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Psychopathic personalities and developmental systems

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ABSTRACT

Is psychopathy born or made? Contemporary psychopathy research shows that there is much wrong with this question. It is increasingly accepted that the development of psychopathy is dependent on multiple causal factors interacting with one another. However, there remains the major theoretical challenge of understanding the relations between these multiple causal factors in the developmental process. In this paper, I argue that the conventional picture of gene-environment interactionism does not offer an adequate account of psychopathy development. Instead, I propose that a theoretical framework from the philosophy of biology, namely developmental systems theory, can facilitate a better understanding of psychopathy development that captures the contingent and dynamic relations between multiple causal factors. Some practical implications of a developmental systems theory approach to psychopathy are also explored.

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1. Introduction

Psychopathy is typically characterized as a behavioral phenotype that is associated with a cluster of interpersonal and affective features. These include impulsivity, aggression, grandiosity, insincerity, superficial charm, shallow affect, remorselessness, and lack of empathy (Cleckley, 1941; Hare, 1991). These features, among others, are operationalized in Robert Hare's *Psychopathy Checklist – Revised* (Hare, 1991). This is a standard assessment instrument for psychopathy that consists of twenty items divided into two factors, which are "callous, selfish, remorseless use of others" and "chronically unstable and antisocial lifestyle". A person is diagnosed with psychopathy if he or she exhibits a sufficient number of items on the checklist. While there is considerable overlap with the diagnosis of antisocial personality disorder in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2013), psychopathy is a different construct that places more emphasis on a particular affective trait, namely callous

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unemotionality. Accordingly, callousness and impulsivity are often hypothesized as being the core features of psychopathy which predict the associated antisocial and criminal behaviors (Hare & Neumann, 2008).

People diagnosed with psychopathy are of legal interest because there are statistics that suggest that they are more likely to commit violent crimes than non-psychopaths, are approximately twenty times more likely than to be in prison than non-psychopaths, and are four to eight times more likely to recidivate than non-psychopaths (Kiehl & Hoffman, 2011). However, not all people with psychopathic personalities come to the attention of the criminal justice system. There is research suggesting that people with psychopathic personalities are disproportionately common in the corporate world and the political world (Boddy, 2011).

Given that psychopathy is often connected with behavior that is considered harmful, the question of what causes someone to develop a psychopathic personality is of considerable interest, not just among researchers and practitioners, but also in popular culture. Commonly, in popular media, this question is couched in the language of the nature-nurture dichotomy, which is the assumption that a trait is either determined by the individual's genes or acquired through the individual's interaction with the environment. For example, an article on psychopathy in the health news resource *Medical Daily* uses with the headline, "Research Indicates that Psychopaths are Made, Not Born" (Brice, 2012), while *The Telegraph* makes the opposite claim with the headline, "Psychopaths are Born Not Bred, According to a New Study" (Alleyne, 2009). And so, the assumption that psychopathy is either innate or acquired remains deeply entrenched in popular culture.

Contemporary research on psychopathy, however, indicates that its causation is more complicated than the picture suggested in popular culture. Genetic determinism is widely recognized to be false, while cultural determinism seems to miss out relevant variations among individuals. Instead, empirical studies have suggested that internal and external influences have much more subtle and complex roles in the development of psychopathy. Accordingly, researchers now generally accept that neither genetic variations nor environmental conditions by themselves are sufficient to account for the development of psychopathy, but rather that psychopathy results from these diverse influences interacting with one another in contingent ways (Beaver et al., 2011; Hyde et al., 2016; Sadeh et al., 2010; Tuvblad et al., 2017).

A major challenge, then, is to understand the relations between these diverse causal factors in psychopathy development. In this paper, I show how a theoretical framework from the philosophy of biology, namely developmental systems theory, can facilitate an understanding of how psychopathy is caused that captures the dynamic and contingent ways in which the various factors interact. Although developmental systems theory is a general theoretical framework with broad applicability, I have two reasons for

focusing specifically on psychopathy. First, the multifactorial causation and developmental complexity associated with psychopathy make developmental systems theory particularly needful for its explanation. Second, a fresh understanding of psychopathy under developmental systems theory could have practical implications for how we intervene on such behavior, which would be welcome in light of the significant moral, legal, and social challenges posed by psychopathy.

