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Why the (gene) counting argument fails in the massive modularity debate: The need for understanding gene concepts and genotype-phenotype relationships

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A number of debates in philosophy of biology and psychology, as well as in their respective sciences, hinge on particular views about the relationship between genotypes and phenotypes. One such view is that the genotype-phenotype relationship is relatively straightforward, in the sense that a genome contains the "genes for" the various traits that an organism exhibits. This leads to the assumption that if a particular set of traits is posited to be present in an organism, there must be a corresponding number of genes in that organism's genome to account for those traits. This assumption underlies what can be called the "counting argument," in which empirical estimates of the number of genes in a genome are used to support or refute particular hypotheses in philosophical debates about biology and psychology. In this paper, we assess the counting argument as it is used in discussions of the alleged massive modularity of the brain, and conclude that this argument cannot be upheld in light of recent philosophical work on gene concepts and empirical work on genome complexity. In doing so, we illustrate that there are those on both sides of the debate about massive modularity who rely on an incorrect view of gene concepts and the nature of the genotype-phenotype relationship.

Keywords: Counting Argument; Evolutionary Psychology; Gene Concept; Genome Complexity; Genotype-Phenotype Relationship; Massive Modularity

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1. Introduction

Assumptions about the relationship between genotypes and phenotypes play a central role in many debates among philosophers of biology and psychology, as well as among biologists and psychologists themselves. One such assumption is that there exists a relatively straightforward relationship between genotypes and phenotypes such that, to the extent that genes play a role in the production of organismal traits, the phenotypic complexity of organisms should be reflected in their genotypic complexity (Bates et al., 1998, p. 593; Buller, 2005, pp. 129–131; Buller & Hardcastle, 2000, p. 314; Cummins, Cummins, & Poirier, 2003, p. 146; Ehrlich, 2000, pp. 124–126; Ehrlich & Feldman, 2003, p. 92; Lewontin, 1991, p. 98). We shall call this assumption the *correspondence assumption*, referring to the assumed correspondence between genotypic and phenotypic complexity. On the basis of this assumption, many of these authors have advanced what we call the *counting argument*. This argument states that in order for a particular set of evolutionarily produced traits to be present in an organism, there must be a corresponding number of genes to serve as their selective bases; estimates of the number of genes in a genome then lead to conclusions about the evolutionary basis (or lack thereof) of particular traits.

At present, the counting argument is most widespread in debates about Evolutionary Psychology (EP) and its Massive Modularity Thesis (MMT), which holds that the brain consists of hundreds or thousands of independently evolved modules.[1] However, instances of the counting argument can be found more widely. For example, Lewontin has argued that human behavior cannot be completely determined by our genes, as there aren't enough genes in the human genome to specify the myriad neuronal connections that would be required to account for the variety of human behaviors that we observe: "there is enough human DNA to make about 250,000 genes. But that would be insufficient to determine the incredible complexity of human social organization if it were coded in detail by specific neuronal connections" (1991, p. 98; see also p. 123). Several authors have used the counting argument to criticize MMT, arguing that the brain cannot consist of hundreds or thousands of separately evolved modules because the human genome doesn't contain sufficient genes to serve as separate genetic bases for all these modules (Bates et al., 1998; Buller, 2005; Buller & Hardcastle, 2000; Cummins et al., 2003; Ehrlich, 2000; Ehrlich & Feldman, 2000).

In this paper, we criticize the counting argument on the grounds that it is based on a misunderstanding of the relationship between genotypes and phenotypes. The most sophisticated version of the argument appears in Buller (2005), thus we focus on this version in order to examine the counting argument in its strongest form. It is important to note that others have offered brief criticisms of the counting argument (e.g., Barrett & Kurzban, 2006, pp. 639–641; Hagen, 2005, pp. 151–152; Marcus, 2004, pp. 131–134, 152–158; Sarnecki, 2007, pp. 531–532; Woodward & Cowie, 2004, pp. 318–319). However, we feel that there is a need for a more detailed analysis, as several aspects of the argument and of its relations to philosophy of biology have so far gone unnoticed.

In section 2, we explicate the counting argument and illustrate that there are at least two instantiations of it, each of which relies on different kinds of data drawn from different fields of investigation. Neither proponents nor opponents of the counting argument have acknowledged this distinction, and in many cases draw on only one of the two instantiations, as we illustrate in section 3. Here we examine criticisms of the counting argument to illustrate the current state of affairs in the debate—a state of affairs that our paper advances both by providing a more detailed analysis of the counting argument as well as by bringing to bear relevant concepts from philosophy of biology. In section 4, we elaborate our criticism of the counting argument, demonstrating that the debate crucially rests on the way genes are conceptualized, and assess the counting argument in light of the large body of literature on gene concepts in philosophy of biology. As we point out, under current molecular gene concepts the relationship between genotypes and phenotypes is not straightforward—there are a number of molecular effects that link genes to multiple products and gene products to multiple genes (e.g., gene overlap, frameshifting, antisense reading, RNA editing, and alternative splicing). Thus, there is no possible one-to-one mapping between particular organismal traits (such as brain modules) and genes, so the number of genes in an organism's genome has no special bearing on the number of traits it can possess. Therefore, the counting argument ultimately fails and is in fact irrelevant to debates about EP and MMT. In section 5, we take a broader view of those debates to show that Evolutionary Psychologists themselves have misunderstood the relationship between genotypes and phenotypes. Thus, our aim in this paper is neither to provide support for or criticism of EP or MMT; rather, it is to examine both sides of the debate around the counting argument, to illustrate a lack of detailed, accurate accounts of the genotype-phenotype relationship in the debate, and to bring to bear relevant discussions of gene concepts from philosophy of biology.

2. The Counting Argument

One of the main characteristics of EP is its endorsement of MMT. Although MMT is usually treated as one thesis, it is actually composed of two claims: first, that the brain consists of hundreds or thousands of domain-specific modules; and second, that each of these modules has evolved independently as a response to particular selection pressures to which our early ancestors were exposed. Here, "independent evolution" does not mean that each module has its own evolutionary trajectory that is completely independent from the evolution of other traits in the organism; rather, each module constitutes a distinct adaptation that has evolved in response to particular selection pressures and has its own hereditary basis (Barrett & Kurzban, 2006).

