mathematical space)." (van Fraassen 1970, p. 327; see also van Fraassen 1972) While I applaud his emphasis on models, I strongly differ with him on the point that a model *always* has to be a mathematical structure. Other proponents of the semantic view, who focus on the presentation of models in a *state space* (e.g., Lloyd, Thompson, and Suppe), also emphasize the mathematical structure of modeling.
[376] See, for example, Morgan and Morrison 1999a, especially the introduction by Morgan and Morrison 1999b, the paper on "paper-tools," that is, material and stoichiometric representations, in chemistry by Klein 1999, and the general papers on mediating models by Suárez 1999 and Cartwright 1999b.
[377] Morrison and Morgan 1999b, p. 10-11.
[378] Clearly expressed in Suppes 1962.
[379] Morrison and Morgan 1999b, pp. 32-35, 18.

"derivable" from theory, in the case of theoretical models, nor (completely) "necessitated" by data, in the case of data models.[380] They have a life of their own, a pragmatic aspect, so to speak, in that they act as instruments. This role is both caused by, and affects, the theoretical and empirical practices of scientists. Through constructing, manipulating, and employing models, scientists learn new theory ("representation") and develop new experimental techniques ("intervention") for collecting data. Thus, models and modeling are the locus of activity from which new theory and data spring.

This "mediating model" view differs from the semantic view in several important respects. First, it does not, *contra* van Fraassen 1970, p. 327, hold that models always have to be mathematical structures: "Models may be physical objects, mathematical structures, diagrams, computer programmes, or whatever, but they all act as a form of instrument for investigating the world, our theories, or even other models."[381] I believe that this is a strength of this view. Second, by using examples of scientific activity, the proponents of the "mediating model" position show that models are independent of theory and that models are intimately tied to the activity of scientists. Morrison complains that,

> the classification of theoretical models suggested by the semantic view does not seem rich enough to capture the many ways in which models are constructed and function. The semantic view characterizes theoretical models as 'models of theory' – there is a basic theoretical structure that does more than simply constrain the acceptable models, it provides the fundamental building blocks from which the model is constructed. …However, there is reasonable evidence to suggest that this represents only a very limited picture of model building in physics; many models are constructed in a rather piecemeal way making use of different theoretical concepts in nothing like a systematic process.[382]

[380] See Suárez 1999, p. 169.
[381] Morrison and Morgan 1999b, p. 32.
[382] Morrison 1999, p. 43.

From this we can glean some of the deeper motives guiding the mediating model view: (1) focus on the disunity of science, and (2) concern with dynamic practice as opposed to static representation. The emphasis on the disparate elements of, and within, scientific (1) theory, (2) modeling, (3) experimentation, (4) data-collection, (5) field activity, and (6) instrumentation have become increasingly popular in the last decade.[383] Furthermore, the notion that philosophers of science should study scientific practice, including the theoretical practice of modeling, instead of obsessing with scientific representation, including the static structure of theories, goes back at least 20 years.[384]

Before I turn to discussing disunity and practice, I need to introduce two distinctions. Theoretical aspects of science are often associated with static product, and empirical aspects of science are often linked with dynamic practice, but I think that these correlations are inaccurate, and the relationships are actually *orthogonal*, as indicated in Table 6. (4.1). In both this and the next chapter, I refer to manipulation happening in either, or both, the theoretical and empirical aspects of science. I also discuss the *outcomes* of such manipulations – the "representations." Note that the outcomes can themselves be input for further dynamic practice.

[383] See particularly Galison and Stump 1996; Cartwright 1999a.
[384] See Hacking 1983 who distinguishes scientific representation from intervention; Pickering 1994 distinguishes between "representational idiom" and "performative idiom." He claims, rightly I think, that philosophy of science—and even much sociology of science using text-based metaphors (e.g., Latour's "inscription devices")—employs the representative idiom. He argues that we should move toward a performative idiom.

	Dynamic Practice[385] (Manipulation-Intervention) ["Proper" Mediating Model View]	**Static Representation** ["Proper" Semantic View]
Theoretical Aspects (Abstract Systems)	Model*ing* (Can include material model manipulation)	Theoretical Models
Empirical Aspects (Concrete Systems)	Experimentation, Data-Collection, Field activity, Instrumentation of the concrete system under study	Data Models or Data Presentation

Table 6. (4.1) The relationship between dynamic activity and static representation for theoretical and empirical aspects of science. The mediating model view is difficult to place in this table as it claims that modeling is *independent* of both theory and concrete systems. To some extent, this view is incoherent *until* it can argue successfully for where and how models reside independently. I favor the idea that modeling is a theoretical *activity* even if it has empirical aspects (e.g., empirical assumptions in it). Manipulating models

[385] The distinction between empirical and theoretical activity (practice) that I employ is not immune to critique – see, for example, Griesemer 1990, p. 3. But I think that Griesemer does not make the important distinction between *content* and *activity*. Theoretical activity can certainly have, and often has, much empirical content. Furthermore, in so far as remnant models and, say, scale models are *representations* of an empirical system (fauna in nature and actual animal parts), manipulation of them, even if material, is a theoretical activity since we are here importing theoretical biases and guiding assumptions regarding what these material models *mean*. I realize that it is counterintuitive to argue that material manipulation (in so far as it is of material *models*) is theoretical, but consider the fact that *any* kind of theoretical manipulation, short of *thinking*, includes material manipulation (e.g., writing formulas or making drawings on a piece of paper or blackboard). Manipulation of data and models using computers does, however, constitute a difficult case to fit under either empirical or theoretical manipulation. I believe that computational manipulation is probably a *genuinely new kind* of scientific activity.

(including *material* models) is a theoretical activity; manipulating the concrete system is an empirical activity (see footnote 385).

Let me now return to the two points regarding disunity and practice, respectively. I want to remain agnostic regarding the disunity of science *in general*. As I argued in Chapter 3, I certainly *hope* that some areas of biology, levels of selection theory and evolutionary developmental biology in *particular*, can be synthesized, or, to use a more philosophical term, unified.[386] Such a unification is, as argued, difficult to achieve given the deeply-entrenched differences in kinds of theory/theorizing and empirical practices, but there are resources to accomplish the unification. Thus, I do not want to adopt *necessary* local disunity in the case study I investigate, even if there is *actual* disunity. It is unclear, however, whether even advocates of the disunity view, which include proponents of the mediating model view, believe in *necessary* local disunity. I will not be concerned further with the issue of unification of scientific domains in general, but in Chapter 5, I will address the notion of unification as an aspect of theoretical explanation in particular domains.

With respect to the second point regarding practice, I want to note that while I do believe that a focus on scientific empirical practice is important and informative, my project focuses on the theoretical aspects of science, dynamical and static alike. These aspects, of course, are not inconsistent with empirical practice.

[386] As discussed in Chapter 3, the unification desired is not just theoretical, it is also methodological (i.e., in terms of experiments, instrumentation, and, if we adopt the mediating model view, modeling).

My comparison between the semantic view and the mediating model view seems, then, to point to three differences between them: (1) the extent of independence between model and theory, (2) the focus on scientific empirical practice, as opposed to static aspects of theory structure, and (3) the theoretical (dynamic and static alike) disunity of science. I think these are *real* differences between extreme interpretations of the two views.

Despite these real differences, a further important point must be noted. The semantic view certainly believes in the *in principle* actual elaboration by scientists, and analytical conversion by philosophers of science, of models from theory. But this is an *ideal* goal. When we actually inspect a case of semantic view analysis, Lloyd's 1988 book, we notice that she too takes a model-centered view and analyzes the models, to some extent, independently of the deep theory of evolutionary biology. That is, the models have a life of their own and they differ, for example, not just in their theoretical resources, but also in their empirical assumptions about, for example, population structure and genetic additivity. Furthermore, Lloyd discusses both the static aspects of theory structure *and* the process of model building. Thus, the models Lloyd analyzes seem to be, as Morrison and Morgan pointed out in note 14 above, "neither just theory nor data, but typically involve some of both (and often additional 'outside' elements)."

Put differently, Lloyd *could have* shown how *every* (or even *any*) model she analyzed could be converted, in principle, into a set of laws, elaborated from theoretical principles, that constrained trajectories in state space. This would seem to be the *proper* ("static representation" in Table 6. (4.1) above) semantic view analysis. But she does not do this anywhere in her analysis. One can argue that that is because it would be too cumbersome and that in analyzing the

assumptions about parametrizations and what variables are important, she is indirectly doing a proper semantic analysis. But this is not a good argument. Conversion into a state space is not too cumbersome – she could even have done a point by point comparison of the laws of coexistence and succession in different models. But, I argue, *this would not have been useful*. The utility of Lloyd's analysis lies, and would lie, in her stringent and cogent comparison of different empirical assumptions and mathematical techniques in the activity of modeling. Furthermore, her (and Wimsatt's) important additivity definition of a unit of selection is a *conceptual resource* (perhaps Morrison and Morgan's "'outside' element") that can be assumed and applied in models of different kinds, both under the gene selectionist and multi-level selection theoretical perspective [see Figure 1. (3.1)]. Her analysis of assumptions (e.g., the assumption of a definition of a unit of selection) could not be done simply by converting the models into state-space trajectories. Conceptual analysis and assessment of the role this assumption plays in the act of modeling must also be taken into account. Thus, I would say that Lloyd also implicitly adopts a "model-centered" notion emphasizing the practice of modeling and including consideration of the role empirical aspects have in modeling.

Lloyd also seems to emphasize the disunity of biology. In fact, one aspect of her research project is to show that different players in the units of selection debate ask *different questions* about biological processes (e.g., What is the replicator? What is the interactor? What is the beneficiary of adaption? What is the carrier of adaptation?).[387] This implies a commitment, on her part, to the idea of fundamental disunity in research projects analyzing units of selection. Thus, if

[387] See especially Lloyd 2000a.

we were to form an *axis*[388], Lloyd falls somewhere in between a "proper" (not meant in a normative sense[389]) semantic view analysis, focusing on a purely mathematical theory structure with an implicit nod toward a unified and neat hierarchy of models, *and* a pure mediating model view, emphasizing the activity of theory construction through modeling and including a commitment to a patchy relationship between models. I think that this intermediary location is a good place to be, and I hope (and believe) that my analysis of models in compositional biology falls in a similar place. There is more to modeling than theory and there is more to theory than models and modeling.

Downes' previous analysis of modeling in science is compatible with my generalized account, to be explored in the next section, of the role models of various types play in scientific theorizing. Downes defends a "deflationary semantic account" in which he rejects the traditional semantic view's account of scientific theories as families of [mathematical!] models.[390] He argues that "there are far greater differences between models in mathematics and logic and models in science than holders of the semantic view have been prepared to admit."[391] In part this is because isomorphism between models and data is a much more difficult relation to define than isomorphism between models at different levels of generality. Thus meta-mathematics cannot provide a clear account of scientific model building. Furthermore, Downes claims that some scientific theorizing does not involve model building, although he does not provide clear examples.

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[388] Although perhaps the notion of *complementarity*, rather than the idea of extremes along an axis, might be more useful.
[389] In fact, a "proper" semantic view would be *without much use* for Lloyd's purposes.
[390] Downes 1992, p. 142.
[391] Downes 1992, p. 144.

In short, Downes argues, although the meta-mathematical strong relationship between theory and model is of limited use in understanding scientific model construction (models as independent from theory), and although theorizing does not always involve modeling (theory as independent from models), "model construction is an important part of scientific theorizing."[392] By focusing on multiple types of models and by allowing for the independence of models and theories, Downes provides a deflationary semantic account compatible with the generalized model account to which I will now turn.

4.2.3 Four General Properties of Models

The model-centered analysis of both the semantic view and the mediating model view provides a useful framework for my project. Models have been and should be an important locus of investigation for the philosophy of science.[393] One of the important differences between formal and compositional biology is precisely the model-types that they employ. I have argued that compositional biology uses primarily a variety of non-mathematical models, such as scale, remnant, diagrammatic, and narrative models. But I have not yet described what these models actually are. In this section I want to finish setting the general stage before I turn to an analysis of the models employed in compositional biology. This stage setting will consist in articulating four general properties any model (not just mathematical models) must have.

(1) *A model is an abstract meaning structure, of various types and at various levels of abstraction, of objects or processes in the world*. Thompson 1988 describes

[392] Downes 1992, p. 151.
[393] As Downes 1992; Griesemer 1990, 1991a have also argued.

van Fraassen's semantic view as one in which models are "meaning structures."[394] I find this to be a useful articulation. Models are abstract representations containing kinds of properties and objects that have particular kinds of relations between them. These can be abstract parameters and variables expressed in different mathematical functions, or they can be material objects with components (e.g., scale or chemical models) standing in particular relations to one another and which, as a whole, *represent* a set of concrete systems, or they can be diagrams with kinds of parts that, also, represent a concrete system. All of these are meaning structures. Meaning structures can exist at various levels of abstraction – for example a mathematical model of gene frequency change with many parameters or a diagram representing many kinds of components are more concrete meaning structures than, respectively, a mathematical model with few parameters or a diagram indexing few kinds of components.

(2) *Models can be articulated, or "hooked up" with other models, subject to the background protocols and assumptions of the pertinent theoretical perspective.* The abstraction relations, rather than the articulation or "aggregation" relations, of models have been emphasized in philosophy of science. Although specifying the abstract-concrete relation between models is crucial, it is also important to study the way models are aggregated and partitioned. For example, how are neutral models and selective models of evolution combined? Or, how are physiological and developmental narrative models of a kind of organism merged? Particularly in compositional biology, where different kinds of parts are studied from different perspectives, this becomes an important question.

[394] Thompson 1988, Chapter 4; van Fraassen uses the term on, for example, p. 327 of his 1970.

(3) Because of their independence as well as their mediating role between theory and concrete system, *models enter into a series of trade-offs*. Richard Levins first articulated a set of three desiderata that any given model cannot maximize simultaneously: realism, generality, and precision.[395] The literature on this topic is significant and I do not want to enter into it here.[396] However, one central trade-off that I will explore is the relation between the desiderata of generality and realism (*sensu* applicability to particular situations). Amount of abstraction (the impetus to generalize) in modeling trades-off with *complete model applicability* to, or *full explanation* of, a concrete set of circumstances.

This is precisely the point that Cartwright made by arguing that truth (i.e., empirical adequacy) and theoretical explanatory power trade off one another: general laws do not have much explanatory power for particular circumstances in which a host of other factors besides the few causes described by the general laws play a role.[397] In her account of scientific activity, Cartwright also presents a model-centered (as opposed to law-centered) approach: "To explain [causally] a phenomenon is to find a model that fits it into the basic framework of the theory and that thus allows us to derive analogues for the messy and complicated phenomenological laws which are true of it."[398] Cartwright further argues that the models serve a variety of purposes and that different models emphasize different (sets of) "realistic" factors. Thus, there is no all-around best model. In so far as a model accounts for an increasing number of factors, and interactions

[395] Levins 1966, 1968.
[396] But see Weisberg 2003. Weisberg makes the point that the fundamental argument Levins made was not so much that there was a trade-off between these three particular desiderata, but rather that there *always* would be trade-offs between any desiderata.
[397] See Cartwright 1983. See also footnote 480 in Chapter 5.
[398] Cartwright 1983, p. 152.

between them, it will be a more realistic and applicable model, but a less general model. In so far as a model accounts for the "additive" (*sensu* averaged) effect of a single (or few) factor(s), it is general, but not causally explanatory of concrete situations. The intended scope of Cartwright's account is definitely physics for which "the mathematical framework" is essential to theory.[399] But her articulation of the fundamental trade off between abstraction and (explanatory) concreteness pertains to all models types.

An interesting disagreement between Cartwright and Friedman, pertinent to my argument, appears. I shall explore Friedman's concerns regarding explanation and unification in Chapter 5, but one way to state his views simply is to say that for him (and Kitcher) generality ("true" laws and models) and explanatory power are deeply and inseparably *connected*. Generality is necessary for explanation in that the former decreases cognitive burden and provides explanatory strength. They thus do *not* trade off one another. The reason for this difference is, at least in part, that for Cartwright explanation is about explanations of concrete phenomena and material causation (i.e., causal explanation) whereas for Friedman explanation is about explanation of laws and theories (i.e., theoretical explanation). Friedman is not concerned with "mechanisms" and material causation. Furthermore, he holds a "neat abstract hierarchy" view of theoretical structure, reminiscent of Suppes'. Interestingly, he also holds that a set of increasingly abstract laws (and models) can be confirmed.[400] While unifiability and subsumption are directly related to

[399] Cartwright 1983, p. 152.
[400] For example: "For a theoretical structure that plays a role in theoretical explanations in many diverse areas picks up confirmation from all these areas." (Friedman 1981, p. 7)

explanation in an approach focusing on explaining laws or models, as we will see in Chapter 5, it seems to be the case that such a relation is, in fact, an ideal rarely reached in science, even when the focus is on theoretical explanation.[401]

Furthermore, because Cartwright analyzes the complexities of (1) *practice* (e.g., by looking at lasers) and (2) causal explanation in physics, she espouses a concretivist trade-off account between unification/generality and explanation. When investigating biology, even mathematical evolutionary genetics, mechanisms, and therefore causal explanation, also come into the picture.[402] In the case of biology, then, the concretivist analysis of desiderata trade-offs, rather than Friedman's abstractivist law or model subsumption account, comes into the foreground. Thus, my emphasis here will be on Cartwright's *trade-off*, rather than Friedman's *complementarity*, relationship between generality and (theoretical or causal) explanatory power in scientific modeling.[403]

(4) *Models are used in a variety of scientific activities, including guiding further modeling and empirical activity, as well as providing explanations.* An abstract model can be used for various scientific activities, such as describing, explaining, or increasing our understanding and control of the empirical world. A model need not do *all* these activities to count as a model. By acting as "needle-eyes" of the empirical world through which relatively few kinds of properties, specified by a

[401] Friedman pers. comm., July 9, 2003.
[402] Some, such as Morrison 2000, make the same argument for much of physics. Here there seems to be a disagreement about whether physics is a theoretical and formal enterprise or, instead, a search for explanations of mechanisms. I do not here seek to engage in this debate. However, the debate will resurface when dealing with the explanatory status of mathematical evolutionary genetics in Chapter 5.
[403] Though in some cases, such as the Price equation of mathematical evolutionary genetics, Friedman's account is a valid and pertinent interpretation, as we shall see in Chapter 5.

theoretical perspective, pass and by being depaupered and constructed representations of nature, they can serve to *expand* our knowledge of nature in that they provide suggestions for experiments, observations, and measurements as well as for new kinds (or more specific) models. Thus, they are in the almost paradoxical state of being impoverished tools for a rich expansion of scientific knowledge. However, their power seems to lie precisely in that they only focus on a few salient features that direct creative manipulation in theory and in concrete systems. In their abstracted state they guide us to unexpected theoretical connections and new empirical observations.

These four general properties of models hold for the mathematical models of formal biology. As I will now show, these properties also apply to the models of compositional biology.

4.3 Five Case Studies of Models in Compositional Biology

In what follows, I will present five case studies of models in compositional biology. For each model, I show how it matches the general properties of models.[404] I start with a material model, Griesemer's notion of remnant models in natural history. It is unclear whether natural history is a compositional biology – but I do think that community ecology and systematics (with the Ghiselin-Hull

[404] The sole exception to this is my discussion of E.J. Corey's "retrosynthetic analysis," which I discuss primarily as a way to undercut any claim that modeling in compositional biology is never abstract and that models are never manipulated independently of empirical activity.

concept of the species as an individual[405]) can be interpreted as investigating organisms as parts of species and species as parts of communities or clades. However, not much rests on this unsubstantiated claim. Here I want primarily to point out that models can be material (consider also scale models in functional morphology) and that bona fide compositional biological sciences such as comparative anatomy and functional morphology certainly use remnant models (e.g., vertebrate fossil skeletons) in their work.

I then follow with two particular models, one from functional morphology (tetrapod feeding) and one from developmental biology (genomic regulatory systems), that are themselves *expressed* through different model types. This leads me, in concluding this chapter, to note another aspect of complexity in my argument: the variety of levels of abstraction in meaning structures. Thus, there are at least two issues at hand regarding models in compositional biology: (1) classifying different model-types (or, "meaning structure type") and (2) distinguishing the level of generality of presentation of the meaning structure.

The last two models that I analyze are from biochemistry. The first (the Krebs cycle) is an actual model (with little actual concrete variation of its various components), whereas the second (the method of "Retrosynthetic Analysis") is a *modeling technique* involving significant amounts of abstract theoretical model manipulation. This is meant to contrast with a number of the other models of compositional biology, where there is a constant interplay between empirical and theoretical activity. I chose to discuss these two models because I believe that it is important to explore a variety of cases of compositional biology. Biochemistry is an important compositional biology, and models and modeling play an

[405] See Ghiselin 1974, 1997; Hull 1975, 1988.

important role in it. Discussing chemical models brings up the important point of how chemical "theory," and manipulation with chemical material or diagrammatic models, *differs* from mathematical "theory," and manipulation with mathematical models.[406]

The model-types pertinent to compositional biology all represent *parts* of a system with particular capacities – these models thus serve a crucial role in the activity of causal explanation. Thus, there is a crucial conceptual relationship between models, modeling, causal capacities of concrete parts, and explanation in compositional biology. I explore this relationship in both this and the subsequent chapter.

4.3.1 Natural History: Remnant Models

In discussing "remnant models", Griesemer shows how the specimens in a museum, though material, actually serve theoretical roles. First of all, these models share multiple properties with the things they model since "they are actually *made from* the very individuals modeled."[407] Of course, though, they are not the object (e.g., populations or species) *in toto* since they are only a representative sample of the individuals in nature. They refer to natural objects at multiple levels of the compositional hierarchy:

> [animal and plant remains preserved in museums] are physical objects, which, for specific scientific purposes, are taken to represent the whole, living individuals of which they were once part. As such, specimens are remnant models of their wholes. Remnants can provide the means for indirectly referring; naturalists sometimes intend talk about specimens in a museum, for example, to be indirect talk about other organisms or other

[406] I thank Sander Gliboff for helping me to see the importance of this comparison.
[407] Griesemer 1990, p. 8.

> more inclusive entities, such as biological species or ecological communities in nature.[408]

As Griesemer shows, these objects can be used for a variety of theoretical activities, such as reconstructing the evolution of different species (in different niches of an area) as a consequence of environmental factors such as temperature and humidity. The remnant models can also be used for determining the ecological relationships between different species (of complementary ecological classes). Thus they are both models in themselves and also serve as the material for constructing further theoretical models about general patterns and processes of the wholes that they represent.[409] Despite their materiality, they play an important role in theory construction and in directing—mediating—further laboratory and field activities. They are also meaning structures in the sense that they serve as *indices* of the fauna in nature, and represent ecological relations between different species, without themselves *being that* fauna. Representing ecological relations is an example of model aggregation.

In terms of trade-offs between, for example, generality and realistic applicability, a remnant model can itself be thought of as a "generalization" since it is an index of objects (species) in nature. Another way in which remnant models could be considered generalizations is that they could represent particularly important representative species for a particular ecological type (e.g., remnant models of a species of bat and a species of hawk as representatives of flying predators). As fewer and fewer remnant models of fewer and fewer species are chosen to represent such ecological types, these remnant models could be thought of as becoming more general in that they stand in for many

[408] Griesemer 1990, p. 8.
[409] Griesemer 1990, p. 12.

other remnant models of other species. Of course, they are distinct from these other remnant models in many respects and do not realistically match them – hence the trade-off between generality and realism.

The modeling and empirical activities of remnant models is found primarily in the museum *practices*. The practices of collecting, labeling, storing, measuring and drawing (and otherwise representing), and sharing remnant models determine how the models are manipulated and further represented. The materiality of remnant models is a strength: "They are robust to some changes of theoretical perspectives because they are literally embodiments of phenomena. If these embodiments are preserved, they may be studied again and again under different [theoretical] perspectives."[410] Manipulation activities are, to a large extent, independent of theory and theoretical perspective.

4.3.2 Functional Morphology: Tetrapod Feeding

I now turn to a case study of a functional morphological general model of the tetrapod feeding cycle, represented with various diagrammatic and narrative models. Bramble and Wake present what they refer to as a "model generalized feeding cycle." They note that "the feeding cycles of such phyletically diverse groups as mammals, reptiles, and amphibians appear to incorporate very similar trains of stereotyped kinematic and motor events, involving homologous sets of muscles…"[411] From this commonality of feeding cycles among such disparate vertebrate taxa, they suggest that the whole feeding cycle was actually present in the ancestor and, therefore, a general model "defining the properties of this

[410] Griesemer 1991a, p. 80.
[411] Bramble and Wake 1985, p. 235.

mechanism" would have "heuristic value for future efforts to understand the evolutionary processes that have led to the array of feeding mechanisms observed among tetrapods, living and extinct."[412] They equate the general pattern with the ancestral pattern – this is reminiscent of 19th century equivalences between archetypes and ancestors. At any rate, the goal of this model was to provide a general meaning structure for generating predictions of feeding behaviors in various *unobserved* taxa as well as noting deviations from the pattern in observed taxa. Various ecological and morphological hypotheses concerning causes of *stability* and *deviation* from the general/ancestral pattern could then be generated. Although the model was generated from careful observation, it was meant to guide both hypothesis development and experimentation and observation to test such hypotheses. This model was intended to itself be a case of a "deductively-based morphology" that was predictively and explanatorily powerful.[413]

In their model, Bramble and Wake partition the motions of the feeding cycle. *These stages become kinds of processual components of the meaning structure.* They divide the opening and closing of the mouth into five stages: slow open I (SO-I), slow open II (SO-II), fast open (FO), fast close (FC), and slow close-power stroke (SC-PS). The first three stages represent what the generalized/ancestral vertebrate does as is it opens its mouth in a sequence and stops briefly at two points before reaching maximum gape breadth (Appendix Figures 4.1, 4.2). Most of the extension of the tongue occurs during the SO-II stage. Once the food item has been secured with the tongue, it is brought rapidly back while the mouth is

[412] Bramble and Wake 1985, p. 235.
[413] David Wake, pers. comm., July 24, 2003.

closing in the FC stage. But the mouth does not completely close until the tongue has been brought completely back. Once this has occurred, the SC-PS stage occurs. Involved in this stage is the initial crushing of the food item (power stroke).

In tandem with measurements of movements of various parts of the head anatomy, the feeding cycle can be represented as movements of various muscle groups (Appendix Figure 4.3) and as *predicted* neuronal activation patterns of various muscle groups (Appendix Figure 4.2; note that these predicted patterns have not to date been robustly confirmed[414]). The diagrammatic and narrative models that Bramble and Wake present *express* the general model of the feeding cycle.

Let us now explore the ways in which this fairly abstract meaning structure meets the general properties of models. First of all, it is clear that this model was used for predictions of behaviors and part capacities in all sorts of taxa. In fact, the model served as a way to focus attention on a particular functional morphological process; a mini-research program ensued from Bramble and Wake's suggestive model.[415] Their theoretical representation of tetrapod feeding was a convenient and powerful starting point for a whole series of investigations. These investigations continue to use the same partitioning (into temporal, structural, and processual units, as described in Chapter 2) of feeding that Bramble and Wake had suggested.

Often models are considered to be idealizations *from* empirical systems. They are meant to capture as many empirical systems as possible. That is, the

[414] David Wake, pers. comm., July 24, 2003.
[415] See, for example, Schwenk's 2000 anthology entitled *Feeding. Form, function, and evolution in tetrapod vertebrates*.

worth of a model stems from the breadth and accuracy with which it describes empirical systems (given the myriad assumptions of the model). One problem is that often models make *false* assumptions for purposes of simplicity and tractability and, in those cases, they may not match many, or even any, empirical systems very well. The idealized pendulum is one such example. Giere has an interesting take on this case. He argues that such models should be considered *definitions* that can either match or not match empirical systems. When they do not match the system, they are not false, they are simply *inapplicable* to that system.[416] Models are thus *postulations* rather than *idealizations*. Others, of course, take false assumptions at face value and then claim that the model is false (e.g., Cartwright, Levins, and Wimsatt). In Chapter 5, I will defend this latter view.

Now, Bramble and Wake's model plays the same sort of role as the idealized pendulum. In both cases an abstract model is presented/postulated that makes a large number of simplifying assumptions such that they strictly match some, but certainly not all, cases. For the cases in which they do not match the real system, reasons for the mismatch can be explored (e.g., ecological conditions in ant and termite-eating mammals are such that, for example, tongue and hyobranchial movement are almost completely decoupled[417] – the fourth column of Appendix Figure 4.1 is such that the two are *coupled*). This can then give us impetus for a more general model from which certain assumptions are simply missing[418] or, for a family of more specific models each using different

[416] Giere 1988.
[417] Schwenk 2000b, p. 51.
[418] There is an important difference between making simplifying assumptions and making no assumptions about a particular state of the system. This corresponds to Cartwright's 1989 distinction between idealization and abstraction made in Chapter 5 of that book. I do not have the space here to

(potentially overlapping) sets of veridical empirical assumptions to match different taxa. The crucial point here is that Bramble and Wake's model is a postulated abstract representation that can be verified/shown to be applicable for particular cases. Despite the fact that the model fails to match some cases, it still serves as an excellent organizing resource for research.

Thus, Bramble and Wake model is a *postulation* of a general feeding cycle, abstracted from observations. It is hypothesized that this model describes the behavior of the ancestor of tetrapods. There is some ambiguity as to how general the model is. In a sense it is very specific in that the curves for each of the five head parts measured have specific values, hence they represent very specific locations during each part of the cycle. A more general model would be one in which there was a spread of possible curves for each of the lines *or* one in which there was a spread of timing for each of the five stages (e.g., SO-I could be broader or narrower than it is), or both. The more specific the curves, and the tighter the stage-definition is, the more concrete the model is. A more concrete model would match fewer cases than the *potential* breadth of matching of a more general model. Bramble and Wake are unclear about exactly what the level of generality is for their model. But it is clear that the more general their model (i.e. the less specific the lines and the more flexibility in stage definition there is), the less it would apply to any particular case, and the less it would *explain* that particular case – i.e., the less it would describe the correlations and functional integrations between the different parts of the head and their capacities. Their

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further discuss this issue. With respect to assumptions, I will argue that either of these moves makes for an increasingly general model.

model enters into the same set of trade-offs between generality and (causal, not theoretical) explanation that occurs in physics as described by Cartwright.

Furthermore, this model also has rules of both theoretical and empirical manipulation. Certain muscle groups (Appendix Figure 4.2) can only be operative during particular stages of the feeding cycle. For example, the depressor mandibulae, which upon contraction brings the posterior end of the mandible close to the posterior (back) end of the skull, can only *open* the mouth. Hence, this part of the muscular system is active during the three stages of gape opening. *Self-consistent* predictions of muscle activity are based on assumptions regarding constraints of movement. Furthermore, recall the discussion above concerning potential levels of generality in this model by leaving the exact values of the lines unspecified. Even when the lines are unspecified, the *relationships* between the lines are constrained (e.g., gape cannot be open with mandible up). Thus, constraints on the consistent manipulation of the model exist. The sum total of these consistent manipulations could be thought to present the total number of allowable states of the model, which describes the total set of possible real world systems.

These constraints are *internal* to the model. There are also constraints regarding how the model can be hooked-up to other models, for example models regarding prey-search behavior (previous to feeding) and swallowing (after feeding). Certain kinds of behaviors are consistent with the model of feeding (e.g., organism must face the prey head-on). Furthermore, the tongue must eventually reach a certain set of positions during food item capture in order to be involved in swallowing. Thus, if models were developed for these other stages of overall feeding, they must be made to articulate ("hook-up") well with Bramble

and Wake's model, which refers to the part-structure involved in the functional morphology of feeding.

Bramble and Wake's model matches most of the general properties of a model. Yet the model actually does not allow for too much informative manipulation. Much of the work comes in testing the model against target concrete systems and seeing how often it is false and inapplicable, and subsequently altering or tailoring it to particular situations at hand.

4.3.3 Developmental Biology: Genomic Regulatory Systems

Another example of the employment of modeling techniques of various kinds (e.g., diagrammatic and narrative) in compositional biology is found in a particular subfield of developmental biology, "functional genomics." Many genes are only expressed in some spatial locations during some periods of development. Of these, some genes are expressed continuously, both in space and in time. Others are expressed discontinuously in space or time, or both. What regulates the timing and placement of gene expression? Answering this question is one of the tasks of functional genomics.

Here I will analyze the models that Eric Davidson presents regarding *cis*-regulatory genetic elements.[419] A *cis*-regulatory element is a segment of DNA near a gene that controls the expression of that gene by serving as a binding site for regulatory proteins involved in repressing or stimulating transcription of the gene. Following convention, Davidson calls such regulatory proteins "transcription factors," which is

[419] Davidson 2001; see also, Arnone and Davidson 1997.

> used… as a neutral term, denoting any protein which displays a high specificity for a particular *cis*-regulatory DNA sequence, and which performs some function that affects transcriptional output. Transcription factors execute a variety of functions, e.g., repression, activation, transduction of external signaling, or architectural alteration of *cis*-regulatory complexes, and they mediate diverse *cis*-regulatory logic functions. For example, a commonly observed *cis*-regulatory format is one in which two different transcription factors responding to two different inputs, perhaps an intercellular signal and a lineage marker, must both be bound in order for there to be any output ("and" logic).[420]

A number of different *cis*-regulatory elements are involved in regulating the expression of any gene. All these elements need to be configured in the correct way (e.g., presence of activation proteins, absence of repression proteins, and appropriate three-dimensional architectural structure of the DNA) if gene expression is to occur. This is a very intricate and piecemeal process, which allows for fine-tuned control of state of gene activation and amount of gene transcript produced.[421]

Davidson emphasizes that these elements tend to be organized in groups, or modules, which, as a unit, activate or repress a gene in the vicinity of the module. These (genetic regulatory element) modules sometimes contain multiple sites for the same type of transcription factor. And modules sometimes have to act in synchrony to activate some kinds of genes. Davidson operationally defines a module as

> a fragment of *cis*-regulatory DNA that, when linked to a reporter gene[422] and transferred into an appropriate cell, executes a

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[420] Davidson 2001, p. 8.
[421] Also discussed in Gerhart and Kirschner 1997; Moss 2002.
[422] Which allows for identification that the transform ("transplantation," to use a vernacular metaphor) of the entire gene complex (reporter + whatever else the reporter is attached to, in this case the *cis*-regulatory module) into a particular cell type has occurred successfully – i.e., it "reports" success by making an *independent* protein-type, such as *B*-galactosidase. (cf. Alberts et al. 1994, p. 424)

> regulatory function that is a subfraction of the overall combined regulatory function executed by the complete system.[423]

As mentioned above, often the same gene is expressed at different times or at different locations, or both. For a particular time and place, only a *sub-set* of the complete regulatory system for that gene (i.e., only some of the modules) is required for gene expression. One reason that there is a mix and match of modules for the *same* gene is that each module responds to the molecular environment present in that time and place; and for different times and places, there will be, for example, *different* co-factors and *different* products of inter-cellular signal transduction events. The same module could not respond to these differences. Hence a gene that has to be expressed at different times and places must be regulated by more than one module.

Now that I have discussed some of the basic details of gene regulation, let me turn to some models that Davidson presents to make his argument. As the most general meaning structure of the regulatory activity of these modules, Davidson presents a "cartoon" of two modules each affected by four kinds of input, "spatial repressors," "signals from adjacent cells," "cell cycle control," and "lineage."[424] The first kind of input serves the role of inhibiting the module from stimulating gene expression when found in a cell *in particular spatial domains*. Most genes are turned off in most places of a developing organism most of the time, so this is an important kind of input. The second input kind concerns transduced molecular signals from adjacent cells (e.g., "signal transduction pathways") that indicate to the module what its state (and if "on," what quantity) of activation it is in (it must produce). The third and fourth input kind could be

[423] Arnone and Davidson 1997, pp. 1851-1852.
[424] See Appendix Figure 4.4.

considered "intrinsic state" recognizers/determiners. That is, the right types of molecules must be present in the cell, contingent on time in the cell-cycle[425] and cell-type (i.e., lineage), to activate the given module. Note that this "cartoon" model is extremely general and does not even specify (1) the relationships, in type and in connectivity, between the molecules of these different kinds of inputs, (2) the nature of the functional architecture of each module, or (3) the relationships among the modules. The cartoon meaning structure is general, i.e., depicts few relations in the meaning structure, and does not explain any particular case. However, it certainly can serve as inspiration for knowing which kinds of objects and processes to look for in a *cis*-regulatory network. It can aid in guiding research and is, thereby, a mediating model.

Let us now turn to a much more concrete instance of this general model, a series of models for the "*cis*-Regulatory logic in the *endo16* gene" of the sea urchin *Strongylocentrotus*.[426] In figure B, Davidson lays out the different modules in their linear order and depicts the transcription factors that bind (as absolute or continuous repressors or stimulators) to the different sites of each module. He does not depict the function of each transcription factor, but he does state what the function of each module is. Recall the more general model now under analysis. The input kinds depicted there inform our understanding of the more concrete model. (1) Modules **DC, E**, and **F** respond to spatial repressors and serve that very function (they repress gene activity in certain spatial domains). (2) Modules **A** and **B** respond to the latter three kinds of input signals of the

[425] I.e., between mitosis events, cells go through different types of phases, and gene expression only happens in some of those phases.
[426] See Appendix Figures 4.5-4.7. Arnone and Davidson 1997; Davidson 2001, Chapters 2 and 3.

more general model. Thus, there is an abstract-concrete relation between the two models. The concrete model pertains to a specific taxon, whereas the general model pertains to any (and thereby, no!) taxon. Note also that there are clear rules of model hook-up in the concrete model – we simply denote more modules and, if we choose to include more genes, we depict *their* regulatory modules.

In figure C, Davidson depicts a very interesting model(-type) that he has developed with some co-workers.[427] The model represents the activity of the two regulatory modules (**A** and **B**) involved in the actual expression of the *endo16* gene in terms of a logic or circuit diagram. Let me try to explain the model in a pithy fashion – the interested reader can consult Davidson's rather lengthy figure caption for further information (see Appendix Figure 4.7). The boxes represent the *cis*-regulatory elements, the lines represent different kinds of output (Boolean as well as continuous time-variant or time-invariant) that produce different intermediary "states" in the circuit. From this wiring a logic-chart can be produced which assigns intermediary values, contingent on which sites actually have their transcription factors present, which when totaled (in i12) depict (relative) *endo16* gene amount expressed. Note that some sites, when they have transcription factors present, actually act as inhibitors [for example R + UI + CG1 + P, which gives (i5)] and then that state, if active, provides an intermediary value of 0. According to the diagrammatic circuit model and the logic chart, if Z and **DC, E**, and **F** are present in the correct ways, giving a value of 1 for (i9), all previous activity is cancelled out. But CG2, CG3, and CG4 will, if all present, still produce *some* gene product. Note that this model does not depict the actual *mechanisms* among transcription factors, but rather depicts the net quantitative

[427] See, for example, Yuh et al. 1998.

relative effect that presence or absence of any transcription factor has on the overall production of *endo16* gene.[428] As Davidson points out, "Each function was established by measurement of the output of the system after mutating one or more target sites, or substituting synthetic target sites for the natural sequence."[429]

It is difficult to say what the nature of these models are. figure B of Appendix Figure 4.5 is a diagrammatic model. But what is figure C of Appendix Figure 4.6? The first diagram, representing the network is a diagram. But it is a *logic* diagram, not a *mechanism* diagram (see footnote 428). So it is not actually representing actual concrete activity, but is portraying *logical consequences* of removing elements of a concrete system. This is a new kind of diagram, which, admittedly, is useful for summarizing the outcomes of interactions among different kinds of parts. It is a useful mediating model for compositional biology. The logic chart in figure C could be seen as short-hand for a narrative model of the logical dependencies between different intermediary states of the overall regulatory system. Writing out the logic chart in fleshed out English would provide a cumbersome narrative model of the overall logical story (although it would not provide the *mechanistic* story!).