Before I proceed, it is worth noting that psychopathy as a construct is not uncontroversial. It has been argued that many of the diagnostic criteria are vague, qualitative, and laden with moral value judgments, which can result in biases and poor reliability in their applications (Martens, 2008). Indeed, many of the items on the diagnostic checklist appear to be behavioral features that we consider to be immoral. It has also been argued that more might be learned by understanding antisocial behavior as an interpersonal interaction rather than decontextualizing it as an individual disposition (Longino, 2019). From a moral philosophy perspective, Jonathan Glover (2014) has argued that the moral and emotional landscapes of people diagnosed with psychopathy are much richer and more intelligible than the items on the diagnostic checklist suggest. For the purpose of scope, I will not be discussing these issues at length in this paper. While I do agree with some of the criticisms, the present paper will largely take the current construct used in psychopathy research for granted, as the aim is to consider some of the philosophical issues that pertain to interpretations of the findings of this research.

The rest of this paper will proceed as follows. In §2, I will argue, with references to conceptual work from the philosophy of biology and empirical research on causal factors, that conventional gene-environment interactionism cannot account for some key features of psychopathy development. In §3, I will present the central themes of developmental systems theory and will show how they can facilitate a more comprehensive account of how causal factors interact throughout the developmental process to produce psychopathy. In §4, I will explore what practical implications a developmental systems theory approach to psychopathy might have for therapeutic interventions.

2. Gene-environment interactionism

A substantial amount of the research into the causal basis of psychopathy has been in the discipline of quantitative behavioral genetics, which seeks to quantify the degrees to which the expression of a given trait in a population correlates with genetic variation and with environmental variation. Studies in quantitative behavioral genetics use a statistical method known as the analysis of variance, which enables calculations and comparisons of

variations in the expression of the trait in different groups of the studied population (Fisher, 1918). For human behavioral traits, the data usually come from family studies, twin studies, and adoption studies. Twin studies allow us to compare the concordance rates for monozygotic twins who share effectively all of their genomes with those for dizygotic twins who share approximately half of their genomes, while adoption studies allow us to compare the concordance rates for adoptive siblings with those for biological siblings. The aim is to obtain an estimate of heritability, which is a measure of how much of the variation in the expression of a trait in the studied population is correlated with genetic variation in that population. The degree to which the variation in trait expression in the studied population is due to environmental variation can then be calculated by subtracting the heritability from the total variation in that population.

In studies involving populations of adults and adolescents, the heritability of psychopathy has been estimated to be approximately 0.4, which suggests that most of the variation in the expression of psychopathy is due to differences in the environments of the individuals in the studied populations. For example, a study of male adolescent twins yielded heritability calculations of o.39 and o.42 for antisocial and detachment traits respectively, suggesting that o.61 and o.58 of the variances in these respective traits were attributable to environmental factors (Loney et al., 2003). Similarly, a study of kinship pairs estimated the heritability of psychopathy to be between o.37 and o.44, suggesting that between 0.56 and 0.63 of the variance in the expression of psychopathy was due to differences in the environments of the participants (Beaver et al., 2011). However, another twin study found the heritability of psychopathy in a population of children to follow a rather different pattern, with 0.25, 0.48, and 0.27 of the variance in the expression of callous-unemotional behavior to be correlated respectively with genetic variation, non-shared environmental factors, and shared environmental factors in the studied population (Tuvblad et al., 2017).

The above results lend support to gene-environment interactionism, which is the view that the expression of a phenotype, such as psychopathy, depends on the interactions between genetic factors and environmental factors. This raises the question of what sort of interactionism is warranted. As noted by Ford and Lerner (1992), the gene-environment interactionism that is often suggested in quantitative behavioral genetics is a form of *static* interactionism, whereby genetic and environmental factors are assumed to be additive components that contribute to the variation in the expression of a phenotype in stable proportions. Moreover, it is assumed that these relative influences can be quantified without reference to the developmental context (Griffiths & Tabery, 2013, p. 83). This is reflected in the suggestion that quantitative behavioral genetics can reveal "the *relative genetic and environmental contributions* to both the interpersonal-affective (Fearless

Dominance) and antisocial (Impulsive Antisociality) traits of psychopathy" (Blonigen et al., 2005, p. 644, emphasis added).