In the debates about EP and MMT, the counting argument and its underlying correspondence assumption have been most clearly elaborated by Buller (2005) in his

Adapting minds:

> If our brains consisted of numerous modules, each of which was ''specified'' by a gene complex, we would expect some positive correlation between brain complexity and the number of genes in a genome. . . . Yet when we look at the . . . data we find no such correlation. (p. 130)

Buller actually makes two separate but related arguments, each of which is an instantiation of the more general counting argument. The first goes as follows: if MMT is true, we would expect the number of genes in the human genome to have increased proportionately with the number of modules in the human brain; humans clearly have more complexly functioning brains (i.e., more modules) than organisms of other species; however, scientific data suggest that humans do not have significantly more genes than organisms of other species; thus, MMT is disconfirmed (pp. 129–131). The expectation here is that differences in brain complexity between distinct species must be correlated with differences in the number of genes in the genomes between those species; thus, we call this the *between-species counting argument*.

In the first step of this argument, Buller reasons that, in order for separate modules to have independently evolved, there must have been at least as many independent mutations in the genome as there are separate modules. Although he admits that a gene complex for one module may have effects on other modules, he also states that since MMT claims that each module evolved independently, these effects could not explain the existence of separate modules:

> First, according to Evolutionary Psychologists, each module (or ''brain circuit'') was ''added'' to the mind at some point in human evolutionary history and then subsequently ''shaped'' by selection to be highly effective at solving adaptive problems in its proprietary domain. . . . Second . . . separate modules evolved in response to independent selective forces and were functionally modifiable independently of other modules.
>
> These two claims entail that each module evolved as a result of numerous mutations over human evolutionary history, each of which added or modified a specialized ''brain circuit,'' and all of which were preserved by selection as the gene complex that regulates the development of the module. In short, *each module must be the developmental product of its own gene complex* [emphasis added]. (2005, p. 129)

Buller thinks that in order for many domain-specific modules to have evolved independently, more and more genes would have to have been ''added'' to the human genome in the course of human evolution, leading to one specific gene complex for each module. Thus, he reasons, since human brains became more complex (in the sense that they came to possess more domain-specific modules), one would expect a correlation between brain complexity and the number of genes in the genome. Buller then suggests that there is no correlation between brain complexity and gene number. He compares estimates of the number of genes in the human genome (30,000–90,000) to that of the mouse (80,000 or more),[2] and argues that because the human brain is clearly more complex than the mouse brain: ''there is *no correlation whatsoever*. Species with very complex brains simply don't have more

'genetic information' available for building those brains than do species with relatively simple brains'' (2005, p. 131). Since, on the correspondence assumption, MMT suggests that there should at least be *some* degree of correlation between brain complexity and genome complexity (which Buller cashes out in terms of number of genes), and since current scientific data indicate otherwise, Buller concludes that MMT is unlikely to be true.

The second of Buller's arguments has a similar form: if MMT is true, we would expect there to be a higher proportion of genes involved in building evolved domain-specific modules than in building other, less complex traits in particular sensory structures; scientific data suggest otherwise; thus, MMT is disconfirmed (2005, pp. 129–130). Here, the expectation is that the amount of complexity of a certain structure within a species (e.g., the modular complexity of the brain as well as the complexity of individual modules) must be correlated with the number of genes devoted to building that structure within that same species. Thus, we call this the *within-species counting argument*.

This argument builds upon some of the same assumptions and reasoning in the previous argument. Buller states that if MMT were true, we would expect there to be at least one specific ''gene complex'' for each module since each module must have evolved independently. If this were the case, then ''the number of genes for features of the human brain would have had to increase proportionately. More and more of the human genome would have had to become involved in building the growing number of modules in the human brain'' (2005, pp. 129–130). According to Buller, if this is true, we would expect a much higher percentage of genes to be devoted to building modules compared to other features of the brain—such as sensory cells—presumably because brain modules are much more complex. Buller then provides statistical data which suggest that this expectation does not hold. According to his figures, about 50% of the genes in the genome are involved in building the brain, with about 30% of these involved in building the sensory organs (p. 130). Assuming there are about 30,000 genes in the human genome, this suggests that about 15,000 genes would be available for building the brain, about 5,000 of which would be involved in building the sensory organs, leaving only 10,000 available for building the hundreds or thousands of modules that Evolutionary Psychologists hypothesize to exist. But, Buller suggests, we would expect *many* more genes to be involved in building a domain-specific module than in building a sensory receptor, as we would assume that brain modules are far more complex than sensory organs. Thus, Buller concludes that scientific data disconfirm MMT.

Most of the previous attempts to advance the counting argument against MMT (or a similar thesis, such as domain specificity or innateness) have focused almost exclusively on the within-species counting argument. For example, Bates et al. (1998) argue against nativism on mathematical grounds: they suggest that there are too many synaptic connections in the brain (which they estimate to be 10^{14}) to be controlled by the relatively small number of genes in the genome (10^6, according to the authors). While they also point to the high degree of genetic overlap between humans and other species, their focus is on the inability of a relatively small number

of genes to provide enough information:

> Genes would need a lot of information to orchestrate a system of this size. Of course, a detailed mapping from genes to cortex would still be possible if genes behaved like letters in the alphabet, yielding up an indefinite set of combinations. But this is not the case; instead, genes operate within a highly constrained spatiotemporal and chemical matrix. (Bates et al., 1998, p. 593)

Buller and Hardcastle (2000; see also Buller, 2005, chapter 4) also focus on the within-species counting argument. Not only do they argue that there aren't enough genes to code for the trillions of synaptic connections in our brain, but they also suggest, as Buller does above, that most of the genes involved in building the brain are dedicated to peripheral structures such as the sensory cells in the nose (Buller & Hardcastle, 2000, p. 314). Ehrlich (2000) and Ehrlich and Feldman (2003) similarly argue that there is a "gene shortage"; i.e., that there are not enough genes to program our behavior given the variety of behavior that we observe: "why, then, couldn't just a few genes have evolved to program millions of our behaviors? In theory they might have, but in that case human behavior would be very stereotyped" (Ehrlich & Feldman, 2003, p. 92). They go on to argue that the relatively low number of genes suggests that genes must often have multiple effects (pleiotropy), because if genes did not have multiple effects it would have been difficult to account for most of the observed traits of organisms with a very small number of genes. Finally, Cummins et al. (2003) argue against innate cognitive modules by citing a "poverty of genetic resources" and alluding to the arguments provided in Buller and Hardcastle (2000).

The main thrust of these criticisms of innate modularity can be broken into three parts: (1) that in order for the hundreds or thousands of modules posited by MMT to have evolved, all the synaptic connections in our brain would have to be genetically specified, (2) that this genetic specification requires a very high number of genes (at least in the millions), and (3) that current estimates of the number of genes in the human genome indicate that there are not nearly enough genes to fulfill the second requirement. In what follows, we argue that both (1) and (2) are not well-justified assumptions, and that while current estimates of the number of genes in the human genome are much lower than was previously estimated, (3) is still problematic insofar as it rests on (2).