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[428] It is unclear what the relationship between mechanism and logical networking is. Does contiguity of logical relationship (a single arrow) imply contiguity of interaction between transcription factors? Does the direction of the arrow (which makes perfect sense in logic) make sense in chemical interactions, which are interactional (molecules A and B have effects on one another)? And what does the overall directionality indicated here represent in terms of mechanisms? Transcription factors are binding and interacting with one another, but they are not actually making products – that is, the various i_n's do not represent actual intermediary products, but rather represent intermediary logically-useful "quantitative states," which often refer to previous quantitative states.
[429] Davidson 2001, p. 61.

Certainly, however, the logical model has predictive power: "The statements afford testable predictions of the output for any given mutation or alteration of the system."[430] That is, if a site is mutated and it cannot bind its transcription factor any more that would, in many cases, be equivalent to an "else" reading. The model can then be checked by systematically mutating different regulatory regions (at different nucleotides, if one desires) and seeing if the effect is what one expects given the previously inferred "else" readings – the model is certainly generative of many new experiments. It would be interesting to further consider the difference between a *mechanistic model* (such as Bramble and Wake's or even Davidson's figure B) and Davidson's *logical model* of a mechanistic system.

4.3.4 Biochemistry: Krebs Cycle and Corey's "Retrosynthetic Analysis"

A model that is not currently guiding research and that is also not, strictly speaking, manipulable, is a diagrammatic representation of the Krebs cycle.[431]

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[430] Davidson 2001, p. 60: Appendix Figure 4.7.

[431] Though it was manipulable in the past (i.e., one could imagine discoverers of the process abstractly considering different types of intermediaries and reactions occurring for each particular step of the reaction). There is a *historical* question here as to the role of models, such as scale models of molecules and diagrammatic sketches/models as technologies of investigation during the process of discovery of the Krebs cycle. A good place to start such an investigation is in Frederick Holmes' (current) two-volume biography of Hans Krebs (Holmes 1991, 1993). Although I suspect that such models (together with experiment) played a role in the discovery of the Krebs cycle [e.g., Krebs writes that in the early stages of the characterization of the cycle, "we tested [for] a large number of other substances which paper chemistry suggested as intermediates." (Krebs 170, p. 155) Krebs' historical paper is full of diagrammatic chemical models describing what was known at different stages of the discovery process], this is not the place to engage in historical research. There is an important point here, though: models of all types (including mathematical models) can serve an important role in scientific discovery. Once knowledge has been established (as happened *in the past* with the Krebs cycle or even with predator-prey

Here knowledge has become "fixed"; the model is an accurate representation of a set of crucial reactions occurring in the matrix (central part) of the mitochondria. This model, in its full detail would indicate the full structure of the organic substrate intermediaries (e.g., isocitrate, succinate, fumarate, etc.), input (e.g., water) and output (e.g., carbon dioxide and electron carriers such as NADH and $FADH_2$) molecules, and, for purposes of *explanation*, to be discussed below, at least the active site, if not the full conformation, of the catalyzing enzymes for each of the 9 reaction steps of the cycle. Such a diagrammatic and narrative model would fully represent all the activity.

Now, different levels of generality of the meaning structure of the Krebs cycle model could be considered here. For example, all enzymatic structures could be left out of the diagrammatic model as is, in fact, often done.[432] Furthermore, the structural formula of the various substrates could be left out and only the molecule *names*, or even the molecule types (indexed by numbers of carbons)[433] indicated. The exact steps in which input and output molecules are involved could also be left out and, if at all, merely indicated in a table showing total numbers of input and output types of molecules. The most *general* meaning structure of the Krebs cycle might be written as:

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mathematical models), the models become a *representation* of knowledge itself rather than tools for further discovery. This is not strictly true in cases where reality is complex (as it often is) and the assumptions of the model are only partly or locally correct. In such cases, the models can serve as investigative tools for a further, more nuanced, investigation into complex "reality." Furthermore, models also serve as investigative resources when knowledge has not yet been "fixed," that is, when the relations between variables or parts of the model have not been fully established. On the role of models in chemistry see also Klein 1999 who discusses the role that symbols for chemical compounds played for stoichiometric and mechanistic purposes in early organic chemistry.

[432] See Appendix Figures 4.9 and 4.10.

[433] See Appendix Figure 4.9.

$substrate_n \rightarrow substrate_{n+x}$ {where x [1,9] and (n=1) = (n+x=10)}. [4.1]

Assumptions in this very general model include that (1) "→" indicates that one substrate is transformed to another by a catalytic enzyme and (2) the term "substrate" instead of "product" is used after the arrow because the product is a substrate for a specific and explicitly known *subsequent* reaction. Such a general model is very distant indeed from the empirical content of the actual activity, and would be a model for any circular 9-step reaction series. Note, then, the trade-off in generality and empirical adequacy/causal explanatory power here.

This trade-off points to how explanations are produced through diagrammatic models in biochemistry, a compositional biology. It is through the presentation of (1) the part articulation of the substrate molecules, with their various functional groups, (2) the part articulation of the reactive enzymes, together with an understanding of (3) the various rules of interaction (including "attacks" and "removals" of electrons, bonds, and, therefore, of functional groups) that explanations of the various reactions (9 types in the Krebs cycle) are produced.

Consider Stryer's narrative model of the formation of L-malate from fumarate (Appendix Figure 4.8; step 8 in Appendix Figure 4.10):

> The next step in the cycle is the hydration of fumarate to form L-Malate. *Fumarase* catalyzes a stereospecific *trans* addition of H and OH, as shown by deuterium-labeling studies. The OH group adds to only one side of the double bond of fumarate; hence, only the L-isomer of malate is formed.[434]

[434] Stryer 1988, p. 377.

Note that his explanation is in terms of movement of particular groups (e.g., hydrogen and hydroxide) into specific parts of the molecule. He does not detail the actual mechanism of this in terms of the active sites and energetics of fumarase, which could also be done; but he does describe an experiment (deuterium labeling) that allowed for determination of the exact method of substrate transformation. He complements his explanation with a diagram showing the two molecules.[435]

This explanation is an example of capacity talk that refers to parts of a system[436]. The fumarase enzyme has the capacity to *convert one molecule into another through hydrolysis and precise placement of H and OH groups*. This is a Cummins' function (see Chapter 5) in that an *overall system behavior* (i.e., Krebs cycle and its production of electron carriers which later relinquish their electrons for the production of ATP in the reactions of oxidative phosphorylation, also known as the electron transport chain) is explained in terms of *compositional capacities of the parts* (e.g., fumarase as the catalyst of fumarate to L-malate). The basis of this explanation is provided in the diagrammatic and narrative models representing the Krebs cycle. Note that such explanations cannot be gleaned from a *too* general presentation of the model.[437] Hence we see the trade-off between generality and explanatory power of this case of diagrammatic and narrative models.

In this meaning structure, particularly at concrete levels of generality, there are criteria of self-consistency. For example, there are very few, often only one, specific reactions that will convert a specific substrate into a specific

[435] See Appendix Figure 4.8.
[436] I will explore this in detail in Chapter 5.
[437] E.g., Appendix Figure 4.9.

product. During the process of discovery, however, there may very well have been various candidate (closely related) molecules and reactions considered for each step of the cycle. Confirmation of these various contrastive models then depended on experimental results, such as characterization of substrates involved and the capacities of enzymes required. Eventually the discoverers settled on a single model of the Krebs cycle, replete with criteria of self-consistency (e.g., particular reactions require particular enzymes, input in number and type of atoms in each reaction must *equal* output in number and type of atoms).

Another property of models is that there are criteria of allowable "hook-up" with other models. For example, consider the criteria of alternative parameterizations for the Price equation[438] and rules for relating selectionist with neutralist models of evolution – these are criteria of model diversification[439] via *abstraction relations*. In the case of narrative and diagrammatic models of objects and processes in compositional systems, the criteria for hook-up refer to *literal* hook-up and do not proceed via abstraction relations. Here the expansion occurs through explicit addition of models of other reactions and reaction series that complement the focal model. For example, a model of the biosynthesis of cholesterol can be added to a model of the Krebs cycle in that they both have acetyl coenzyme A in common. This model expansion is indicated in Appendix Figure 4.11, which also shows "diagrammatically" about 500 common and interrelated reactions in a cell. Note that this figure can be decomposed into reaction parts; an "articulation of parts" mechanistic explanation of the different

[438] Kerr and Godfrey-Smith 2002a, 2002b.
[439] I will explore this term in great detail in Chapter 5.

nodes can be provided by accounting for the previous nodes and the reaction type connecting the two nodes. Reaction model partitioning and articulation is clear in this diagrammatic model.

Let us turn to a different model, or, rather a modeling technique. There is an interesting case of the uses and extent of model *manipulation* in biochemistry, or rather, organic chemistry, which is, arguably a compositional science (perhaps not quite a compositional *biology*). E.J. Corey, a Harvard organic chemist who won the Nobel Prize in 1991, developed, in detail, the practice of "retrosynthetic analysis." Before him:

> most syntheses were developed by selecting an appropriate starting material, after a trial and error search for commercially available compounds having a structural resemblance to the target of synthesis. Suitable reactions were then sought for elaboration of the chosen starting material to the desired product. Synthetic planning in most instances was strongly dependent on an assumed starting point.[440]

The reigning methodology was trial and error through a series of reactions that eventually provided the desired end product.

Corey's insight, simple as it may sound, was to *start* with a chemically accurate model, either physical or diagrammatic, of the *end product* and, using chemical models, subject it

> to a deconstruction process which corresponds to the reverse of a synthetic reaction, *so as to convert that target structure [end product] to simpler precursor structures, without any assumptions with regard to starting materials*. Each of the precursors so generated is then examined in the same way, and the process is repeated [stepwise backwards] until simple or commercially available structures are arrived at.[441]

[440] Corey 1991, p. 456.
[441] Ibid.

This "deconstruction process" was *not* carried out in experiments, but, rather, detailed knowledge of organic chemistry was used to infer, using physical or diagrammatic models, how particular kinds of reactions would produce end products from initial reagents. One "attractive" pathway at a time is "selected and validated by experimental execution."[442] (See Appendix Figure 4.12.) Of course, computers were eventually used (starting in the late 1960s) to develop many possible pathways and to choose the most attractive ones. Present in the computer software is

> The chemical knowledge base, written so as to be intelligible to a practicing chemist, [and] contain[ing] all the types of information required for generation and evaluation of retrosynthetic changes, for example, data on individual transforms and their mechanisms, scope, and limitations.[443]

Corey's methodology is a clear case of the uses and prevalence of model manipulation directing empirical research in a field that is focused on parts.

Let me now compare chemical to mathematical theory. Chemical theory can provide the basis for significant material model manipulability independently of (continuous) empirical verification, either through the use of physical chemical models or diagrammatic chemical models (as we saw in the case of Corey's work). For both kinds of theory, we can manipulate symbols or bits of matter in ways that are informative and highly predictive. Yet there are at least two important differences between the two kinds of theory, the role of

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[442] Ibid.
[443] Corey 1991, p. 458. Earlier in the paper (p. 457), Corey had listed six "major types of strategies which are of value in retrosynthetic analysis." They include: (1) structure-goal strategies, which focus on the structure of the reagent and investigate whether one can get from the reagent to the product – this is the "actual" chemical reaction direction!, and (2) functional group-based strategies, which focus on the effects of functional group removal or interchange.

empirical generalizations and the existence of exceptions, both of which I will now examine.

Fundamentally, chemical theory seems to be an elaborate collection of empirical generalizations, much of which, admittedly, does have an internal consistency, given background knowledge. There is an "internal" logic, but it has been inferred from data. *Today's chemical theory, on which we can base theoretical manipulation, is the product of yesterday's empirical manipulation*. This is very different from the mathematical theory of, say, population genetics, where a few axioms together with the rules of, for example, algebra, matrix algebra, differential equations, etc. can be used to consistently model the results of the behavior of gene frequency change under the existence of particular evolutionary forces at particular magnitudes. Here theory is not *inferred* or built up empirically, although it may (and should!) be *verified* empirically. To exaggerate: chemical theory is the product of empirical manipulation, formal mathematical theory is the product of theoretical manipulation. This gets to deep issues surrounding the *a priori*, necessary, and definitional aspects of mathematics and the relations between mathematics and science. Further exploration of these issues would be fruitful.

Related to the issue of empirical generalizations (chemical theory) versus "definitional" postulation (mathematical theory) is the idea of exceptions in chemical theory. Clearly the exceptions are many, as any student just beginning to learn chemistry (especially organic!) will complain. There are, *sensu stricto*, no exceptions in mathematics. Furthermore, empirical exceptions to a mathematical functional relationship can be attributed to lack of knowledge of all the relevant factors (i.e., *ceteris paribus* condition fails). Mathematical laws and models, if

postulated consistently and correctly, cannot have empirical exceptions. They can just fail to apply.

Thus, while chemical theory allows for *very large* amounts of independent theoretical manipulation of chemical meaning structures, there seems to be a significant difference in the empirical vs. (purely) theoretical aspects of (1) theory, (2) models, and (3) modeling in formal and compositional biology. Models and the theoretical activity of modeling is much more "*a priori*" in formal biology and empirical/material in compositional biology, even though we should not discount the importance of theoretical activity in discovering new knowledge in (especially some fields of) compositional biology nor should we ignore the theoretical *content* of the components of the meaning structures of compositional biology.

4.4 On Models as Meaning Structures and Model-Based Explanation in Compositional Biology

In Chapter 1, I noted that manipulation in compositional biology occurred primarily in the empirical realm of the concrete systems under study, through activities such as experimentation and observation. This I contrasted with formal biology where manipulation occurs primarily in its mathematical models. I believe this to be a fairly accurate depiction, within limits, of the *activity* that occurs in each of the two respective kinds of biology.

But we should *not* take this to suggest that the two kinds of biology are, respectively, observational and theoretical. Theoretical modeling, through the

use of remnant, scale, diagrammatic or narrative model types, is a crucial aspect of compositional biology. Such models are used to abstractly represent the compositional structure and process-function (capacities) of, for example, organisms and their hierarchically-organized component parts. These models present depictions of systems, including the structures and capacities of their constituent parts, in order to both (1) suggest loci for further experimentation and observation (including inter-taxa comparisons) as well as (2) make theoretical claims, and provide *causal explanations*, regarding the general characterizations of the structures and capacities of parts. Put differently, without these models, a clear theoretical representation of the systems and activities under study by large areas of biological science would simply not be possible.

It might be argued by some that the models prevalent in compositional biology are not (sufficiently) theoretical because (1) they cannot be formulated on the basis of simple and few laws and axioms of any sort, or because (2) clear predictions and "work" cannot arise from them simply by manipulating them independently of the concrete systems of which they are meant to be representations.[444] With respect to the first criticism, it is clear that a number of models in particularly mathematical ecology are also not theoretical in that sense – simulations are not always based on simple and few laws and axioms.[445] Of course, it could be further claimed that simulations are not theoretical and do not match the "gold-standard" form of analysis of law-like population genetics. But I

[444] Michael Weisberg, a recent PhD graduate from the Stanford Philosophy department who wrote his dissertation on model-building in ecology, has suggested the latter criticism to me.
[445] For a philosophical analysis of simulations in biology see, for example, Dietrich 1996.

do think that a number of practitioners of formal biology would agree that simulations count as theoretical and as acceptable model-types of formal biology.

More importantly, while it is true that the models of compositional biology tend to not be mathematical, they are often based on some kinds of simple and few theoretical principles. For example, Davidson and co-worker's diagrams of genomic regulatory systems follow the conventions of flow charts, which include drawing objects such as boxes and arrows indicating (types of) activity. Rules of geometry, and even aesthetic rules pertinent to ease of cognitive presentation[446], also govern such depictions. Such conventions and rules are, or could be presented, axiomatic(ally). They are certainly *theoretical* principles that govern the mode of representation and that bring representational order to the chaos of mechanistic material causality. Mathematical representations of the sort held dear by mathematical evolutionary genetics and theoretical population ecology are merely one form of theoretical order-imposition; multiple other modes of enforcing order, stemming from different kinds of "axiomatic" rules, in different media of model presentation, exist. Different theoretical perspectives, particularly ones very different in kind, can include assumptions regarding different modes of representation – these are theoretical assumptions guiding the activity of model-building.

It is true that many of the models of compositional biology are not, by themselves, subject to significant and useful independent theoretical manipulation – a case like Corey's retrosynthetic analysis, or chemical theory more generally, is the exception rather than the rule. Furthermore, we have seen how chemical and mathematical theory differ. Constant referral to the concrete

[446] Cf. Tufte 1983.

system is necessary in generating most of the meaning structure types of compositional biology. But this does not make the models *less* theoretical (although, as we shall see in Chapter 5, model development, that is, theoretical explanation, requires continuous confirmation).

The models still contain much theoretical *content* in that they contain assumptions about *what* the models are supposed to represent and *how* the background concepts are supposed to be defined. For example, any diagrammatic model portraying the structure or development of inter-taxon parts that are supposed to be homologous is, whether the claim is accurate or not, an extremely theoretically-rich model. This is because the very concept of homology is a significantly theory-laden one for which there is a large amount of discussion and disagreement.[447] A model that makes robust claims about homology uses a stabilized and robust notion of homology (although not vice-versa in the case for which the model is empirically inaccurate – i.e., false). Furthermore, the model summarizes, in an abstract fashion, the shape, relations, and capacities of the parts it portrays. This is a theoretical representation of the system under study.

With respect to theoretical *activity*, some manipulation of the model is possible independently of empirical manipulation of the concrete system[448] and this often suggests numerous places in which a scientist could further investigate the concrete system. Models and modeling point toward new experiments that could be performed (e.g., is there a causal relation of any sort between these two

[447] Cf. Bolker and Raff 1996; Brigandt 2002; Raff 1996; Roth 1991, 1994; Wagner 1989, 1994, 1995, 1996; Wray 1999.
[448] Recall the distinction made above in footnote 385 between (1) material models as theoretical entities in that they represent a concrete system and (2) the materiality of the concrete system itself.

genes for which there is no line in the model? Is there a relation of neural activation between this and that muscle group during the process of mastication?), and it also intimates at new ("mere") observations that could be made (e.g., how would my model of the evolution of function of this set of parts in related taxa look if I added these characters (of the parts) to my character matrix?).

Thus modeling is a theoretical activity that furthers scientific knowledge. It may be true that such activity is, in the models of compositional biology, closely tied to, and constantly constrained by, empirical observations, but the formalistic distinction between theory/observation statements does not and should not map onto a more robust distinction between theoretical and empirical activity. That is, the fact that theoretical activity relies on empirical observations does not make the activity *less* theoretical, as we shall also see in Chapter 5. It is clearly theoretical in the sense that it generalizes, organizes, represents, and provides abstract suggestions for intervention in, concrete systems.

In this chapter, I have shown how model-types such as scale models, remnant models, diagrams, narratives—linguistic and "logical"—are employed in compositional biology. The point is not that these models are absent from formal biology, but they play a prevalent and powerful role in compositional biology precisely because they can accurately represent the compositional *structure* and *process-function* (causal capacity) of the components, at various integrated levels (determined by the theoretical perspective employed), of hierarchical biological systems. They can be used to precisely depict the causal capacities of the parts of the system, as I will also discuss in Chapter 5 (they thus serve crucial roles in causal explanation in compositional biology).

But while mentioning these model-types, and showing how they can be meaning structures at different levels of abstraction (e.g., tetrapod feeding and genomic regulatory systems), I have not provided an in-depth analysis of each model-type. I definitely think that such an analysis is important. There is some excellent literature on remnant models (Griesemer), diagrams[449], and narratives[450], independently of the context of the framework I have been developing in this dissertation. (There seems to be scant literature on the role of scale models.) I believe that further analysis of these kinds of models in the context of compositional biology would be very fruitful and future work will address the nature and role of each of these model-types in the context of compositional biology.

These model-types (e.g., diagrams and narratives) can serve as fairly concrete meaning structures in which to present more general meaning structures, or frameworks, of, for example, tetrapod feeding and genomic regulatory systems. The general meaning structure is concretized, in Bramble and Wake's model, for example, in diagrammatic and narrative meaning structures specifying a particular set of behaviors common to many taxa. The sets of tetrapod behaviors are a "heuristic model that serves as a hypothesis for testing rather than a fixed reality of feeding kinematics in tetrapods."[451] Note, though, that the general model can, and has, survived testing which showed its *inadequacy* for particular taxa. It continues to guide research and is, therefore, a mediating model that further advances theory and experiment/data.

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[449] See, for example, Goodman 1976, 1978; Lopes 1996.
[450] See, for example, Danto 1985; Griesemer 1996; Hull 1975, 1981, 1992; Richards 1981, 1992.
[451] Sckwenk 2000b, p. 49.

In this chapter I hope to have shown that a *general* picture of the nature and role of models and modeling in scientific practice can be gleaned using the semantic view and the "mediating model" view, complemented with Cartwright's and Friedman's physics-based analysis of abstraction as well as Griesemer's and Downes' analysis of non-standard models, as philosophical input. The picture of models articulated by these philosophers was initially designed for the mathematical models of physics and formal biology. But, as I hope to have shown, the general properties that I have articulated also definitely apply to the models prevalent in compositional biology.

I have argued that models have four general properties: (1) models are meaning structures of various types and at various levels of abstraction, (2) models can be "hooked up" with other models, subject to the background protocols and assumptions of the theoretical perspectives guiding the articulation of models, (3) models are always subject to trade-offs among pragmatic desiderata – for example a general model is rarely a realistic one, and (4) models are used in a variety of scientific activities, including guiding further modeling and empirical activity, as well as providing explanations.

In the following chapter I will turn to how models serve a crucial role in the activity of explanation. There are crucial conceptual relationships between models, causal capacities, and explanation in both formal and compositional biology leading to two different *pictures* of explanation in the two kinds of biology.

Chapter 5: Explanation in Formal and Compositional Biology

5.1 Framing and Goals

In this dissertation, I have explored a multitude of differences between two kinds of theorizing in biology, formal and compositional biology. In Chapter 1, I alluded to a fifth distinction between the two kinds of biology, differences in the goals of explanation. In Chapter 3, I discussed differences in explanatory resources between the two kinds of theoretical perspectives analyzed therein, levels of selection theory and evolutionary developmental biology. One of these resources is precisely the goal or aim of an explanation. In both chapters, I mentioned that I would address this issue head on in Chapter 5. Furthermore, in Chapter 4, I explored the general properties of models and investigated a few case studies of meaning structures (models) that *present* them, in compositional biology. Thus, there is an outstanding issue of *how these models assist scientific explanations*. In this chapter, I will therefore discuss the differences in aim of explanations in the two kinds of biology as well as the way that models particular to each kind of biology assist in achieving these diverse aims.

In order to do this, I must place my analysis in the context of the philosophy of science literature on explanation. I will show how explanations, both causal and theoretical, are employed in the two kinds of biology. The formalistic (i.e., statistical and mathematical) accounts of causal explanation

present in philosophy of science, such as those of Cartwright, Glymour, and Salmon, are relevant to formal biology. Part-based versions of causal explanations, such as those of Cummins, Kauffman, and Wimsatt are pertinent to compositional biology. The contrast in patterns and goals of these two overall projects regarding explanation are striking, as I will show. Furthermore, I also want to show that theoretical explanation differs remarkably between the two kinds of biology in that law and model articulation occurs neatly and, to a large extent, independently of empirical activity in formal biology, whereas modeling and empirical activity are intricately tied in compositional biology. This is also why theoretical unification, as a form of theoretical explanation most ably explicated by Friedman, pertains only to formal biology. Thus, the pictures, figuratively and literally, of explanation in the two forms of biology are quite distinct. In order to draw, conceptually and actually, these pictures, I must also bring in considerations regarding confirmation.

We can thus get a much better handle on the methods and goals of different accounts of explanation in the philosophy of science literature (including the models and model-types that they favor) by comparing the two *families* of accounts *within* my framework contrasting formal and compositional biology. Conversely, we can, not surprisingly, understand the differences between formal and compositional biology much better if we analyze their adoption of different pictures of explanation. Table 7. (5.1) provides the framing for my analysis.

In formulating my argument, I show that there is a *close* relationship between the view of explanation endorsed and the model-types employed. Models can be taken to represent formal dependencies among mathematically-

defined properties of simple kinds[452] of objects (formal biology) or material-mechanistic interactions among kinds of parts of a hierarchical and integrated complex system (compositional biology). Models can also be taken to represent the causal capacities of the objects, sometimes parts, under consideration by the two types of biology (causal explanation) or to represent the theoretical inter-relation among models at different levels of abstraction (theoretical explanation).

I will proceed by first motivating *what* an explanation even is and how one can even distinguish causal from theoretical explanation. Of course this is a deep question, but by analyzing van Fraassen's views on explanation, and different interpretations of them, I will motivate my position on this. I will then continue by analyzing formal and compositional biology, respectively, vis-à-vis their commitments to causal and theoretical explanation. This I do by explicating some *case analyses* for each area: Cartwright, Cummins, and Friedman. I also assess Kitcher's view on theoretical unification. I could also have evaluated the views of other philosophers on these issues, but I choose these five because I believe that their views are clear and paradigmatic instances of the explanatory patterns that I wish to explore.

5.2 Does Van Fraassen Have an Anti-Metaphysical Deflationary, or an Empiricist Positive, Account of Explanation?

One influential account of scientific explanation is van Fraassen's pragmatic version. Van Fraassen is an anti-realist who believes that science

[452] In the sense of Chapter 1.

produces "empirically adequate" theories with no metaphysical realist content about unobservables. His view, as developed in *The Scientific Image*, is one he calls "constructive empiricism" which foregoes hypotheses about "truth" and the "real" state of the world. The following paragraph provides a pithy summary of his view:

> …[W]e can distinguish between two epistemic attitudes we can take up toward a theory. We can assert it to be true (i.e. to have a model which is a faithful replica, in all detail, of our world), and call for belief; or we can simply assert its empirical adequacy, calling for acceptance as such. In either case we stick our necks out; empirical adequacy goes far beyond what we can know at any given time. (All the results of measurement are not in; they will never all be in; and in any case, we won't measure everything that can be measured.) Nevertheless there is a difference: the assertion of empirical adequacy is a great deal weaker than the assertion of truth, and the restraint to acceptance delivers us from metaphysics.[453]

I interpret his anti-realism to stem, at least partially, from the myriad incompatible realist interpretations that can be and are produced of quantum mechanical phenomena.[454] In quantum mechanics, his specialty, unobservable or theoretical entities are ubiquitous – almost, by definition, so. It is no surprise that the study of such a field would produce an anti-realist philosopher.

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[453] Van Fraassen 1980, pp. 68-69. This position mirrors the operationalist position of Duhem in his *The Aim and Structure of Physical Theories*, which is both anti-model (in the sense of mechanical material models such as those developed by Kelvin of the aether; see Part I, Ch. 4) and anti-metaphysics (metaphysics should be, according to Duhem, but contra Quine, clearly distinguished from science; see Part I, Ch.1). For Duhem, the aim of science was to produce symbolic, formal laws, which, he argued, were approximate and *ceteris paribus* laws (see Part II, Ch. 5). Van Fraassen shares Duhem's anti-metaphysical stance, but emphasizes symbolic and formal models rather than laws (recall, as I argued in the introduction, that I do not think that the two are that different, provided we interpret the notion of "law" correctly). Van Fraassen, like Cartwright, is highly critical of the validity of laws of any sorts and, more so than Cartwright, emphasizes the role of models in scientific work. At any rate, there is much in common, albeit in different ways, between Duhem and both van Fraassen and Cartwright.

[454] See van Fraassen 1989.

The issue that I want to address here is the nature of van Fraassen's view on explanation, including its relationship to his anti-realism. There are two general interpretations of his views. The first one asserts that *because* of his anti-realism, he adopts a very deflationary account of explanation, claiming that it is not a proper part of scientific investigation or that, at best, it amounts merely to empirical adequacy.[455] The second one argues that he does have a substantive version of explanation that is relativistic and pragmatic, and that it is *simply* "a three-term relation between theory, fact, and context" as van Fraassen himself claims.[456] Commentators partial to the first interpretation, think that van Fraassen does not give "explanatory power" sufficient importance, whereas proponents of the second interpretation claim that he gives it the appropriate amount of importance. I do not wish to adjudicate between the two views in so far as determining van Fraassen's actual view; but I do want to state that I am more sympathetic to the concerns underlying the first interpretation. Explanatory patterns are importantly different in the two kinds of biology, and I think that this is due to *more* than empirical adequacy *within* an explanatory context. Let us turn to a brief analysis of the two interpretations.

For van Fraassen, explanations are the attainment of empirical adequacy in an answer to a why-question[457]. A typical request at an explanation is a why-

[455] For example, see Kitcher 1991 (1981), p. 330-332; Strevens 2004 (in press). So does Cartwright 1983, implicitly, in her arguments against van Fraassen's criticisms of inference to the best explanation. Cartwright sees a deep link between van Fraassen's anti-realism and his skepticism regarding this form of explanation. What she believes about van Fraassen's *general* view on explanation remains, however, indeterminate.
[456] Elisabeth Lloyd clarified van Fraassen's views to me. The quote from van Fraassen is in 1980, p. 156.
[457] This is also the view of Hempel and Oppenheim in their classical and much-critiqued essay. They wrote: "To explain the phenomena in the world of our

question (e.g., "Why did allele X increase in frequency in the population?"). Answers to such a question *are* the explanation. Note that there can be different contrast classes of answers: (1) because the allele produced functional rather than dysfunctional hemoglobin (a kind of "material" contrast class), or (2) because the allele had a higher fitness than alternative alleles (a kind of "formal" contrast class). Or, to use an example from van Fraassen, "Why did Adam eat the apple?" is a question demanding an answer *relative* to three different contrast classes, each of which defines different sets of relevant answers: (1) Adam as opposed to somebody else, (2) eating as opposed to some other activity, (3) an apple as opposed to another object.[458] An explanation, then, is an empirically adequate answer, within the causal framework and set of accepted contrast classes and relevance conditions of a scientific theory, for a why-question.[459]

Empirical adequacy is itself determined by the "matching" (a concept explored in more detail below) between theoretical model and data model. But this matching occurs, importantly, within the context of the scientific theory. Thus, explanation

> is a three-term relation between theory, fact, and context…. So to say that a given theory can be used to explain a certain fact, is always elliptic for: there is a proposition which is a telling answer, relative to this theory, to the request for information about certain facts (those counted as relevant for *this* question) that bears on a

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experience, to answer the question "why?" rather than only the question "what?" is one of the foremost objectives of all rational inquiry; and especially, scientific research in its various branches strives to go beyond a mere description of its subject matter by providing an explanation of the phenomena it investigates." (1948, p. 135)

[458] Van Fraassen 1980, p. 127. See also Garfinkel 1981.

[459] This is also the account of scientific explanation that Lloyd adopts in her work – see especially Lloyd 1988, 2000a. It remains a bit unclear how to relate the notions of contrast classes and relevance relations to the state space articulation of models.

> comparison between this fact which is the case, and certain (contextually specified) alternatives which are not the case.[460]

It would seem, then, that explanation is simply empirical adequacy within an explanatory context. It is the import of this claim about which commentators disagree.

One camp (the "deflationary" one) notes that this is a very weak view of explanation. They remind us that, for van Fraassen, using a theory to provide an answer does not imply that you believe that the theory is *true*, either in capturing laws or in capturing causes. The following paragraph describes some of the attitudes van Fraassen wants to distance himself from:

> Once you decide that explanation is something irreducible and special, the door is opened to elaboration by means of further concepts pertaining thereto, all equally irreducible and special. The premises of an explanation have to include lawlike statements; a statement is lawlike exactly if it implies some non-trivial counterfactual conditional statement; but it can do so only by asserting relationships of necessity in nature. Not all classes correspond to genuine properties; properties and propensities figure in explanation. Not everyone [especially not van Fraassen!] has joined this return to essentialism or neo-Aristotelian realism, but some eminent realists [e.g., Armstrong, Dretske, Tooley, Salmon, and, perhaps, even Cartwright] have publicly explored or advocated it.[461]

The deflationary camp argues that this thoroughly anti-metaphysical account of explanation is overly driven by worries about what sorts of information are epistemically justified in science. Put differently, van Fraassen fights "metaphysics" with "anti-metaphysics," that is, with an appeal to a highly tempered epistemology. But if we move *away from* the particular philosophical bout of realism-anti-realism and explore the nature of actual scientific theories,

[460] Van Fraassen 1980, p. 156.
[461] Van Fraassen 1980, p. 155.

the deflationists argue, we see that while van Fraassen's account of explanation as context-dependent answers to why questions is certainly a component of what scientists are doing, it also seems clear that scientific explanations are doing much *more* – they seem to have independent patterns and structures that are clearly recognizable and that provide us with particular types of *claims* that differ from other types of explanatory claims produced by other (kinds of) theories. Thus, we should judge explanations not just by their empirical adequacy, but also by the consistency and pattern of their theoretical structure and aim (e.g., are we explaining causes or theories?). They have "explanatory power" over and above empirical adequacy.

Now, the second camp, which I will call the "actualists," believe that van Fraassen does hold that explanation plays an actual and important role in science.[462] They hold that van Fraassen's view on explanation is a positive one and that it is a *sufficient* view. Van Fraassen, they say, does not deny that explanation exists, he simply holds that there is no independent virtue of "explanatory power" over and above empirical adequacy within a theoretical and pragmatic context. Actualists seem to claim that van Fraassen would allow for genuine explanatory differences between contexts, but that the power, meaning, structure, or aim of an explanation is always judged and fleshed out on the basis of empirical adequacy and not on the basis of the intrinsic nature and goal of either the theoretical structure, or the *pattern* of relation—rather than the actual empirical adequacy of a particular instance—between the theory and the data. In short, the actualist seems to believe that if models, laws, or statements are

[462] I take Elisabeth Lloyd to be a key "actualist." Note that my terms refer to the nature of the *interpretations* of van Fraassen's views, not to the role or nature such commentators believe scientific explanation *in itself* has or should have.

empirically adequate, then they could explain depending on the question. The deflationist (vis-à-vis interpreting van Fraassen's view) claim that models, laws, or statements have internal properties that give them explanatory structure and goals over and above empirical adequacy and that, therefore, van Fraassen's view on explanation is deflationary.

This is not a debate that can be further clarified, yet alone solved, here. Perhaps van Fraassen did not say enough in his 1980 book to determine which interpretation *of him* is appropriate. However, I am more sympathetic to the views *motivating* the deflationist, as opposed to the actualist, interpretation of van Fraassen. While I agree with much of van Fraassen's general framework in which context plays a central role, I do believe that we can inspect *just* the theoretical structure and aim of particular *kinds* of explanations to articulate what is special and characteristic of that form of explanation. In fact, the two kinds of biology that I am exploring, while not necessarily making claims about "reality," do use different *kinds of explanatory patterns*. That is, the different models and model-types developed by each kind of science provide the grounds for different forms of explanations that, although they can certainly be seen as answers to why-questions within a context, also have more specific properties that differentiate one explanation picture from another in important ways, independently of the empirical adequacy of particular explanations. Furthermore, given the prevalence of "observational entities" in biology (in contrast to quantum mechanics), we are even less justified in biology to discard the view that there is more to explanation than empirical adequacy within a context. And we do not need to adopt realism to adopt meaty views regarding explanation – appealing to the differences in structures and goals in biological

model-based explanatory practices, as I will show in this chapter, is sufficient. Switching metaphors, biological explanations have some metaphysical and theoretical bite, even if the jaw that does the biting is scientific *theoretical practice* rather than metaphysical *realism*. I will now turn to an analysis of the different kinds of explanation in formal and compositional biology.

5.3 Distinguishing Between Causal and Theoretical Explanation

The distinction between causal and theoretical explanation, commonly recognized by philosophers of science, is that between explaining a concrete event such as the movement of a planet around the Sun or the catalysis of some substrate to make a product *and* explaining a law or general pattern, such as Kepler's laws or the general pattern of hierarchical organization (both of these latter two can also be thought of as theoretical models).

With respect to explaining concrete cases, when we explain the particular orbit of a planet, as discussed above, we appeal to subsumption of the concrete case under some law or, more generally, the matching of some bit of a concrete system to a theoretical model (e.g., Kepler's or, more generally, Newton's laws); here we are showing that a concrete system is an instantiation, or, more generally, stands in a particular relation of similarity to the theoretical assertions produced by the law or model[463].

[463] In what follows, I will repeatedly mention "law" and "model" together. As I argue in Appendix A, the two are intimately related in formal biology. For example, laws, properly formulated as "equations of the model," can be considered to produce models, in the mathematical sense of the semantic view,

On the other hand, when we explain a particular concrete case of catalysis, there is also a sense in which we match that particular case under a more general model of that catalysis-type[464]. But this matching (subsumption) activity is not the *central* aspect of explaining a chemical reaction; the crucial aspect is that we employ compositional and functional analysis, sensu Cummins, in both the general model and manipulation of the empirical case. That is, we explain the reaction by referring to the dispositions of the different parts of the reactive system.

I will explore these two importantly distinct forms of causal explanation, pertinent respectively to the two types of biology I examine, by looking at two particular suggestions for each of the two causal explanatory patterns, Cartwright's and Cummins' analysis of causal capacities. These are *not* the only ways of explicating causal explanation in the two forms of biology (e.g., Glymour and Salmon for formal biology, Kauffman and Wimsatt for compositional biology), but despite their limitations they are good candidates for

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that produce predictions that are then compared in degree of matching to the empirical evidence. [Even an extreme syntactic view, which does have an inappropriate, by my account, version of Laws, can still be interpreted as producing a (syllogistically-driven, in this case) prediction that is then *compared* to actual data – degree of similarity between prediction and data is assessed. Thus, the semantic view's emphasis on theoretical model <-> data model matching is not unique to it. Both the syntactic view of theories and related versions of it, honest to scientific practice, such as Friedman's (but, as we shall see, not Kitcher's), also contain the idea of matching.] For further arguments regarding the deep relation between laws (properly understood) and (mathematical) models, as described by the semantic view, see Appendix A.

[464] This is something Cummins denied in 1977, but remained agnostic about in his 1983 book. In 1977 he claimed that subsumption, which, as we shall see, he called the strategy common to formal biology, "[is a word that] captures one central element in the strategy [common to sciences premised on formal laws and models] which is *absent from*, and *irrelevant to*, the other strategy I want to discuss [which is the "analytical" explanatory strategy common to compositional biology]." (1977, p. 272, emphasis mine)

understanding what causal explanation looks like in each of the two forms of biology.

The other, complementary, sort of explanation, *theoretical* explanation, is particularly clear in theoretical physics. The goal of this sort of explanation is for a more general law or model to account for more specific laws or models. How, for example, do we "explain" Kepler's laws or hierarchical organization in biology? Here we seek to use models to explain other models, rather than to explain concrete cases (the latter amounting to causal explanation).

Often theoretical explanation in formal biology is fairly clear. General models can be *diversified* (that is, in my language to be explicated below, either *elaborated* or *derived*) into more specific models using fairly precise mathematical concretization procedures (such as adding precisely-defined variables to capture more of the law-like causal structure of formal biology). In this case, the general model is involved in explaining the more specific model. Furthermore, model *unification* (a special "deductive" form of model *merging*), explained most clearly by Friedman, plays a particularly important but rare explanatory role in formal biology, for example with the Price Equation.

Theoretical explanation in compositional biology is, for a variety of reasons, fairly rare. The theoretical content of compositional biology is much less structured, in many ways, than that of formal biology. It is difficult, often impossible, to diversify general models (when they even exist) with primarily theoretical manipulation (we saw this most clearly in the case, explicated in Chapter 4, of Davidson's general "cartoon" model of *cis*-regulatory genetic elements) – empirical manipulation is crucial. Kitcher's general linguistic account of theoretical explanation via unification will also not, as I show, work for

compositional, or, even, formal, biology. Despite the frequent lack of relevance, as well as the absence, of theoretical explanation in compositional biology, there is one sense, at least, in which such explanation exists: (the model of) compositional hierarchy is explained through the theory (model) of evolution. The rarity of theoretical explanation suggests that a different picture of explanation pertains to compositional biology – one in which empirical and theoretical activity are intimately related.