However, the static interactionism associated with quantitative behavioral genetics has been criticized for failing to capture the causal process of trait development. Notably, Gilbert Gottlieb suggests that the "population view of behavioral genetics . . . is based on the erroneous assumption that the quantitative analysis of the genetic and environmental contributions to individual differences sheds light on the developmental process of individuals" (Gottlieb, 2003, p. 338). In this spirit, I argue that the conventional picture of gene-environment interactionism does not offer an adequate account of how psychopathy is caused. Contrary to static interactionism which characterizes genetic and environmental influences as stable and independent components, I propose that the understanding of psychopathy development requires a form of *dynamic* interactionism, according to which the trait develops through the contingent and reciprocal interactions of multiple resources whose contributions to development cannot straightforwardly be decomposed into additive components. The critical discussion will begin by underscoring a general problem with static interactionism concerning the interpretation of heritability, before drawing on empirical evidence to show how this problem specifically applies to psychopathy.

Let us proceed with the general problem concerning the interpretation of heritability in quantitative behavioral genetics. As noted above, heritability is a measure of the degree to which the *variation* in the expression of a trait *in the studied population* is correlated with genetic *variation* in that population. If understood correctly, this is a useful statistic. However, it is important to be clear about what this statistic does not tell us. Heritability neither reflects the degree to which the presence of the trait *in an individual* is caused by the individual's genes, nor the degree to which the trait *in general* is caused by genetic factors. A heritability of 1 for a given trait does not mean that the trait is genetically determined and a heritability of 0 does not mean that it is environmentally determined.

The non-entailment from heritability to genetic causation in general is articulated by Richard Lewontin (1974). As noted above, heritability is a measure of how much genetic variation is correlated with the variation in trait expression *in the studied population*. Crucially, that studied population is associated with a particular environmental context, and so its members are exposed to a certain range of environmental influences. If the environmental context is changed so that the members of the population are exposed to a different range of environmental influences, then the measure of heritability is also likely to change. For example, in a homogeneous environmental context where nutritional resources are equally distributed among the members of the population, the variation in height in that population is likely to correlate strongly with genetic variation

in that population, thus yielding a high heritability. However, in a more heterogeneous environmental context where nutritional resources are unequally distributed among the members of the population, the variation in height is likely to be influenced by this environmental variation, thus yielding a lower heritability. The upshot of Lewontin's argument is that heritability is not a measure of genetic influence *tout court*, but is relative to the particular environmental context in which it is measured. Insofar as the method for calculating heritability implicitly takes for granted the environmental context of the studied population but does not take into account how heritability measures differ across different environmental contexts, it obscures the actual causal contribution of the environmental context to the expression of the trait.

The non-entailment from heritability to genetic contribution in the individual case is given detailed philosophical treatment by Elliott Sober (1988). The kind of interdependence exhibited by genetic and environmental factors in development makes it conceptually incoherent to decompose the expression of a trait in a given individual into additive genetic and environmental components. To clarify why this is the case, Sober imagines a fictional scenario in which it would make sense to decompose the relative causal contributions of genetic and environmental factors into such additive components:

> Suppose height were the result of the accumulation of height particles, which organisms could obtain from their environment and also from their genes. Imagine that an individual's height is some increasing function of the number of height particles obtained from all sources. If so, we could look at local facts about Jane and say whether her genes or environment contributed more. (Sober, 1988, p. 312)

In this fictional scenario, the genetic and environmental influences are additive, because they are commensurable in the ways they produce their effects. The quantity of height particles serves as a "common currency" that would allow us to decompose Jane's height and to measure how much of it was obtained from her genes versus how much of it was obtained from the environment (Sober, 1988, p. 312). The genetic and environmental influences in this fictional scenario are also independent, and so it would make sense to ask how much of Jane's height is solely due to genetic factors and how much is solely due to environmental factors.

However, in the actual world, genetic and environmental factors do not accumulate in this way. Rather, they are interdependent in such a way that neither kind of factor on its own would produce any portion of the trait in the absence of the other kind of factor. Genes cannot produce anything on their own without the relevant environmental resources, while environmental resources influence development by interacting with genes. Therefore, it is not possible to decompose Jane's height in a way that allows

us to say that that *this* much of it is caused by genetic factors and that *that* much is caused by environmental factors. Each kind of factor in isolation would produce no height. It is only through their joint interplay that they enable any height to develop at all.