It is interesting to note that these assumptions are specific to the within-species counting argument. Not surprisingly then, most of the criticisms of the counting argument have only targeted the within-species version, as we show in the next section. Subsequently, in section 4, we examine both versions of the counting argument, illustrating that neither version can be upheld on any of the available gene concepts and that, furthermore, many of the responses to the counting argument have missed this point.

3. Responses to the Counting Argument

Previous responses to the counting argument have identified some of the assumptions underlying it, either calling them into question or outright refuting

them (Barrett & Kurzban, 2006, pp. 639–641; Hagen, 2005, pp. 151–152; Marcus, 2004, pp. 131–134 & 152–158; Sarnecki, 2007, pp. 531–532; Woodward & Cowie, 2004, pp. 318–319). In this section, we review these responses. While we believe these authors are on the right track, their discussions have not analyzed the counting argument in its strongest form—examining all of its assumptions in both versions—nor have they considered how the use of different gene concepts affects the argument, a key issue in our view.

Both Woodward and Cowie (2004) and Hagen (2005) criticize the second part of the within-species counting argument—which states that genetic specification of the brain requires a very high number of genes—simply by pointing to one of the interactive effects of genes. According to Woodward and Cowie, ''the role of regulatory genes and networks in governing the expression of structural genes probably generates many more combinatorial possibilities than the figure of 30,000 genes suggests'' (2004, p. 319). While the authors do not find the counting argument to be decisive, they do take it as ''suggestive'' in that it draws attention ''to the need for evolutionary psychologists to explain, consistently with what is known about brain development, how cognitive modules could be genetically specified'' (p. 319). Hagen also rebuts the counting argument, as it appears in Ehrlich and Feldman (2003), by pointing out that adaptations arise from interactive gene effects. According to his calculations, ''to produce an adaptation ... more than two genes interact. The number of 25-gene combinations is around 10^{82}...'' (Hagen, 2005, p. 152). Furthermore, he suggests that the counting argument, taken to its conclusion, would imply that there aren't enough genes to specify the thousands of physical adaptations in humans, other than properties of the brain, and that such an implication is problematic. These authors are certainly right to point out that the counting argument seems to assume a non-interactive view of genes—a view that is certainly not compatible with current scientific evidence. However, they do not discuss in any detail the many kinds of interactions that occur in the genotype-phenotype relationship in order to elucidate what genes are in the first place, as we do here (section 4.2).

Marcus (2004) provides a more detailed response to the counting argument, targeting Ehrlich (2000) and Ehrlich and Feldman's (2003) discussion of a ''gene shortage.'' Marcus argues against the assumption that a very high number of genes are needed to specify a massively modular brain, though he also seems to disagree with the first assumption, that massive modularity requires direct genetic specification of synaptic connections. Marcus makes four main points. First, he says that genes specify a process for building structures rather than a bitmap or picture of the structure itself (p. 156). This suggests a different view of how genes ''specify'' neuronal connections—rather than specifying the trillions of connections directly, they specify the process for building them. Second, genes do not work independently, but in combination with one another. As a result, the effects of new genes are exponential rather than linear. Third, one gene can have many effects. Marcus points out that changing one gene in a fly (*Pax6*) can result in the development of a new eye on the fly's antenna, which illustrates that one gene can

cause the formation of millions of cells into a complex structure. Finally, Marcus points out that the same genes get used again and again, resulting in different structures depending on their expression. Thus, ''the sheer number of genes underestimates the amount of information in the genome because a single gene can have multiple functions associated with multiple regulatory regions'' (p. 154).

These points undermine a number of the assumptions in the counting argument, not the least of which is the idea that in order for a domain-specific brain to have evolved each module would require its own ''gene complex.'' Marcus argues against this assumption, suggesting a great deal of overlap between evolved structures:

> Specialized biological structures need not be, and perhaps never are, made up entirely, or even in large part, of wholly novel materials. Consider, for example, the differences between an arm and a leg. Thousands of genes contribute to each, but probably only a handful are special to the arm. (p. 132)

While these arguments target the within-species counting argument, they can also be used to cast doubt on assumptions underlying the between-species counting argument. For example, it may be a mistake to assume that only evolutionary ''novel'' genes (that is, genes that do not have homologues in other species) can account for uniquely human traits, as Buller seems to do. In principle, any of the genes in the human genome that are homologous with those in other species could have been modified during the course of human evolution, and thus underlie traits that are specific to humans.[3]

Barrett and Kurzban (2006) and Sarnecki (2007) draw from Marcus's work, emphasizing and expanding certain aspects of his criticism. Sarnecki reiterates Marcus's first and fourth points, that genes can regulate processes of building structures even if they don't specify the details, and that they can be recursively employed, citing Marcus's example of genetic specification of human arms and legs. In addition, he calls into question the idea of direct genetic control that seems to underlie the counting argument (the version of which he cites from Buller & Hardcastle, 2000). He says:

> While Buller and Hardcastle do not suppose that genetic control amounts to a direct one to one correspondence between genetic structures and neuronal ones, they do require some suitably low multiple of genes to neuronal or axonal/dendritic structures—and this seems unlikely. (Sarnecki, 2007, p. 531)

Again, while this response is on the right track, it does not discuss current scientific views or data that would support the conclusion. An issue that remains open is exactly *why* it would be unlikely that the ratio of genes to neuronal structures is low, i.e., how a comparatively low number of genes can produce complex structures. Specifying the underlying reasons involves explicating the ways in which DNA is expressed in the cell, a discussion we provide in section 4.2. Barrett and Kurzban (2006) also draw from Marcus (2004), focusing on his third and fourth points, namely that few genetic mutations are needed to produce large phenotypic effects (and hence new modules), and that many of the genes involved in specifying a new module are likely to be conserved, also citing the arms and legs example.

Thus, they conclude, ''the answer to the question, 'Does each module need 'its own' set of dedicated genes?' is no'' (p. 640).

In addition, like Woodward and Cowie, Barrett and Kurzban point out that there are some unanswered questions about massive modularity, namely regarding the number of modules that exist and the pathway from genes to modules. However, unlike Woodward and Cowie, they do not take this to be a problem with Evolutionary Psychology, but rather with criticisms of it: ''absent the answers to these questions, constraining hypotheses about modularity with reference to numbers of genes seems insurmountably problematic'' (Barrett & Kurzban, 2006, p. 639). They go on to suggest that without understanding the genotype-phenotype relationship, critics cannot specify the genetic requirements for massive modularity. As we've tried to show here, neither the critics nor those who seem to be defending MMT have provided a thorough and accurate picture of the genotype-phenotype relationship such that the genetic requirements *can* be specified. We discuss this further in section 5. In the next section, we take these responses one step further: what we *do* know about the genotype-phenotype relationship suggests that the requirements critics have laid out—namely that there must be a very high number of genes, certainly much higher than exist—are not supported by current understandings of genetics.