The distinction between theoretical models and (sets of) concrete cases is a difficult one to make. When do (sets of) concrete cases become a model? The minute we abstract away from a single case? The minute we formalize concrete system behavior in mathematical notation? The minute we use "theoretical" or "non-observational" vocabulary to describe the concrete cases? (Is it ever possible not to use such vocabulary?)

There are no easy answers to these, and other related, questions and they have been extensively discussed in the literature by important figures such as Hempel, Sellars, van Fraassen, Friedman, and Salmon, among many others. I will not attempt the impossible task of delimiting or providing criteria for exactly what sorts of explanations count as causal and which count as theoretical. I appeal to intuitions we presumably have about which (cases of!) explanation count as causal and which as theoretical. I also hope that the discussion in this chapter makes the difference clearer. Certainly, the *pictures* of explanation that I develop for each of the two forms of biology is based on, and, in conceptual feedback fashion, help to clarify the distinction.

	Formal Biology	**Compositional Biology**
Causal Explanation	*Subsumption* under Law and Law-Based Models (E.g., Cartwright Capacities)	Compositional and Functional Analysis *Represented* by Models (E.g., Cummins Capacities)
Theoretical Explanation	Model Elaboration *Or* (rarely, but importantly) Model Unification sensu Friedman (E.g., Price Equation)	Rare by itself (*Always* related to confirmation) *Or* Important for the particular case of Hierarchical Organization being "explained by" Theory of Evolution

Table 7. (5.1) Table summarizing causal and theoretical explanatory strategies in the two kinds of biology. I will explore each cell in turn.

5.4 On Causal Capacities as a Form of Causal Explanation

I will now turn to two particular suggestions for how to assess causal explanation in science each of which is pertinent, respectively, to formal and compositional biology: Cartwright's and Cummins' view. Note that these views have limitations that I will mention (e.g., there are disanalogies between Cartwright's view of causation and exactly what the models of formal biology are trying to capture, and both Cartwright's and Cummins' views are "causally

monadic" and "causally reductionist" in a sense to be explicated at the end of this section, 5.4), but my presentation of them is intended as a way to provide *suggestive* rather than *definitive* accounts of causal explanation in the two kinds of biology.

5.4.1 An Account of Causal Explanation in Formal Biology

There are a variety of views on causal explanation, including Salmon's classic treatment in Salmon 1984. Here I will focus on Cartwright's view as I think that it is a paradigmatic case for how causal explanation can be fleshed out in formal biology. After explicating her view, I will show how it can be applied to the methodology and theoretical structure of formal biology.

For Cartwright objects have a peculiar kind of property(ies): capacities. This allows objects to engage in certain processes in particular reliable ways with other kinds of objects that also have their own capacities. She

> maintain[s] that the most general causal claims—like 'aspirins relieve headaches' or 'electromagnetic forces cause motions perpendicular to the line of action'—are best rendered as ascriptions of capacity. For example, aspirins—because of being aspirins—can cure headaches. The troublesome phrase 'because of being aspirins' is put there to indicate that the claim is meant to express a fact about properties and not about individuals: the property of being an aspirin carries with it the capacity to cure headaches.[465]

These capacities are part of the ontology of the world and underlie the causal (mathematically-presented) laws, which are phenomenological descriptions of the behavior of different kinds of objects. For Cartwright, the idea of a "capacity" is a "genuine descriptive concept[], and [is] not in any way to be reduced to more

[465] Cartwright 1989, p. 141.

primitive ideas."[466] Other concepts that fall in this category of concepts describing basic ontological and epistemic primitives are: "[interaction], enabling conditions, precipitating factors, triggers, inhibitors, preventatives, and the like." Yet, "capacity" has primacy in Cartwright's view of the world: "[The other concepts] are the kinds of concept that will have to go into a proper account of what capacities are and how they operate; that is, in some sequel to this book."[467] For Cartwright, the point of science is the search for capacities.

Formal models mediate this search. Cartwright, for example, describes the use of causal modeling and path analysis in econometrics as a case of "probabilistic measures of causality."[468] Note that these mathematical techniques are the same as those in formal biology (in the case of path analysis, they even *originated in* Wright's work on heredity[469]). The diversification and confirmation of (probabilistic and statistical) mathematical models allow us to *infer* the capacities of the objects under study. (1) Causal laws are "deduced" from probabilities (about the effect a factor has on an outcome) and (2) "causal claims are claims about capacities."[470] Given the way we measure these capacities (under experimental randomization and control[471]), we can establish the situations when the capacity will appear and not be washed out by interactive effects. A strong capacity is one that makes itself felt in all backgrounds. Such a capacity has "contextual unanimity."[472] Due to interaction (another primitive term that

[466] Cartwright 1989, p. 166.
[467] Cartwright, 1989, p. 166.
[468] Cartwright 1989, p. 148 and ff.
[469] Provine 1986; Griesemer 1991b.
[470] Cartwright 1989, pp. 148 and 142, respectively.
[471] See sections 2.4.1 "The Randomized Experiment" (pp. 62-66) and 2.4.2 "The Totally Controlled Experiment" (pp. 66-71), respectively, of Cartwright 1989.
[472] Cartwright 1989, p. 143.

Cartwright says remarkably little about in her book), contextually unanimous capacities are not themselves frequent.

Cartwright's program, then, is *to infer causes, and ultimately (general) capacities, from statistical regularities using mathematical models*. In her formal condition *CC* she defines a factor as a cause iff, by either randomizing or holding fixed *all other possible causes*, we note that the effect has a higher probability of occurring *with* the cause than *without*.[473] Of course, the idea that all other possible causes can ever be postulated or found, let alone measured, can be criticized. I think, however, that Cartwright means this to be a discussion of what we *should* strive for. Cartwright's formulation is a way to move from a Humean notion of cause as "mere regularity" to a modal claim of cause that will support a specific (and peculiar!) kind of counterfactuals. That is, with her definition, formalism, and method of calculating cause, she provides a way of abandoning *local* Humean skepticism:

> [Condition *CC*] justifies a very local kind of causal claim: if in a given test population we see the increase in probability that we are looking for, that guarantees that *C*s cause *E*s *there in that population*. But it does not tell us any more. Since it is probabilities and not mere frequencies that are involved, it is assured that the causing of *E*s by *C*s will happen regularly—but regularly in that kind of population. Who knows what happens elsewhere?[474]

Capacities thus support local counterfactual claims. They, ontologically, *underlie* causal laws and models, but they are *inferred* from such models [see Figure 2. (5.1)].

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[473] For Cartwright's formalism, see 1989 pp. 55-56. It is important to note that Cartwright claims that this is a *simplified* version of a more accurate complex condition, which, in addition, accounts for causes that "screen off" the focal cause. See Chapter 3 "Singular Causes First," especially pages 95-98.
[474] Cartwright 1989, p. 144.

Note that there is an unclear connection between her notion of capacity and her earlier notion, in the 1983 book, of phenomenological and fundamental (that is, abstract) laws. The search for "causes" is common to, and central in, both works. However, discussions of explanation are present in the earlier book, but absent in the later one. In the earlier book she praises explaining by reference to causes: "Although philosophers generally believe in laws and deny causes, explanatory practice in physics is just the reverse."[475] It is unclear why she dropped discussions of (causal) *explanation* in the later book. It is also unclear what the relation is between causal laws, grounded by capacities (in 1989), and phenomenological laws (in 1983).

But there are hints about how the two books tie together. My argument is that the first book is a vindication of the study of causal laws and causal explanations, which are best described through phenomenological laws: "The causal story uses highly specific phenomenological laws which tell what happens in concrete situations."[476] The second book is then an attempt to account for what *grounds* those causal laws. In 1983 she writes,

> The emphasis on getting the causal story right is new for philosophers of science; and our old theories of explanation[477] are not well-adapted to the job. We need a theory of explanation which shows the relationship between causal processes and the fundamental laws we use to study them, and neither my simulacrum account [of explanation] nor the traditional covering-law account [of explanation] are of much help.[478]

This "theory of [causal] explanation" she searches for in 1983 is, I suggest, her account of causal capacities developed in her 1989 book. Such capacities, as we

[475] Cartwright 1983, p. 86.
[476] Cartwright 1983, p. 11.
[477] Based on subsumption under laws, e.g., Friedman's "theoretical explanation," to be explored below.
[478] Cartwright 1983, p. 162.

have seen, are developed through the use of *mathematical models* (including regression and variance analyses), or what she, in 1989, calls causal models: "given the kinds of very strong assumptions that go into causal models, it is possible to extract causal information from statistics."[479] As I shall explore below, the more concrete models of quantitative and population genetics, as well as those of theoretical mathematical ecology, can also be interpreted as causal models replete with capacities of kinds of objects such as genes, organisms, and populations.

There is a last piece to the puzzle of how the two books relate: what of the relation between causal models (using causal laws grounded in capacities) and phenomenological laws? As we saw, in 1983 (footnote 476 above) she seems to believe in a direct link between the two. Furthermore, due to her skepticism about the explanatory status of fundamental laws for concrete situations, which is captured in the idea of the trade-off between generality and (causal) explanation discussed in my Chapter 4, we already know that fundamental laws are not going to be much use in, or be descriptions for, causal models. Given her 1983 skepticism of the (causal) explanatory power[480] of fundamental laws and her

[479] Cartwright 1989, p. 13.

[480] It is important to note that Cartwright is ambiguous in her use of "explanatory power." Sometimes she uses it to refer to *theoretical* explanation as in her essay "The Truth Doesn't Explain Much," where unifying and organizing fundamental laws are taken to explain more concrete laws, but are, strictly speaking false. At other times, she takes it to mean *causal* explanation as when she writes: "Although philosophers generally believe in laws and deny causes, explanatory practice in physics is just the reverse." (1983, p. 86) I believe that Cartwright's ambiguity is mitigated by the fact that she is at least clear in *each* use of "explanatory power" and "explanation." Given her *own* focus on causal explanation, unless otherwise stated, *I will use "explanatory power" and "explanation" to refer to causal explanation*. I can, therefore, legitimately contrast it with generality and unification, which are endemic to theoretical explanation. I thank Elisabeth Lloyd for discussion on this point.

relating of causal models and phenomenological laws, one is surprised to find that in 1989 she claims that the phenomenological laws attached to concrete representations of a laser[481], both in the form of a diagram and in the form of "a highly complex" phenomenological law ("equation") involve "*no causality*."[482]

What is the solution to this inconsistency between the two books? The purity of the link between causal models and phenomenological laws is already suspect in 1983. There is an inconsistency: in motivating her simulacrum account of explanation, she makes a distinction between "phenomenological" terms in laws and "a more detailed 'causal' construction."[483] So there is actually already a tension in Cartwright's 1983 book: causal models rely on phenomenological laws in their articulation and presentation (p. 11), but there is also something deeper or different about causal explanation (pp. 225-226). The relation between phenomenological laws and causal explanation thus remains opaque. Despite this inconsistency, which spills over into her wholesale ignoring of phenomenological laws in 1989, it is clear that, at bottom, Cartwright wants to get at causal explanation through causal modeling of causal capacities.

Let us now turn to how compatible this account of capacities and causal modeling is with the practice of modeling in formal biology. Cartwright's account of capacities is meant to be a *general* account of capacities, but it is undoubtedly true that it applies best to those sciences that *measure*, and *infer*, capacities in a mathematical manner using probabilistic effects of simple kinds of objects in a rich mathematical theoretical framework with relatively few and

[481] Cartwright 1989, pp. 224-230. Page 225, to the best of my knowledge, is the only place in this book that she mentions phenomenological laws.
[482] Cartwright 1989, pp. 225-226, emphasis mine.
[483] Cartwright 1983, p. 152.

mathematically well-articulated forces, expressed in the form of laws. Cartwright's account of capacities does *not* suit compositional biology well since assessment of causal capacities in terms of probabilities to change certain outcomes is not even *an aspect* of explanation there. Furthermore, Cartwright does not in any way appeal to the importance of cause in a hierarchically-structured system. Hence, we can rule out the applicability of her account to compositional biology, and hence the complete *generality* of her account.

In the models of formal biology capacities are inferred, in a justified manner, when conditions of the model are met, and its predictions confirmed. Consider a model in which the presence of a particular object (allele D) will increase the probability of survival, in a particular amount, given particular background conditions (e.g., randomization or fixation of other genes which interact epistatically with D; assumptions of constant environmental conditions). If this model is confirmed, we will have captured one capacity of allele D (i.e., its fitness effect on the organism). Given the probabilistic interpretation(s) of fitness (and even mutation and genetic drift), the mathematical structures of evolutionary genetics are compatible with Cartwright's probabilistic framework. In Cartwright's framework, a capacity is expressed in mathematical terms of *increasing the probability of an event occurring*, such as the capacity of a gene type to affect individual survival. Other capacities include the mutation rate of a gene type, which measures its proclivity to change during a particular amount of time (or, operationally, out of a particular sampling population).

When the model proposes the capacity, and when the model is confirmed, then the causal capacity serves an explanatory role, in the causal sense, in the dynamics of the theoretical model of formal biology and in that bit of the

concrete system. That is, the capacity(ies) of the referent kinds of objects provide(s) *an account* of why the objects in the model behave the way that they do. Note that this manner of speaking could be interpreted as having metaphysical realist import[484], but I prefer to think of it as a legitimate way to understand the theoretical structure, and theoretical claims, of formal biology. Their explanations do aim for something more than just empirical adequacy –

[484] Cartwright seems to be a realist, both in her own right and certainly as compared to van Fraassen, who is explicitly an anti-realist. Although she distances herself from either a defense or an attack of scientific realism (1989, p. 162) and although she endorses a "radical empiricism" in that every "cause and capacity" has to be measured and tested (1989, p. 167), she also notes that "capacities are much like essences. If you are committed to the assumption that all the internal properties of electrons are essential, this makes science a lot easier for you. You can measure the charge or mass on one, and you know it on all the others." (1989, p. 146). [Note that this is a version of my claim of the *equivalence* of the simple kinds of objects of formal biology, which all have the same *kinds* of properties, such as fitness values and mutation rates of mathematical evolutionary genes (though, unlike the simple kinds of objects of physics, the actual values are not equivalent).] Furthermore, she claims that we should "take capacities seriously as things in the world, and not just particularly strong modalities, [and] they should remain intact from one kind of situation to another. But that does not mean that there can be no exceptions; it means that any exception requires a reason [for example, the existence of interaction between capacities]." (1989, p. 162) Now, determining the *exact* extent and interpretation of Cartwright's realism is distracting for my purposes. I do think that Cartwright's formalized notion of capacity nicely captures what formal biologists do when they elaborate mathematically-idealized models and attempt to capture and explain the causal structure of the world *as they see it*. Her analysis also suggests that scientists attempt to do more in their explanations than merely have their models be empirically adequate within a context (van Fraassen's account still remains an important *component* of an analysis of scientific explanation). Ultimately, all I want to take away from Cartwright's, van Fraassen's, Cummins', and Friedman's distinct views on explanation is a set of distinct conceptual resources to describe the varying practices of causal and theoretical explanation in the two different kinds of biology explored in my dissertation. I remain agnostic about the merits or demerits of particular ontological attitudes that either may be, or are, correlated with these views, for advocacy of any particular ontological attitude on my part would distract from my analysis of the *practice* of providing scientific explanations in very different contexts.

they want to understand what the mathematically idealized objects and processes of their theories *do*.

Although the matching between Cartwright's framework and other formal biological sciences, such as theoretical mathematical ecology, is less straightforward, in those sciences particular objects types, such as individual organisms do also have *particular statistical capacities* (e.g., average number of offspring, average lifespan, impact on resources), which, for particular conditions, affect the probability of certain outcomes (e.g., the total population size having a certain value). That is, even in the models of ecology, individual organisms have capacities that will affect the properties of the effect being measured (i.e., properties of the population).

Cartwright's capacity talk can therefore also be read, without too much difficulty, into the mathematical idealized models of ecology, where "functional"[485] relations between (1) independent and (2) dependent variables are sought. The "effect" can be interpreted as a particular value for a particular dependent variable and then the value(s) of the independent variables, represented as properties of the cause (e.g., average lifespan of an individual organism), are the ones that affect the probability of that particular effect (e.g., population size) occurring. The mathematical models of both mathematical evolutionary genetics and theoretical mathematical ecology are built from theoretical resources regarding the mathematical idealization of the *causes* and *objects* of the theory. The causal structure of the theory is contained in mathematical formalizations from which the models are built. It is these

[485] In the mathematical sense.

idealizations that can be matched with Cartwright's formulation of causal capacities. These causal capacities are *explanatory* of the outcomes of the model.

There are some imperfections in the comparison that I want to make. Although the formal models of population genetics do try to get at the causal structure of the concrete system under study, they are not *explicitly* concerned with causes, and formulations such as *CC*, in the way that Cartwright is. Their models can be reinterpreted and reconstructed, to a large extent, in this way, but doing this would, admittedly, take some work. Furthermore, as we shall see in Appendix A, sometimes the models of formal biology cannot *actually* give us causal information since sufficient empirical information is sometimes simply lacking – the parameters of a certain model cannot be filled in and, therefore, adjudicating between various contrasting models cannot be done (Cartwright too admits that limited data is often a problem in determining causes). In those cases, the correct causal model cannot be confirmed. Also, although formal biologists do want to get at causal structure, it is unclear whether they would accept and adopt Cartwright's fairly strong (and unique) version of causal structure in terms of causal capacities. They might prefer to adopt another set of concepts regarding causation that would also be consistent with their models.

Despite this, it is clear that there is significant overlap in methodology and goals, regarding causal explanation, between Cartwright's research program of finding causal capacities and the research program of formal biology to produce models capturing the causal structure of mathematically-interpreted concrete systems. As I said above, it is beyond the scope of this dissertation to further compare, for example, Glymour's or Salmon's statistical accounts of cause with

Cartwright's and, more generally, with formal biology. This certainly should be done.

5.4.2 An Account of Causal Explanation in Compositional Biology

In compositional biology, a very different explanatory pattern exists. As discussed in the section on van Fraassen above, the aim and structure of causal explanations in formal and compositional biology differ radically. The clearest and most detailed version of causal explanation pertinent to compositional biology can be found in Cummins' explication of causal capacities.[486] However, other scholars such as Kauffman and Wimsatt have also analyzed causal explanations in a manner pertinent to compositional biology (Kauffman's "articulation of parts" explanations and Wimsatt's account of "functional explanation"). Future work should include an explication of Kauffman's and Wimsatt's views on explanation. Here I focus on Cummins because his view ties in clearly to, and is *explicitly* contrasted with, the received philosophy of science literature on explanation, which pertains, almost exclusively, to formal biology. Cummins view applies to different systems and sets of problems; it also appeals to very different contrast classes and relevance relations, to use the language that van Fraassen usefully articulated.

Cummins, in the philosophy of biology literature, is best known for his "causal role" analysis of function. But he considers this analysis to be an aspect of a more general explanatory pattern. Cummins' account is extremely important and topical for my purposes. It is therefore worthwhile spending some time unpacking it.

[486] Cummins 1975, 1977, 1983.

Cummins claims that there are two explanatory strategies, subsumption and analysis pertinent to two kinds of theory, transition and property theories.[487] Transition theories seek "to explain changes of state in a system as effects of previous causes—typically disturbances in the system."[488] And the way such explanations are effected is by "subsumption under causal law."[489] This is done by trying "to fix on a set of state variables for the system that will allow one to exhibit each change of state as a function of a disturbing event and the state of the system at the time of the disturbance."[490] These functions, or transformations, are the causal laws. Note that Cummins analysis of transition theories and subsumption is a *semantic theoretic* one since he is essentially appealing to laws of succession. Cummins distinguishes between subsumption of concrete phenomena under causal laws (causal explanation) and subsumption of a causal law under a more general causal law (theoretical explanation).[491] Cummins is insensitive to the multiplicity of analyses on causal laws and the differences

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[487] See Chapter 1 of Cummins 1983.
[488] Cummins 1983, p. 1.
[489] Cummins 1983, pp. 1-2.
[490] Cummins 1983, p. 2.
[491] Cummins 1983, pp. 5-6, where he distinguishes "explanation of individual events" from "explanation of event-types." Here he adds that subsumptive explanation fundamentally requires analytical explanation, to be discussed below, since subsumptive explanation alone does not explain the dispositions (capacities) that the objects referred to by causal laws have. I would add, as argued below, that analysis also requires subsumption, even if that is not the *central* relation. Either explanatory strategy, appeals to causal capacities of its objects and the constituent parts of the objects. Both Cartwright and Cummins argue that at some level capacities (dispositions) become irreducible and "primitive." No further explanation can be provided. What is different between the two explanatory strategies is (1) where the capacities lie, (2) what they do, (3) how they are theoretically expressed, and (4) how they are structured. In the subsumptive explanatory strategy they lie in the object and are described in formal terms. In the analytical explanatory strategy, dispositions of an object are explained in terms of dispositions of its (hierarchically-organized) parts. Such dispositions are often described in qualitative terms.

between semantic and syntactic accounts of theories, for which the subsumptive (or "matching" – see footnote 463 above) pattern of explanation is central. Part of the reason for these lacunae is that he is not particularly concerned with the subsumptive pattern of explanation, which, he claims in a massively understated manner, is "more familiar" to "philosophers and methodologically minded scientists."[492] Good examples of causal subsumption are explanations of pendulum or planetary motion or explanations of changes in gene frequencies in populations. Cartwright's account of causation is also a case of this pattern. It is, in fact, the explanatory pattern that has completely dominated philosophy of science analysis.

Property theories[493], and the analytical strategy of explanation that they involve, are the subject of Cummins' book (and 1975 article). Let me present Cummins' own wording:

> The characteristic question answered by a property theory is: What is it for system S to have property P?
>
> The natural strategy for answering such a question is to construct an analysis [including the pertinent model(s)] of S that explains S's possession of P by appeal to the properties of S's components and their mode of organization. The process often has as a preliminary stage an analysis of P itself into properties of S or S's components. This step will loom large when we come to discuss complex dispositional properties such as information-processing capacities. Analysis of a *system* will be called compositional analysis, to distinguish it from analysis of a *property*, which will be called functional analysis when the property is dispositional, and property analysis when the property is not dispositional. Analysis is "recursive," since a given analysis may appeal to properties or components that themselves require analysis.[494]

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[492] Cummins 1983, p. 1.
[493] I will return to transition and property theories, and the problematic distinction between them, as well as the mismatch between them and the two explanatory strategies, at the end of this section on Cummins.
[494] Cummins 1983, p. 15.

It is precisely in the context of analytical explanations that Cummins developed his account of functional explanation – these are cases where the property being explained has a disposition, a *capacity*, to do something within the system (the whole) under investigation from a particular theoretical perspective. Note the overall pattern of explanation in analytical explanations: a system is broken down into components at the next lowest level. These components have a particular organization vis-à-vis one another. This "compositional analysis" is to be distinguished from, but is a necessary preliminary for, "functional/property analysis" which involves the explanation of the system property *in terms of* the properties of its components. Both the partitioning of the system into *components*, and the ascription of hierarchical *properties* (capacities) to the system and its components, have to be done appropriately for an analytical explanation to work successfully: "Successful analysis yields an explanatory payoff when we come to see that something having the kinds of components specified, organized in the way specified, is bound to have the target property."[495]

Note that the analysis is "recursive." Once we explain a system in terms of the properties of its components one level down, we might desire to continue going down levels recursively until we reach some sort of atomic parts. In his article, he opaquely and provocatively claims that we should continue "until pure physiology takes over, i.e. until the analyzing capacities are amenable to the subsumption strategy."[496] This sentence is conspicuously absent from an otherwise *identical* passage on page 29 of Cummins 1983. In the book, he merely hints that we should stop at the "elementary parts" that are deemed as such by

[495] Cummins 1983, p. 17.
[496] Cummins 1975, p. 761.

the theory in question: for example the elementary parts of "the theory of bonding" are "atoms."[497] The recursions of compositional and functional/property analysis end where the theoretical perspective coordinating the analysis suggests that they should. Furthermore, the theory is the one that *picks out* the system capacity of explanatory interest. These aspects of Cummins' view are highly naturalized – they appeal to the scientific theory to perform both the compositional, and the property, partitioning.

One point I would like to add to Cummins' account is that subsumption (matching) is not "absent" or "irrelevant" to compositional and functional analyses, as Cummins claims in 1977. In fact, theoretical models suggest and motivate the partitioning of both organization and capacities. Furthermore, the models representing this partitioning are changed in response to empirical findings regarding the validity and utility of the partitioning. Although theoretical manipulation rarely happens independently of empirical manipulation in compositional biology, there is a matching between theoretical models and concrete systems in compositional biology (it is a much less important relation compared to the one in formal biology since in compositional biology, models can be changed with impunity). Subsumption is thus not absent and irrelevant here, although it plays a different and smaller role than in formal biology. I also believe that when Cummins talks about an *actual* analytical explanation, he refers to a *model* that serves an important explanatory role. He is not referring to a partitioning in the concrete. For Cummins, models are related in important ways to explanation.

[497] Cummins 1983, p. 27.

The power of an analytical explanation lies in the relationship between the causal capacities of the components and the capacity of the system. Cummins argues:

> The explanatory interest of functional analysis is roughly proportional to (i) the extent to which the analyzing capacities are less sophisticated than the analyzed capacities, (ii) the extent to which the analyzing capacities are different in kind from the analyzed capacities, and (iii) the relative sophistication of the program appealed to—i.e., the relative complexity of the organization of the component parts/processes that is attributed to the system. (iii) is correlative with (i) and (ii): the greater the gap in sophistication and kind between analyzing capacities and the analyzed capacity, the more sophisticated the program must be to close the gap.[498]

Cummins' picture of a powerful analytical explanation is this: a system capacity, chosen by the theoretical perspective at hand[499], is partitioned into simpler, and different in kind, component capacities. The simpler and more different in kind part capacities are, the more complex the program for integrating these interacting capacities must be.

A number of problems do arise from Cummins' account. First of all, what is the nature, status, and role of the "program"? Cognitive capacities and cognitive information-processing are the investigative domain of Cummins' research program. His book, most of which I shall not discuss, is replete with information-centered language and analysis. He distinguishes between "descriptive" and "interpretive" analysis.[500] Descriptive analysis, which he barely

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[498] Cummins 1983, p. 30. For an almost identical passage, see Cummins 1975, p. 764.

[499] For example: "Unlike the states of a digital computer, the states of natural systems do not have standard or intended interpretations. They are *assigned* semantic significance in order to explain the system's capacities." (Cummins 1983, p. 41) Theoretical perspectives assign the significance and the system capacities to be explained.

[500] Cummins 1983, p. 32 ff.

spends any time on, but which I believe is the central form of analysis in *compositional biology*, is analysis in terms of the *material mechanisms* of the system. Interpretative analysis is done in terms of symbols that have semantic interpretations and import. Cummins believes that this is the proper form of analysis for *cognitive information-processing* systems.

Here I part ways with him. Although I have little to say about cognition, I believe that a proper functional analysis (to use his term) of biological systems should rely on *descriptive* analysis. Such a descriptive analysis involves the models pertinent to compositional biology. Thus, returning to the question regarding the nature, status, and role of program, I do not believe that a "program" is a useful metaphor, yet alone an explanation, of how capacities of system components aggregate to form a system capacity in biology. Information-integration will not, for example, explain how the parts of the circulatory system, with their different capacities [e.g., pumping (heart), distributing nutrient and oxygen rich blood (arteries), distributing nutrients and oxygen *by* distributing blood (capillaries), and returning, using one-way valves, depaupered blood to the pump (veins)], contribute to the overall capacity of the circulatory system of distributing nutrients and gases to the body.

This then leads to the question of which metaphor or explanation will allow us to account for how part-capacities, less sophisticated and different in kind, are *integrated* to form a system capacity. Unfortunately, I think that no good philosophical account of this exists and I am not ready to offer one. Cartwright already provides a view to this general problem when she notes that interaction (of capacities) is a primitive term or process, and that she too does not have an

account of capacity interaction.[501] Note, though, one difference between Cartwright's and Cummins' notions of capacities: unlike Cartwright's, Cummins' capacities are *explicitly* hierarchical. Thus, under Cummins' picture, (lower-level) capacities are *always* going to interact – they have to do this in order to produce a higher-level property.

There is, however, an ambiguity in the term "interaction": it can be additive or non-additive. Cummins captures this ambiguity by distinguishing between "morphological" (additive interaction) and "systematic" (non-additive interaction) analysis, which is meant to be orthogonal to his "descriptive" and "interpretive" distinction.[502] Regardless of the existence or absence of additivity, however, (lower-level) capacities have to "interact" on Cummins hierarchical picture, but not on Cartwright's potentially-sole-object-expressing-its-capacity (e.g., a mass in a gravitational field) picture, where a single capacity can explain object behavior.

Although there is no satisfactory philosophical account of capacity integration (which includes, in my vocabulary, both additive and non-additive capacity interaction), one person who has provided some grounds for such an analysis is William Wimsatt.[503] But he also simply *assumes* that there is going to be some relation between part capacities and system capacity, without giving an account of what that relation would philosophically amount to, whether it be an additive or non-additive interactive relationship. For example, Wimsatt writes:

> Suppose that system and parts' properties are related in a theory by a function, F:
> $P_j(S) = F[p_1, \ldots, p_n(s_1);\ldots;p_1, \ldots, p_n(s_m)]$,

[501] See Cartwright 1989, p. 166.
[502] Cummins 1983, pp. 31-34.
[503] See Wimsatt 1972, 1974, 1986, and 1997.

> Where $P_j(S)$ is a system property, s_1 through s_m are parts of system S under an exhaustive and exclusive decomposition, and p_1 through p_m are properties possessed by these parts.[504]

Nowhere does Wimsatt give a general philosophical account of how the function *F* actually *does* what it is supposed to do: integrate part properties (capacities) to produce the system capacity. Wimsatt has provided an account of functional loops which is a beginning for explaining how part capacities, at a particular level *depend on* one another[505]; but this dependence does *not* explain how a new kind of, under Cummins' view, *more* sophisticated higher-level capacity, comes about.[506] Nor does Wimsatt give an analysis of how the multiple capacities ascribed to each part kind relate to one another for each part kind, or even how and whether part kinds overlap in capacities. Furthermore, while Wimsatt does talk about "cooperative or inhibitory interactions," he assumes them (as a way to find system non-aggregativity) and does not describe them philosophically.[507]

These criticisms are not intended to undermine the utility of Wimsatt's analysis for describing when a system can be considered aggregative and when it cannot. But I also think that capacity integration from a lower level to a higher one is an important outstanding philosophical problem necessary for discussing causal capacities pertinent to compositional biology. Cummins' informational "program" explanation will not do the biological trick; Cartwright has no account (and is not committed to describing hierarchical systems anyway); Wimsatt has provided grounds, but has not provided an analysis.

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[504] Wimsatt 1986, p. 260.
[505] See, for example, Wimsatt 1997, pp. 107-108.
[506] Despite the strengths of his analysis, Craver 2001 also does not provide such an account.
[507] Wimsatt 1986, p. 269.

Let me, then, provide a few brief suggestions for such an analysis. First, the integration of the capacities requires an explicit causal story, a narrative model, of exactly how the causal capacities fit together ("articulate," to use Kauffman's term) and how, through this precise causal articulation, a higher-level causal capacity arises. That is, careful attention must be paid to the precise *qualitative dispositions* of the parts. This can best be done through a narrative model complemented by diagrams such as functional flow charts and more concrete structural diagrams representing material structures, such as arteries, capillaries, and veins. The qualitative nature of the system capacity must be carefully and intuitively partitioned into a series of qualitative part capacities that can clearly account for the system capacity. What I have just provided is not an account; it is a sketch, or at least a "functional specification"[508] of what must go into an account of how part capacities become system capacities under particular theoretical perspectives. Note that I stress the unique qualitative nature of part capacities and the role of models pertinent to compositional biology, such as narrative and diagrammatic models.

With regard to propositional non-mathematical models, Cummins too emphasized their utility for compositional and functional analysis:

> We can easily imagine biologists expressing their analyses in a form analogous to the schematic diagrams of electronics, with special symbols for pumps, conduits, filters, and so on. Indeed if transplants and implants ever become commonplace, this is the only sort of description that would achieve real generality.[509]

[508] In the computer science design sense.
[509] Cummins 1983, p. 29. For an almost identical passage, see Cummins 1975, p. 761. For a direct application of Cummins' view, see how he interprets Freud's theories using intricate flow charts on pages 142-161 of his 1983. Eric Davidson's logical model, discussed in Chapter 4, is a very pertinent example of this too.

Given the intimate relation between form (and organization) *and* function, the role of diagrammatic models expressing form in providing representations for the production of analytical explanations using (functional) causal capacities, is clear. For example, in the Krebs cycle case described in Chapter 4, the chemical models describing the structure of the molecules involved provide the grounds for displaying the capacities of the molecules (including the catalytic enzymes). These individual part capacities explain how the Krebs cycle occurs and how system behavior, such as production of ATP and production of carbon dioxide from molecular oxygen, ensue.

An analogous hierarchical story could be told for the diagrammatic models representing structures and, ultimately, capacities, in Bramble and Wake's model of feeding. As considered in my discussion of the general properties of models in Chapter 4, these models allow for the discovery, as well as the representation, of the capacities of the objects that, ultimately, explain, in an *analytical* way, the processes occurring. These models attempt to capture the qualitative unique capacities of numerous object types, organized in a hierarchical and integrated fashion. The mathematical models of formal biology make no assumptions about hierarchical organization and certainly make no claims about hierarchical integration; furthermore, these models are mathematically idealized and capture the properties of the objects in terms of a few key variables that specify trajectories through an idealized state space. Cummins' analysis, including the models it requires, pertains to compositional biology, whereas Cartwright's analysis of causal modeling and capacities underlying it, and inferred from it, pertains to formal biology.

I have elaborated on Cummins' framework for two reasons: (1) his account of the analytical explanatory strategy, which includes his account of functional explanation, directly pertains to explanation via models in compositional biology; this is so despite the fact that I disagree with him fairly early on in his account – I adopt neither informational analysis or language, nor the notion of a program. (2) His contrast between subsumptive and analytical[510] explanatory strategies *exactly instantiates* the distinct explanatory strategies in the two kinds of biology I distinguish: formal and compositional. Thus, I think that his analysis of two general explanatory strategies bolsters my distinction between formal and compositional analysis – this corroboration occurs despite the fact that he glosses over important differences (e.g., syntactic and semantic views) in the "subsumptive (matching) explanatory strategy" camp.

I do not, however, agree with Cummins that these two explanatory strategies correlate neatly with transition and property theories, respectively. While there certainly is a case to be made that transition theories [which, under a semantic interpretation, are specified by a state space and laws of coexistence and succession (or "transition")] are often amenable to subsumption and that property theories can be adequately explained by analysis, the correlation is not strict. For example, a number of transition theories, such as a theory of biological development (a transitional process par excellence), are certainly better dealt with by (compositional and functional) analysis, while transition theories of physics subject to subsumption can, under Cartwright's view of capacities, be *reinterpreted* as property theories with (mathematically modeled) capacities as primitives. The distinction between transition and property theories is not

[510] Which, I argue, does include subsumption!

necessarily a neat one and it certainly does not map on cleanly onto subsumption and analysis explanatory strategies (although there is a correlation). However, subsumptive and analytical strategies are distinct and describe accurately the causal explanatory efforts, through the use of different general kinds of causal modeling, of formal and compositional biology.

One weakness common to the causal explanatory pattern of both Cartwright and Cummins is their emphasis on *capacities* and their sidestepping of the process of *interaction*. They have a quintessentially *partitioned* way of explaining causality through the modeling of particular causal factors or capacities of objects (or parts) of concrete systems. Their views are causally "monadic" and "reductionist"[511] in this sense. Note that their views stand in stark contrast to views that could emphasize the interaction terms in formal equations, or mechanistic interaction between parts of a system. Oyama's or Lewontin's philosophically-oriented views on these matters provide material for a view that *starts* from interaction (see footnote 163). What would causal explanation in formal and compositional biology look like if, instead of averaging or controlling the capacities across contexts to derive "additive" overall capacities, we *started* with context and looked at interaction in each kind of context (i.e., we did not attempt to find "contextually unanimous" Cartwright capacities and we focused on *integration* among Cummins capacities)?[512] This would be a "holistic"[513]

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[511] In footnote 177, I mentioned the different meanings of reductionism explicated by Sarkar 1998 and Zucker 1996.
[512] Goodnight and Wade 2000 explicitly suggest this: "If we were to offer a prescription for constructing such [genetic] models, we would suggest building a model with *only* interactions between genes and, from it, *derive* the mean additive effects of the component genes and the variances about the means." (p. 319)
[513] See Winther 2003 in press for an argument for replacing "holism" and "reductionism" with, respectively, "articulation" and "partitioning."

alternative to the "reductionism" of Cartwright and Cummins. It remains unclear, however, how this would be done. The point, though, is that Cartwright's and Cummins' view, respectively, are *one* way to flesh out causal explanation in each kind of biology.

5.5 Theoretical Explanation and Confirmation in Formal Biology

I now want to turn to the relation between laws and law-based models at different *levels of generality* in formal biology. That is, I want to turn to how these theoretical components of formal biology are explained. This is a complex topic for which I will develop a taxonomy of different ways of concretizing and abstracting. Modeling as an activity independent of empirical activity is of crucial importance in formal biology. This is why, for example, semantic analyses are so powerful in understanding population genetics, which has a rich mathematical structure. This is also why theoretical explanation, confirmation, and causal explanation can be neatly distinguished in formal biology (but, as we shall see, not in compositional biology).

5.5.1 An Account of Theoretical Explanation via Theoretical Unification

First I want to turn to Friedman's views on theoretical unification as a way to explain laws and models. Friedman has developed a detailed account of how unification guides theoretical explanation.[514] Here I want to first explicate the pertinent aspects of Friedman's view and then, in the next section, show they apply to the modeling activity of formal biology.

[514] Friedman 1974, 1981, 1983.

5.5.1.1 Friedman's "Theoretical World" and My Theoretical Perspectives

How exactly does theoretical explanation occur? Consider Friedman's notion of a "theoretical world" into which, he believes, we embed concrete cases.[515] Causal explanation[516], then, would be the direct embedding of concrete cases into some theoretical world, whereas theoretical explanation involves finding *embedding*—and other sorts of—relations between abstract entities (i.e., laws and models) in the theoretical world. Friedman defines a theoretical world in the following way: "some larger, relatively abstract and unobservable theoretical [formal mathematical!] structure [of theoretical physics]."[517] This is a fairly rigid and precise definition appealing to the formal mathematics of physical theory. Friedman's definition is itself embedded in the context of a theoretical and static representational orientation in philosophy of science [see Table 6. (4.1)]. For simplification and theoretical reasons, it glosses over difficulties in *individuating* such a theoretical world. That is, how do we account for theoretical "practice," and what relationship do we see between theoretical and empirical aspects?[518] Furthermore it is also unclear how we should discuss the role *models* play in this theoretical world, particularly if we in any way consider them as independent from theory, as is the case in the mediating models view. These concerns are not Friedman's – he is focusing on the (highly) theoretical structure of relativity theory.

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[515] E.g., Friedman 1981, 1983.
[516] Friedman is not interested in this kind of explanation.
[517] Friedman 1981, p. 1.
[518] Again, Table 6 (4.1) is an attempt to address such questions.