The above has implications for how we understand causation in interactions between genetic and environmental factors. A common idea in the philosophical literature on causation is the idea that causes are difference makers (Mill, 1843; Woodward, 2003). A particularly influential example of such an account in contemporary philosophy of science is James Woodward's (2003) interventionist theory of causation, which states that for *C* to count as a cause of *E* is for there to be a regular, though not necessarily universal, response of *E* to an intervention on *C* in at least some background circumstances. This is usually coupled with the notion of modularity, which is the notion that the system consists of distinct causal components that are "independently disruptable" (Hausman & Woodward, 1999, p. 550). Paradigmatic examples of modular causes are those that satisfy John Stuart Mill's (1843) principle of the composition of causes. This states that "the joint effect of several causes is identical with the sum of their separate effects" (Mill, 1843, p.426). For example, in a closed system consisting of a particle, a downward gravitational force, and a downward electrical force, the downward acceleration of the particle would equal the sum of the acceleration due to the gravitational force and the acceleration due to the electrical force.

With respect to the development of a trait in an organism, Sober's (1988) analysis shows the genetic and environmental influences do not satisfy the principle of the composition of causes. As noted above, a trait in a given individual develops through the joint interactions of genetic and environmental factors, but genetic factors in isolation have zero magnitude and environmental factors in isolation have zero magnitude. Hence, the magnitude of the total effect does not equal the sum of the magnitudes of the individual effects. Nonetheless, there are ways in which genetic and environmental factors may still be *modular*, even if they are not *additive*. For example, Sober argues that we can make counterfactual inferences about how Jane's height would have been different had her genes been different but her environment the same and how her height would have been different had her genes been the same but her environment different. These would not be inferences about the *proportions* of Jane's height that are produced by her genes and by her environment, but they would still be inferences about the *differences* that changes in certain factors are likely to make to the overall outcome when all other factors are kept fixed.

This suggests that we may still be able to analyze genetic and environmental factors as difference makers under the interventionist theory of

causation, even though they are not additive components. However, in order to see whether a certain kind of intervention on *C* yields a regular response of *E* in at least some background circumstances, it is important to specify what these background circumstances are. A problem for static interactionism is that these background circumstances are not stable, but dynamic. In some cases, the dynamic ways in which different factors interact with one another may result in changes in the background circumstances, and so the effects of intervening on particular factors may not be consistent across different developmental contexts. For example, the effect of increased nutrition on Jane's height will vary depending on whether this intervention is introduced in infancy or in adolescence. As we shall see, this can be partly overcome by assuming a form of dynamic interactionism which acknowledges the context sensitivity of the system. By taking into account these different developmental contexts, dynamic interactionism can allow us to specify more precisely the range of background circumstances over which particular interventions have their effects.

Having laid out the general problem with static interactionism, let us now relate this back more specifically to psychopathy. The complex and non-additive ways in which genetic and environmental causes interact have been highlighted by recent studies in molecular genetics. One study suggests that being raised in a socioeconomically disadvantaged environment is associated with an increased risk of psychopathy in adolescents who are homozygous with the long allele of the serotonin transporter (5-HTTPLR) gene, but not so much in adolescents who are homozygous with the short allele or who are heterozygous (Sadeh et al., 2010). Another study reports that participants with a certain variant of the dopamine receptor (DRD4) gene who had experienced poor caregiving in infancy developed more psychopathic traits in adolescence (Nikitopoulos et al., 2014). Furthermore, participants with the same genotype who had experienced good caregiving in infancy exhibited the fewest psychopathic traits in adolescence, while there was no significant association between poor caregiving and psychopathic traits in participants without this genotype.

What is significant about these studies in molecular genetics is that they highlight different ways in which genetic variants and environmental conditions can interact with one another in development. Both of the above studies indicate that the genetic variants on their own are insufficient to produce psychopathic traits and that such traits also depend on certain environmental conditions being present in order to develop. However, each study suggests a different interaction pattern. The study on 5-HTTPLR and socioeconomic disadvantage is suggestive of a diathesis-stress pattern, whereby adverse environmental conditions increase the risk of the trait developing in individuals with a certain genetic variant (Sadeh et al., 2010). By contrast, the study on DRD4 and

caregiving in infancy is suggestive of differential susceptibility pattern, whereby individuals with a certain genetic variant may be more sensitive to both positive and negative early environments than those individuals without that genetic variant (Nikitopoulos et al., 2014). In both kinds of pattern, the phenotypic outcomes are not the sums of independent additive components, but the contingent effects of joint interactions between different factors at certain developmental stages. Furthermore, the differential susceptibility pattern in the latter study highlights the complex ways in which these factors interact. Not only do the environmental factors interact with genetic factors in the development of the trait in the individual, but the genetic factors also influence the sensitivity of the individual to particular environmental factors. And so, the interactions are dynamic and reciprocal.