4. Analyzing the Counting Argument in Terms of Gene Concepts

As illustrated above, both the between-species counting argument and the within-species counting argument rest on the correspondence assumption, i.e., the idea that brain complexity (understood here as the number of modules in the brain) and genome complexity (understood as the number of genes in the genome) correspond. However, this assumption and its underlying notions of complexity rest on a particular understanding of the nature of genes and their relationship to phenotypes. In order to illustrate this, we again focus on Buller's version of the counting argument. We demonstrate that the gene concept Buller uses is inappropriate given the particular fields he draws upon for the scientific data in his argument, and that the gene concept that is appropriate, namely the molecular gene concept, undermines the counting argument.

4.1. Buller's Concept of the Gene and the Classical Gene Concept

Buller's description of what genes are and how they relate to phenotypes suggests that he is using a simple ''gene for'' view. This view (which is widespread, especially in popular literature and the media) conceives of genes as segments of DNA that serve as important causal factors directly responsible for the presence of a particular trait. On this view, the presence of a gene is a necessary prerequisite for the organism to exhibit the associated trait; that is, while the presence of the gene G for trait T doesn't guarantee the presence of T, absence of G—or other genes with the same

effect—guarantees the absence of T. Buller describes his view as follows:

> To say that a gene or genotype *G* is "for" a phenotype *P* means first of all that, other things being equal, an organism with *G* is more likely to have *P* than is any organism without *G* (that is, with possible rival allele of *G*). . . . Thus, to say that *G* is "for" *P* means, second, that *G* must play a causal role in the development of *P* (in those organisms with *P*). When these two conditions are met, it is perfectly sensible to speak of genes or genotypes as being "for" phenotypes. (2005, p. 25)

On this view, a new trait cannot arise without the addition of at least one new gene in the genome.

This view is sometimes identified with the gene concept that was held by classical geneticists in the early 20th century; thus, we might think that Buller is drawing on the classical gene concept. However, Waters (1994) has argued that classical genetics did not encompass such a simple understanding of the nature of genes as is represented in the common "gene for" view. In classical genetic explanations, genes did not feature simply as the underlying causes of organismal phenotypes in statements of the form "the presence of gene *x* causally contributes to the presence of trait *T*." Rather, genes featured only indirectly in the explanation of organismal phenotypes in the way that *differences* between two organisms' phenotypes were explained by *differences* in their genotypes. As Waters writes, "the basic dogma of classical genetics was that gene *differences* [emphasis added] cause phenotypic *differences* [emphasis added]. . . . What were studied were character *differences* [emphasis added], not characters, and what explained them were *differences* in genes, not the genes themselves" (1994, p. 172). In classical genetics, then, it was not the case that explanations related single genes (the explanantia) to single organismal phenotypes (the explananda). According to Waters, the traits of individual organisms weren't even what classical geneticists wanted to explain. Rather, what needed explanation were observed differences in phenotypes between organisms of the same species, and what did the explanatory work were postulated differences in those organisms' genes. The explanatory relation thus was not of the form "Organism O has trait T because it has gene G," but rather "Organisms O and P exhibit a difference in trait T because of an underlying difference in their genes." On this "difference view," phenotypic differences can be produced in several ways that do *not* require the presence of an additional gene—for example, by way of differences in interactions among or between genes, or differences in interactions between genes and environmental factors. This is a much broader view of how genomic differences can lead to phenotypic differences than is implied in the counting argument, as that argument clearly requires the addition of new genes for the production of new traits.

In contrast to the simple "gene-for view," the "difference view" takes into account one of the important lessons of genetic research, namely that there are many different kinds of genomic differences that can bring about differences in phenotype. The difference view recognizes that genes do not typically function in isolation but usually work in various combinations and in various contexts to produce organismal phenotypes. Changes in the context in which a particular stretch of DNA is

expressed—either changes in environmental factors or changes in other genes—can thus lead to changes in phenotype. For example, phenotypic differences can arise from differences in *interactions* between genes; the fact that a gene is involved in the production of one brain module does not mean that it cannot be involved in the production of one or several other brain modules. It is therefore not necessarily the case that the addition of a new trait via evolutionary processes, such as a new brain module, is *always* accompanied by an increase in gene number; new modules can in principle also arise from new combinations of genes that are already available or by their being differently expressed in new contexts.

Research and theorizing in comparative genomics adds to this argument. According to Moss, for example:

> Genome sequence analysis suggests that it does not appear likely that differences between mammals will be reflected at all in gross gene number . . . . While the earlier realization that organismic complexity does not scale with overall genome size has been referred to as the *C-value paradox* . . . the more recent realization that organismic complexity does not scale with gene number has been referred to as the *N-value paradox*. (2006, p. 936)

Of course, there must be *some* kind of genetic difference in order to account for the phenotypic differences that MMT postulates. Moss discusses two such differences that have been shown to account for phenotypic differences between species, including humans: differences in gene density and differences in repetitive DNA sequences. Scientific data drawn from studies of over 25 different species suggest that the human genome has a very low density of genes per total amount of DNA relative to other species: compared to eubacteria and archaea, the human genome has a lower gene density by a factor of 100, and the coding sequences of the human genome only account for 1% of its genome while for *E. coli* the coding sequences account for over 90% (Moss, 2006, p. 937). Moss concludes, ''whereas gene numbers do not correlate with organismic complexity in particularly striking ways, the separation of coding regions by noncoding regions does appear to correlate with organismic complexity in a very striking way'' (ibid). With respect to repetitive DNA sequences, Moss comes to a similar conclusion: ''where humans thus far appear to be most distinct at the molecular level is not with respect to unique gene sequences but rather with respect to DNA sequences which are found to be repetitive'' (ibid p. 938).

While we are not arguing that new brain features actually come into being in any of the ways discussed above (this is in need of empirical investigation, after all), or that these features would count as modules according to EP, the possibility that new features arise in the course of evolution without a corresponding increase in the number of genes cannot be ruled out in advance as those making the counting argument would have it. As we've shown, Buller's view, and the counting argument more generally, seems to rely on a gene concept that does not match the one actually featured in classical genetics—and if it is reinterpreted using the correct gene concept, the counting argument no longer holds.