Despite the legitimacy of the notion of "theoretical world" at distinguishing causal from theoretical explanation in theoretical physics, I seek something broader. I am concerned with the messy practice, theoretical and empirical alike, of biology.[519] Recall the notion of theoretical perspective discussed in Chapter 2. Theoretical perspectives are broad and are—in a manner anathema to Friedman's ultimate goal of finding ways to theoretically unify—often unrelated and sometimes even in tension with one another. But I do think that the idea of theoretical perspectives as *coordinating* models, methodologies, and theories, via biases and guiding assumptions, for local purposes, encompasses the theoretical, humanly-constructed, world that Friedman, for a particular discipline, captures with his notion of "theoretical world." My account of theoretical perspectives seeks to incorporate practices and also non-mathematical theoretical accounts, such as Cummins' analytical explanatory strategy. I thus think of Friedman's notion of "theoretical world" as an instantiation[520] of my notion of theoretical perspective.

The contrast between causal and theoretical explanation is thus, respectively, that between (1) explaining the behavior of concrete systems (i.e., using a model to provide an explanation – this involves empirical activity) and (2) explaining the relations between theoretical structures at different levels of generality (i.e., this is done primarily through theoretical activity). This

[519] Certainly *concrete* physics is complex and rich too – I take that to be one of the points of Cartwright's work. Friedman and Cartwright, as we have and shall see, stand at opposite ends of many spectra. I think that both of their views can be of use, in different ways, in my analysis of formal biology.
[520] It is also a partial instantiation – Friedman does not consider experimental methodologies that are related to the theoretical world he describes.

difference can be clearly seen using Friedman's notion of a theoretical world. However, the difference, as we shall see, is not clear in compositional biology.

5.5.1.2 Theoretical Unification

Now that I have provided further motivation for the distinction between causal and theoretical explanation, let us turn to Friedman's particular version of theoretical explanation. In his first attempt at developing his views, he claimed that there was an important connection between theoretical explanation and scientific understanding – namely that when a unification, that is, a subsumption of a series of different laws under one more general law, occurs, the scope and depth of our understanding increases (i.e., cognitive burden decreases) and this process *is* a theoretical explanation.[521] "Science increases our understanding of the world," Friedman writes, "by reducing the total number of independent phenomena that we have to accept as ultimate or given."[522] That is, if we can show how more specific laws or models can be embedded into a more general one, the empirical consequences of each previously independent law or model can be shown to be related in that they can now be *derived* from a more general law or model.

Given his primarily (but not exclusively nor even importantly!) syntactic view of theories, with which he can interpret empirical consequences as sentences, Friedman claims that such a generalization relation implies that we have then effected a *reduction* in the total number of "independently acceptable

[521] Friedman 1974.
[522] Friedman 1974, p. 15.

sentences."[523] Such a reduction implies that we require fewer distinct explanatory theoretical resources (laws, models, assumptions, etc.) and, thus, we have increased our (unified) understanding of the world. Friedman provides an example:

> The kinetic theory [of gases] effects a significant *unification* in what we have to accept. Where we once had three independent brute facts[524]—that gases approximately obey the Boyle-Charles law[525], that they obey Graham's law [of diffusion][526], and that they have the specific-heat capacities they do have—we now have only one—that molecules obey the laws of mechanics.[527]

The kinetic theory of gases reduces the total number of empirical consequences, or sentences, that we have to take as independent. It has unifying power, and therefore increases our scientific understanding.

In later work, Friedman fleshed out his view of the relations between theoretical explanation, unification, understanding, and reduction in terms of a meta-model of *hierarchical model reduction* which appeals to (1) realism, and which provides further grounds for (2) analyzing the confirmation relation between evidence (about concrete systems) and theory (the "theoretical world").

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[523] Friedman 1974, p. 16.
[524] By "brute facts," Friedman simply means *independent* phenomena (or generalizations of phenomena). He does not mean this to contrast with empirical generalizations ("phenomenological laws") – the first two in his list *are* precisely such generalizations. (Friedman, pers. comm., September 2, 2003)
[525] $PV = nRT$.
[526] "Under the same conditions of temperature and pressure, rates of diffusion for gaseous substances [r] are inversely proportional to the square roots of their molar masses [M]." i.e.:
$r_1 / r_2 = (M_2 / M_1)^{1/2}$ (See Chang 1994, p. 190)
[527] Friedman 1974, pp. 14-15. Friedman further claims that the kinetic theory can be unified with planetary behavior and falling body behavior under the laws of mechanics, according to which *all* bodies behave. There are, however, *other* forces involved in the behaviors of real gases besides the laws of mechanics, such as Van der Waals forces. In the 1974 article (but not the 1983 book), Friedman glosses over this.

Briefly, Friedman contrasts *reductive* and *representational* theoretical inferences (explanations). Reductive theoretical inferences are ones where *realism* is assumed and that, therefore, relating observable objects (and their respective properties/relations) of one kind of theory (e.g., gas and gas laws) with the objects (and their respective properties/relations) of *another* kind of theory (e.g., molecules and chemical laws) can be done in a powerful and unifying way:

> The important point here is that, on a literal construal of $\mathcal{A}$ [i.e., the general theoretical model], our theory *evolves by conjunction*. Certain assumptions about $\mathcal{A}$ play a role in the explanation of the gas laws. These same assumptions, in conjunction with further assumptions about $\mathcal{A}$, play a role in the latter explanation of chemical combination.[528]

It is the overall postulation of realism that allows us to "carry-over" or conjoin the assumptions and models of one observational domain into another observational domain. We are assuming that different theoretical models are describing the same objects, properties, and relations. When we unify or conjoin objectively here, we *reduce* the number of independently acceptable empirical consequences and the number of independent observational domains. Note that "reduction" is a loaded term – here the strength of unification lies not in reduction to

[528] Friedman 1983, p. 245; see also Friedman 1981, p. 4. Morrison 2000, pp. 38-52 critiques Friedman's account of reduction by claiming that unification is primarily a theoretical claim not tied to mechanism in any way. By Morrison adopting Cartwright's trade-off between (causal) explanatory power and (theoretical) generality, she endorses a general framework anathema to Friedman's. As I have discussed in Chapter 4 and here immediately below, because of their differing assumptions regarding abstraction, Cartwright and Friedman do not, and cannot, see eye to eye on the matter of theoretical reduction.

microstructural mechanism, but in *theoretical* reduction to a broadened observational scope of a higher-level theoretical structure.[529]

If we do not postulate realism, Friedman claims, then we cannot objectively conjoin distinct models and expect to infer a more general model. In such merely *representational* cases (observational objects, together with their properties/relations are only represented by a mathematical structure), "all initial assumptions about $\mathcal{A}$ have been 'trapped' within the scope [of the representation]." No objective, non-arbitrary, conjoining with other models, either at the same or higher level of generality, could then occur: "Under this … construal, $\mathcal{A}$ can be thought of as a purely mathematical object. It is no longer necessary to attribute physical reality to $\mathcal{A}$, since we no longer literally identify elements of the concrete [system] with elements of $\mathcal{A}$."[530]

In the context of the interpretation of mere representation, Friedman's model of explanation is riddled with the problem of "arbitrary law-conjunction" that was one of the downfalls of the positivist's deductive-nomological model of explanation. This is why Friedman prefers the realist interpretation. Recently, however, Friedman has desired to downplay the strongly realist interpretation while still maintaining the importance of both the unification of models and the reduction of independently acceptable empirical consequences.[531] His recent position works well in the context of my agnosticism about ontological attitudes

[529] Friedman explained this to me in conversation (July 9, 2003).
[530] Friedman 1983, pp. 236-237.
[531] Friedman, pers comm., July 9, 2003.

(see footnote 484). It is not clear, however, how such agnosticism is to be implemented in the context of Friedman's earlier account.

Let us now turn to confirmation. Friedman's emphasis on theoretical explanation is so large, that for him the world comes in primarily as a way to *confirm* the laws and models at various levels of generality. He is not at all concerned with causal explanation – i.e., with subsuming concrete situations under particular laws in order to explain—to focus—on the concrete situation itself. *The world merely confirms, it is not itself in need of explanation*. Put differently, Friedman is concerned with the formal relations of the "theoretical world" rather than with causal relations of, and in, the "concrete world" (explained by the theoretical world). This, of course, stands in stark contrast to Cartwright's and Cummins' concerns with causal explanation.

Although Friedman does not have a clear account of the confirmation relation[532], he does know what such a relation is *supposed to do* in the context of the theoretical world. The important point Friedman wants to make is that "a total theory rich in higher-level structure is likely to be better confirmed than a total theory staying on the phenomenological level, even though the latter theory may have precisely the same observational consequences as the former."[533] This is because

> the theoretical description [of the phenomenology of a gas] receives confirmation from indirect evidence—from chemical phenomena, thermal and electrical phenomena, and the like—which it then 'transfers' to the phenomenological description. If the phenomenological description is removed from the context of

[532] In conversation he noted that to a "0^{th} approximation," the positivist's hypothetico-deductive method of confirmation was accurate.
[533] Friedman 1983, p. 244,

> higher-level theory, on the other hand, it receives confirmation only from direct evidence—from the behavior of gases themselves.[534]

Formal models at different levels of abstraction thus receive confirmation directly as well as indirectly, through their theoretical relationships with other models.[535]

5.5.1.3 An Incremental and Creative account of Abstraction

Before I evaluate the utility of theoretical unification as an account of theoretical explanation for biology, I will analyze a crucial point Friedman makes vis-à-vis abstraction and how the abstract laws of physics do not only *not* lie, but actually tell and construct *much* of the truth. For Friedman, abstract laws have rich explanatory content. Perhaps his view stems from the idea that such laws have context and assumptions – they borrow, and are intertwined with, other very abstract areas of the theoretical world. In discussing the "analogous relationship" between two specific pairs of laws in two domains, Newtonian theory of motion and the kinetic theory of gases, he writes, in a passage that serves as counterpoint to Cartwright's claims:

> In both cases we start with a phenomenological law formulated in terms of more or less observational quantities; relative acceleration… [and] p, V, and T…. It turns out that this *phenomenological law is false*[536]; no actual concrete reference frame is

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[534] Friedman 1983, p. 244. See also Friedman 1981, p. 7: "For a theoretical structure that plays a role in theoretical explanations in many diverse areas picks up confirmation from all these areas."
[535] For Friedman, but not for Cartwright, confirmation is "inherited upwards" (pers. comm., September 2, 2003)
[536] This contrasts starkly with Cartwright's claim that the *abstract* laws of physics lie! For Friedman, phenomenological laws, in general, are false, in that they hold under much fewer conditions and take into account fewer relations and properties.

> inertial; no actual gas is ideal. In both cases *we can see just what is missing from the point of view of a higher-level theory*; Newtonian theory tells us that we have to take account of *B*'s absolute acceleration and rotation… [and] kinetic theory tells us that we have to take account of the sizes and interactions of the molecules constituting our gas…. In both cases we correct an initial phenomenological law by relating relatively observable entities to higher-level theoretical entities: we correct [one formula] by relating *B* to the Newtonian's unoccupied inertial trajectories [and] we correct [the other formula] by relating our gas to the kinetic-theorists unobservable molecules.[537]

Friedman's view here contrasts quite starkly with Cartwright's skepticism of the veridical status of abstract physical laws. For Friedman, abstract laws provide crucial information in the practice of theoretical explanation. Friedman has an "incremental" view of abstraction as compared to Cartwright's "removal" view. Here explanation (and abstraction!) occurs when a specific law/model is embedded or subsumed under a more abstract law/model – this is subsumption in the theoretical world rather than subsumption from concrete cases to the theoretical world. Note that Cartwright is concerned with the former and downplays the latter. Friedman's philosophical strategy and goals are the opposite.

But even if we focus solely on theoretical explanation (which Cartwright also discusses, as in the chapter entitled "The Truth Doesn't Explain Much" in 1983), we see that very different philosophical consequences follow from the view of abstraction (e.g., law and model abstraction) as removal of properties or Aristotelian causes (Cartwright's view) and abstraction as the filling in of theoretical richness and texture (Friedman's views). For a follower of Cartwright's view of abstraction, abstract laws of physics have relatively little content and even in the domain of theoretical explanation, they can have

[537] Friedman 1983, p. 240, emphasis mine.

relatively little explanatory import; they certainly cannot, by themselves, be used for derivations of lower-level (increasingly "phenomenological") laws – assumptions about particular conditions must be added. For Friedman, abstract laws have significant content: they allow us to "see just what is missing" in a phenomenological law. They also provide their own "conditions of application," something a phenomenological model, according to Friedman, cannot do.[538]

One reason for their differing views is that, for Friedman, abstract laws are individuated differently than for Cartwright. She only focuses on the statement of the law itself, while Friedman may hold that all the theoretical assumptions and context related to the law should be somehow considered "part of" the law – the statement of the law is individuated together with this context to make the "abstract law." But even with such an individuation, it still seems that a Cartwrightian would emphasize the (theoretically) explanatory impotence of abstract laws, whereas Friedman would claim explanatory power. *For Cartwright the abstract is depauperate, whereas for Friedman the abstract is rich.*

This notion of the *explanatory power* of theoretical abstract laws is related to the important idea of the *creativity* (rather than "mundanity"[539]) of abstraction. Although, as we saw in Chapter 1, Cartwright's removal view does not imply a mundanity view, her position on abstraction could easily lead an advocate to underplay the creative (i.e., "tying together," "seeing new connections," "formulating new *kinds* of hypotheses") role that abstraction, and abstract laws

[538] Friedman pers. comm., September 2, 2003.
[539] I.e., that abstraction does not do very much for us scientifically. Perhaps it helps us to organize the world a bit, but the main "action" lies in empirical research and in phenomenological laws.

and models play. This creative role, however, can be recognized quite easily under Friedman's account. Furthermore, Friedman writes,

> If [my] present treatment [of theoretical explanation] adds anything to Whewell's notion of consilience it is perhaps the thought (itself borrowed from Boyd and Putnam[540]) that the whole point of *theoretical* structure is to facilitate this process of inductive "jumping together."[541]

Abstraction, and model construction, provides the grounds for scientific creativity.

One could make the *further* argument that abstraction, in its creative aspect, is also tied to scientific understanding. Creativity and understanding, then, would be related. But here some problems appear. Friedman provides arguments for the relationship between unification and scientific understanding, but once we bring in creativity and the role it plays for model *derivation* or *elaboration*, the relationship is no longer so neat. Friedman assumes some sort of epistemic Ockham's razor: fewer (but simplified?) assumptions, laws, and models are supposed to give us more scientific understanding.[542] This undercuts the role of *comparative analysis* and the importance of diversity of (sometimes competing) models and theoretical perspectives. As can be seen at least in biology, there is tremendous creative strength in approaching the same problems

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[540] Here I think that Friedman is not giving himself credit that he deserves.
[541] Friedman 1983, p. 242.
[542] Barnes 1992 and Humphreys 1993 consider the problem of equating unification with understanding from points of view different from mine. Barnes 1992, in particular, provides a discussion of the issue of why derived phenomena/statements should be considered less mysterious, or better-understood, than underived "independent" phenomena. He argues that they can only be considered less mysterious if one adopts a "local understanding" view, that is, if one brings in causal explanation as well. I think that he underestimates the power of derivation and the important independent role that theoretical consistency plays in science, especially theoretical physics and, for some theoretical activity in formal biology.

or objects from various points of view, both when the various theoretical perspectives do not complement each other's analyses very much (as discussed for the various areas of organismal biology in Chapter 2), and when they literally are conjoined in an analysis of a complex phenomena[543].

Furthermore, sometimes we may not want to reduce different kinds of objects to fewer and more general kinds of objects – that may abstract away from irreducible complexity and, in fact, obscure and destroy the creative process of hypothesis formation regarding the causally-potent variety of complex objects in biology (especially, compositional biology, with its complex abstraction, as explored in Chapter 1). Thus, we cannot underestimate the importance of variety and the creativity of comparison of various object types and theoretical perspectives in biology. We cannot completely accept the relationship, implicit in Friedman's work, between creativity and understanding/unification.[544] However, we should also be wary of adopting a mundanity view of abstraction. In compositional biology, creativity and diversity (not unification!) are linked!

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[543] With regard to the second point, Kitcher 1976 provides the useful example of explaining "why lightning flashes are followed by thunderclaps" as an example of hybridizing two different theories (p. 210). Kitcher's article is intended to show "why Friedman's theory goes wrong, and it [Kitcher's account of unification] may point the way to something better." (p. 212) Below I will attempt to show that Kitcher's account has weaknesses and that Friedman's account is closer to scientific practice.

[544] Frederick Schmitt made an interesting and related point: the relationship between unification and explanation may not be one of *identity*, but may be one in which unification "contributes to something else" related to explanation, such as facilitating "comparison of competing alternatives." In this case, unification may be finding of similarities across competing (or even mostly independent) explanatory alternatives. So perhaps unification and diversity are not, as I argue, opposed. This suggestion deserves further consideration. But, for the time being I will hold that such unification seems to be very different from Friedman's theoretical embedding relations. It would seem to have more in common with Wimsatt's analysis of robustness across perspectives, a theme explored in the context of compositional biology in Chapter 2.

5.5.2 Theoretical Explanation (Model Elaboration and Unification) and Confirmation in Formal Biology

Let us now turn to the practice of how models are related to one another in formal biology (i.e., theoretical explanation in formal biology). I have spent considerable effort articulating Friedman's views because I believe that they can be used to explain a rare, but important, case of relating models in formal biology – model unification (a case of model merging). I will discuss the Price Equation in this context as a paradigm example. But, most modeling in mathematical evolutionary genetics, the paradigm case of formal biology, occurs by *elaborating* models – that is, by developing models of ever-increasing levels of specificity and complexity for increasingly realistic situations. For example, much of population genetics consists in making ever-elaborate models with increasing numbers of parameters and variables, which capture different forces (e.g., genetic drift, natural selection, mutation) under particular organizations, including population structure, and for particular assumptions. The primary drive in mathematical evolutionary genetics is to *diversify*, not *merge*, the models.

	Model Merging (Abstraction)	Model Diversification (Concretization)
Deductive	Unification	Derivation
Tinkering	Tinkering Merging	Elaboration

Table 8. (5.2) The relationships between the different forms of model merging and model diversification.

In Appendix A, I describe model construction in formal biology quite abstractly as (1) starting with certain equations ("input equations"), (2) manipulating them in a theoretical way (i.e., mathematical in formal biology), and then (3) formulating the equations that determine the behavior of allowable values in the state space ("model equations" – laws of succession and coexistence). But I left open what it meant to "manipulate" the input equations—the laws—to form the model equations, which are the laws emphasized by the semantic view. I also left open *how* the input equations were chosen and which other entities were involved in model manipulation. In order to provide even the sketchiest of accounts of model elaboration and unification in formal biology, we have to address these issues.

Here I want to make a few distinctions important to my argument [see Table 8. (5.2)]. There is a difference between model *merging* and model *diversification*. The former is a matter of increasing levels of generality, whereas the latter is a matter of decreasing levels of generality. The two are thus opposite and complementary in direction of abstraction. Diversification can happen by either adding parameters or variables of the *same kind* (e.g., adding more loci, each with a particular fitness structure, including epistatic fitness effects) or of *different kinds* (e.g., adding stochastic elements of genetic drift to a model that had only included natural selection as a force). Merging happens in the opposite direction by removing parameters or variables. This does not imply, however, that the more general model has *less* theoretical content as one could claim by focusing on Cartwright's account of abstraction – recall Friedman's "incremental" account of abstraction (although for Friedman, incremental abstraction involves *adding* parameters or variables).

There is now a further question as to how the concretization occurs. Is it a matter of tight mathematical "deductive" *derivation* or is it a sort of "tinkering"[545] *elaboration*? Note that these are *both* cases of diversification! This distinction is indeed difficult to make in practice. Some model diversification in mathematical evolutionary genetics has more of the sense of necessary and definitional specification—a deduction, if you will—such as the relation between the Price Equation and models of group, kin, soft, and hard selection.[546] Here empirical assumptions used to move from the former to each of the four latter are few and explicit – the derivation is precise and mathematically tight. In many ways, this can be described as *theoretical embedding* in Friedman's sense, although it is in the opposite *direction*. Friedman's account of unification can be usefully and precisely applied in this case, as I hope to show in future work with Wade. Moving from a one-locus to a two-locus Hardy-Weinberg model is similar in terms of its mathematical tightness.

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[545] This word is *not* intended to carry perjorative normative weight. It is meant to capture a style of modeling techniques as well as a kind of abstraction relationship between models at different levels of generality.

[546] Wade 1985 writes: "The models of soft selection, hard selection, kin selection, and group selection can be represented as variations of a common general model that expresses the total gene frequency change, itself a covariance, as the sum of two covariance components: (1) the covariance within groups between individual relative fitness and individual gene frequency averaged over all groups; and (2) the covariance between group mean relative fitness and group mean gene frequency." (p. 72) See also Frank 1997, who writes: "A general framework is presented to unify diverse models of natural selection. This framework is based on the Price Equation…" (p. 1712) and "The Price Equation subsumes the particular results, by Fisher, Hamilton, Lande and Arnold, and many others [including, presumably, Michod and Wade], and generalizes these results to arbitrary systems of inheritance and selection." These are strong and clear statements for the unifying role the Price Equation (or, more generally, a covariance approach) plays. Both Wade and Frank support their claims with rigorous mathematical models.

Other cases of model diversification have more of a sense of tinkering, with multiple kinds of assumptions, empirical and mathematical alike, involved. The model generated is extremely sensitive to the assumptions made, and different investigators make very different kinds of assumptions. For example, the very definitions of epistasis depend on whether one adopts the additive definition of independence of quantitative genetics or the multiplicative definition of independence of population genetics.[547] The notion of epistasis is an extremely complex one with multiple meanings and epistatic models are diversified in many different ways.[548] To some extent, the amount, and validity, of derivation present in a particular case of model diversification can be measured by the amount of conceptual debate surrounding that case. If the derivation is clear (assuming that the assumptions are also accepted, which they sometimes are), then there will be relatively little discord. The validity of the Price Equation unification (opposite direction of derivation) is an example; this equation is accepted as a valid, but perhaps not useful, rendition of selection. The contention surrounding both the definition and prevalence of epistasis is an indicator of the non-derivational status of these models and their sensitivity to assumptions made.

The account of derivation (or, in the opposite direction, "unificatory merging," or "unification" for short) vs. elaboration (or, in the opposite direction, "tinkering merging") that I have sketched here[549] [see Table 8. (5.2)] can be placed

[547] Wade et al. 2001.
[548] See Wolf et al. 2001
[549] Lloyd, pers. com. (July 28, 2003), has argued that most of the modeling activity of population genetics is (non-deductive) elaboration. I agree with her. But I also think that the (deductive) unification case of the Price Equation is an important case of model-merging in formal biology precisely because of the theoretical

in the account of abstraction I developed in Chapter 1. Recall that I there differentiated between the simple and complex abstraction of formal and compositional biology, respectively. The "simple" mathematical abstraction of formal biology—for which there is yet no good philosophical account, but for which John Locke and C.I. Lewis provide good linguistic analogues—is used in both unification and tinkering merging even if, in the latter there is much more sensitivity to overall modeling assumptions, empirical and mathematical alike. But when we look at the *pattern* of abstraction, it is in terms of removal of parameters and variables in well-specified ways (even if the specified ways are contingent on assumptions). Note also that because of the important background role the assumptions play, the removal of parameters and variables need not be equated with a removal (and mundanity) account of abstraction.

Thus far I have differentiated between the direction of model-construction (i.e., concretization-diversification vs. abstraction-merging) and the mathematical "tightness" of model-construction (deductive vs. tinkering). Now I want to make the point that both derivation and elaboration of models is a theoretical activity with some *empirical* content.

Much of the literature, however, claims that model diversification is primarily analytical. Lloyd and other proponents of the semantic view emphasize that models are "*constructed*, not discovered."[550] Model construction happens in an idealized theoretical domain and is then related to data models of concrete systems, in an attempt to find empirical adequacy that will confirm the

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power of derivation. Friedman, pers. com. (July 9, 2003), has noted that he believes that even in theoretical physics, unification is rare, even if it is a desired outcome—a regulative ideal—among models at different levels of generality.

[550] Lloyd 1988, p. 21.

models. Lewontin argues that "population genetics begins with the undoubted facts of Mendelism, of chromosomal recombination, of mutation, of inbreeding, and builds a theoretical structure that is unassailable in its general outline."[551] Sober states that

> …when one tries to state an evolutionary law precisely, the result seems always to be an *a priori* model in mathematical biology… [I]t is time to investigate the possibility that biology has no empirical laws of evolution because of the strategies of model building that biologists have adopted.[552]

These strategies are presumably the *a priori* strategies employed in model diversification. Friedman also concurs that model construction occur in the "theoretical world" – this is precisely what he *means* by theoretical explanation.

While I agree that model diversification in formal biology is a mathematical *activity* and occurs in a theoretical (sensu mathematical) domain, it is not without *empirical content*. The laws, for example, are suffused with empirical assumptions – for example (1) that parents have pairs of genes for each locus and that these pairs are, in normal systems, segregated (for each locus) and assorted with varying levels of linkage (across loci) and (2) that relative fitness can be measured in terms of the relative numbers of offspring left by different types of organisms of a species. Of course the laws, and concomitant models, are represented and manipulated mathematically, but we cannot ignore the

[551] Lewontin 1985, p. 3. He further argues, as he did in 1974, that it is difficult, if not impossible, to estimate the parameters that specify the magnitude of the different forces of population genetics. Thus, multiple kinds of models can sometimes produce the same empirical results. This lack of model confirmation and truth-evaluation, does not detract from the mathematical theoretical consistency of the models. Nor does it necessarily imply, as the semantic view has proposed, that there is an in principle difference between applicability and truth. In cases of multiple realizability of models to a particular empirical case we know *neither* the applicability nor the truth of any particular model. Again, as argued in Appendix A, applicability and truth are related!
[552] Sober 1997, p. S467.

empirical content that massively constrains them. *Theoretical activity does not imply absence of empirical content*.

But, empirical *activity* is a separate matter and when the applicability, empirical adequacy, truth-content, and causal explanatory power (four *highly* related concepts) of the models are evaluated, we are concerned with *confirming* the models. Here we have moved from model-diversification, a theoretical activity, to model testing, which involves empirical activity. Lloyd provides a good account of what is involved in establishing isomorphism, or near-morphism[553], between theoretical models and data models, (using also models of the experiment, although she does not "distinguish between models of the theory and models of the experiment"[554]).[555] She claims that there are "three distinct factors in the evaluation of the relation between the model and the data: fit between model and data; independent testing of aspects of the model; and variety of evidence… ."[556]

An open question here is at what level of abstraction the models have to be in order to be tested. Phenomenological laws and models in physics, that are directly tested, tend to be at a very concrete level. Lloyd appeals to Suppes' hierarchy of models and notes that a model that is directly tested has to be "more specified, more concrete than the abstract theory itself."[557] This, then, is

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[553] There are *degrees* of truth and applicability, that is, matching, between a theoretical model and a data model.
[554] Lloyd 1988, p. 146.
[555] Lloyd 1988, Ch. 8.
[556] Lloyd 1988, p. 145. Something like this kind of scheme can be used for compositional biology, but there model manipulation is not purely a theoretical activity (even in the non-mathematical sense of theoretical). It is difficult in compositional biology to distinguish the activity of model-data *fit* from model *construction* – the two are intimately related.
[557] Lloyd 1988, p. 146; Suppes 1962.

compatible with the case in physics. However, Lloyd also hints at degrees of isomorphism in discussing models in which, according to her, only "the state variables are empirically interpreted."[558] The concept of "degrees of isomorphism"[559] implies that fairly abstract models can also be tested against data models, to a limited extent. I will not here further adjudicate the level of abstractness a model must have in order to be confirmed, either under Lloyd's view or in the actual practice of mathematical evolutionary genetics. But I do want to emphasize the difference in formal biology between diversifying (or even merging) models, which is a theoretical activity (*theoretical explanation is a theoretical activity!*), *and* model testing (or, as the semantic theorists prefer it, theoretical hypothesis evaluation), which crucially involves, and is contingent on, empirical activity. [This difference is also indicated in Figure 2. (5.1).]

Note that this whole discussion of the distinction between the confirmation and explanatory relation, as well as the various distinctions concerning model diversification and merging, hold *specifically* for formal biology.[560] I do not think that such neat separations can always be made in the case of compositional biology. This is already discussed, for example, in Griesemer's work on remnant models who does not accept an *a priori* distinction

[558] Lloyd 1988, p. 148.
[559] "Degree of isomorphism" could mean either degree of actual similarity, or degree of being able to even assess similarity in a particular respect (this will depend on level of model concretization – a non-empirically concretized model cannot, in some respects, be evaluated against empirical data). Here I use "degree of isomorphism" in the latter sense.
[560] Note also that theoretical content is required for empirical activity, such as knowledge of how to set up an experiment or how to even *interpret* data and make it useful for confirming a model or for deciding between models.

between "theoretical and observational *activities*."[561] I will return to this point below in the section on theoretical explanation in compositional biology.

In ending this section, I want to note that confirming models is indeed a *very difficult* task in formal biology.[562] In conversation, Lloyd has mentioned this. Lewontin has also stated this in various ways.[563] He has pointed out that population genetics is not the "computational science" that it would ideally like to be: give me as input the initial gene frequencies and the forces involved in changing them, and my model will give, as output, the resulting gene frequencies.[564] The reason population genetics cannot do this is because it lacks empirical information about the force parameters that would specify the movements through state space. Rather, the task of empirical population genetics is to estimate these parameters and to provide (different kinds of) models that link (occasionally) known initial and final gene frequencies. As discussed above, however, there are often multiple (kinds of) models that match the same data set, thus confirmation is impossible given the lack of empirical information regarding parameter settings of different evolutionary forces.

Thus, some have argued that the purpose of models in mathematical evolutionary genetics (and even theoretical evolutionary ecology) is not so much to provide "how actually" confirmable and confirmed explanations, but rather to provide a host of other more open-ended types of explanation which cannot be strictly confirmed. For example, Brandon contrasts "how-actually" with "how-

[561] Griesemer 1990, p. 3. I defended this distinction in Chapter 4 for many cases of formal biology.
[562] In compositional biology confirmation is *deeply related* to generating (or theoretically explaining) a model.
[563] Lewontin 1974, 1985.
[564] Lewontin 1985, p. 5.

possibly" explanations. Speculative "how-possibly" explanations "show[] how known evolutionary mechanisms *could* produce known phenomena."[565] This is better than not having *any* account of how a particular character could have been produced; of course, the problem is that often there are multiple (sometimes conflicting) accounts among which we cannot decide due to lack of empirical information – the models cannot be confirmed. Plutynski provides examples showing that "mathematical and laboratory models function as plausibility arguments, existence proofs, and refutations in the investigation of questions about the pattern and process of evolutionary history."[566] Although the former two are cases of "how-possibly explanations" and the latter *constrains* the "how-possibly explanation" options available, as Plutynski admits[567], her analysis is a contribution to our understanding of the broad role models play in population genetics because she provides a taxonomy and articulation of "how-possibly explanations" and she also ties together different kinds of models – mathematical and material (in the laboratory). These models provide open-ended explanations that cannot, strictly speaking, be confirmed.

In formal biology, model unification is a rare achievement, but is highly respected and is an important form of model merging. Model elaboration (diversification in a "tinkering" way) is a much more common form of model generation – it is also very useful and realistic. Models produced in evolutionary genetics are sometimes confirmed, but more often they simply provide grounds for "how-possibly explanations." Admittedly, *this* kind of work in formal biology is very far from an unificationist account of theory development.

[565] Brandon 1990, p. 180
[566] Plutynski 2001, p. S225.
[567] Plutynski 2001, p. S227.

5.6 Theoretical Explanation and Confirmation in Compositional Biology

Let me now turn to the role of theoretical explanation and confirmation in compositional biology. This is an understudied area and here I can at best provide a sketch of a full analysis. A complete analysis would require an exploration of the relationships between (1) models, (2) modeling (model articulation), and (3) theoretical and empirical (3a) activity and (3b) content in compositional biology. Investigating these relationships would provide us with information about how model evaluation (confirmation) and model application (explanation) occurs in compositional biology. I will address a number of these relations. First, I will evaluate one possible account of theoretical explanation for compositional biology, Kitcher's version of theoretical unification. I will show that for a variety of reasons, this version, though interesting, does not work. After this, I will turn to particular examples from compositional biology to provide some general lessons that can be gleaned regarding explanation and confirmation in compositional biology.

5.6.1 A Syntactic Version of Theoretical Unification

Now that we have discussed theoretical explanation in formal biology at some length (as model merging and diversification), let us turn to theoretical explanation in compositional biology. Is there anything analogous to unification or even (theoretically-motivated) model elaboration in compositional biology? This is not an easy question, considering especially that theoretical activity in

compositional biology is closely related to empirical work. My argument is that there are general (although not "unified") models in compositional biology that guide empirical activity, but that the bulk of the model diversification work lies in working out the causal details empirically and then representing them in specific theoretical models. Thus, there is a constant feedback effect between theoretical model explanation (i.e., diversification) and confirmation. Furthermore, propositional and material models differ in their properties in this case. In this way, then, although there are theoretical "resources" for model diversification and causal explanation in compositional biology, there is relatively little "pure" theoretical explanation (which is a theoretical activity). Before I provide my view, however, I need to consider one last view on theoretical unification in science, which could perhaps have been, but turns out not to be, pertinent to compositional biology, Kitcher's account.

Kitcher adopted Friedman's idea of the close relationship between theoretical explanation and unification. He was critical of Friedman's exact account already in 1976 where he claimed that his own sketch "enables us to see why Friedman's theory goes wrong, and it may point the way to something better."[568] I will argue that Kitcher's account is the weaker one for a variety of reasons[569]: (1) it is far from scientific practice, (2) it does not provide a sharp account of unification, and (3) it does not provide us with precise conditions of

[568] See Kitcher 1976, p. 212.
[569] Friedman's account of unification is, as we have seen, useful for a variety of other reasons besides fleshing out the relationship between theoretical explanation and unification, such as providing a counterpoint to Cartwright's account of abstraction. I hope to have shown that a comparison of Friedman's and Cartwright's respectively cogent and scientifically-honest positions on this and other matters (e,g., modeling and the role and nature of laws), while designed for problems in physics, are highly instructive for thinking about biology, formal and compositional alike.

application that would indicate to us how to actually *employ* it in particular cases. Before I address these problems, I will provide an outline of Kitcher's sketch.

Succinctly put, Kitcher's thesis is that "a theory unifies our beliefs when it provides one (or more generally, a few) pattern(s) of argument which can be used in the derivation of a large number of sentences which we accept."[570] Kitcher thus needs, and has, an account of what "argument patterns" and what the "sentences" are. Note that his focus is on these *logical* structures, rather than on mathematical model structures of *actual* sciences, such as formal biology. Thus, although as Lloyd has pointed out in conversation, his argument patterns can be thought of as "model-types," they are formalized in syntactic rather than semantic terms. An argument pattern is a particular reasoning "mold" that allows us to represent a particular theory (say Newton's laws of motion, or Darwin's theory of natural selection). This reasoning mold consists of sentences with (1) logical operators and (2) non-logical dummy variables standing for observational states. The highly abstract sentences of the argument pattern themselves stand in a particular relation to one another (often in a sort of nested syllogistic structure). The intra-, and inter-, sentential logical operations, when "filled in" with particular conditions and observations for a particular case, provide the *explanations* of that case – i.e., the observational sentences can be deductively inferred from the argument pattern. And, according to Kitcher, a unified and unifying argument pattern is one that allows us to derive a large number of observational sentences. Kitcher calls the set of argument patterns the "explanatory store" (abbreviated "*E(K)*") and the set of observational sentences,

[570] Kitcher 1991 (1981), p. 333.

that should be inferred (subsumed?) by this explanatory store, the "accepted sentences" (abbreviated "*K*").[571]

Kitcher's account does have weaknesses. First of all, it will not seem to work for formal biology since there the language of mathematics, as we have seen in great detail, is employed rather than the language of logic. This is why a semantic interpretation of theories works well for, say, mathematical evolutionary genetics. One of the advances of the semantic view is that it provided strong and cogent arguments for abandoning the logical formulations of the logical positivists for mathematical expressions of actual science.[572] But perhaps Kitcher's view could work for compositional biology. Are non-mathematical "logical" arguments not common there? Explanations, causal and theoretical alike, are not formalized in axiomatic logic in compositional biology, although non-mathematical models are prevalent. It is these *models*, not *logical argument patterns*, that are ubiquitous in the explanatory activity of compositional biology. Put differently, if we pay close attention to the scientific practice of theory development in compositional biology, focusing on models and modeling (tied closely to empirical activity) seems more useful than emphasizing logical reconstruction.

Second, Kitcher's point is to claim that explanation is about *unifying* explanatory resources. There certainly are prevalent explanatory resources in compositional biology (such as basic assumptions about how mechanisms work, or what genealogy is), but there is often debate about what terms and resources should be employed in particular explanations (e.g., "homology"); this debate

[571] Kitcher 1991 (1981), p. 332, cf. 1993, pp. 82-84.
[572] See also Lloyd 1988, pp. 22-23 for her criticism of Kitcher's endorsement of the Syntactic View.

stems from the different explanatory strategies employed by different theoretical perspectives of compositional biology (e.g., comparative morphology, physiology, and developmental biology in organismal biology). Thus, there is little *unification* of explanatory resources, even narrative ones, in compositional biology. Unification of explanatory patterns, whatever that exactly would mean, is not a goal in compositional biology.

Furthemore, Kitcher's account of unification is vague. He claims that a theory unifies our beliefs when it employs one or a few argument patterns. The beliefs are presumably the observational sentences that we have come to accept. The argument patterns are probably those that allow us to derive large numbers of observational sentences, hopefully in a very unified way. So a theory unifies our observational sentences by employing argument patterns that unify our observational sentences. The question now arises as to how this unification actually takes place. If it is simply the arbitrary union of many observational sentences, then it is unclear how that happens. Perhaps it occurs by independent derivability of sentences, but it is unclear how we would go about reconstructing science in terms of logical derivability of sentences – this was precisely the failed project of the positivists. Or perhaps Kitcher has some logically "weightier" notion of unification in which argument patterns with more unifying power have some intrinsically more robust logical properties. It is unclear what these would be. Whether Kitcher defines unification in terms of achieving a union of many[573]

[573] How many? Unified in what way? Simply that they derive from the same argument pattern? Then we have a circular definition of unification – sentences are unified when they derive from the same pattern, an argument pattern unifies when it derives many sentences.

observational sentences or in terms of the logical "strength(s)" of argument patterns, or both, his account is, admittedly, vague.[574]

Kitcher's account is unclear both in terms of its central concepts and in how philosophers should actually *implement* it in particular cases. An important aspect of this problem is that it is unclear *how* we should actually individuate the sentences and argument patterns to which he refers. There are many ways to carve up the world into observational sentences and there are many, indeed infinitely many, logically-equivalent argument patterns. One way in which the semantic view solved this problem was by appealing to scientific practice, by *naturalizing* the model interpretations in the actual practice of science and, thereby, picking the valid from the invalid model interpretations (i.e., state space interpretations and interpretations of laws). Kitcher cannot appeal to such a solution because his analysis is not naturalized in scientific practice. Unfortunately, other advocates of Kitcher's account have not succeeded in specifying his views.[575] For these problems, among others, Kitcher's syntactic account of the role of unification in theoretical explanation, though interesting and general, does not seem to work for either formal or compositional biology.

[574] In contrast, Friedman provides a clear account of unification in that (1) it is tied to decreasing "cognitive burden" (an independent psychological notion) and (2) it has a direct and clear relation to scientific practice (i.e., mathematical modeling) and the actual implementation of unification in theoretical physics.
[575] See, for example, Jones 1995, 1997; Skipper 1999. Skipper's account brings in the notion of mechanism and "mechanism schema." Skipper accepts Kitcher's program, while criticizing some aspects of it.