4.2. The Counting Argument and the Molecular Gene Concept

In addition to the classical gene concept, contemporary molecular biologists regularly make use of what is commonly called the "molecular gene concept" (Waters, 1997, 2004), also known as the "classical molecular gene concept" (Stotz, Griffiths, & Knight, 2004, p. 649) or the "neoclassical gene concept" (Portin, 1993). As Waters (2007, section 4.2) has argued, it is not the case that the molecular gene concept has replaced the classical gene concept, but rather that both of these concepts are still in use in contemporary molecular biology. Since the counting argument pertains to how genes are conceived of in molecular biology (i.e., in the context of counting the number of genes in a genome), it would seem that it could draw on either the classical gene concept (which, as we argued in the previous section, severely weakens the counting argument) or the molecular gene concept. In this section, we argue that the molecular gene concept is incompatible with the counting argument as well.

The molecular gene concept reveals two ways in which the proposed correlation between brain complexity and gene number is broken. First, the relationship between genotype and phenotype is not as direct as the counting argument suggests. According to Waters:

> The fundamental concept underlying the application of "gene" in molecular biology is that of *a gene for a linear sequence in a product at some stage of genetic expression* . . . called the "*molecular gene concept*." . . . Genes are for linear sequences in products of genetic expression. (1994, p. 178)

On this concept, genes don't directly produce phenotypes, but rather are identified by the molecular products they are involved in making. Even the relationship between molecular genes and their molecular products is not straightforward, as one particular segment of DNA can be involved in bringing about several different products, which in turn can play a role in the development of many different traits; in other words, there is no possible one-to-one mapping between particular expression products and continuous linear stretches of DNA.

The following are examples of the kinds of effects that can complicate the relationship between molecular genes and their products.[4] (1) Molecular genes can *overlap* with one another and can even be nested within one another in the DNA sequence, which means that a particular sequence of nucleotides may be part of more than one molecular gene. (2) In the transcription from DNA to mRNA, *frameshifting* can occur in which the reading of a DNA sequence begins one or more nucleotides to the left or right of where it normally begins, and results in a wholly different expression product than would otherwise be the case. (3) *Antisense reading* can also occur: DNA sequences can be read in both the 'normal' and the reverse directions, leading to different mRNA sequences and thus to different expression products. (4) A fourth effect is *RNA editing*, which is the insertion, deletion or substitution of single nucleotides in the transcription from chromosomal DNA to mRNA. (5) Finally, there is *alternative splicing*. When DNA is transcribed into mRNA via an intermediate primary RNA transcript, the parts of the primary RNA transcript that correspond to non-expressed parts of the DNA (i.e., introns) are spliced out while those parts that

correspond to the DNA that will ultimately be expressed (i.e., exons) are transcribed, spliced together, and subsequently used in the production of a polypeptide or RNA molecule. Alternative splicing means that a particular DNA segment can be transcribed into mRNA in a number of different ways, given that exons can be spliced together in various combinations, leading to the production of different expression products. (For example, not all exons that make up a gene are always transcribed into mRNA. For a more extensive discussion, see Ast, 2005.)

Extreme examples of how one gene can lead to many different products can be found in instances of alternative splicing. For example, the *Dscam* gene in *Drosophila* can produce up to 30,016 different expression products depending on the way in which it is spliced when it is expressed (Knight, 2007, p. 297; Wieben, 2003, p. 583). Also in *Drosophila*, alternative splicing and RNA editing in the expression of the *para* gene can theoretically lead to over a million different RNA transcripts, each of which encodes for a slightly different protein (Szathmáry, Jordán, & Pál 2001, p. 1316). Alternative splicing can also *directly* lead to major phenotypic differences. A nice example of this is again found in *Drosophila*: whether a *D. melanogaster* embryo develops into a male or a female fruitfly depends on the way in which the molecular *sex lethal* gene (*Sxl*) is spliced, where one way of splicing leads to a functional protein product and a female fruitfly, and other ways lead to a non-functional protein and a male fruitfly (Ast, 2005, p. 63).

What is most important with respect to our present discussion is that, despite disagreements in exact estimates, there is widespread agreement that alternative splicing is common in humans: Szathmáry, Jordán, & Pál (2001) state that at least 35% of gene transcripts in humans are subject to alternative splicing, while other authors provide figures of 50% (Brett, Popsil, Valcárcel, Reich, & Bork, 2002, p. 29; Stetefeld & Ruegg, 2005), 60% (Keller & Harel, 2007, p. 10), and 75% (Ast, 2005, p. 60). According to Ast, ''alternative splicing has emerged as the pivotal process that permits a small number of genes to generate the much larger assortment of proteins needed to produce the human body and mind'' (2005, p. 65). Thus, it is clearly an important factor that disrupts the presumed correspondence between phenotypic complexity and gene number.

Ever since the human genome project showed that there are far fewer genes in the human genome than was expected, there has been a tendency among scientists to cease measuring biological complexity in terms of the number of genes in a genome; rather, the relevant measure has become the degree of connectivity of the networks in which genes function. Szathmáry et al. write:

> We need to distinguish between two forms of genomic complexity: one measured by the number of genes and the other by the connectivity of gene-regulation networks. The complexity of organisms (in terms of morphology and behavior) correlates better with the second definition. (2001, p. 1316)

Other scientists and philosophers have made a similar point (e.g., Claverie, 2001; Miklos & Rubin, 1996, p. 522; Moss, 2006, p. 936). The gene concept that fits these notions of complexity, the molecular gene concept, connects genes to their molecular

products rather than to organismal phenotypes. Thus, adopting this concept further undermines the correspondence assumption and the counting argument more generally.

5. The Failure of the Counting Argument and its Consequences for Debates About MMT

The specific aim of this paper has been to show that the counting argument is not successful. We have argued that a central assumption in this argument is inconsistent with accepted scientific accounts of how genotypes relate to phenotypes. Current scientific evidence does not rule out the possibility that a relatively small number of genes in the human genome could *in principle* build a brain structure that consists of hundreds or thousands of evolved modules; whether or not this is actually the case has yet to be determined.

We have also shown that the counting argument cannot be upheld on any of the currently accepted gene concepts. From the perspective of both the classical and molecular concepts there are clear problems for the counting argument since there is no reason to expect phenotypic complexity to be captured by gene number. In other words, due to the aforementioned effects as well as the phenomenon of gene interaction, there is no reason to expect the correspondence assumption to hold. The counting argument thus fails because it rests on an erroneous view of how organisms' genotypes relate to their phenotypes. We have drawn on the gene concept literature in order to illustrate this point, as well as to bring the philosophy of biology literature to bear on debates about MMT.