5.6.2 On Theoretical Explanation and Confirmation in Compositional Biology

Now that I want to turn to whether theoretical explanation is at all possible in compositional biology (whether it be in terms of unification or even in terms of model diversification, as discussed above for formal biology). In order to address this issue, I want to turn to examples of pairs of models (general and specific) in each of two research areas the genomic regulatory systems described by Davidson and the Krebs cycle.[576] I deal here particularly with propositional non-mathematical models. As we saw in Chapter 4, the status of remnant models as clear cases of models is unclear.

I explained these two pairs of models in detail in Chapter 4. I discussed how these four models satisfied the general properties of models I had inferred from considering the semantic view, the mediating model view, Friedman's and Cartwright's views on abstraction, and non-standard views regarding models, such as that of Griesemer and Downes. Here I want to elaborate on how the two models, general and specific, *relate* to one another. Can the specific model be *derived* or, even *elaborated*, from the former in any way analogous to model diversification in formal biology? That is, can theoretical explanation be used to specify details of the more concrete model that are then tested against the empirical world?

Davidson provides a general "cartoon" model as well as a specific model, for the *endo16* gene, of *cis*-regulatory systems. The cartoon model introduces some general objects (e.g., adjacent cells, lineages, modular genetic regulatory elements, basal transcription apparatus) and processes (e.g., spatial repression,

[576] Appendix Figures 4.4-4.7; 4.9, 4.10, respectively.

cell signaling, cell cycle) and notes how these objects and processes interrelate and respond to one another. The cartoon model captures *general* aspects of transcription regulation via structural DNA modular elements. It informs the reader what kinds of objects and processes biologists look for when they investigate the concrete causal processes of gene regulation. But the actual research occurs not primarily by model manipulation, but rather by empirical research activity in the laboratory. Model *diversification* occurs primarily by empirical manipulation (which confirms the models and greatly biases their further development).

For example, the details of the structure of the *endo16* gene are discovered by empirical research. A specific model of that gene is then produced, with 6 modules.[577] However, this model is not derived or even elaborated from the more general model. Certainly there are basic types of processes (e.g., spatial repression or cell-cycle control) that apply here as theoretical resources [see Figure 3. (5.2)], but the details of the extent, nature, and interaction among these processes could not in any way have been inferred from the general model without empirical research. That is, there are no theoretical manipulations that could have produced the more specific model, as there are in formal biology.

Let us now consider the opposite direction of theoretical explanation, model merging. Is there unification in any way here? I would venture to say that although there is useful abstraction of concepts and terms in the models as we generalize, there is no sense in which a *useful* unification of the *actual* models occurs. That is, there is no way in which we can say that the most general model (the cartoon model of Appendix Figure 4.4) somehow *embeds* the more specific

[577] Appendix Figure 4.5, B.

model (of *endo16* or whatever gene we are considering). There is no relation of necessity or "enriched theoretical information," such as theoretical assumptions, in the higher-level model. The cartoon model does not somehow contain, or refer to, information that will allow us to derive the more specific models, as was the case on Friedman's account of model embedding, and incremental and creative abstraction in which the general model contained its own criteria of applicability.

Let us now turn to the pair of models of the Krebs cycle. Here the two are really just different levels of resolution of *presentation* of a process that is already known. In the case of Davidson's model the more general model both guides empirical research and does, in a sense, include or describe, a whole host of distinct specific models. In the Krebs cycle, however, the "general" model simply depicts the number of carbons associated with the molecules present in it and the energy-rich products of the cycle. The specific model shows every step in great detail. But there is no difference in process-type referred to between the two models. Perhaps the more general model has didactic advantages in showing a beginning student the overall structure of the model, but it is not a more general model in the sense that it unifies or embeds, in any way (actually or potentially) the more specific model.

Having said this, the models of the Krebs cycle do depict, in different ways, the causal capacities of the components to which they refer. The general model provides a sense of the "flow" and general partitioning of the whole processual system. The particular model indicates, complemented with textual narrative, the causal capacities of specific components, though not the enzymes.

We have seen that one way in which unification in model merging *fails* to happen in compositional biology is because the "theoretical world" does not

behave in a way analogous to formal biology. In part this is because it has less structure, mathematical *or* otherwise, and does not exist independently as it seems to do in formal biology.[578] That is, modeling as a theoretical activity independent of empirical activity almost never occurs in compositional biology (recall, though, Corey's methodology in biochemistry), where constant reference to the concrete part-organization of integrated and hierarchical systems is the modus operandi. Another reason for the failure of unification in compositional biology is that objects have many different causal capacities and there are many kinds of objects (even different theoretical perspectives that individuate the objects). It is not possible to *usefully* unify objects, properties, and relations of different types under one (or a few kinds of) type(s) – too much of the qualitative uniqueness of the objects, properties, and relations (and capacities as properties of objects) is lost. In formal biology, there are relatively few kinds of objects, properties, and relations; there, unification of them is useful and—this is a different point—*precise* and *clear*. Recall that the absence of useful unification is one issue that I referred to when I stated that "complex" abstraction prevailed in compositional biology.

There is, however, one, important case in which one model explains another model: some of the hierarchies appealed to in compositional and functional explanations arise from descent with modification. That is, the theory of evolution, to an extent, explains biological hierarchy. Of course, there are hierarchies that are not directly a product of lineages (e.g., while cells and organisms are genealogical units, *organs* are not direct genealogical units), but

[578] Furthermore, it is also difficult to think about how *material* models would be unified or embedded. What would unification of material entities be?

many hierarchies are. Furthermore, *all* biological hierarchies can be given an evolutionary narrative (explanation?) at some level of removal. There is the question of whether we here have a theoretical explanation: the theory of evolution, in general, explaining the existence of biological hierarchies. To an extent it seems that it is. But it certainly is not a unification or an embedding of one theory into another in a clear theoretical space. It is a different kind of theoretical relation: one theory *explains*, by explicit appeal to a material-historical process, the existence of the phenomena of the other theory.

Let us now turn to the issue of model confirmation in compositional biology. This is a difficult issue, and here I will only provide a sketch of it. Given that *theoretical model manipulation in compositional biology is intricately tied, in a feedback process, to manipulation of concrete systems* (which continually *confirms*, or *disconfirms*, the models developed/diversified), propositional models are not built completely and independently (like Diana from Zeus' head, or like most models in formal biology) to be subsequently confirmed by concrete systems. Models already contain the empirical information that allows them to "match" the world. And they can be altered with epistemic impunity.[579] That is, there is no cost to changing representations, in an ad hoc fashion, in compositional biology. Certainly this cannot be done in formal biology, where all sorts of theoretical assumptions, including those regarding rules of mathematical manipulation, must be met. Although, in compositional biology, many theoretical assumptions regarding kinds of objects and processes also *aid* in the construction (and initial lay-out) of the model, at various levels of meaning structures, informative

[579] On the other hand, altering a model in formal biology requires examining the assumptions of the model and searching for new theoretically-consistent ways to alter it.

presentations of how parts are structured and articulate processually are constructed (i.e., theoretical explanation) based on knowledge, acquired empirically, of causal mechanisms (i.e., confirmation through empirical manipulation). The representations can be changed without much cost. Independent theoretical model development, followed by empirical testing (confirmation), is not the operative image of confirmation in compositional biology.

But *aspects* of the models are developed, or at least *postulated*, abstractly and subject to confirmation. For example, the robustness of the various theoretical assumptions [e.g., the theoretical organization of the diverse kinds of (structural and processual) parts abstracted in a particular science, such as developmental biology], is subject to empirical verification and revision. For example, consider how the discovery of *Hox* genes changed our theoretical notions of what regulatory genes were and how they worked. Before the discovery of those genes we certainly did have strong and independent theoretical notions of gene regulation.[580] But they were altered in the face of new empirical information. Recall also Bramble and Wake's tetrapod feeding model. Fifteen years after its initial articulation, the *basic* framework (i.e., the periodization of the feeding process and the partitioning into the muscle groups and head parts) is still used.[581] This is a case where the theoretical assumptions (i.e., the framing) of the model have been confirmed.

In general, model confirmation is not the same concern in a science where the details of the theoretical models are *unavoidably* worked out empirically,

[580] E.g., the work of Waddington and Goldschmidt, as well as Davidson's early work in the 60s and 70s.
[581] Schwenk 2000b.

rather than mathematically. But *aspects* of the model, such as the assumptions that go into the model-building are subject to confirmation.

5.7 Consequences of My Analysis: Two Different Pictures of Explanation and Confirmation

Models clearly play an important role in both forms of biology. In their roles as representational theoretical objects that serve as grounds for explanations and for guiding further research, they are crucial in scientific theorizing. As we saw in Chapter 4, the models of both formal and compositional biology share the same general properties (although material models do not perfectly fit a *classic* account of models). In both cases, for example, there is a matching or "subsumption" relation between models and the world – that is the *definition* of causal explanation. But the nature, extent, and emphasis on this relation varies widely between the two types of biology. This relation is central to formal biology, while it pales in light of a more central feature of the picture of explanation favored by compositional biology. I will conclude this chapter by summarizing, and presenting diagrammatically, the respective pictures of explanation favored by each type of biology.

In formal biology, causal explanation occurs when a concrete case can be shown to be subsumed under a law or shown to match a (law-based) model. There are important similarities between the D-N model of explanation,

Cartwright's and, say, Salmon's causal accounts of explanation[582]. In all cases, a law or model, expressed mathematically, is shown to *pertain* to a particular case (whether it be in a subsumptive or a matching relation). Despite its deflationary attitude to explanation, this "matching" picture of explanation is also central to the semantic view on models. The (phenomenological) law or model at hand has itself been articulated theoretically, by manipulation, from assumptions and mathematical relations among simple properties. The important point is that the models are interpreted as movements of "simple" mathematically-defined properties of (simple) objects through state spaces. Cummins captured an important and large subset of such theories in his notion of "transition theories." The explanatory act lies in showing that the concrete situation is indeed a *case* of that law or model – for, if so, then the theoretical structure (a bit of the "theoretical world") involved in deriving the law or model has been shown to be relevant to, account for, and "rule over," the particular case (a bit of the "empirical world"). Despite an extraordinarily rich set of differences in the ways of actually cashing out this *picture* of explanation, there is a basic common pattern to it [See Figure 2. (5.1)].

In formal biology, theoretical explanation can occur in a variety of ways. Most of the time there is a non-deductive diversification ("elaboration") of models. Sometimes, however, a formal embedding and a theoretical unification of models is possible (e.g., Price's Equation and various particular models of natural selection); such cases are important theoretically in formal biology.

[582] This is not the place to explore Salmon's causal account, but he clearly believes that there is an important relation between causes and laws. For example, he notes that "Causal processes and causal interactions seem to be governed by basic laws of nature…" (Salmon 1984, p. 179)

Whether diversification is deductive or not, theoretical explanation clearly happens in formal biology in the form of *theoretical mathematical* model diversification.

In compositional biology, models (that are not laws-based!) play a very different role. They basically *represent* parts of hierarchical systems (a crucial aspect of compositional biology) and summarize the causal capacities of parts. Explanation does not (primarily) happen in showing how the model rules over particular concrete systems. Although there is a "matching" relationship between theoretical models and concrete systems, this relationship is much weaker and less important in compositional biology as compared to formal biology in large part because in the latter, but not the former, there is (1) much theoretical manipulation and (2) there are theoretical constraints on the theoretical manipulation. Models in compositional biology are highly flexible and responsive to empirical discoveries – they can be changed with epistemic impunity. There is no rigid theoretical structure, mathematical or otherwise, to which they must conform.

In compositional biology, causal explanation happens when the hierarchically-organized concrete parts with their respective capacities have been captured in the appropriate way in the model. This happens *after* the interplay of (limited) theoretical explanation (model articulation) and confirmation of models. Although theoretical models mediate the explanatory act, the models are developed primarily through the manipulation of concrete systems, rather than of theory. That is not to say, however, that models do not *guide* research – they do, but capturing the right causal structure is informed primarily by empirical work, not in formulating the right theoretical models. Furthermore, the "right

theoretical model" is one that accurately represents the structure and interactions of the parts and their capacities [See Figure 3. (5.2)].

Explanation, both causal and theoretical, as well as the confirmation of the *models* that perform such explanations, are central to both kinds of biology. The structure of explanation, and the role confirmation plays, is *very* different in the two, as I have argued, but in both formal and compositional biology, models and modeling, which share general properties across the two kinds of biology, are absolutely crucial.

Figure 2. (5.1) The *Picture* of explanation in formal biology.
(Influenced by Cartwright and Friedman)

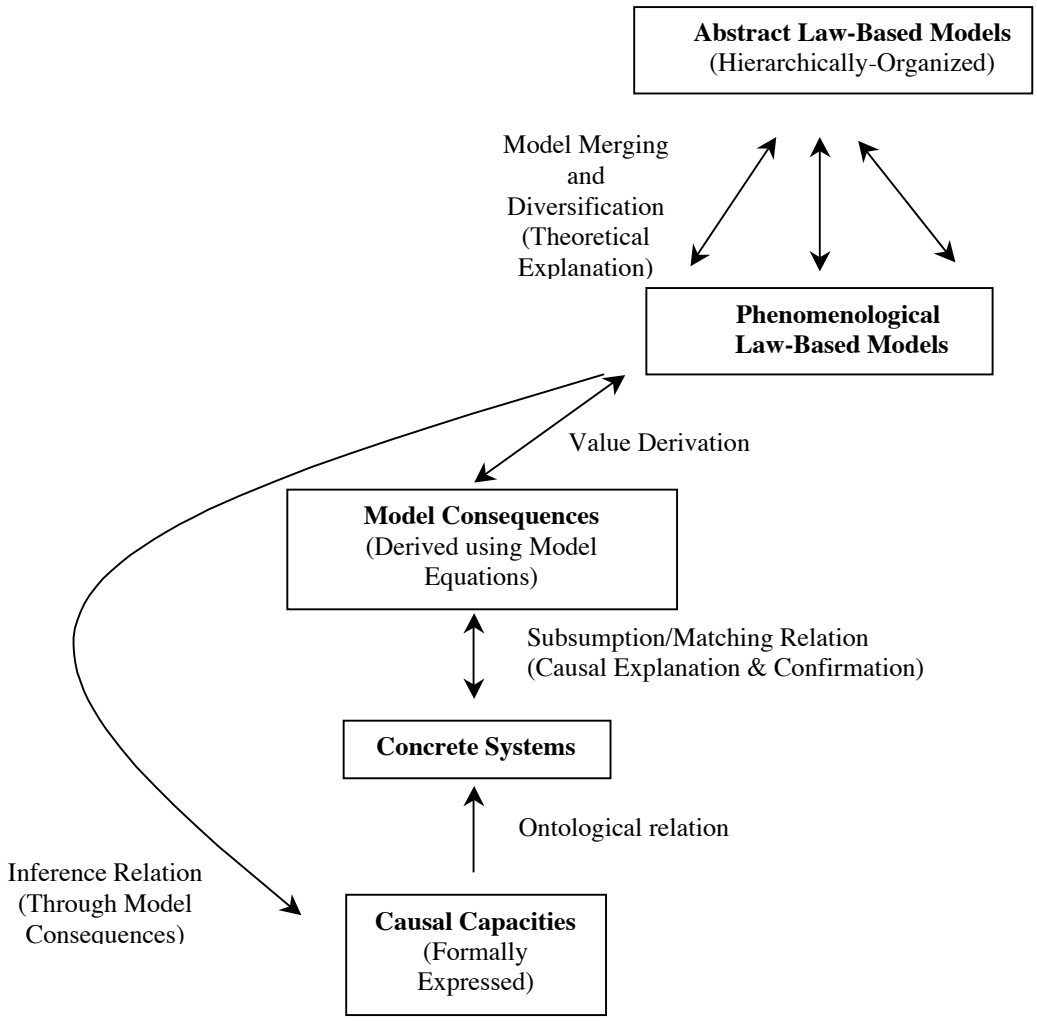
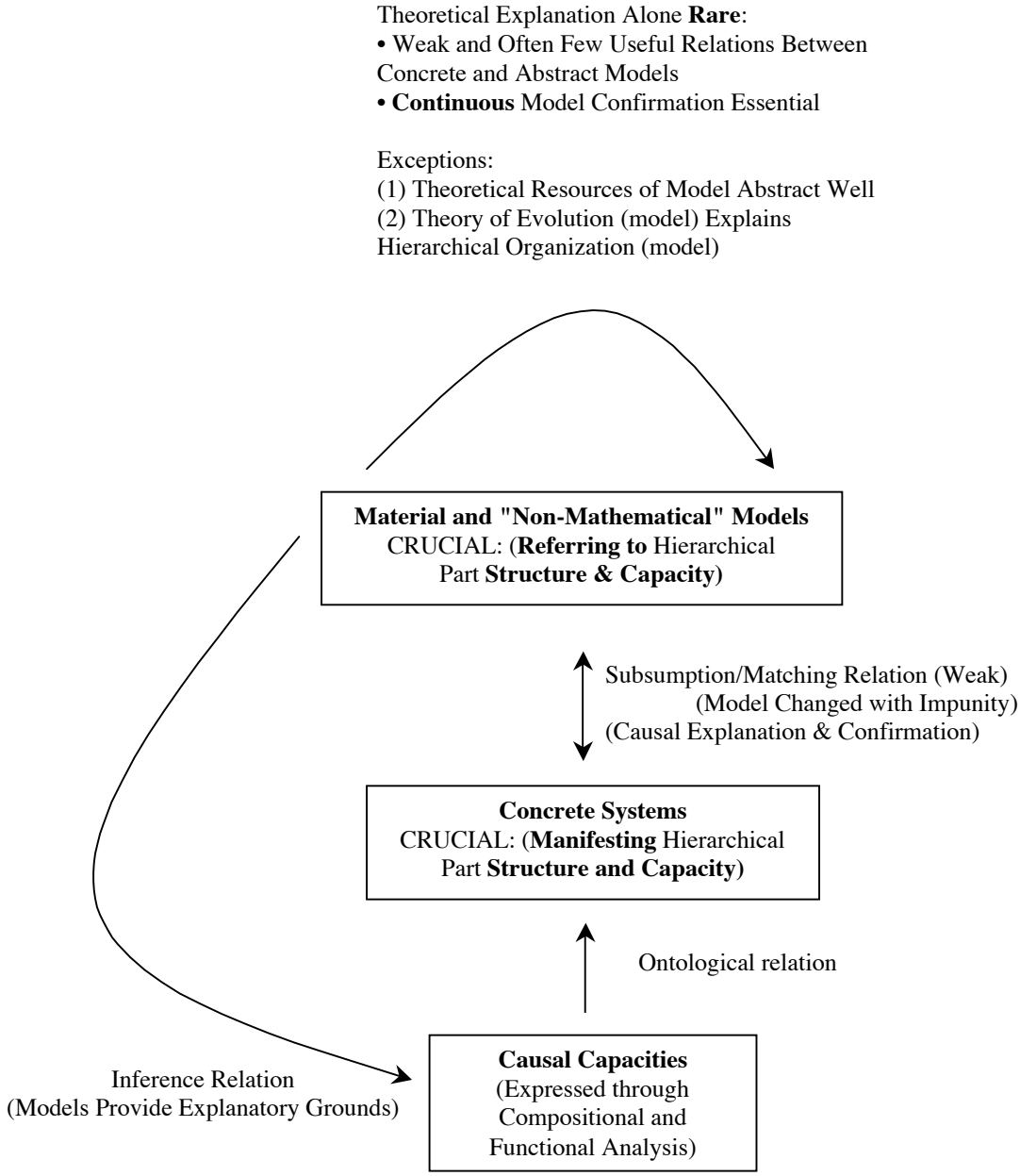


Figure 3. (5.2) The *Picture* of explanation in compositional biology. (Influenced by Cummins as well as Kauffman and Wimsatt)



Conclusion

In this dissertation I hope to have developed a philosophical framework with which to analyze compositional biology. Abstraction, models and modeling, explanation, and confirmation are so clearly different in compositional biology that we require a new mode of expressing them in philosophy of science. Furthermore, this new picture can then be employed to comprehend disciplines in biology rarely studied and understood. I hope to have engaged in both directions (i.e., science to philosophy and philosophy to science) in this dissertation.

In my "Introduction" to this dissertation, I motivated and presented, generally, my analysis and its conclusions. Throughout the dissertation, I have also consistently reiterated the conclusions, at a finer level of detail. Thus, I do not here want to restate my conclusions, but instead desire to point the way toward new directions and questions.

I believe that it is important to analyze compositional biology from a philosophy of biology vantage point, which I have not done fully in this dissertation, due to space constraints. Philosophy of biology and philosophy of science are concerned with distinct topics and my analysis of issues in the latter did not explicitly address problems pertinent to the former. I will list three outstanding issues highly pertinent to compositional biology and philosophy of biology. First, it is important to understand how mechanisms and functions

interrelate. No systematic analysis of this relationship exists in the philosophy of biology literature. Second, a more detailed exposition of hierarchy theory using the distinction between formal and compositional biology would also be useful. Hierarchies can be, and have been, presented, and analyzed, in a variety of ways and I believe that my distinction and framework can give us purchase in explicating, and categorizing, these various ways. Third, analyses of extremely understudied theoretical perspectives in compositional biology, such as physiology, functional morphology, and biochemistry would provide welcome comparative studies to complement the current emphasis (in so far as attention *is* devoted to compositional biology) on developmental biology and molecular genetics. In the context of my distinction, then, there await a host of projects in philosophy of biology.

There are also some outstanding deep problems regarding the relationship between mathematics and concrete biological phenomena. First, we need a rigorous and detailed account of mathematical abstraction, which I have only hinted at here. Many unanswered questions remain. What must the world (including our minds and social organization and social practices) be like in order for us to be able to abstract mathematical properties from the objects around us? What exactly have we captured when we find relations between these (kinds of) properties? What is the hierarchical structure of mathematical properties, and meaning structures built from such properties?

Second, we require a clear account of the relationship between two theoretical structures—mathematical and "qualitative"—pertinent, respectively, to formal and compositional biology. As in theoretical physics, mathematics clearly provides the grounds for a rich theoretical structure in formal biology.

But I hope also to have shown that there is a large amount of theoretical structure in compositional biology, captured in, for example, the compositional and functional analysis (sensu Cummins) of a system. Furthermore, there is a clear set of theoretical concepts and explanatory resources in compositional biology, even if the theoretical structure is almost certainly *less* organized than a mathematically-driven structure (and why should *that* be the case?).

Third, the different types of models and causal concepts in the two kinds of biology indicate that there is *elision* inherent in the oft-asked question: "What is the relationship between mathematical models and causal mechanisms?" First, there are many kinds of models, or meaning structures, present in the theories pertinent to formal and compositional biology. Second, what is considered a cause, and what is deemed an appropriate *presentation* of a cause, varies tremendously between (and even within) the two kinds of biology. Furthermore, "mechanism" also has multiple meanings and instantiations in the two kinds of biology. Hence, we cannot simply assume that models are mathematical, while causes and mechanisms are concrete; the question above is too simple! We still need a complete account of the various possible relationships between modeling practices *and* the conceptualization of causes and mechanisms in biology.

Now that I have discussed the possibilities of future work (1) in philosophy of biology and (2) topics surrounding mathematics, let me turn to an "internal" problem concerning my framework. I have presented five distinctions pertinent to the two kinds of biology (the fifth one is their different views on explanation). There is a question concerning whether these are *a priori* criteria that allow us to differentiate a particular science as either formal or compositional, *or* whether we already have an idea of which sciences are formal

and which are compositional, and then from that we infer the properties that sciences of a kind share. That is, do we use the criteria as *a priori* tools for judging a biological discipline as compositional or formal, or do we already know what kind of biology it is and, instead, abstract the criteria as properties of that discipline as a prototype? Although my answer may not be satisfactory and clearly requires further evaluation, it seems to me that *both* of these aspects are important. In developing this dissertation, I have certainly employed both epistemic directions (i.e., used the criteria in an *a priori* manner, as well as abstracted them as properties from prototypes). This is consistent with my view on science – in both science and philosophy, epistemology/philology/sociology interact intimately and inseparably with ontology.

The last point for future work that I want to allude to here concerns issues of relativism and disunity in science. Given these two forms of biology, we need to examine the possible relationships *between* them. Are they unifiable? If they are, then under which conditions are they unifiable? If they cannot be unified, what does this tell us about the nature of scientific theorizing? If we can really construct and define theories and theory-types that are irreducibly different, is the cause to be found in our epistemic and social efforts, or in some sort of ontological disunity, or both? And, in considering, more specifically, the varieties of compositional biological sciences, what do we make of its different and incommensurate perspectives existing side-by-side? What lessons regarding realism and relativism s can we draw from this? Does variety give us strength and robustness? Despite, or maybe precisely *because*, of variety in theory and theorizing, can we still dream of an ontologically grounded grand unified biological theory?

Appendix A: On Laws and the "Law-Model" Equivalence in Formal Biology

A.1 The Presence of Laws in Formal Biology

In an important set of criticisms against the logical positivists, proponents of the semantic view of theories have argued against the notion of laws both in science in general, and certainly in biology. In his important discussion on the topic, van Fraassen argues against the views that laws (1) are universal in temporal and spatial scope, (2) state what is necessary (and, perhaps, are themselves necessary), (3) are required for explanations, (4) provide the grounds for prediction and, therefore, through experimentation, are the theoretical units that are (dis)confirmed in science, (5) hold counterfactually, (6) are objective.[583] I will not here rehearse van Fraassen's generally convincing arguments. It certainly seems that the *traditional* conception of laws as held by metaphysicians and epistemologists such as Armstrong, Dretske, Tooley and, more recently, Leckey and Bigelow[584] will not stand up to scrutiny. Such laws are also often written in first-order logic rather than in the scientifically-honest language of mathematics. Do these criticisms mean that we must abandon all desire and expectations to find a scientifically useful and potent notion of law?

Other proponents of the semantic view, besides van Fraassen, certainly think so. Let us look at a few other arguments against the notion of laws. Giere argues that the relationship between the equations of any domain of science and

[583] Van Fraassen 1989, pp. 25-36.
[584] Armstrong 1983; Dretske 1977; Tooley 1977; Leckey and Bigelow 1995.

the world is "indirect" – it is mediated by an abstract system, a model.[585] A model in the semantic view, as we saw at the beginning of Chapter 4, provides for a specification of a family of curves through a particular state space. Which model then *applies* to the situation at hand is determined by the empirical facts. So, Giere argues, models (and the equations from which they are constructed) are not so much true or false, they are, rather, applicable or not to the particular empirical situation.[586] The *theoretical hypothesis* linking the model and the world may be true or false, but the model is not, by itself, true or false.[587]

This may be a useful way of analyzing modeling, but I do not think that it is a consistent argument against the role of laws in science. Let us first analyze Giere's claim that the model is neither true nor false. There is a fine line between breadth of applicability and the degrees of restriction of truth. Let us say that a model turns out to have *no*, or *next to no*, applicability. Would that not, intuitively at least, give us some sense that the model is "false"? Certainly on a pragmatic definition of truth it does – a model that does *no useful work* in scientific theory, including in theoretical or causal explanation, is false on a pragmatic evaluation. But even on a correspondence definition of truth, the model seems to be false: it simply fails to match any, or a significant number, of (data models of) real systems and, thus, does not *represent the world accurately*. Under either criterion of truth, then, an inapplicable (or very narrowly applicable) model is false. Note, though, that a false model does not imply a *meaningless* model – a false model still has empirical meaning, it is just that the meaning did not capture the right

[585] Giere 1999, p. 92.
[586] Giere 1999, p. 92.
[587] See also Giere 1997.

data model structure.[588] Thus, Giere's claim the irrelevance of truth evaluation of models is problematic and, though I can see the point that it plays in his picture, I shall not endorse his analysis.

My position, then, gives us the opportunity to directly compare the model—including the assumptions, methods, and equations that went into making it[589]—with the world (or, more precisely, a data model of the world). So we can *directly* assess the veracity (in pragmatic or correspondence terms, or both) of the claims that went into making the model. Now, it is clear, and Giere admits this, that an important aspect of numerous models is the "equations," such as "Newton's *equations* of motion [not his] *laws* of motion."[590] The issue under investigation, then, is whether such equations can be usefully called laws. We have already seen that the equations have empirical content that allows for evaluation of their truth (utility or correspondence to nature/data models of nature) – I argued against Giere's separation of applicability and truth. So the equations can be, and are, (relatively) true or false, from the point of view of causal explanation. As idealizations, we can only test the models constructed from the equations against the world in terms of the limited state spaces used in the models themselves. But these models can certainly match, or fail to match, to different degrees, cases they purport to causally subsume or explain within particular state spaces.

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[588] Using Giere, Thompson 1989 argues that "the empirical meaning of a theory is separate from the empirical application of a theory." (p. 72) This is true, but the *truth* of a theory (in terms of correspondence or utility) is distinct from the meaning and is related to the application, I would argue.
[589] All of which Friedman would claim is part of the "theoretical world" and which I claim is part of the dynamics occurring within the particular theoretical perspective guiding research in that field.
[590] Giere 1999, p. 91.

Furthermore, from the point of view of theoretical explanation, an issue that Giere does not discuss, the equations are "true" or "generative"— perhaps a better word for Giere's deflationary attitude toward explanation—when they are involved in generating a fairly abstract set of curves (in a state space with relatively few variables) which can *further* specify a whole family of more specific models with more elaborate state spaces (the same variables of the abstract model in addition to distinct sets of possibly overlapping variables). Thus, the "truth" of equations can be evaluated for both causal and theoretical explanation.

What we then have, under this view of both causal and theoretical explanation in a state space approach, is a view of the equations as relatively restricted to particular cases for which they are true (i.e., confirmed) – these equations are precisely the phenomenological laws that Cartwright discusses and from which phenomenological (sometimes considered "causal," at least in her 1983 book) models are built. The general equations that specify models that are highly generative of further models are what Cartwright and Friedman both call abstract laws. Equations can produce models, with laws of succession and coexistence as model properties, that match or fail to match the empirical domain.

The notion of "matching" is admittedly a difficult one. For Friedman, model confirmation happens to models at all levels of generality; general models, however, can only be tested with the few variables and parameters they include and, thus, a variety of concrete situations would be consistent with the model (although some might not be). Furthermore, he thinks that such confirmation is cumulative. Cartwright emphasizes that only highly specific models are tested. General models "lie" in so far as they do not allow us to match rich concrete

situations very easily to the model. As idealized models (with few parameters and variables and all kinds of *ceteris paribus* conditions), they are basically useless at matching and explaining reality or data models of reality. The locus (i.e., level of generality) of model confirmation is one real difference between Cartwright and Friedman. Note, though, that the notion that general models (and the laws which underlie them) cannot match reality is not itself necessarily an argument for the trade-off between explanatory power and generality – as Friedman points out, general laws may have *theoretical* explanatory power and thus, indirectly once they generate other more specific models, causal explanatory power (even if you believe that explanation is causal subsumption of concrete situations).

What we should take away from this is that the equations that go into constructing models can be directly evaluated in terms of truth content. Furthermore, there are some equations that build models at various levels of generality that actually cover a broad range of empirical cases, or that are also highly generative of further models that turn out to match many empirical cases, or both. These equations are true in many cases. They may not be *universal* in the sense that there may be cases for which they should hold but for which they do not (e.g., Mendel's laws of inheritance, although they are supposed to be "laws of inheritance" certainly don't hold for all cases of inheritance – consider the single bacterial chromosome). Furthermore, they may not be *necessary* – they may themselves have evolved, as Beatty correctly claims that Mendel's laws did.[591] And it is also difficult to assess their *counterfactual* explanatory power in fields like biology with so many explanatory factors and background assumptions. Let me now refine my question from above: does this mean that the equations whose

[591] Beatty 1980.

truth-value we use, and evaluate, through model building cannot be considered laws?

In recent years there have been a number of philosophers who have made cogent arguments in favor of the notion of considering these equations laws of at least some sort. For example, Robert Brandon has argued that the "contingent regularities of evolutionary biology have a limited range of nomic necessity and a limited range of explanatory power even though they lack the unlimited projectibility that has been seen by some as a hallmark of scientific laws."[592] The regularities of evolutionary biology serve more often in "how possibly" explanations rather than the more precise "how actually" explanations.[593] In the former kind of explanation, explanatory weight and the necessity of laws in deducing phenomena are fairly limited.

Concerning empirical generalizations in biology, Waters has made an important distinction between *distributions* and *causal regularities*. Mendel's laws or principles would be an example of the latter, whereas a listing of the taxa for which these laws or principles actually pertain would be an example of the former. Waters notes that causal regularities "exhibit many of the features traditionally attributed to scientific laws."[594]

Sober has, with most force, argued that there are indeed laws, and causal forces, in evolution.[595] Recently he has argued that *a priori* modeling in mathematical biology (e.g., the "definitional" status of Fisher's Fundamental

[592] Brandon 1997, p. S444.
[593] See Brandon 1990, pp. 176-184.
[594] Waters 1998, p. 6.
[595] Sober 1984.

Theorem or the Price Equation) is what provides us with the laws of evolution.[596] Thus, while only Sober endorses the usage of the term "law," these three authors all argue for the importance of some derivate of the concept of law in evolutionary biology.[597]

This too is what I hold. In order to count as scientifically useful and desirable, (true) equations, or "laws," need not be necessary, universal, and have full counterfactual-supporting weight. Admittedly the traditional metaphysical, as well as the classical positivist, conception of law does have to be abandoned if we want to understand, at least, biology, and possibly also physics (as van Fraassen argues). *But we would be throwing out the scientific baby with the*

[596] Sober further claims that "…when one tries to state an evolutionary law precisely, the result seems always to be an *a priori* model in mathematical biology. Why has biology developed in this way, whereas physical processes seem to obey laws that are empirical?" (Sober 1997, p. S467) I am not sure what to make of Sober's argument. Certainly there are numerous cases in evolutionary biology in addition to these, such as the Hardy-Weinberg equilibrium principle or the definitions of inter-generational gene frequency change due to selection or genetic drift, where mathematical *definitions* seem to play an important role in theorizing and model-building. But Mendel's laws or principles seem to be empirical generalizations. Furthermore, in some cases in physics, *empirical* generalizations are the law-like content of the theory, such as the law of universal gravitation which is an inverse-square law which could *possibly* have been an inverse-linear or inverse-cube law. But what about Maxwell's equations of electromagnetism or Einstein's time-dilation equation of special relativity? They also seem to be almost mathematical definitions (derived, admittedly, using some empirical content and assumptions – but that too is the case for all the biological models which have definitional *a priori* status). How stark is the difference between physical and biological (mathematical) model building here? And how stark is the difference between *a priori* and empirical models, laws, and generalizations? I am not sure.

[597] See Weber 1999 for cogent arguments for the presence in ecology of "evolutionary invariant generalizations which are law-like and at the same time distinctively biological." (p. 91) He, together with the three authors just discussed, are all arguing against Beatty 1995 who argues that all generalizations in biology are either those of physics and chemistry *or* are contingent outcomes of evolution lacking the universality and counterfactual-supporting power of laws. As implied, I think that Beatty endorses too strict a notion of laws.

metaphysical bathwater if we gave up entirely on the concept of "law." We need a new concept of law[598], and we are on the way to developing one.

Thus far, I have shown how Giere's separation of applicability and truth does not work and that we, therefore, *can* evaluate the veracity of laws (equations), and the models that they are involved in producing, directly against the world or data models of the world. There are different intuitions as to how such matching would occur, including intuitions regarding at what levels of generality it occurs, and whether we should focus on a confirmation relation or an (causal or theoretical) explanatory relation, or both. But the point is that the matching can occur, and is routinely done in (formal!) science, and it evaluates the utility or correspondence, or both, of laws and law-based models and the world. I have also provided further evidence, from the current philosophy of biology literature, for the importance of ("tempered"[599]) laws in biological theory and model-building.

There is another important set of reasons for maintaining a (revised) concept of law in biology, which stems from my contrast between formal and compositional biology. I believe that *laws, in their mathematical and, often, simplified, form that they exhibit, are a crucial aspect of formal biology, in direct analogy to much of theoretical physics, and in direct contrast to compositional biology.* Although the mathematical biological laws of formal biology are restricted in scope, necessity, and modality, they describe, allow for the modeling, and explain the behavior of the simple objects of formal biology. An aspect of the simple abstraction of that kind of biology, as explicated in Chapter 1, is also precisely

[598] See Cooper 1996 who argues that we must rethink our conception of law *and* our conception of theoretical modeling.
[599] To use a term Ken Waters has brought into the literature.

that there are well-defined and clear methods of abstracting models and laws in formal biology using necessary and sufficient abstraction (i.e., removing parameters and variables one at a time, or varying specific, and mathematically-defined precise assumptions). Such abstraction can happen precisely because the mathematical methods of formal biology are well defined and allow for simple functional (in the mathematical sense) relations between different (properties of) kinds of objects. There are few kinds of objects and properties (and relations) in formal biology. Furthermore, each kind of object and property (and relation) pertains to many equivalent concrete cases.

Keeping a concept of "laws" is also important for causal and theoretical explanation in formal biology. Causes can be precisely, neatly, and *sufficiently* captured in the equations of formal biology. Causal explanation occurs, in this kind of biology, as in physics, when the concrete system, or a data model thereof, is *subsumed under a law (or law-based model).* As I argue below, causal subsumption under law is analogous, even synonymous, for my purposes, to matching of data model and theory model in the semantic view – in both cases a matching is done and an explanation of empirical causes is produced. Another way to put the point is that the semantic view is a good way to *implement* a law-based view of scientific causal explanation – it is also closer to actual scientific practice. With respect to theoretical explanation, models can be *carefully* and *precisely* generated/derived (i.e., theoretical explanation) using these equations in a cumulative and interactive fashion. The equations of formal biology play very analogous roles to the equations of theoretical physics (and certainly in contrast to the methodology of model-building in compositional biology). As long as we are aware of, and avoid, the *metaphysical* burden, I do not think that it causes

philosophical damage to call such equations laws – at any rate, it reminds us of their *function* in model and theory building. This is why Friedman's model of theoretical unification is indeed pertinent to formal biology.

Laws play no role in compositional biology. In part this is because the complex objects of that form of biology have very different and unique kinds of causal capacities. The large number of distinct causal capacities of these complex objects cannot be captured by mathematical simplification – their qualitative variety is too "multidimensional." Furthermore, mathematical formalization is not the appropriate strategy to capture the complex qualitative abstraction pertinent to this form of biology.

Furthermore, the explanatory strategy in this kind of biology, as discussed in the Cummins section above, is very different from causal subsumption. Here compositional and functional analysis are crucial. Here the explanatory burden lies in the unique causal capacities of different parts of a system rather than a law or mathematical model that is matched to the data. This is not to deny that models, of some form or other, play an explanatory role in analytical explanation. It is just that the models are of a very different form and function than the models of formal biology. The models of compositional biology capture the hierarchical organization and the unique causal capacities of the parts of that system.

A.2 The "Law-Model" Equivalence in Formal Biology

I have thus far remained vague about the relationship between models and laws, mostly because I do not think that the difference is that significant with respect to my argument. That is, the arguments about matching theoretical structure to empirical structure, assessing truth, abstracting to different levels, and the like are not significantly different between a model-picture and a law-picture of theoretical structure. I fully realize that this is a highly contentious statement. As I will argue below, though, a law-based view is *not the same* as the "syntactic" or "received" approach to theory structure. I believe that a law-based view, where the laws are written in the *language of mathematics*, the scientifically actual formal language, *rather than* laws written in first-order logic with the inclusion of only logical operators and "theoretical vocabulary," is a perfectly reasonable way to describe theoretical structure in formal biology. I believe that the failure to distinguish between these two senses of a law-based view has led to much confusion in the literature and needless battering of the scientifically useful and respectable concept of (formal mathematical) law. Furthermore, this interpretation of a law-based view is perfectly compatible with the semantic view and with the views of some "syntactically-oriented" thinkers such as Friedman.