The specific criticisms that we have made against the counting argument point to a more general concern: a failure of those engaged in the debates surrounding the counting argument to present a *complete and accurate* account of the relationship between genotypes and phenotypes, a relationship that is crucial in trying to understand how brain modules may have evolved. We've already shown that proponents of the counting argument (who criticize MMT) fail to understand this relationship. In what follows, we broaden our analysis of the state of affairs in debates about MMT—as they relate to gene concepts and the genotype-phenotype relationship—by illustrating that Evolutionary Psychologists (who defend MMT) also seem to misunderstand the nature of genes and their effects. First, in section 5.1, we examine Tooby's (2001) attempt to rebut the counting argument, an attempt that rests on reconceiving genes in an unjustified manner. In section 5.2, we consider developmentalist critiques that are parallel to our own. While we have criticized the counting argument based on current understandings of molecular biology, developmental psychologists and psychobiologists have been criticizing Evolutionary Psychologists for their account of how genes lead to cognitive outcomes. Finally, in section 5.3, we discuss a recent paper by some Evolutionary Psychologists (Confer et al., 2010) who argue that they are simply *not required* to provide an account of the genetic basis underlying MMT; thus, they try to shift the burden of providing such an

account off of Evolutionary Psychologists. These discussions serve to show that it is not only critics of MMT, but Evolutionary Psychologists themselves, who seem to misunderstand gene concepts and the nature of the genotype-phenotype relationship.

5.1. An Evolutionary Psychologist's Response to the Counting Argument

Despite the fact that the counting argument fails based on current scientific theories about the genotype-phenotype relationship, Tooby (2001, p. 263), one of the main proponents of MMT, has acknowledged the counting argument as a "genuinely interesting argument" that might pose a valid problem for the position. In an attempt to circumvent this problem, Tooby has adopted an extremely liberal view of what genes are. For example, he suggests that, "for Darwinians . . . a gene is any nucleotide sequence whose modification would lead to a different developmental outcome. By this definition, the number of genes in our genome is presently unknown, but orders of magnitude higher [than only 30,000]" (Tooby, 2001, p. 263). Tooby appears to be endorsing a suggestion similar to the one given by Marcus (2004, p. 154), that not only protein-coding sequences, but in principle also regulatory sequences, etc., should be counted as genes. This would be a much more liberal understanding of genes than either the molecular gene concept or the classical gene concept, in any of their varieties, encompasses.

However, there are two problems with this move. First, it goes against much of what biologists have thought and currently think about genes. Tooby's assertion is simply not a commonly held view among biologists or philosophers of biology, and may not be held by anyone other than Tooby himself. Second, this move implies an acceptance of the correspondence assumption, and tries to refute the counting argument by attacking the second premise, which claims that there are an insufficient number of genes available to build a massively modular brain. The attack proceeds by redefining genes in an ad hoc manner so as to increase the estimated number of genes in the human genome to a sufficient number, which seems to be a rather weak strategy, especially as the resulting definition does not correspond to any of the gene concepts currently in use. But, irrespective of whether or not this move works, we have argued that the counting argument already fails on the correspondence assumption such that there is no need to resort to this weak strategy.

5.2. Developmental Criticisms of EP and MMT

Several authors have criticized EP on the grounds that contributions of developmental processes and environmental factors to the formation of organisms have not been taken seriously by Evolutionary Psychologists. For example, both Karmiloff-Smith (2006) and Lickliter and Honeycutt (2003) have criticized Evolutionary Psychologists for presuming a one-to-one relationship between genotypes and phenotypes.

Karmiloff-Smith (2006) notes that that people who defend MMT think that one can easily map genes and their protein products on cognitive outcomes. In contrast, she argues that a direct mapping from genes and their protein products to cognitive

outcomes is almost impossible because these cognitive outcomes are the result of very complex interactions taking place at the level of developmental processes. She points out that, ''domain-specific end states can stem from more domain-general start states'' (Karmiloff-Smith, 2006, p. 9) and therefore that it is very difficult to say, for example, that there is a specific gene responsible for language. Although she does not deny that ability to speak language has a genetic basis, she insists that such ability cannot be mapped onto a single gene or gene complex.

Lickliter & Honeycutt (2003) advance a more general argument against the view in classic evolutionary biology that genes are primarily responsible for phenotypic outcomes and that genetic and environmental factors can be neatly partitioned in the production of developmental outcomes. Evolutionary Psychologists, Lickliter & Honeycutt argue, assume that there is a direct road from genes and their products to so-called modules. However, recent studies in biology show that organismal development has multiple determinants, such as genes, hormones, and environmental variables, and that these contributing factors cannot be neatly separated. Some morphological and physiological traits are not pre-specified by genes in the way assumed by Evolutionary Psychologists. For example, some female members of certain species of coral reef fish change sex when all males from the group are removed (Lickliter & Honeycutt, 2003, p. 821). This example, according to Lickliter & Honeycutt, shows that even sex is not pre-specified by genes alone and that environmental cues and other developmental factors make important contributions to these traits. Buller (2005) makes a similar argument against EP, pointing out that, for example, the immune system consists of specialized systems for different kinds of diseases that are not determined in advance by genes, as they require environmental input in order to realize this specialization.

These arguments do not target the counting argument directly, but are complementary to those presented in this paper. They aim to show that the relationship between genotype and phenotype is very complex and that relating genes to phenotypic outcomes is extremely difficult as such a mapping also requires incorporating other factors besides genes. As these authors illustrate, Evolutionary Psychologists make assumptions about how genotype and phenotype are related that conflict with recent results in biology.

5.3. Must Evolutionary Psychology Specify the Genetic Basis of Traits?

Some Evolutionary Psychologists have dealt with these issues not by specifying the genetic basis of MMT, but by arguing that they need not do so. For example, Confer et al. (2010) emphasize that Evolutionary Psychology does not presume genetic determinism (that is, it does not presume a tight connection between genotype and phenotype); yet, the way in which they clarify this point is by pointing to the role of the environment. While their paper includes a section entitled, ''What role do genes play in the framework of evolutionary psychology?'' they do not discuss the complex relationship between genotypes and phenotypes. Rather, Confer et al. just don't believe they have to identify the genetic basis of traits (or presumably modules) in

order to make arguments to the effect that human behaviors are the results of adaptations (see especially 2010, p. 120), which would allow them to avoid the problem that Tooby (2001) tries to address. However, this seems too easy a way out. Proponents of Evolutionary Psychology assume that each of the hundreds or thousands of modules in the brain has been molded by natural selection in response to particular environmental demands. Given the centrality of this assumption, they should either present a plausible account of how new modules can arise genetically, or ground their assumption in biological/genetic research that shows how modules can or do actually arise. Confer et al., however, do neither: they deny that they have to identify the genetic basis of mental modules and fail to present an account of how modules actually arise that is rooted in the latest biological knowledge. Thus, their assumption that brain modules have arisen by means of natural selection stands unsupported.

The considerations we have presented in this paper suggest that, if Evolutionary Psychologists want to ground this assumption, they should not approach the issue by trying to identify the genetic basis of mental modules, as mapping mental modules on genes or gene complexes is not possible. Rather, they ought to come up with a convincing account of how mental modules arise in real organisms that is well grounded in current biological knowledge. Such an account, however, is still lacking.

6. Conclusion

In this paper, we have argued that current scientific knowledge about the relationship between genotypes and phenotypes does not rule out the possibility that a relatively small number of genes in the human genome could build a brain structure that consists of hundreds or thousands of evolved modules. Thus, the counting argument fails. However, this should not provide comfort to proponents of MMT. First, although the arguments we've provided here undermine the counting argument, they are merely consistent with MMT and do not provide independent support for it. This can be seen, for example, in the fact that our considerations are also consistent with a view that opposes MMT, the ''generalist genes'' hypothesis, evidence for which supports a non-modular view of the brain (see Kovas & Plomin, 2006a; 2006b; Marcus & Rabagliati, 2006). Second, as Woodward and Cowie (2004, p. 319) point out, and as we discussed above, a plausible scientific account of how new brain modules come into being is still lacking, as is a concrete understanding of what modules are to begin with. We thus believe that our conclusion that the counting argument fails to undermine MMT should be taken *not* as support for MMT or EP, but rather as an indication that the assumptions involved in the counting argument are *irrelevant* to the debate about the tenability of MMT and hence the feasibility of EP. Therefore, attention in this debate should be redirected to other, more promising criticisms that have been offered (e.g., Bechtel, 2003; Dupré, 2001; Richardson, 2007; Samuels, 1998; Woodward & Cowie, 2004).

Our analysis also reveals that people on both sides of the debate have misunderstood the complexity of the genotype-phenotype relationship. Counting genes is not an effective strategy for criticizing MMT, nor for defending it. Certainly, if the correspondence assumption were correct then EP could not have gotten off the ground at all, as its main core thesis, MMT, would have been falsified. However, the converse does not hold: one cannot infer that EP is supported because the correspondence assumption fails. Our argument is thus similar in spirit to that found in Samuels (1998), who argues that much of the evidence that purportedly supports MMT is consistent with another hypothesis as well (in that case, the "Library Model of Cognition"). Whether or not MMT is a well-supported thesis is an issue that must be addressed separately from the considerations presented here (i.e., in ways other than counting genes). We hope to have shown, however, that in addressing this issue, one must attend to the complexity of the relationship between genotypes and phenotypes, and that looking to gene concepts is a helpful way to begin.

Notes

[1] Following Buller (2005), we distinguish between evolutionary psychology as a general approach to studying the mind from an evolutionary perspective (lower case 'ep'), and the specific research program of 'Evolutionary Psychology' (capitalized 'EP') defended by Barkow, Cosmides, and Tooby (1992), Buss (1998), Cosmides and Tooby (1997), and Pinker (2002). Bechtel (2003, p. 211) draws a similar distinction using the terms 'broad evolutionary psychology' and 'narrow evolutionary psychology', respectively. We explicate MMT in section 2; for debates about MMT, see Carruthers (2004), Fodor (2001), Frankenhuis and Ploeger (2007), Machery (2007, 2008), Machery and Barrett (2006), Samuels (1998), Silvers (2007), Sperber (2002), and Woodward and Cowie (2004).

[2] These estimates are the only data Buller offers with respect to the relative complexity of the human genome, and he doesn't provide references for those data. Strangely, as it would seem to strengthen his position, he does not discuss recent literature that puts the size of the human genome at a much lower estimate of about 25,000-30,000 genes. This much smaller number of genes was quite unexpected and was addressed in a wave of publications that appeared after the publication of the first drafts of the human genome in 2001 (Claverie, 2001; International Human Genome Sequencing Consortium, 2001; Szathmáry et al. 2001; Venter et al., 2001).

[3] We thank H. Clark Barrett for this point. See also Barrett & Kurzban (2006, pp. 639–641).

[4] It is generally accepted that the complications mentioned here occur quite commonly. Recent overviews can be found in Ast (2005), Griffiths & Stotz (2007), Portin (1993), Stotz et al. (2004), Waters (1994, 2007), Weber (2005, pp. 188–228), and Wieben (2003).

References

Ast, G. (2005). The alternative genome. *Scientific American*, *292*, 58–65.

Barkow, J. H., Cosmides, L., & Tooby, J. (1992). *The adapted mind: Evolutionary psychology and the generation of culture*. New York: Oxford University Press.

Barrett, H. C., & Kurzban, R. (2006). Modularity in cognition: Framing the debate. *Psychological Review*, *113*, 628–647.

Bates, E., Elman, J., Johnson, M., Karmiloff-Smith, A., Parisi, D., & Plunkett, K. (1998). Innateness and emergentism. In W. Bechtel & G. Graham (Eds.), *A Companion to cognitive science* (pp. 590–601). Oxford: Blackwell.

Bechtel, W. (2003). Modules, brain parts, and evolutionary psychology. In S. Scher & F. Rauscher (Eds.), *Evolutionary psychology: Alternative approaches* (pp. 211–227). Dordrecht, NL: Kluwer.

Brett, D., Popsil, H., Valcárcel, J., Reich, J., & Bork, P. (2002). Alternative splicing and genome complexity. *Nature Genetics*, *30*, 29–30.

Buller, D. J. (2005). *Adapting minds: Evolutionary psychology and the persistent quest for human nature*. Cambridge, MA: MIT Press.

Buller, D. J., & Hardcastle, V. G. (2000). Evolutionary psychology, meet developmental neurobiology: Against promiscuous modularity. *Brain and Mind*, *1*, 307–325.

Buss, D. M. (1998). *Evolutionary psychology: The new science of the mind*. Boston: Allyn & Bacon.

Carruthers, P. (2004). The mind is a system of modules shaped by natural selection. In C. Hitchcock (Ed.), *Contemporary debates in philosophy of science* (pp. 293–311). Oxford: Blackwell.

Claverie, J-M. (2001). What if there are only 30,000 human genes? *Science*, *291*, 1255–1257.