By laws, as discussed above, I mean the *equations* that *motivate* the models and which capture aspects of theoretical structure. These laws use variables that specify the state space employed and the parameters determine the shape of the curves through the state space. Now, the laws give the laws of succession,

coexistence (and interaction, which are relatively rare in formal biology). In most cases, the input laws (the equations) are manipulated and aggregated to form the "model equations" that describe the motion of the curve through the space (laws of succession)[600]. Some formalization, built from these model equations, of the allowable combination of variable values, under the particular parameters under consideration constitute the laws of coexistence. Hence there is a difference between the input equations and the model equations, but the latter is derived from the former together with various assumptions about the empirical system and about mathematical methodology. Thus, it is clear that the model structure is based on equations, or what could be called "laws."

Proponents of the semantic view happily use the (non-logical and non-metaphysically-loaded) notions of laws I too am proposing, although they do not make a distinction between input equations and model equations, which I think is important to capture the modeling practice of (1) starting with certain equations ("input equations"), (2) manipulating them, and then (3) forming the equations that determine the behavior of allowable values in the state space ("model equations" – laws of succession and coexistence). For example, Suppe argues that "The behaviors of physical systems are represented by various configurations imposed on the phase space in accordance with the laws of the theory."[601] He then provides examples of deterministic and statistical laws of succession, coexistence, and interaction. Lloyd concurs with, and uses, Suppe's analysis.[602] Thompson argues similarly by noting that "Physical laws, in this [the semantic] analysis, serve to select the…[laws of coexistence, succession, and

[600] Note the clear analogy here to Cummins' formulation of "transition theories."
[601] Suppe 1977, p. 226.
[602] Lloyd 1988, pp. 19-20.

interaction]."[603] He further notes, following Giere, that "laws do not describe the behavior of objects in the world; they specify the nature and behavior of an abstract system."[604] Laws, as I have described them, are certainly involved in model-specification. I differ with many proponents of the semantic view, however, as we have seen, with respect to the nature of the "truth" of the models and laws on which such models are based. Advocates of the semantic view claim that the relationship is indirect and that models are never "falsified," they are just shown to be inapplicable[605]; I claim that this is an unfair characterization of science and of language. Models, and laws, are falsified when they are shown to be inapplicable.[606]

An excellent example of a biologist capturing the relationship between laws and models can be found in Lewontin's 1974 book. He writes,

> [T]he problem of constructing an evolutionary theory is the problem of constructing a state space that will be dynamically sufficient, and a set of laws of transformation in that state space that will transform all the state variables. It is not always appreciated that the problem of theory building is a constant interaction between constructing laws and finding an appropriate

[603] Thompson 1989, p. 81.
[604] Thompson 1989, p. 72.
[605] Lloyd does not seem to be completely consistent here. Lloyd 1988, pp. 21-22, concurs with Giere's argument and notes that "This distinction [between theory and theoretical hypothesis] will play an important role in our discussion of units of selection." (p. 22) However, in her excellent discussion of the confirmation of evolutionary models she notes three ways of *confirming* a model – i.e., of showing isomorphisms between the theoretical models and data models [goodness of fit between model and data; independent testing of aspects of the model; and variety of evidence]. This seems to be an argument of the validity and truth content (here I am *not* using truth in a strong realist sense – I bracket that issue) of the model. Certainly her insightful discussion of confirmation implies that she is *evaluating* the correspondence relation between the theoretical model and the data model. This seems, on many accounts (even deflationary ones) of truth, to be a concern with the *truth* of the model.
[606] I take this to be the force behind, for example, Cartwright's idea (1983) of the laws of physics "lying" (i.e, being false) and Wimsatt's idea (1987) of false models as "means" to truer theories.

> set of descriptive state variables such that laws can be constructed.[607]

Lewontin takes the constant shift between laws and descriptive state variables to be an oscillation between theory and data. I do not think that this is necessarily the correct way to interpret the construction of a state space, which, as advocates of the semantic view point out, is itself a theoretical activity. It is interesting, however, that Lewontin's explication, compatible with the semantic view in a number of respects, immediately brings in the relationship between theory and data, thereby undercutting some of the claims that model-construction is completely *ideal* and *a priori*, and that applicability is a completely distinct notion from truth evaluation.[608] But what I primarily want to point out here is that, for Lewontin, laws are clearly important and he even defines their "construction" as a crucial, if not central, aspect of model-building.

Now, the notion of law I have been defending, and that the semantic view and Lewontin also use, is explicitly differentiated from the logical positivist notion of law, which is loaded with metaphysical baggage (cogently argued against by van Fraassen) and written in the language of first-order logic. These laws fall prey to a number of problems associated with the "received" view of scientific theories: the (in fact, infinite) underspecification of models by any set of laws, the inability to deal with unpleasant consequences of the arbitrary conjunction of laws as well as with the problem of relevance, the apparent explanatory symmetry of laws, the contentious distinction between observational

[607] Lewontin 1974, p. 8.
[608] He does, however, claim that: "The delineation of the prohibited and the possible is the function of population genetic theory. The revelation of the actual is the task of population genetic experiments… ." (Lewontin 1985, p. 11) Again, though, the actual is a *subset* of the possible and must be stated in the same currency or metric. Model generation is not completely *a priori*.

and theoretical terms and entities [laws, for the positivists, were supposed to contain only *theoretical* terms and logical operators], and the lack of fit between these logical formalizations and how scientists actually represent theoretical knowledge in their day-to-day theoretical activity. These problems arise as a consequence of the "laws" being written in first-order logic and being interpreted as directly related to the causal properties of concrete systems. As I hope to have shown, laws are related through *models* to the world, and first-order logic together with an advocacy of a strong distinction between observational and theoretical terms is invalid.

Appendix Figures

AF.1 Figures for Chapter 2

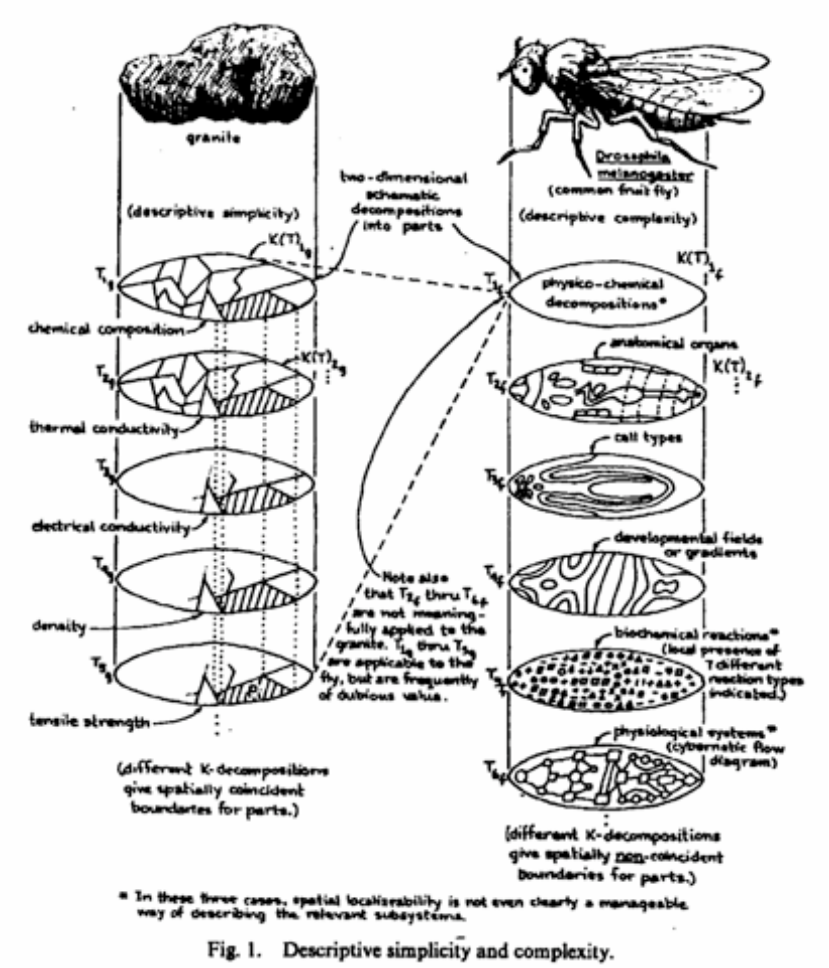


Fig. 1. Descriptive simplicity and complexity.

Appendix Figure 2.1. Wimsatt 1974, p. 71.

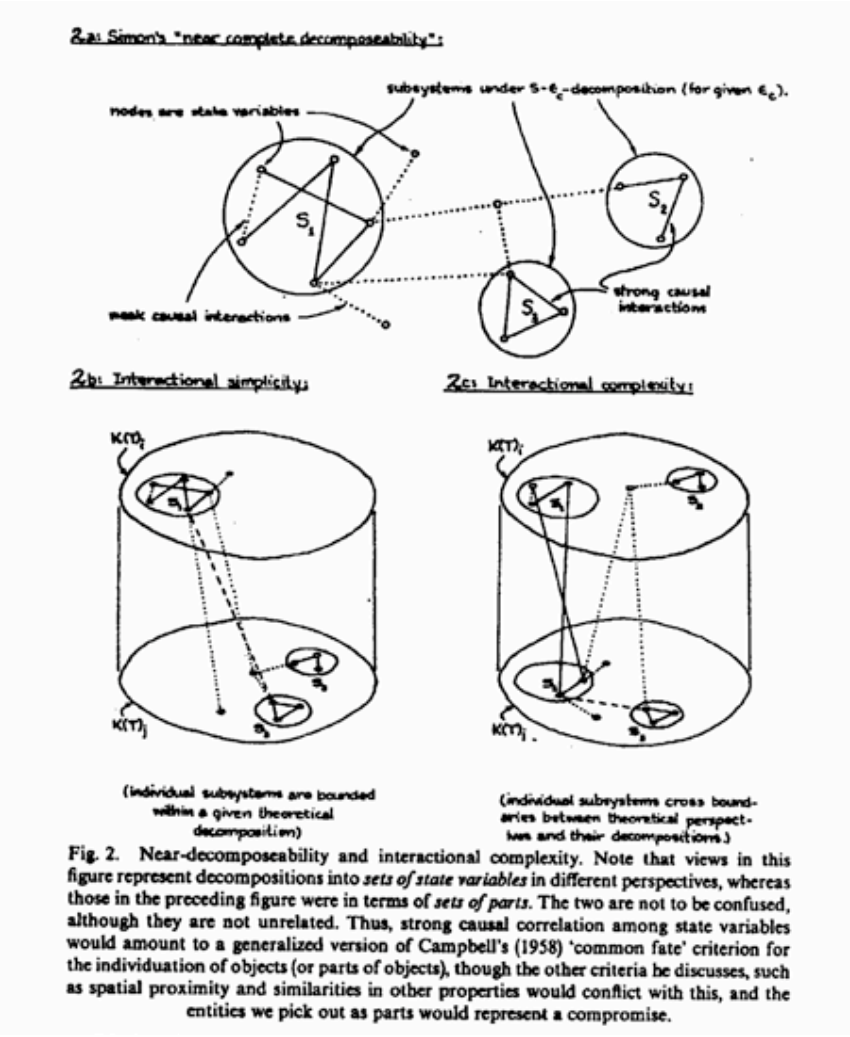


Fig. 2. Near-decomposeability and interactional complexity. Note that views in this figure represent decompositions into *sets of state variables* in different perspectives, whereas those in the preceding figure were in terms of *sets of parts*. The two are not to be confused, although they are not unrelated. Thus, strong causal correlation among state variables would amount to a generalized version of Campbell's (1958) 'common fate' criterion for the individuation of objects (or parts of objects), though the other criteria he discusses, such as spatial proximity and similarities in other properties would conflict with this, and the entities we pick out as parts would represent a compromise.

Appendix Figure 2.2. Wimsatt 1974, p. 73.

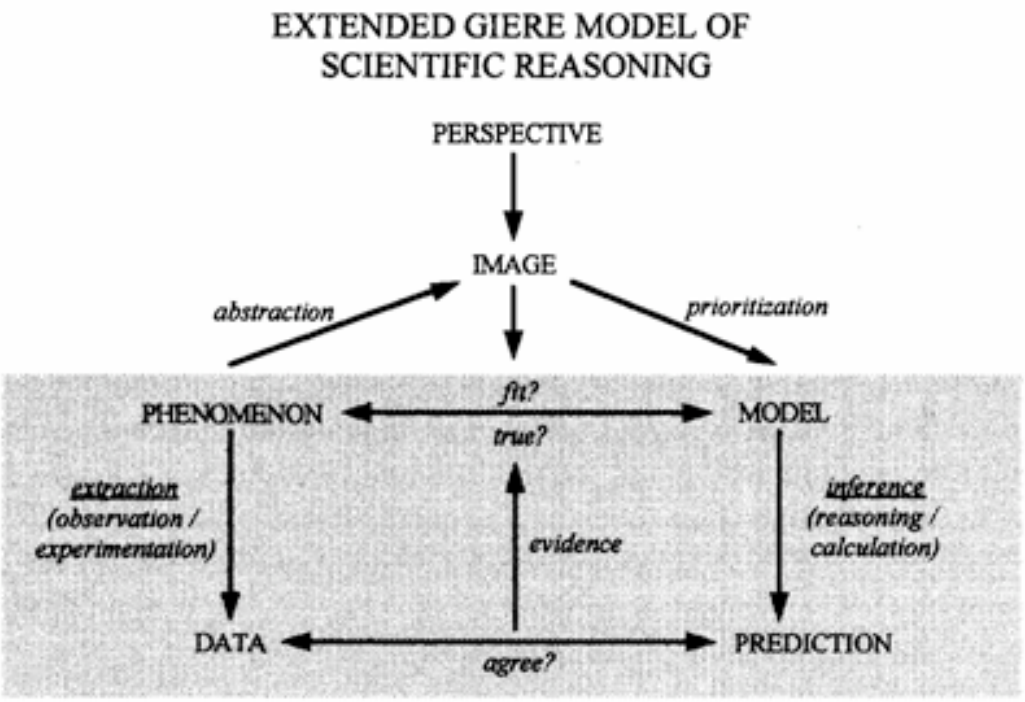


Figure 1. Extended Giere Model of Scientific Reasoning. Perspectives and Images are added to Giere's model of scientific reasoning. Shaded portion redrawn after Giere 1997, Fig. 2.9. Images, the concrete expressions of theoretical perspectives, coordinate phenomena and models by specifying relevant respects and degrees of fit. Coordination is important in both representation of phenomena by models and in planned interventions into nature to produce experimental and observational data and predictions. Images abstract from phenomena and also prioritize principles that go into explanatory and phenomenological components of models.

Appendix Figure 2.3. Griesemer 2000, p. 349.

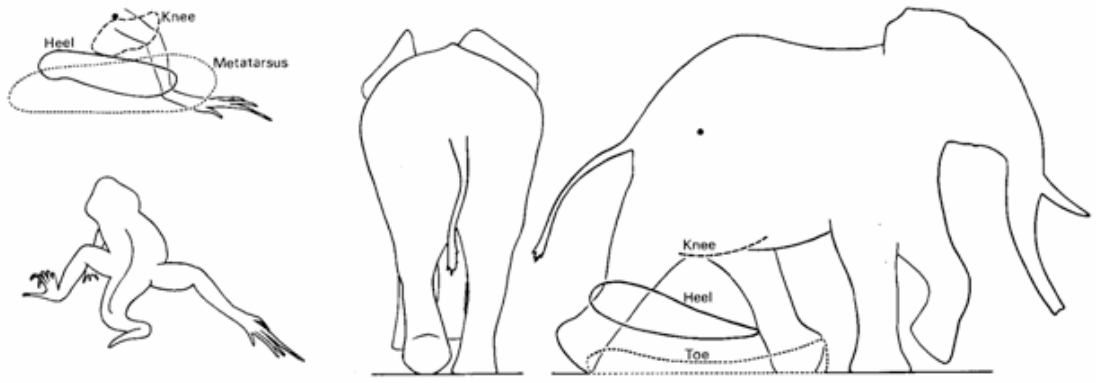


Figure 3-1 Posterior view of sprawling and upright postures and right lateral view of the arcs described by knee, heel, and toe (elephant) or distal metatarsus (lizard). (Lizard redrawn from Snyder, 1962.)

Appendix Figure 2.4. Hildebrand 1985, p. 39.

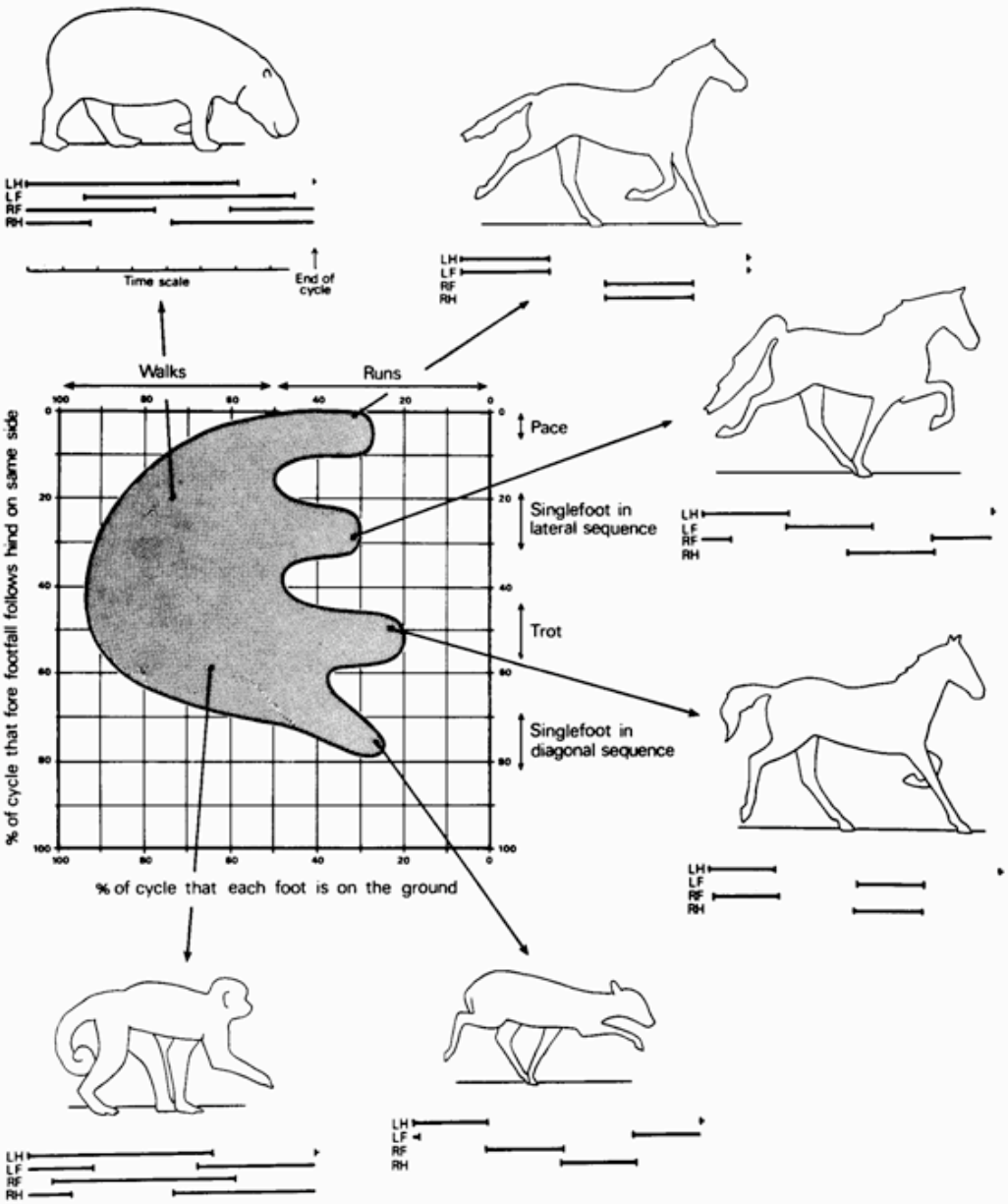


Figure 3-2 Symmetrical gaits as defined by the duration of contacts with the ground and the phase relationship of fore and hind feet. The outlined area encloses nearly 1200 plots for 156 genera, including amphibians, reptiles, and 16 orders of mammals. For the seven gaits recorded, a pigmy hippopotamus, three horses, a duiker, and a monkey are shown at the instant the left hind foot strikes the ground. Gait diagrams indicate the timing and durations of the respective contact intervals. Time scales *(upper left)* for the different animals are independent. H = hind; F = fore; R = right; L = left.

Appendix Figure 2.5. Hildebrand 1985, p. 41.

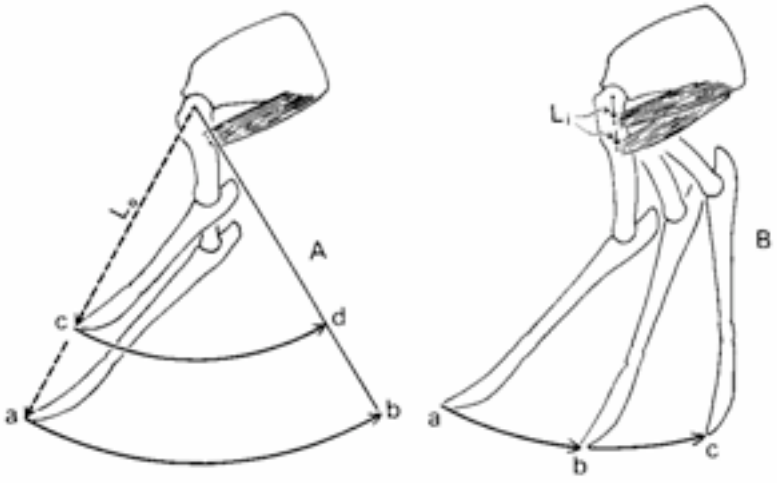


Figure 3-6 Some consequences of lever mechanics. In A, the foot of the longer leg travels from *a* to *b* in the same time that the foot of the shorter leg travels the shorter distance from *c* to *d*. In B, the muscle having the shorter in-lever moves the foot from *a* to *c* with the same shortening (hence time) that the other muscle needs to move it from *a* to *b*. For clarity of illustration the shoulder is shown as the pivot. The same principles apply as the muscle draws the shoulder over the stationary foot.

Appendix Figure 2.6. Hildebrand 1985, p. 46.

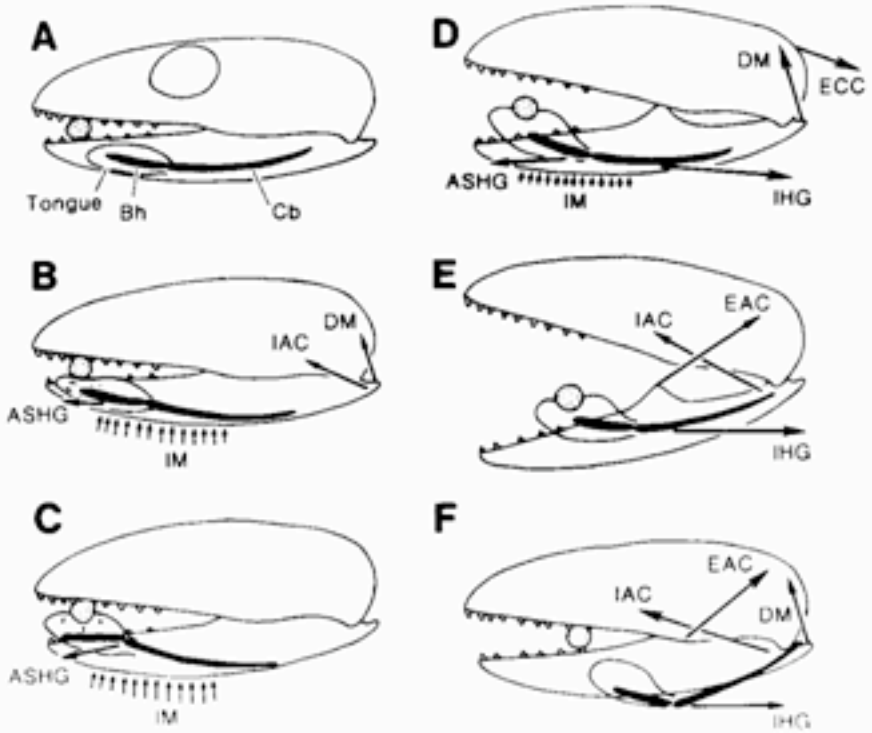


Figure 13-5 The mechanism of intraoral transport in the model generalized tetrapod. A: System at rest with food object *(stippled)* in jaws. B: Slow open I, hyolingual unit advancing beneath food. C: Slow open II, tongue being fitted to food. D: Middle of fast open with tongue cradling food and cranium and mandible accelerating in opposite directions. E: Early in fast close with cranium descending, mandible elevating, and tongue and hyoid rapidly accelerating rearward. F: Slow close stage of slow close–power stroke, showing food fixed by teeth and tongue and hyoid at maximally retracted position. *ASHG* = anterior suprahyoid group; *Bh* = basihyal; *Cb* = ceratobranchial; *DM* = depressor mandibulae; *EAC* = external adductor complex; *ECC* = epaxial cervical complex; *IAC* = internal adductor complex; *IHG* = infrahyoid group; *IM* = intermandibularis; arrows indicate direction of muscle force.

Appendix Figure 2.7. Bramble and Wake 1985, p. 238.

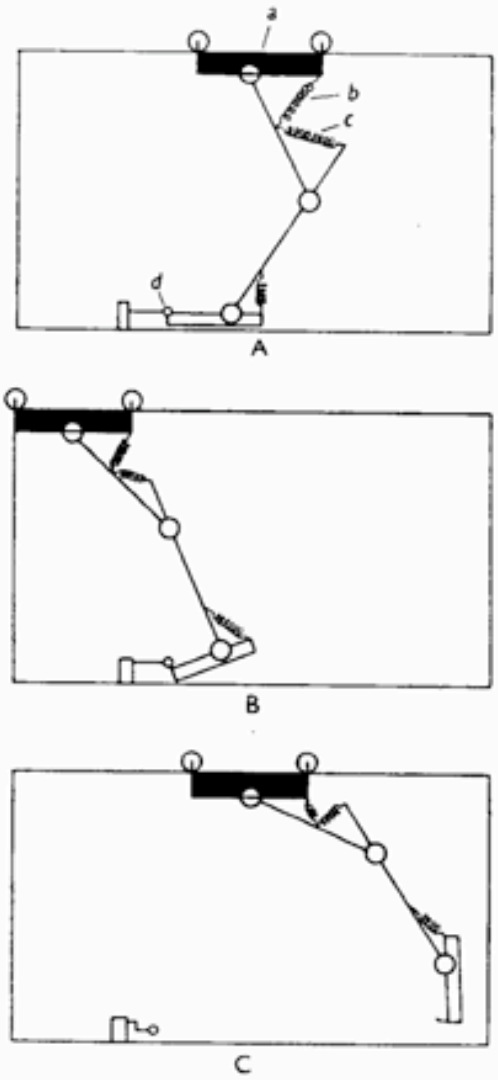


Fig. 4. Vertebrate hind limb. The body is represented by a carriage (*a*) on a rail. If it is held back towards the right (4A), the springs (*b*) and (*c*) are in tension (active muscles). The foot is restrained at (*d*) (ground friction). When the carriage is released (B) it moves to the left as the springs shorten. If, however, the attachment (*d*) is released (C) the limb swings back (ground friction is necessary for propulsion).

Appendix Figure 2.8. Brown 1960, p. 75.

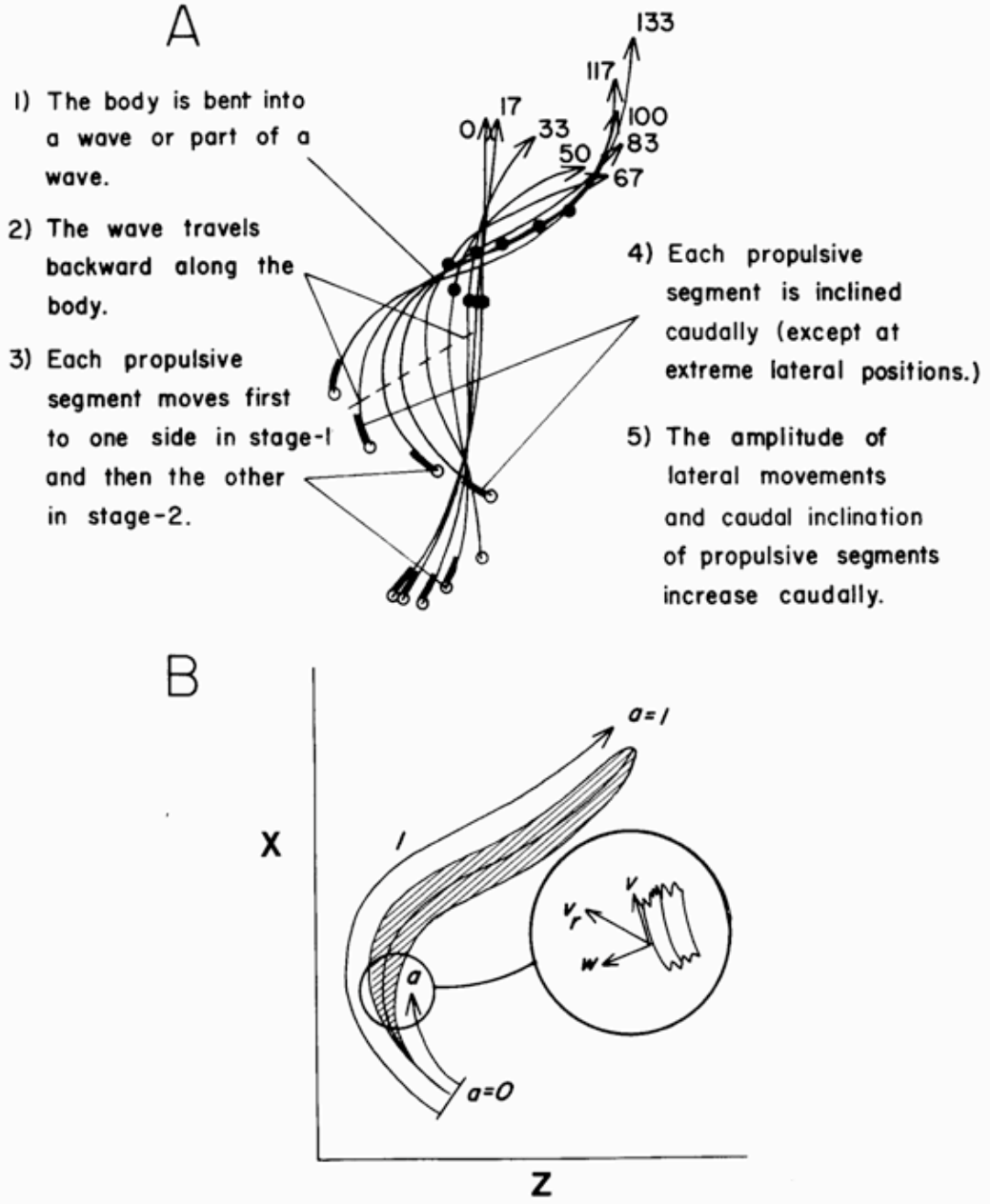


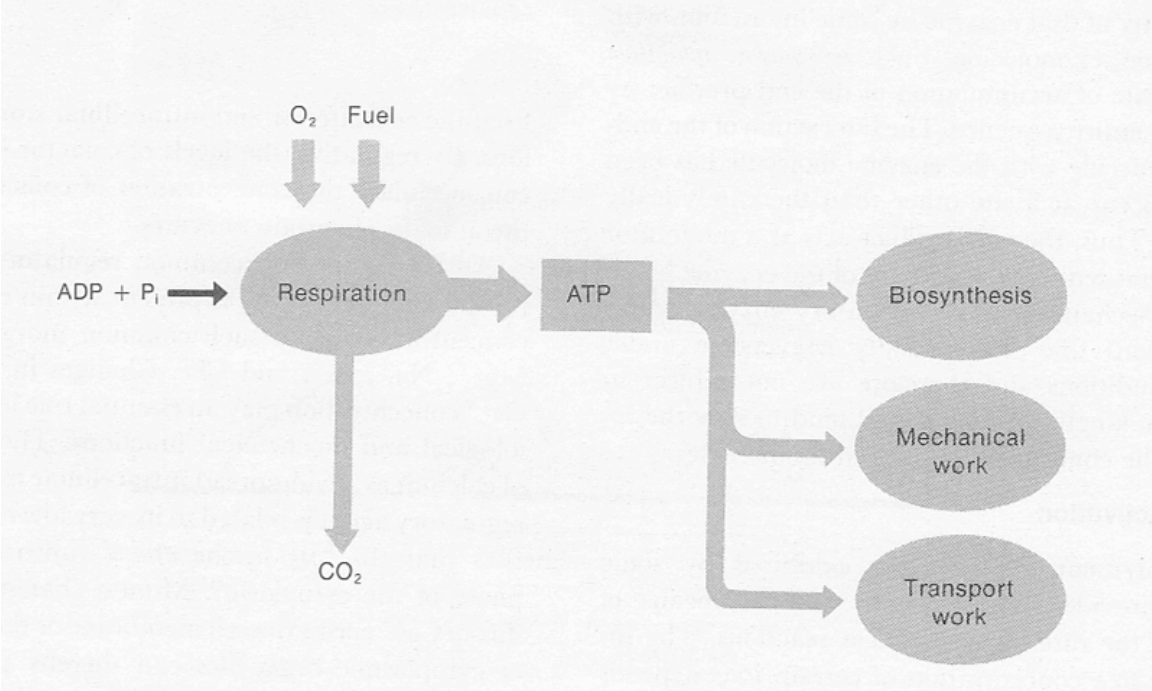
Figure 7-2 The kinematic features involved in thrust generation. A: Tracings of the center line of the body of a trout, *Salmo gairdneri*, taken from cine film of a fast start. Times between tracings are shown in msec. Shaded circles show the location of the center of mass. B: Convention for coordinates used to analyze locomotor movements such as those in A, based on Weihs (1973). Note the body is arbitrarily divided into infinite propulsive segments from the trailing edge ($a = 0$) to the tip of the nose (a = length). One of these segments is shown at a, along with the directions of its forward, lateral, and resultant velocities (V, W, and V_r).

Appendix Figure 2.9. Webb and Blake 1985, p. 114.

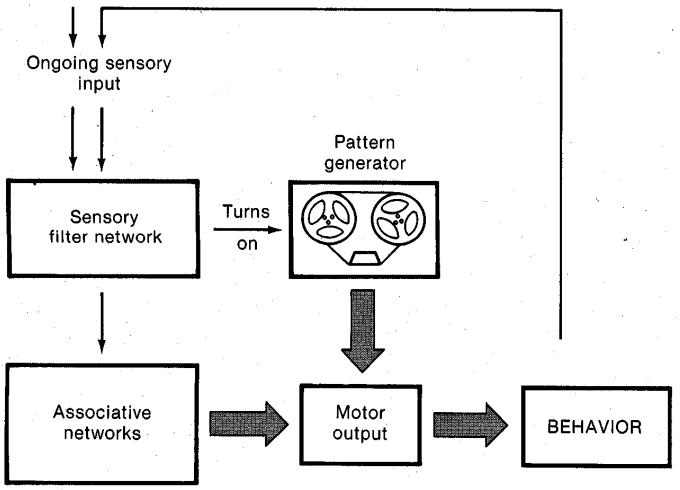
Table 7-2 Symbols used in text.

Symbol	Definition	Symbol	Definition
C_D	Drag coefficient	T	Thrust
C_n	Normal drag coefficient	$\overline{T}$	Mean thrust
c	Wave velocity	t	Time
D	Drag force acting on a body or paddle	t_p	Duration of the power phase of the beat cycle
D_p	Drag during power phase of beat cycle		
D_r	Drag during recovery phase of beat cycle	t_o	Duration of the beat cycle
		u	Speed of a paddle
d	Body depth	V	Forward velocity of animal, propulsive segment, or blade element
dr	Spanwise length of an element of a paddle blade	v_r	Resultant velocity of a propulsive segment or blade element
E	Total work done during the power phase of the beat cycle	v_n	Normal velocity of an element of a paddle blade
$\overline{E}$	Mean work done	v_s	Spanwise velocity of an element of a paddle blade
E_D	Work done in overcoming drag	W	Lateral velocity of a propulsive segment
E_o	Total work done during a beat cycle	w	Velocity of water accelerated by a propulsive segment
F	Force	β	Added mass correction for the effect of body
F_a	Acceleration force	γ	Angle between a paddle and the body axis
F_n	Normal force	η	Efficiency
L	Lift force	θ	Angle between the trailing edge of a propulsive segment and the swimming axis
m	Added mass		
N	Number of paddles	ρ	Density of water
n	Normal coordinates of a propulsive segment	ω	Angular velocity
P	Total power output		
P_k	Rate of loss of kinetic energy		
P_T	Thrust power		
R	Radius		
S	Surface area		

Appendix Figure 2.10. Webb and Blake 1985, p. 115

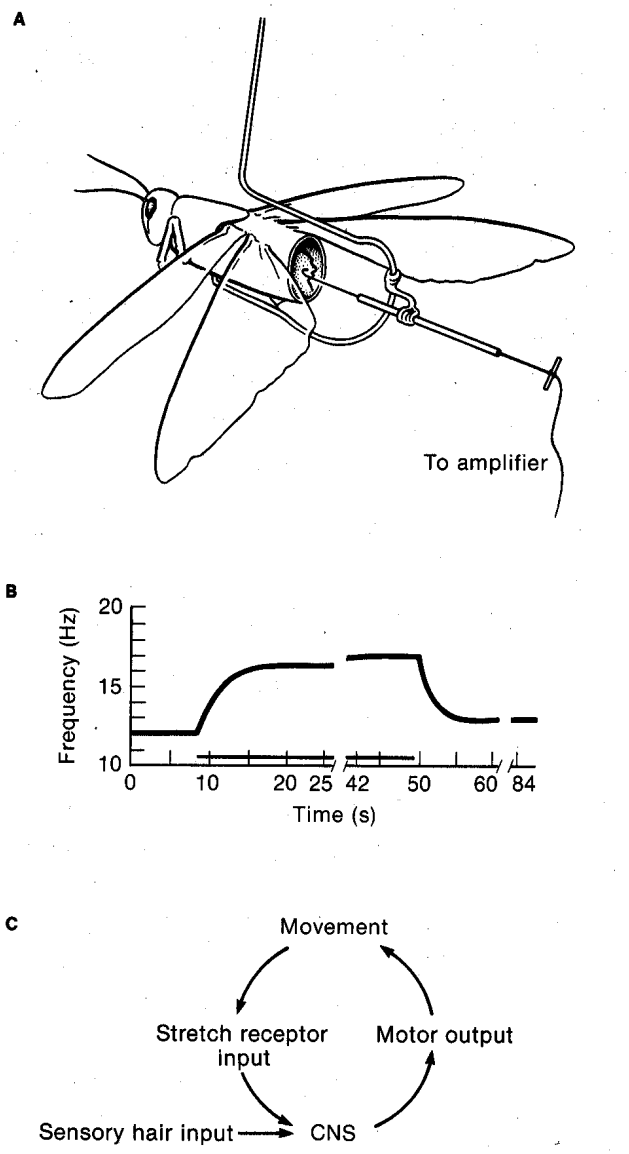


Appendix Figure 2.11. Eckert et al. 1988, p. 52. Caption 3-29 for the figure in the text reads "Uses of ATP in biological systems. The ADP produced by hydrolysis is recycled to ATP by rephosphorlyation energized by the oxidation of foodstuff molecules to CO_2 and H_2O. [Lehninger, 1971.]



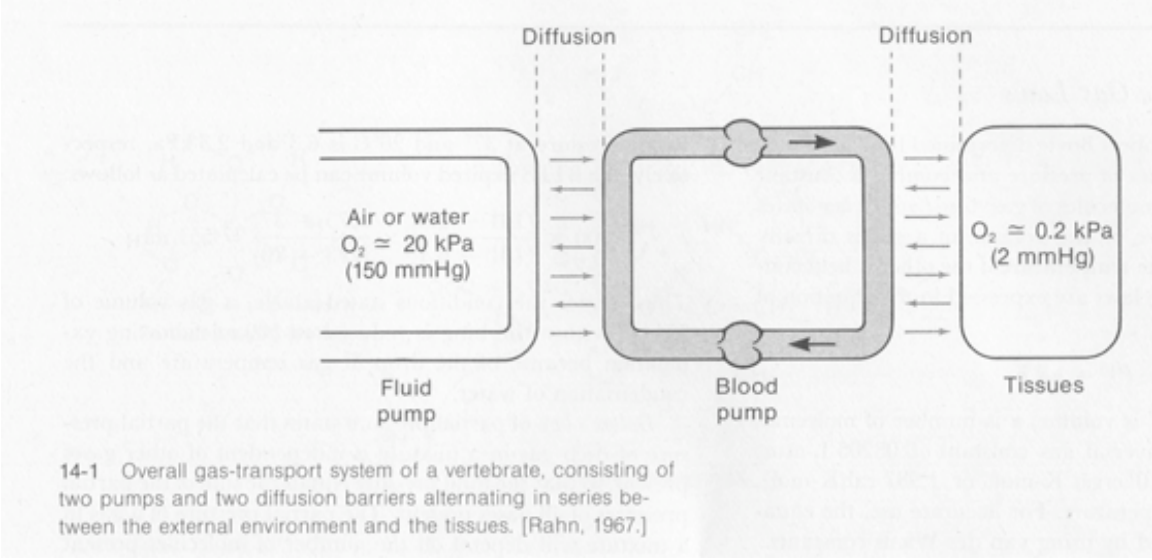
8-30 Block diagram of the circuitry between sensory input and motor output. Part of the sensory input arises from the motor activity of the animal. Note that the tape machine plays a loop of tape that represents the "motor score," namely, the endogenous pattern of motor signals. The blocks are somewhat arbitrary, and in reality probably overlap to some extent.

Appendix Figure 2.12. Eckert et al. 1988, p. 242.



8-38 Role of proprioceptive feedback in grasshopper flight. (**A**) Experimental arrangement. An eviscerated grasshopper or locust is mounted so that it can flap its wings when stimulated by air blowing on facial receptor hairs. Electrodes for recording motor output and for stimulating receptor nerves are fixed in place. (**B**) The result of sensory input is to increase the frequency of the endogenous rhythm of motor output. The duration of receptor nerve stimulation is indicated by the black line. (**C**) Cyclic organization of behavior. External sensory input (puff of air on hair receptors) stimulates behavior (flying). The wing movements activate stretch receptors that provide further input that stimulates the flight motor. [Wilson, 1964, 1971.]

Appendix Figure 2.13. Eckert et al. 1988, p. 249.



14-1 Overall gas-transport system of a vertebrate, consisting of two pumps and two diffusion barriers alternating in series between the external environment and the tissues. [Rahn, 1967.]

Appendix Figure 2.14. Eckert et al. 1988, p. 475.

Box 14-3 *Ventilation-to-Perfusion Ratios*

The actual ratio of ventilation to perfusion is affected by a number of factors. The oxygen uptake by the blood as it passes through the respiratory surface is given by

$$\dot{V}_{O_2} = \dot{Q}(C_{aO_2} - C_{vO_2}) \quad (1)$$

where $\dot{V}_{O_2}$ is oxygen uptake per unit time, $\dot{Q}$ is blood flow per unit time, and C_{aO_2} and C_{vO_2} are, respectively, the oxygen content of blood leaving (oxygenated) and entering (deoxygenated) the respiratory epithelium.

Similarly, the amount of oxygen leaving the medium is given by

$$\dot{V}_{O_2} = \dot{V}_A(C_{IO_2} - C_{EO_2}) \quad (2)$$

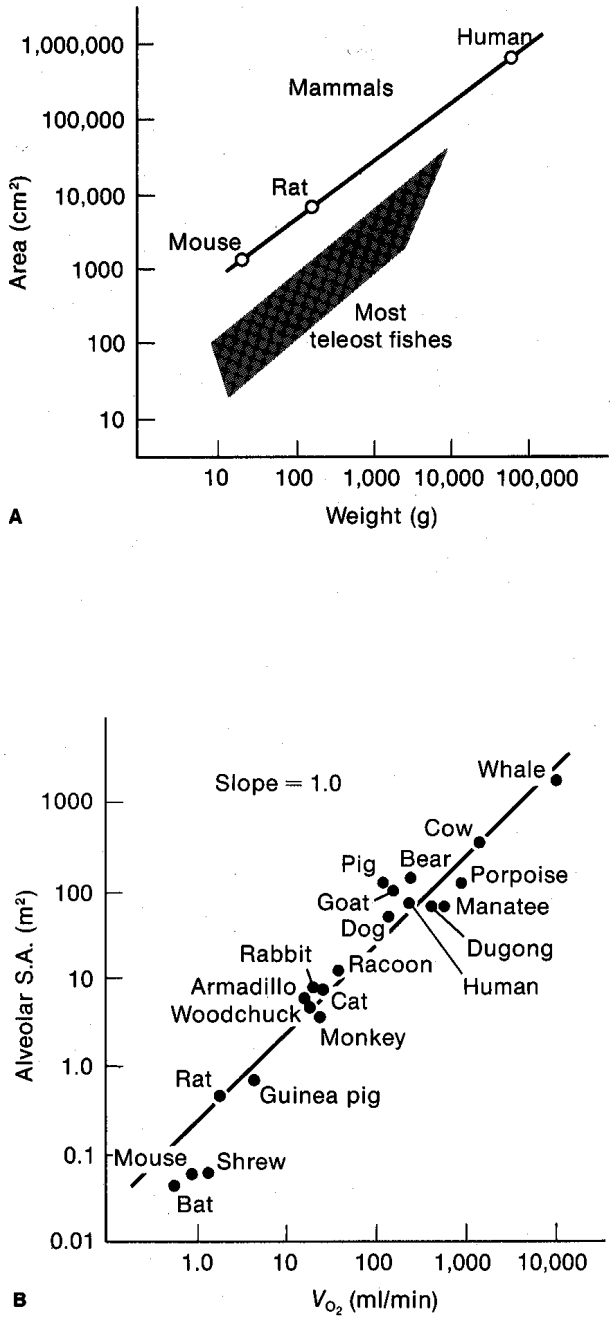
where $\dot{V}_A$ is alveolar ventilar volume and C_{IO_2} and C_{EO_2} are, respectively, the O_2 content in inhalant and exhalant medium. Equation 2 is true only if inspired and expired volumes are equal, which they generally are not in air-breathing animals. Therefore, mean values from replicates of samples taken during several breathing movements should be used. From Equations 1 and 2, we obtain

$$\dot{Q}(C_{aO_2} - C_{vO_2}) = \dot{V}_A(C_{IO_2} - C_{EO_2})$$

and

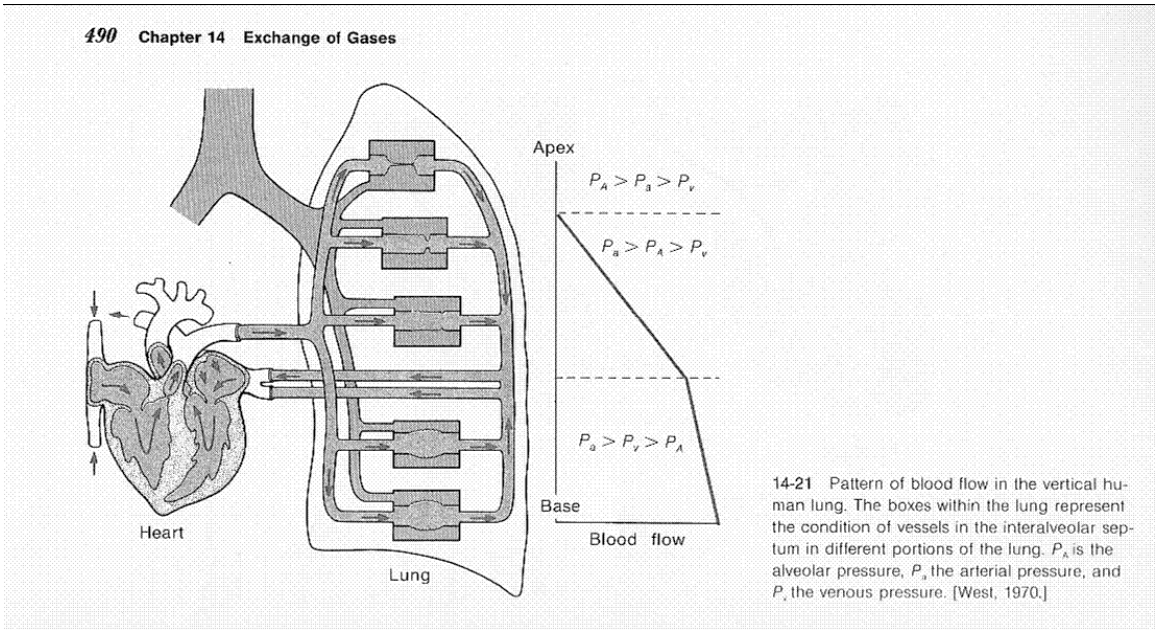
$$\dot{V}_A/\dot{Q} = (C_{aO_2} - C_{vO_2})/(C_{IO_2} - C_{EO_2})$$

Appendix Figure 2.15. Eckert et al. 1988, p. 499.

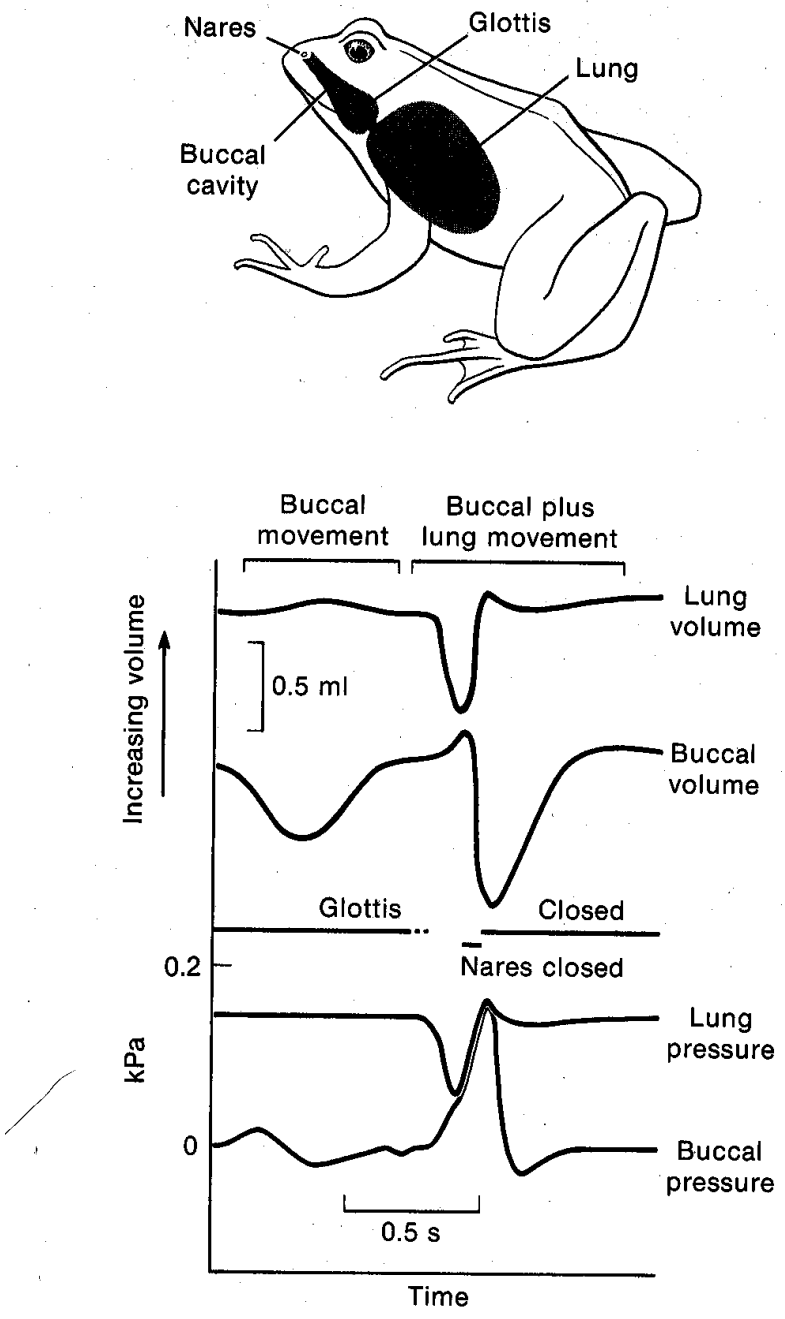


14-14 (**A**) Relationship between respiratory surface area and body weight for a number of vertebrates. [Randall, 1970.] (**B**) Relationship between alveolar surface area (S.A.) and oxygen uptake in mammals. [Tenney and Temmers, 1963.]

Appendix Figure 2.16. Eckert et al. 1988, p. 485.



Appendix Figure 2.17. Eckert et al. 1988, p. 490.



14-27 Changes in pressure and volume in the buccal cavity and lung of a frog during buccal movements alone and during buccal and lung movements. [West and Jones, 1975.]

Appendix Figure 2.18. Eckert et al. 1988, p. 495

AF.2 Figures for Chapter 3

Selection Scenario	**Interactor**	**Replicator**
Origins of life	Lengths of RNA	Lengths of RNA
"Selfish genes"	Lengths of DNA	Lengths of DNA
Meiotic Drive	Chromosome (or a part thereof)	Chromosome (or a part thereof)
Developmental or somatic selection	Parts of organisms	Genes or genome
Organismic selection:	Organism	Genome (or organism?)
asexual reproduction		
sexual reproduction	Organism	Genes
Intrademic group	Group	Genes
Interdemic group	Group	Group
Avatar selection	Avatar	Avatar
Species selection	Species	Species
Clade selection	Clade	Clade

Appendix Figure (Table) 3.1. Table entitled "Hierarchies of Interactors and Replicators," taken directly out of Brandon 1990, p. 97.

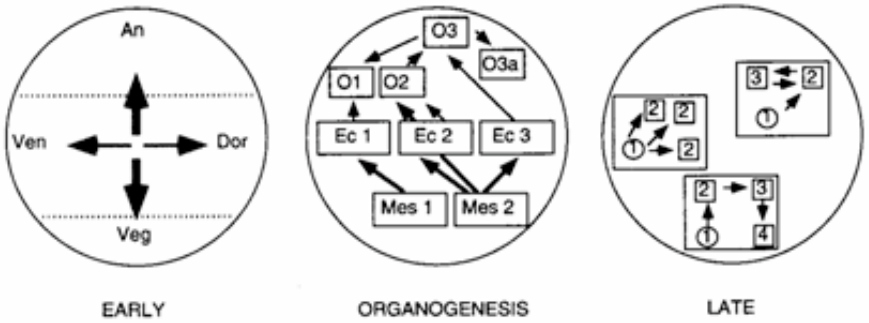


Figure 6.6. Qualitative changes in modularity over early, middle, and late stages of development. The model shown here is based on general features of deuterostome development, particularly of echinoderms and amphibians. Events of early development are dominated by axial information systems (animal-vegetal and dorso-ventral), which establish initial patterning processes and the first localized gene action as cells divide. In these embryos, these axes are global, and allow considerable developmental flexibility. Late development also shows considerable developmental flexibility, which arises because the body is highly modularized by division into separate organ primordia. Signaling events (small circles and boxes with arrows) within the primordia (shown as large boxes) are little influenced by events in other primordia. Mid-development, however, exhibits a high interconnectivity between elements that will later come to represent separate modules. In this diagram, mesodermal tissues signal ectodermal tissues, which in turn interact with sensory placodes. Clearly this represents only a few of the inductive processes involved in the formation of the phylotypic stage. (From R. A. Raff, in *Early Life on Earth*, edited by S. Bengtson. Copyright 1994 by Columbia University Press. Reprinted with permission of the publisher.)

Appendix Figure 3.2. Raff 1996, p. 204. This figure represents Raff's *processual* hypothesis for why there is significant morphological and developmental conservation during the *middle* stages of development, when organogenesis occurs. This mechanism is supposed to account for the hourglass *pattern*, or developmental and phylogenetic conservation, described below in Figure 3.3.

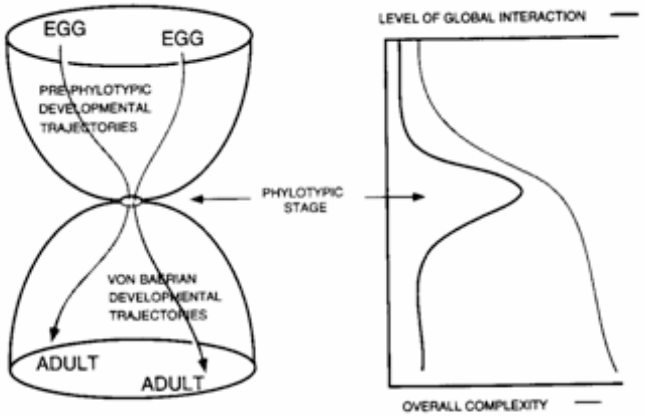


Figure 6.7. The developmental hourglass. The graph at the right plots the rise in overall complexity of an individual embryo moving through the hourglass. It also plots the level of interaction among modules. Interaction rises as the embryo approaches the phylotypic stage, and then declines as modules become more autonomous in later development.

Appendix Figure 3.3. Raff 1996, p. 208.

Theory	Cause	Criteria
Owen	Ideal morphology	Anatomical structure and position
Darwin	Phylogenetic descent	Anatomical structure and position
Developmental genetics	Phylogenetic descent	Gene expression patterns

Appendix Figure (Table) 3.4. Table entitled "Causes and Criteria of Homology," taken directly out of Bolker and Raff 1996, p. 490.

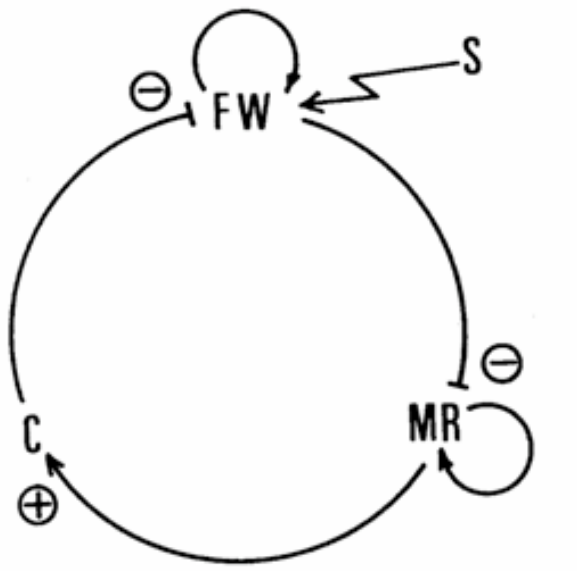


FIG. 6. Hypothetical causal relationships among the characters of a Blenniid pectoral fin hook during morphogenesis. Both the fin web (FW) and the mesodermal ridge (MR) are assumed to have a tendency toward spontaneous self-differentiation, which is symbolized by the small circles originating and terminating at the respective characters. The fin web is assumed to inhibit the transformation of the mesenchymal condensation into a proper mesodermal ridge. The mesodermal ridge is assumed to modulate the epidermis to form a cuticula (C), and the cuticula is assumed to inhibit the growth of the fin web on the free edge of the fin ray, leading to the deep incision of a mature fin hook. The initial regression of the fin web is assumed to be triggered by an unknown stimulus (S). Note that this model constitutes a positive feedback loop, comprising two inhibitory interactions and one inductive interaction.

Appendix Figure 3.5. Wagner 1989, p. 1168. Wagner's model for the mutual stimulation and inhibition of the three parts of the fin during the development of the fin hook.

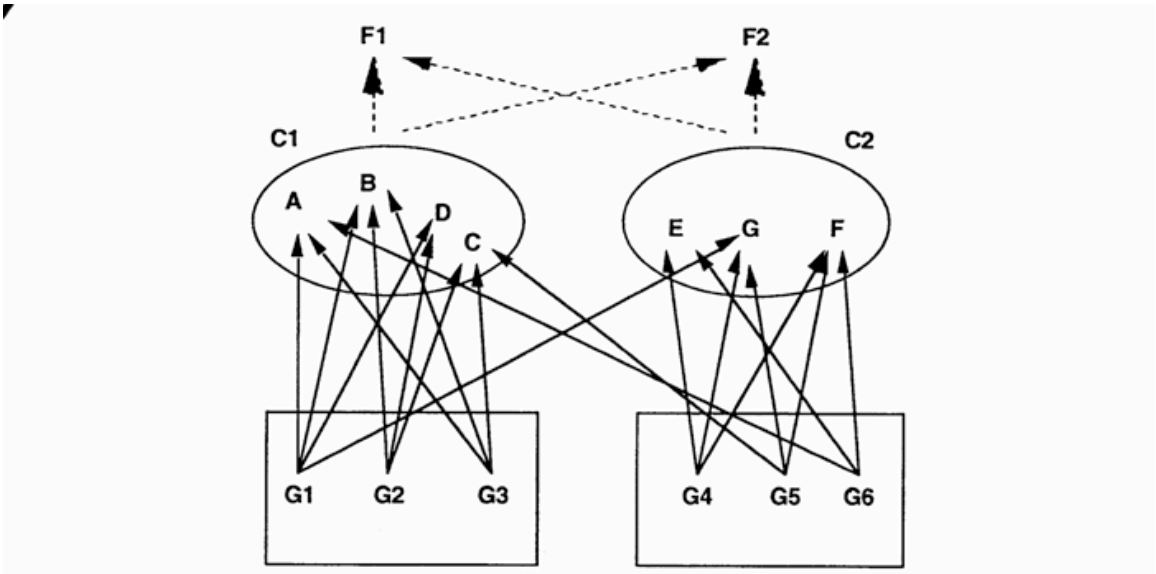


FIG. 1. Example of a modular representation of the character complexes C1 = {A, B, C, D} and C2 = {E, F, G} which serve to functions F1 and F2. Each character complex has a primary function, F1 for C1 and F2 for C2. Only weak influences exist of C1 on F2 and vice versa. The genetic representation is modular because the pleiotropic effects of the genes M1 = {G1, G2, G3} have primarily pleiotropic effects on the characters in C1 and M2 = {G4, G5, G6} on the characters in complex C2. There are more pleiotropic effects on the characters within each complex than between them.

Appendix Figure 3.6. Wagner and Altenberg 1996, p. 971. A representation of how genes are involved in causing modules that then have particular functions. Note that the genes are not represented as interacting with each other, which is odd given that Wagner is also known for his work on, among other things, genetic epistasis in mathematical evolutionary genetics. Although he does do mathematical modeling of evolutionary genetics, his models tend to be very complex and tend not to appeal to the axioms of population genetic theory.

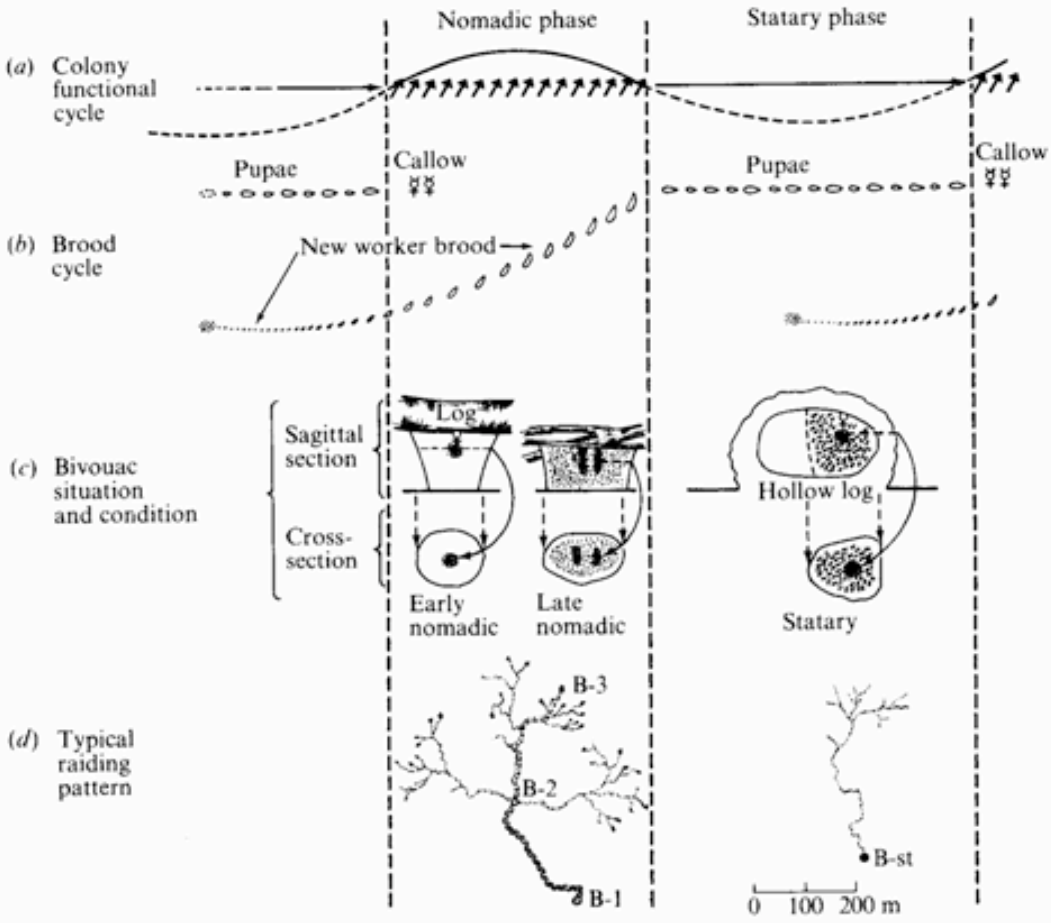


FIGURE 1.1
Functional cycle of the army ant *Eciton hamatum*. (*a*) Phases in the cycle indicated by a sine curve; arrows: large daily raids and nightly emigrations. (*b*) Concurrent development of successive (coordinated) all-worker broods, from eggs (*at left*) to larvae, to pupae, and finally to callow workers (☿ ☿ , *at right*). (*c*) Types of bivouac in each functional phase, indicating typical placement of brood in each. (*d*) Patterns of raiding systems typical of the functional phases; B-1, B-2, and B-3: successive bivouac sites; B-st: statary bivouac site.

Appendix Figure 3.7. Schneirla 1971, p. 2. A diagram depicting aspects of the development, behavior, and ecology of both the individuals and the colonies of the army ant *Eciton hamatum*. Note that the colony is depicted as possessing (1) a reliable series of (physiological) cycles in that it alternates between two phases, (2) integrated movement through space, (3) and favored habitats. It is an individual unit.

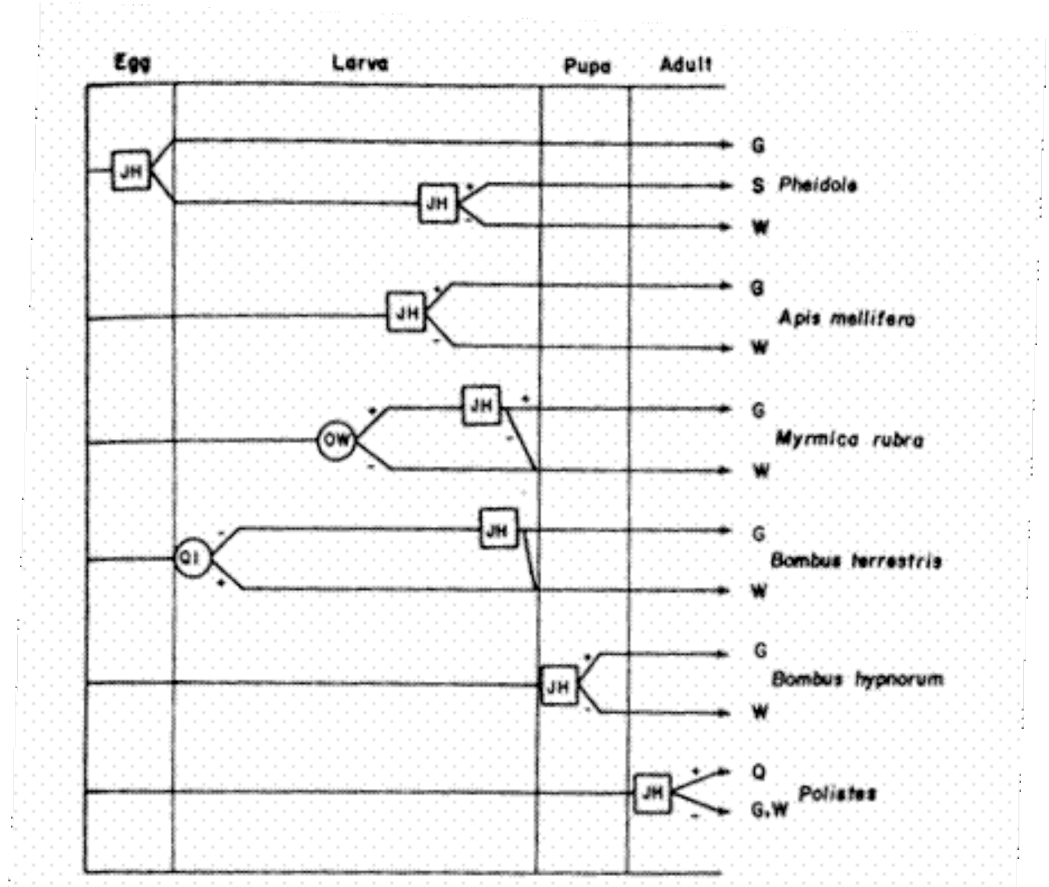


FIG. 3.—Developmental control points where juvenile hormone is known or suspected to play a role in caste determination. Those in larval or early pupal development are probably associated with nutritional switches (from Wirtz 1973; Brian 1974; Röseler 1976, 1977; Passera and Suzzoni 1979; Wheeler and Nijhout 1983; Röseler et al. 1984). JH, juvenile hormone; OW, over winter; QI, queen inhibition; G, gyne; S, soldier; W, worker; Q, queen.

Appendix Figure 3.8 Wheeler 1986, p. 19. A diagram indicating the different phases of development during which the nutritional switch works in a variety of hymenoptera species.

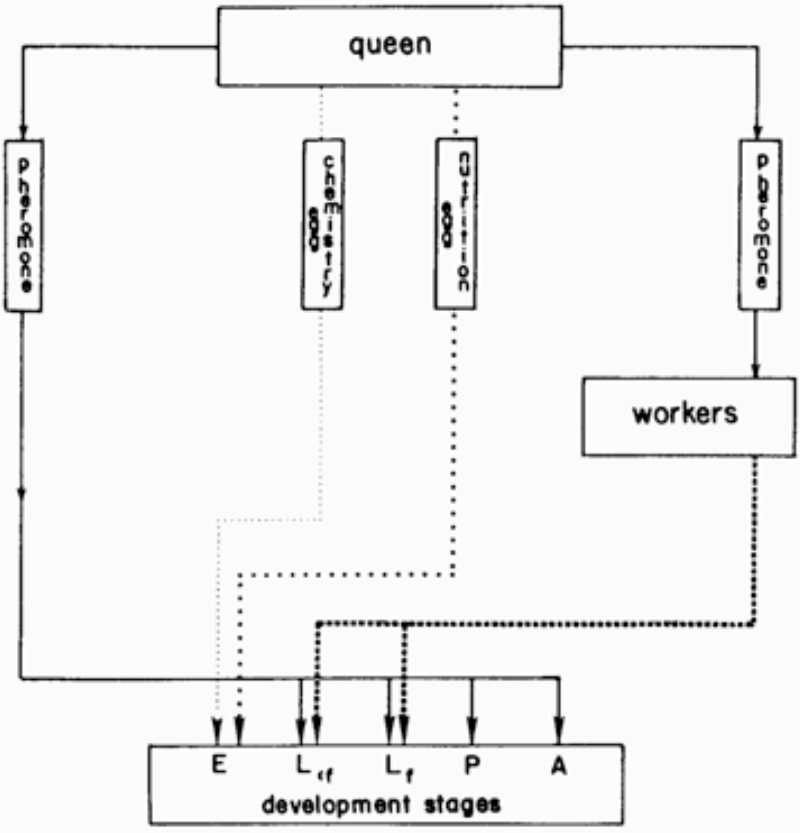


FIG. 6.—Control mechanisms for gyne determination in social Hymenoptera. *Right*, type 1 control, in which regulation of nutrition directs the developmental decision, while the nutritional switch functions similarly in all individuals. Nutritional bias of eggs may be considered a variation of nutritional control. *Left*, type 2 control, in which inhibition appears to preempt or impair the switch mechanism itself, making it impossible for the gyne program to be initiated. Effects of maternal hormones on eggs may be considered a variation of type 2 control. E, egg; $L_{<f}$, all larval instars except last; L_f, final larval instar; P, pupa; A, adult.

Appendix Figure 3.9. Wheeler 1986, p. 28. Diagram indicating the control mechanisms for gyne determination by queens and workers, which are (kinds of) parts of a colony. Note that this diagram is structurally very similar to the Figure 3.5 portraying developmental influences among (kinds of) parts of a fin.

AF.3 Figures for Chapter 4

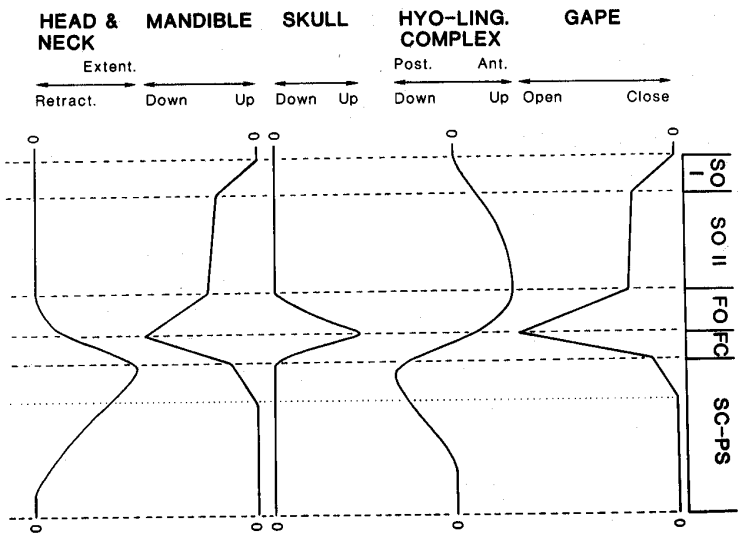


Figure 13-3 Composite kinematic profile of a theoretical model feeding cycle as would be expected in a primitive, generalized tetrapod. Major kinematic stages of the cycle are slow open I (*SO*-I), slow open II (*SO*-II), fast open *(FO)*, fast close *(FC)*, and slow close–power stroke *(SC-PS)*. The dotted line indicates the boundary between the *SC* and *PS* intervals of the final stage. For each profile the zero point is the baseline or resting position.

Appendix Figure 4.1. Bramble and Wake 1985, p. 236.

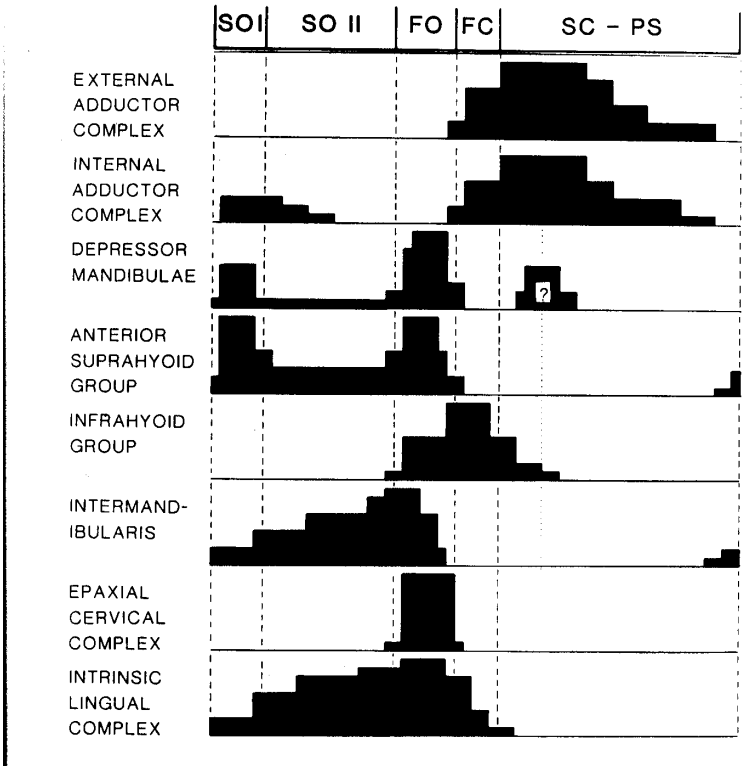


Figure 13-4 The expected electromyographic activity patterns of eight selected muscle units during the model feeding cycle. Width between dotted lines indicates timing of stages. External adductor complex = chiefly M. adductor mandibulae; internal adductor complex = chiefly M. pterygoideus; anterior suprahyoid group = Mm. geniohyoideus and genioglossus; infrahyoid group = chiefly M. rectus cervicis; epaxial cervical complex = muscles linking neck with occiput.

Appendix Figure 4.2. Bramble and Wake 1985, p. 237.

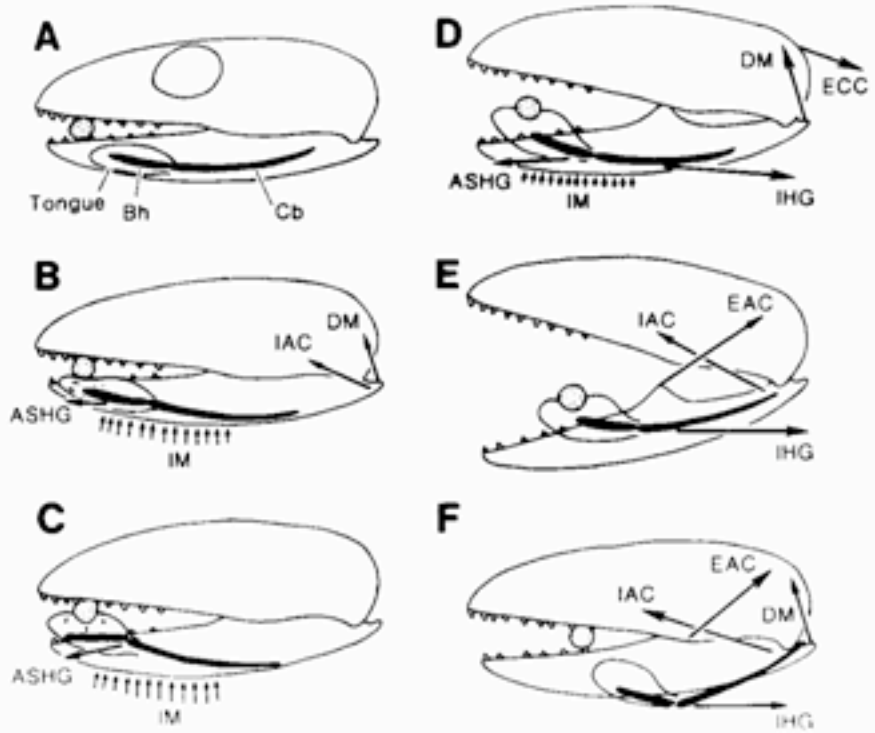


Figure 13-5 The mechanism of intraoral transport in the model generalized tetrapod. A: System at rest with food object *(stippled)* in jaws. B: Slow open I, hyolingual unit advancing beneath food. C: Slow open II, tongue being fitted to food. D: Middle of fast open with tongue cradling food and cranium and mandible accelerating in opposite directions. E: Early in fast close with cranium descending, mandible elevating, and tongue and hyoid rapidly accelerating rearward. F: Slow close stage of slow close–power stroke, showing food fixed by teeth and tongue and hyoid at maximally retracted position. *ASHG* = anterior suprahyoid group; *Bh* = basihyal; *Cb* = ceratobranchial; *DM* = depressor mandibulae; *EAC* = external adductor complex; *ECC* = epaxial cervical complex; *IAC* = internal adductor complex; *IHG* = infrahyoid group; *IM* = intermandibularis; arrows indicate direction of muscle force.