Confer, J. C., Easton, J. A., Fleischman, D. S., Goetz, C. D., Lewis, D. M. G., Perilloux, C., et al. (2010). Evolutionary psychology: Controversies, questions, prospects, and limitations. *American Psychologist*, *65*, 110–126.

Cosmides, L., & Tooby, J. (1997). *Evolutionary psychology: A primer*. Retrieved August 25, 2011, from University of California, Santa Barbara, Center for Evolutionary Psychology Web site Retrieved August 15, 2011, from: http://www.psych.ucsb.edu/research/cep/primer.html

Cummins, D., Cummins, R., & Poirier, P. (2003). Cognitive evolutionary psychology without representational nativism. *Journal of Experimental & Theoretical Artificial Intelligence*, *15*, 143–159.

Dupré, J. (2001). *Human nature and the limits of science*. Oxford: Clarendon Press.

Ehrlich, P. R. (2000). *Human natures: Genes, cultures, and the human prospect*. Washington, DC: Island Press.

Ehrlich, P. R., & Feldman, M. (2003). Genes and cultures: What creates our behavioral phenome? *Current Anthropology*, *44*, 87–107.

Fodor, J. (2001). *The mind doesn't work that way: The scope and limits of computational psychology*. Cambridge, MA: MIT Press.

Frankenhuis, W. E., & Ploeger, A. (2007). Evolutionary psychology versus Fodor: Arguments for and against the massive modularity hypothesis. *Philosophical Psychology*, *20*, 687–710.

Griffiths, P. E., & Stotz, K. (2007). Gene. In D. L. Hull & M. Ruse (Eds.), *Cambridge companion to the philosophy of biology* (pp. 85–102). Cambridge: Cambridge University Press.

Hagen, E. H. (2005). Controversial issues in evolutionary psychology. In D. M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 145–173). Hoboken, NJ: John Wiley & Sons.

International Human Genome Sequencing Consortium (2001). Initial Sequencing and Analysis of the Human Genome. *Nature*, *409*, 860–921.

Karmiloff-Smith, A. (2006). The tortuous route from genes to behavior: A neuroconstructivist approach. *Cognitive, Affective, & Behavioral Neuroscience*, *6*, 9–17.

Keller, E. F., & Harel, D. (2007). Beyond the gene. *PLoS ONE*, *2*, e1231.

Knight, R. (2007). Reports of the death of the gene are greatly exaggerated. *Biology and Philosophy*, *22*, 293–306.

Kovas, Y., & Plomin, R. (2006a). Generalist genes: Implications for the cognitive sciences. *Trends in Cognitive Sciences*, *10*, 198–203.

Kovas, Y., & Plomin, R. (2006b). Response to Marcus and Rabagliati 'Genes and Domain Specificity'. *Trends in Cognitive Sciences*, *10*, 398.

Lewontin, R. C. (1991). *Biology as ideology*. New York: Harper Perennial.

Lickliter, R., & Honeycutt, H. (2003). Developmental dynamics: Toward a biologically plausible evolutionary psychology. *Psychological Bulletin*, *129*, 819–835.

Machery, E. (2007). Massive modularity and brain evolution. *Philosophy of Science*, *74*, 825–838.
Machery, E. (2008). Modularity and the flexibility of human cognition. *Mind & Language*, *23*, 263–272.
Machery, E., & Barrett, H. C. (2006). Debunking *Adapting minds*. *Philosophy of Science*, *73*, 232–246.
Marcus, G. (2004). *The birth of the mind: How a tiny number of genes creates the complexities of human thought*. New York: Basic Books.
Marcus, G. F., & Rabagliati, H. (2006). Genes and domain specificity. *Trends in Cognitive Sciences*, *10*, 397–398.
Miklos, G. L. G, & Rubin, G. M. (1996). The role of the genome project in determining gene function: Insights from model organisms. *Cell*, *86*, 521–529.
Moss, L. (2003). *What genes can't do*. Cambridge, MA: MIT Press.
Pinker, S. (2002). *The blank slate: The modern denial of human nature*. New York: Viking.
Portin, P. (1993). The concept of the gene: Short history and present status. *Quarterly Review of Biology*, *68*, 173–223.
Richardson, R. (2007). *Evolutionary psychology as maladpated psychology*. Cambridge, MA: MIT Press.
Samuels, R. (1998). Evolutionary psychology and the massive modularity hypothesis. *British Journal for Philosophy of Science*, *49*, 575–602.
Sarnecki, J. (2007). Developmental objections to evolutionary modularity. *Biology and Philosophy*, *22*, 529–546.
Silvers, S. (2007). Adaptation, plasticity, and massive modularity in evolutionary psychology: An essay on David Buller's *Adapting minds*. *Philosophical Psychology*, *20*, 793–813.
Sperber, D. (2002). In defense of massive modularity. In E. Dupoux (Ed.), *Language, brain and cognitive development: Essays in honor of Jacques Mehler* (pp. 47–57). Cambridge, MA: MIT Press.
Stetefeld, J., & Ruegg, M. A. (2005). Structural and functional diversity generated by alternative mRNA splicing. *Trends in Biochemical Sciences*, *30*, 515–521.
Stotz, K., Griffiths, P. E., & Knight, R. (2004). How biologists conceptualize genes: An empirical study. *Studies in History and Philosophy of Biological and Biomedical Sciences*, *35*, 647–673.
Szathmáry, E., Jordán, F., & Pál, C. (2001). Can genes explain biological complexity? *Science*, *292*, 1315–1316.
Tooby, J. (2001). Is human nature hidden in the genome? *Nature Genetics*, *29*, 363.
Venter, J. C., Adams, M. D., Myers, E. W., Li, P. W., Mural, R. J., Sutton, G. G., et al. (2001). The sequence of the human genome. *Science*, *291*, 1304–1351.
Waters, C. K. (1994). Genes made molecular. *Philosophy of Science*, *61*, 163–185.
Waters, C. K. (2007). Molecular genetics. In E. N. Zalta (Ed.), *The Stanford encyclopedia of philosophy*. (Spring 2007 edition) Retrieved August 25, 2011, from http://plato.stanford.edu/archives/spr2007/entries/molecular-genetics/
Weber, M. (2005). *Philosophy of experimental biology*. Cambridge: Cambridge University Press.
Wieben, E. D. (2003). Primer on medical genomics part VII: The evolving concept of the gene. *Mayo Clinic Proceedings*, *78*, 580–587.
Woodward, J., & Cowie, F. (2004). The mind is not (just) a system of modules shaped (just) by natural selection. In C. Hitchcock (Ed.), *Contemporary debates in philosophy of science* (pp. 312–334). Oxford: Blackwell.