Appendix Figure 4.3. Bramble and Wake 1985, p. 238.

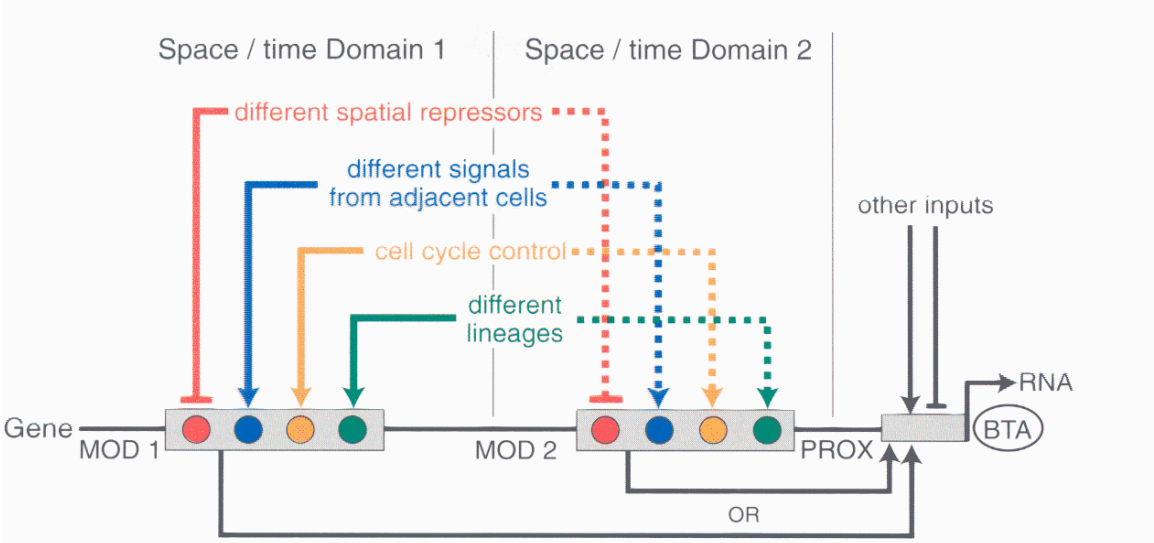
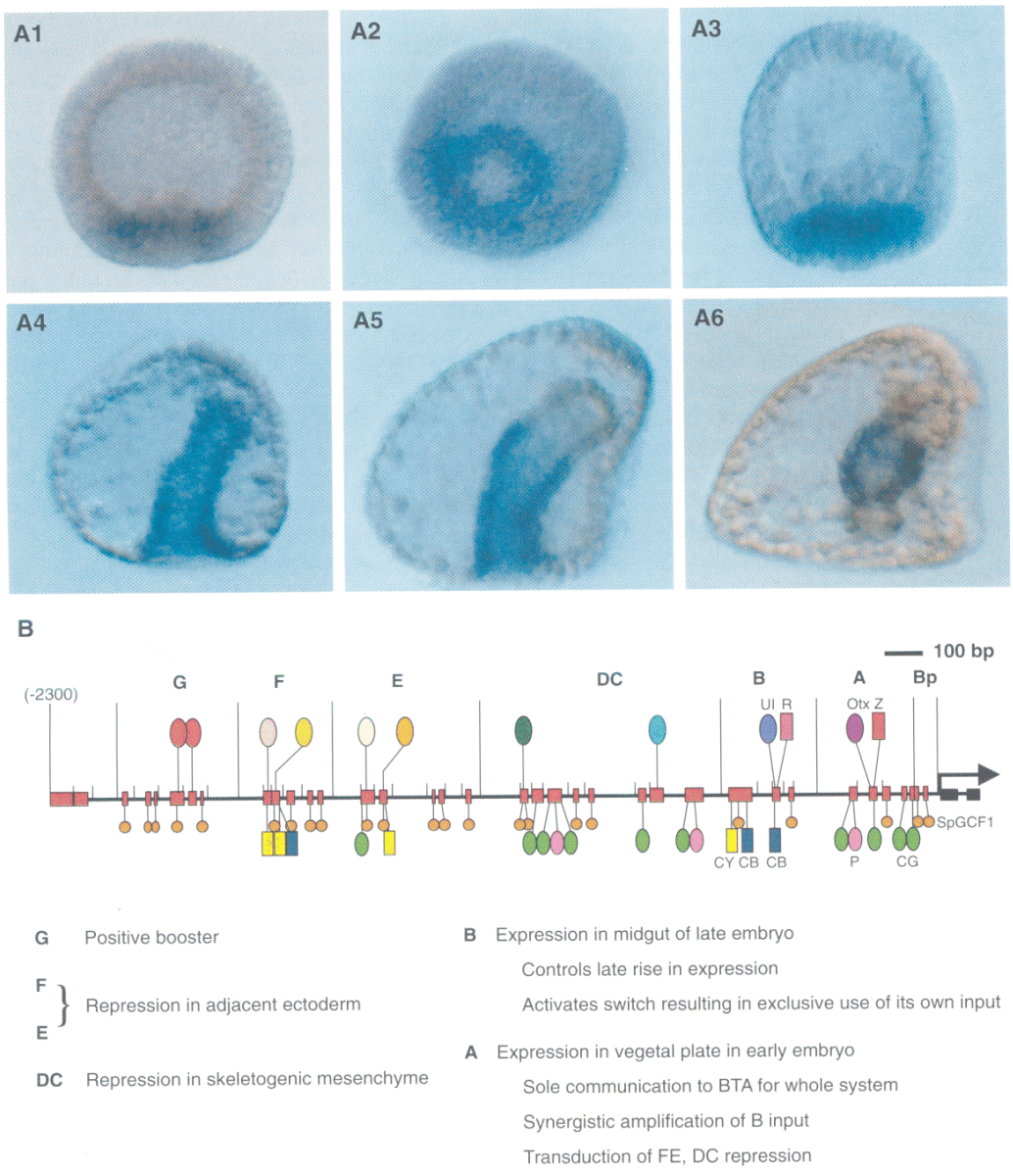
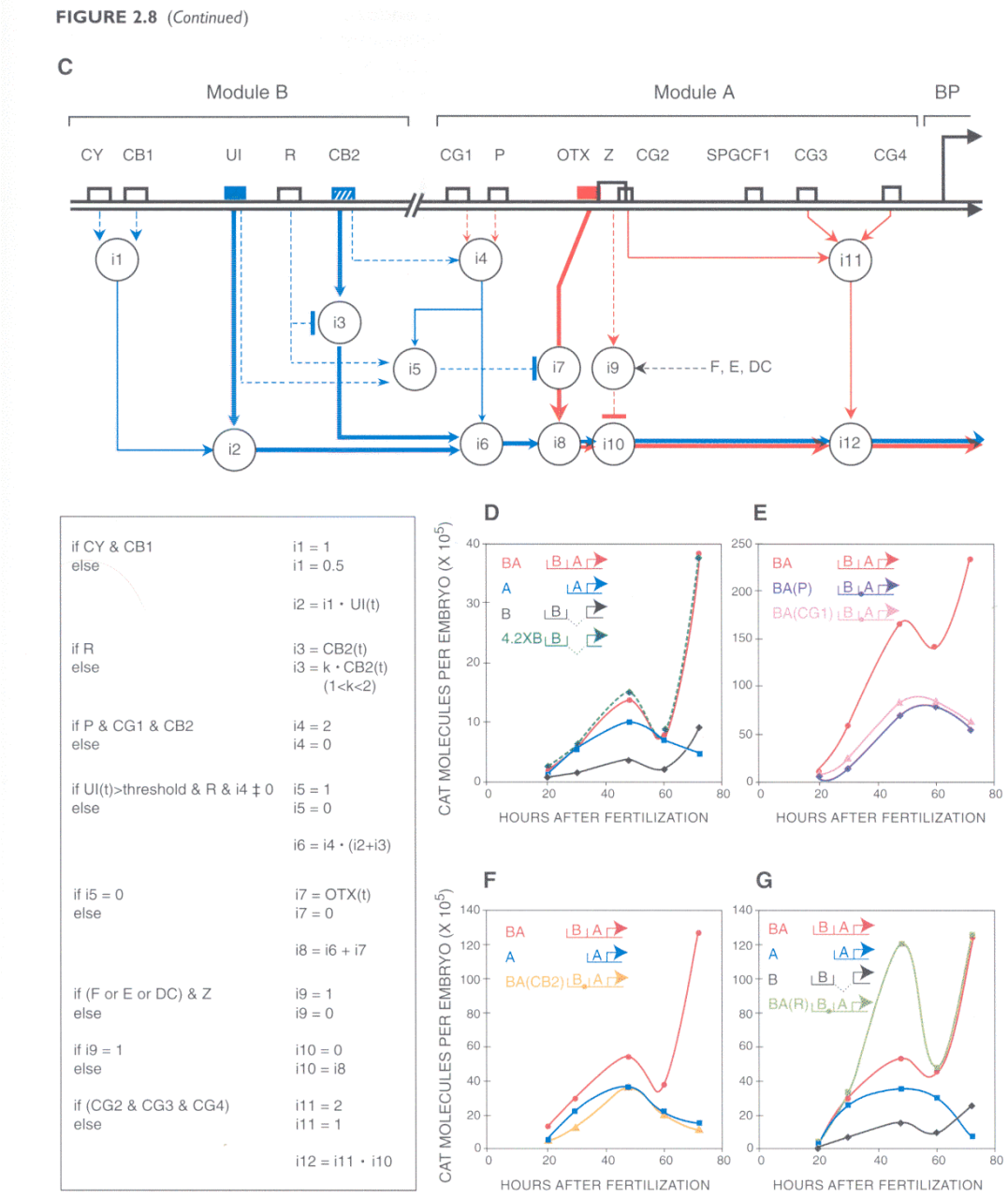


FIGURE 1.3 Modular *cis*-regulatory information processing. The cartoon shows several kilobase (kb) upstream of a gene operating under the control of two *cis*-regulatory modules (MOD1 and MOD2), each of which is operative in a particular spatial domain at a particular time in development. Each module receives multiple parallel inputs (arrows). The diagram shows examples of the kinds of inputs each module might receive and of course is not meant to imply that every module utilizes these same particular inputs. The specific inputs of each kind will be different for MOD1 and MOD2, symbolized by the solid vs. dashed colored arrows. The inputs are of two types, positive and negative. The red barred inputs denote different spatial repressors which are utilized in each module to set boundaries of expression in the spatial domain where that module functions, i.e., these inputs repress the gene across the relevant boundaries. The blue activators are downstream of different intracellular signaling pathways; the tan activators turn on the gene when cells are in cycle; the different green activators are present in cells of the respective lineages that constitute the fields in which the gene will be active, but only when all inputs are present. These inputs can be thought of as bringing the indicated kinds of situational biological information to the gene. Each module acts by communication of its output to the proximal *cis*-regulatory module (PROX), which may receive other inputs; and may further process (e.g., amplify) the output of MOD1 or MOD2. PROX then communicates directly with the basal transcription apparatus (BTA). Note that the major spatial information processing occurs in the developmentally regulated, upstream *cis*-regulatory modules, and that the BTA simply responds to the various alternative outputs of the two upstream regulatory modules, as transmitted to it by PROX.

Appendix Figure 4.4. Davidson 2001, p. 10.



Appendix Figure 4.5. Davidson 2001, p. 58.

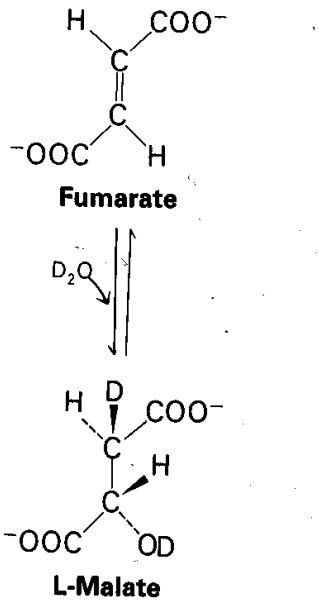


Appendix Figure 4.6. Davidson 2001, p. 59.

FIGURE 2.8 *cis*-Regulatory logic in the *endo16* gene. (A) *In situ* hybridization display of *endo16* expression pattern in *Strongylocentrotus* embryos (Ransick *et al.*, 1993). The gene is expressed in the vegetal plate (A1), but not the skeletogenic cells and small micromeres located at the vegetal pole in the early blastula (A2), nor is it active in the skeletogenic cells as they ingress (A3). After gastrulation it is expressed throughout the archenteron (A4). Expression is later shut off in the foregut and secondary mesenchyme (A5), and the hindgut (A6). The terminal pattern of expression is confined to midgut. [(A) From Ransick *et al.* (1993) *Mech. Dev.* **42**, 117–124; copyright Elsevier Science.] (B) Map of protein interactions in the *endo16 cis*-regulatory system. The 2300 bp DNA sequence indicated as the horizontal line is necessary and sufficient to provide accurate expression of a reporter construct. Proteins that bind at unique locations are shown above the line, and proteins that bind at several locations are indicated below (Yuh *et al.*, 1994). Different colors indicate distinct proteins. "G–A" indicate the functional regions or modules discussed in text. Below the map the functions of each module are briefly indicated (Yuh and Davidson, 1996). [(B) Adapted from Yuh *et al.* (1994) *Mech. Dev.* **47**, 165–186; copyright Elsevier Science.] (C) Logic model for Modules B and A (Yuh *et al.*, 1998, 2000). The regulatory DNA of *endo16* is shown as a horizontal strip at the top of the diagram. The individual binding sites are indicated by labeled boxes. Module B and its effects are shown in blue; Module A and its effects are shown in red. Logic interactions (i) are indicated by numbered circles. Each represents a specific regulatory interaction modeled as a logic operation. Note the two types of regulatory input: time-varying interactions (colored boxes) which determine the temporal and also spatial pattern of *endo16* expression, and time invariant interactions (open boxes) which affect the level of expression and control intra-system output and input traffic. In the diagram interactions that can be modeled as Boolean are shown as dashed lines; those which are scalar as thin solid lines; those which are time-varying quantitative inputs as heavy solid lines. The individual logic interactions are defined in the set of statements below the diagram. Here statements of the form "If X," where X is the name of a target site, means that this site is present and occupied by the respective factor. If the site has been mutated (or if the factors were inactivated or eliminated) this is denoted by zero; or as the alternative ("else") to the site being present and occupied. The statements afford testable predictions of the output for any given mutation or alteration of the system. Briefly, the system works as follows: CB1 and CY1 interactions together (at i1) synergistically increase the output of the positive spatial and temporal regulator of Module B which binds at the UI site. The output of the U1 subsystem is at (i2). An additional smaller time-varying positive input, which peaks at about 40 h, is generated by the interaction at CB2 (i3). An interaction at R is required for the BA intermodule input switch, which shuts off Otx input (i5, i7), but this switch operates only if there is input from UI, and the CB2 site is present and occupied (i5). Furthermore, the proteins binding at the adjacent R and CB2 sites apparently interact, in that if the R site is mutated CB2 input (at i5) is somewhat enhanced. In Module A the CG1 and P sites together with CB2 in Module B are all required for linkage of Module B to Module A (i4), and for synergistic amplification (by a factor of about 2) of the Module B input (at i6). If the switch mediated by R does not function (i.e., in an R mutation) the summed input of Modules A and B at i8 is observed. If CB2, CG1, or P are mutated Module B is unlinked from Module A. That is, $i4 = 0$, so $i6 = 0$ and in this case i8 is just the output of the Otx interaction (at i7). If

the gene is in a location where the repressive interactions in any of the upstream F, E, or DC modules are operating, the sytem is shut off via an interaction at the Z site of Module A (i9, i10). Finally, the CG2, CG3, and CG4 sites combine (at i11) to boost whatever output is present (at i12) by an additional factor of 2. The result at i12 is transmitted to the basal transcription apparatus. [(C) From Yuh *et al.* (2000). In preparation.] (D–G) Expression of CAT reporter as a function of time after fertilization, in batches of 100 eggs per time point. The eggs were injected with expression constructs consisting of Modules B and A linked to one another and to the basal promoter as in the normal gene (BA; red curves); with a construct driven by Module B alone (B; black curves); with a construct driven by Module A alone (A; blue curves); or with constructs in which specific target sites in Modules B or A were mutated, as indicated by parentheses. In the color-coded cartoons in each figure the dots indicate the location of the mutated target sites given in the parentheses. (D) Wild-type constructs. The dashed line shows the time function generated if the B curve is multiplied by the factor 4.2 at each point. (E) CAT activity profile in batches of eggs injected with BA, with BA in which the P site had been mutated [BA(P); violet]; or with BA in which the CG1 site had been mutated [BA(GC1); pink]. The latter two time curves are almost identical to that generated by wild-type A module in isolation from any other modules. [(D, E) From Yuh *et al.* (1998) *Science* **279**, 1896–1902; copyright American Association for the Advancement of Science.] (F) CAT activity profiles generated by BA control; and by BA in which the CB2 site was mutated [BA(CB2); orange]. The profile of expression generated by this construct can be seen to be almost identical to that produced by Module A alone. As predicted in C: if CB2 = 0, i4 and i6 are 0, and the only input is from Module A, i.e., at i8 = i12). (G) Derepression of Module A when the intermodule switch function is cancelled by mutation of the R site of Module B [BA(R); green]. The output is the sum (at i8) of the enhanced CB2 input (i3), Otx input (at i7), plus the amplified input from Module B, including (at i3) the enhanced CB2 input: i.e., [A + i4 · (i2 + i3)]; see logic table. [(F, G) from Yuh *et al.* (2000). In preparation.]

Appendix Figure 4.7. Davidson 2001, pp. 60-61. Davidson's own captions to the Appendix Figures 4.5, 4.6.



Appendix Figure 4.8. Stryer 1988, p. 377. This depiction shows where the deuterium (an isotope of hydrogen) is placed in the molecule of L-malate in the eighth step of the Krebs cycle.

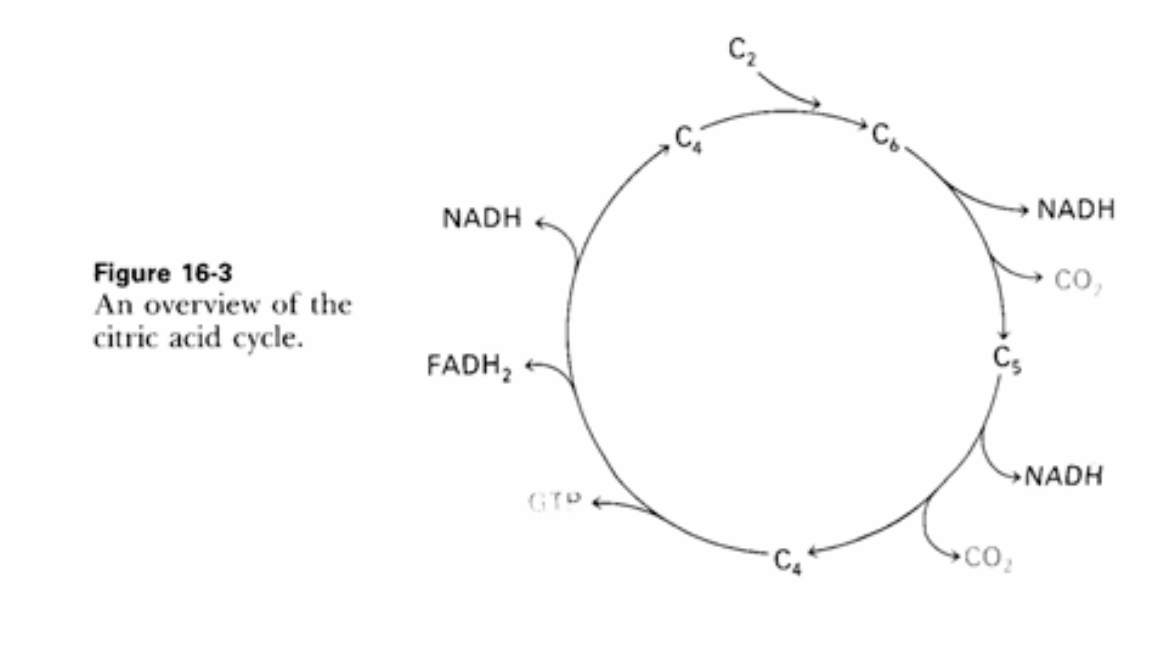


Figure 16-3
An overview of the citric acid cycle.

Appendix Figure 4.9. Stryer 1988, p. 374.

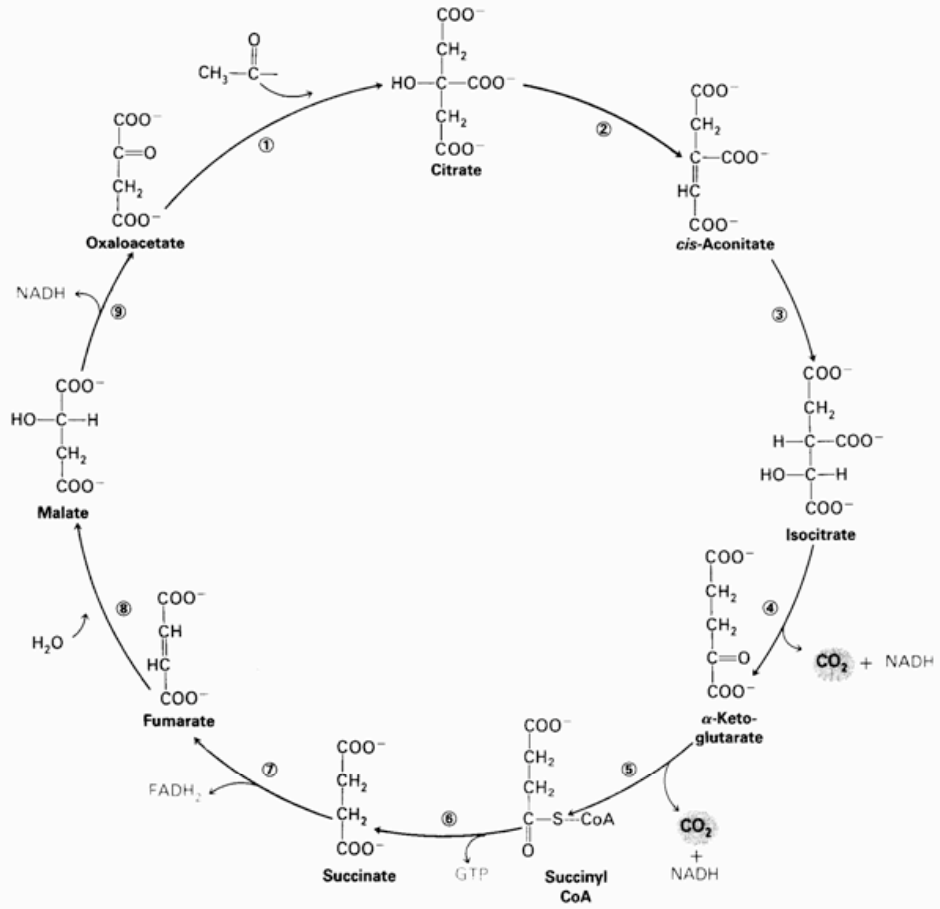


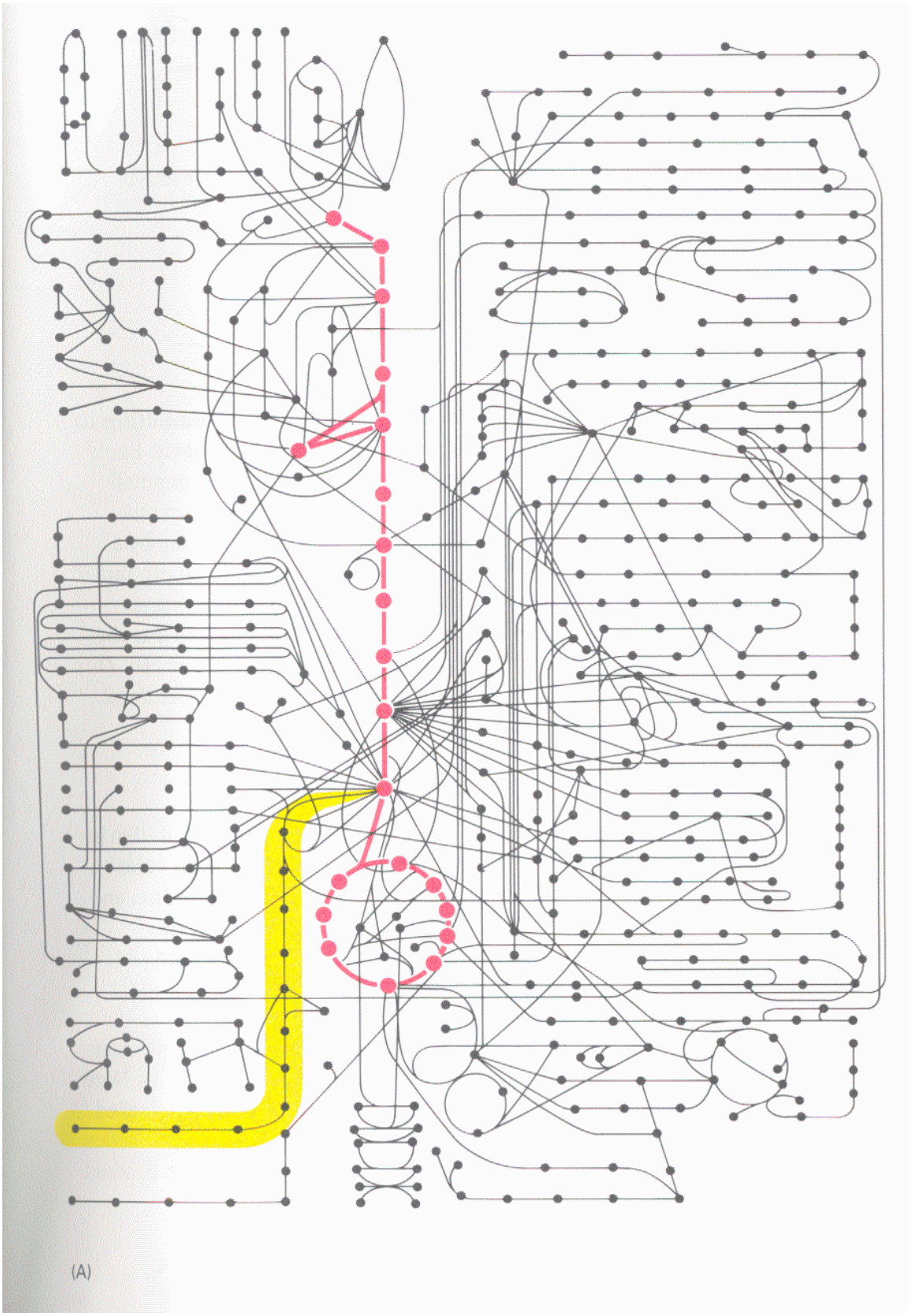
Figure 16-6
Citric acid cycle.

Table 16-1
Citric acid cycle

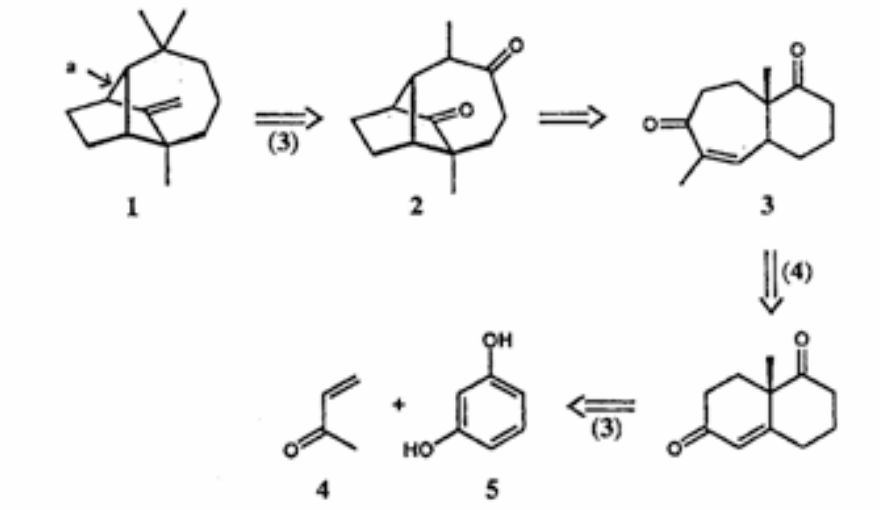
Step	*Reaction*	*Enzyme*	*Prosthetic group*	*Type**	$\Delta G^{\circ\prime}$
1	Acetyl CoA + oxaloacetate + H_2O ⟶ citrate + CoA + H^+	Citrate synthase		a	−7.5
2	Citrate ⇌ *cis*-aconitate + H_2O	Aconitase	Fe-S	b	+2.0
3	*cis*-Aconitate + H_2O ⇌ isocitrate	Aconitase	Fe-S	c	−0.5
4	Isocitrate + NAD^+ ⇌ α-ketoglutarate + CO_2 + NADH	Isocitrate dehydrogenase		d + e	−2.0
5	α-Ketoglutarate + NAD^+ + CoA ⇌ succinyl CoA + CO_2 + NADH	α-Ketoglutarate dehydrogenase complex	Lipoic acid FAD TPP	d + e	−7.2
6	Succinyl CoA + P_i + GDP ⇌ succinate + GTP + CoA	Succinyl CoA synthetase		f	−0.8
7	Succinate + FAD (enzyme-bound) ⇌ fumarate + $FADH_2$ (enzyme-bound)	Succinate dehydrogenase	FAD Fe-S	e	~0
8	Fumarate + H_2O ⇌ L-malate	Fumarase		c	−0.9
9	L-Malate + NAD^+ ⇌ oxaloacetate + NADH + H^+	Malate dehydrogenase		e	+7.1

*Reaction type: (a) Condensation (b) Dehydration (c) Hydration (d) Decarboxylation (e) Oxidation (f) Substrate-level phosphorylation

Appendix Figure 4.10. Stryer 1988, p. 378.A detailed depiction of the nine types of chemical reaction of the Krebs cycle. The "citric acid cycle" is a synonym for the Krebs Cycle.


(A)

Appendix Figure 4.11. Alberts et al. 1994, p. 83. "About 500 common metabolic reactions are shown diagrammatically, with each chemical species represented by a filled circle. The centrally placed reactions of the glycolytic pathway and the citric acid cycle are shown in *red*. A typical mammalian cell synthesizes more than 10,000 different proteins, a major proportion of which are enzymes. In the arbitrarily selection segment of this metabolic maze (shaded *yellow*), cholesterol is synthesized from acetyl CoA." (caption on p. 82)



Scheme 1. Retrosynthetic analysis for longifolene **1** (1957) (see text).

An abbreviated form of the 1957 retrosynthetic plan for the synthesis of longifolene (**1**) is shown in Scheme 1. Changes in the retrosynthetic direction are indicated by a double arrow (⇒) to distinguish them from the synthetic direction of chemical reactions (→) and the number below indicates the number of transforms required for the retrosynthetic change if greater than one. The selection of transforms was initially guided by a topological strategy (disconnection of bond *a* in **1**). The Michael transform, which simplifies structure **2** to precursor **3**, can be found by general transform selection procedures.[5, 9] The starting materials for the synthesis which emerge from retrosynthetic analysis, **4** and **5**, have little resemblance to the target structure **1**.

Appendix Figure 4.12. Corey 1991, pp. 457-458. A reaction series and a piece of Corey's text. Note that the double arrow indicates the *retrosynthetic* direction, which is in a direction opposite to the chemical reaction.

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EDUCATION

Indiana University, Bloomington, IN.
History and Philosophy of Science Department, Ph.D. program. 8/98 – 10/03
Biology Department, Masters program in Ecological and Evolutionary Biology. 8/99 – January 2004 (expected)

Stanford University, Stanford, CA. 9/94 – 6/96
Philosophy Department, M.A.

Stanford University, Stanford, CA. 9/90 – 6/94
Philosophy Department, B.A. with Honors in History and Philosophy of Science.

Colegio Internacional de Caracas, Caracas, Venezuela. 9/87 – 6/90
High School International Baccalaureate (I.B.) Diploma.

AREAS OF SPECIALIZATION

Philosophy of Science, Philosophy of Biology, History of Biology, Evolutionary Theory

AREAS OF COMPETENCE

Metaphysics and Epistemology, History of Modern Philosophy

HONORS, PRIZES, AND FELLOWSHIPS

Victor Thoren Research Fellowship, History and Philosophy of Science Department, Indiana University, Bloomington, IN, August 2002 – August 2003.

Marjorie Grene Graduate Student Essay Prize, International Society for the History, Philosophy and Social Studies of Biology, for "August Weismann on Germ-Plasm Variation," 2001.

Norwood Russell Hanson Graduate Student Essay Prize, History and Philosophy of Science Department, Indiana University, Bloomington, IN, for "Varieties of Modules: Kinds, Levels, Origins and Behaviors," 2001.

Summer 2001 Research Fellowship through the Biology Department, Indiana University, Bloomington, IN, May – August 2001.

Chancellor's Fellowship through the History and Philosophy of Science Department, Indiana University, Bloomington, IN, August 1998 – August 2002.

PROFESSIONAL ACTIVITIES

Member

American Philosophical Association
Philosophy of Science Association
International Society for the History, Philosophy and Social Studies of Biology

Referee

Philosophy of Science
Journal of Experimental Zoology (Molecular and Developmental Evolution)
The Biologist

Recent Conferences Attended

Philosophy of Science Association, Milwaukee, WI, November 2002.

Dibner Institute Summer Seminar, "From Embryology to Evo-Devo," Woods Hole, MA, May – June 2001. Graduate student discussion leader.

Dibner Institute Summer Seminar, "Putting Humans Into Ecology," Woods Hole, MA, May – June 2000.

Dibner Institute Summer Seminar, "Why Ernst Haeckel?", Woods Hole, MA, June 1999.

Philosophy of Science Association, Kansas City, KS, October 1998.

TEACHING AND RESEARCH EXPERIENCE

Ph.D. Dissertation, Indiana University, IN. 10/03
Chair: Elisabeth A. Lloyd
Committee: Sander Gliboff, Victor Goodman, Frederick Schmitt, Michael J. Wade
Title: Formal Biology and Compositional Biology as Two Kinds of Biological Theorizing.

Instructor, Indiana University, Bloomington. 1/00 – 8/02
"Ants, Apes and Humans: Genes, Behaviors, and Societies" (self-designed course).
"Introduction to Scientific Reasoning" (pre-existing course; syllabus reworked).

Associate Instructor, Indiana University, Bloomington. 8/99 – 12/99
"Voyages of Scientific Discovery: From Captain Cook to Captain Kirk."

Laboratory Researcher, Indiana University, Bloomington. 1/01 – 8/02
Acquired molecular biology techniques in a bacteriology lab (Carl Bauer's lab).
Acquired population biology techniques in a Tribolium lab (Michael J. Wade's lab).

High School Biology Teacher, French-American International School, San Francisco. 9/96 – 6/98

Research Assistant, California Academy of Sciences, San Francisco. 7/96 – 8/98; 6/94 – 8/94

Masters Thesis, Stanford University, CA. 6/96
Supervisor: Peter Godfrey-Smith.
Title: Constructions of Form in Biological Systems.

Undergraduate Philosophy Club, Stanford University, CA. 1/93 – 6/94

Undergraduate Honors Thesis, Stanford University, CA. 6/94
Supervisor: Peter Godfrey-Smith.
Title: Charles Darwin's Views on Inheritance and Variation.

PERSONAL INFORMATION

Native speaker of English, Spanish, and Danish; working knowledge of French.

Danish Citizen.
Born in Aabenraa, Denmark on March 20, 1972.
Lived in Caracas, Venezuela from September 1972 until June 1990.
Lived in the USA from September 1990 until October 2003.
Currently living in Mexico City.

PUBLICATIONS

Refereed

Michael J. Wade, Rasmus G. Winther, Aneil F. Agrawal, and Charles J. Goodnight. 2001. "Alternative Definitions of Epistasis: Dependence and Interaction," Trends in Ecology and Evolution, 16: 498-504.

2001. "August Weismann on Germ-Plasm Variation," Journal of the History of Biology, 34: 517-555.
[Winning essay for Grene Prize.]

2001. "Varieties of Modules: Kinds, Levels, Origins and Behaviors," Journal of Experimental Zoology (Molecular and Developmental Evolution), 291: 116-129.
[Winning essay for Hanson Prize.]

2000. "Darwin on Variation and Heredity," Journal of the History of Biology, 33: 425-455.

Robert J. van Syoc and Rasmus Winther. 1999. "Sponge-Inhabiting Barnacles of the Americas: A New Species of *Acasta* (Cirripedia, Archaeobalanidae), First Record from the Eastern Pacific, Including Discussion of the Evolution of Cirral Morphology," Crustaceana, 72: 467-486.

Invited

In press, 2003. "Evolutionary Developmental Biology Meets Levels of Selection: Modular Integration or Competition, or Both?", to appear in Modularity: Understanding the Development and Evolution of Complex Natural Systems (W. Callebaut and D. Rasskin-Gutman, eds.), MIT press.

2001. Review of Ants at Work: The Organization of a Social Insect Colony by Deborah Gordon, Philosophy of Science, 68: 268-270.

2000. "William Morton Wheeler," Encyclopedia of the Life Sciences (website) – http://www.els.net

Submitted

"An Empirical Analysis of Theoretical Perspectives in Compositional Biology," Biology and Philosophy

PRESENTATIONS

"Parts and Kinds in Evolutionary Developmental Biology," delivered at a Graduate Student Workshop, Philosophy Department, Stanford University, CA, December 2002.

"Mereology and Modules: Parts and Wholes in Evolutionary Developmental Biology," delivered to the Philosophy Department, Notre Dame University, South Bend, IN, March 2002.

"On Integrated Wholes and Competing Parts," delivered to the History and Philosophy of Science Department, Indiana University, Bloomington, IN, November 2001. Hanson Prize Lecture.

"Selectional, Instructional and Maturational Theories in Evo-Devo and Behavior," co-authored with Susan Oyama, delivered to the International Society for the History, Philosophy and Social Studies of Biology, Quinnipiac University, CT, July 2001.

"Evolutionary Developmental Biology Meets Levels of Selection: Modular Integration or Competition, or Both?", delivered at a symposium entitled "Modularity: Understanding the Development and Evolution of Complex Natural Systems" at the Konrad Lorenz Institute, Vienna, Austria, October 2000.

"Multi-Level Selection or Modular Integration? Two Perspectives on Biological Individualization," poster presentation at a symposium entitled "Modularity in development and evolution" at the Hanse Wissenschaftskolleg, Delmenhorst, Germany, May 2000.

"Weismann's Lamarckism: On the Inheritance of Acquired Germinal Characteristics," delivered to the International Society for the History, Philosophy and Social Studies of Biology, Oaxaca, Mexico, July 1999.

"Darwin on External Sources of Heritable Variation," delivered to the International Society for the History, Philosophy and Social Studies of Biology, Seattle, WA, July 1997.