This dynamic complexity is further emphasized by studies on the social environmental causes of psychopathy. These studies have yielded robust results indicating that poor caregiving in infancy and childhood trauma causally contribute to psychopathy development. In a recent community study, poor parental bonding, childhood physical abuse, and early separation from caregivers were strongly associated with subsequent psychopathic traits in adulthood (Gao et al., 2010). This cross-sectional study relied on the retrospective reports of adults with and without psychopathic traits. However, the presence of a causal connection between poor caregiving in infancy and psychopathy has also been supported by longitudinal studies. These assessed the quality of caregiving received by infants at different stages in time and prospectively measured psychopathic traits in these individuals while controlling for other confounding factors (Pardini et al., 2007; Waller et al., 2015; Willoughby et al., 2013). For example, a study of children and their mothers showed that harsh parenting, measured at regular intervals from ages two, was associated with the subsequent development and maintenance of callous-unemotional behavior from age three onwards (Waller et al., 2015). Interestingly, another longitudinal study showed that only those children who experience harsh parenting in *early* infancy are more likely to develop callous-unemotional traits, whereas those who experience harsh parenting in *later* toddlerhood are more likely to develop oppositional-defiant traits (Willoughby et al., 2013).

Other longitudinal studies have, in addition to measuring the effects of poor caregiving, also measured the effects of interventions which mitigate against this poor caregiving, including positive interactions with adoptive parents and specific parenting therapies. These have shown that secure parental attachment in infancy protects against the development of psychopathy and can decrease callous-unemotional behavior (Hyde et al., 2016; McDonald et al., 2o11). The results support the role of poor caregiving in

infancy as an important causal factor in the development of psychopathy. In addition, recent studies have shown that parental mental state talk, which refers to the caregiver's use of language that acknowledges the subjective experience of the child, has an important role in the development of empathy in early childhood, which protects against subsequent psychopathy development (Centifanti et al., 2016; Wagner et al., 2019).

What is significant about these studies on the social environmental causes of psychopathy is that they show that the phenotypic outcome is contingent on interactions between the relevant causal factors occurring at the appropriate stages of development. A common finding is that harsh parenting specifically in *early* childhood is associated with subsequent psychopathy development, while harsh parenting in *later* childhood is associated with the development of a different sort of externalizing behavior (Gao et al., 2010; Waller et al., 2015; Willoughby et al., 2013). Furthermore, positive changes in the caregiving environments, such as bonding with adoptive parents, can prevent trajectories to psychopathy when implemented specifically in *early* childhood (Hyde et al., 2016). These findings suggest that the development of psychopathy is not simply a matter of stable risk factors measured before development combining with one another to yield the phenotype, but is contingent on the continually changing ways in which these resources interact with one another through different developmental stages. The influence of development is further supported by the study mentioned earlier which showed the heritability measures for callous-unemotional traits to be smaller and the correlations with shared environmental factors to be greater in study populations of children than in study populations of adults (Tuvblad et al., 2017). And so, genetic and environmental factors are not static components whose relative influences remain stable over time, but are dynamic resources whose interactions are contingent on the developmental context.

To sum up so far, the considerations explored throughout this section underscore the inadequacies of static interactionism as a theoretical framework for the causation of psychopathy. First, the critical discussions of heritability by Lewontin (1974) and Sober (1988) show that genetic and environmental factors, while being difference makers, are not components that contribute in additive proportions to the expression of a trait in a given individual. Rather, they only produce the trait through their joint and reciprocal interactions, which can follow different patterns depending on the particular genes and environmental conditions involved (Nikitopoulos et al., 2014; Sadeh et al., 2010). Second, the empirical evidence on the social environmental causes of psychopathy shows that the ways in which these genetic and environmental factors interact are not fixed, but change across different developmental stages (Wagner et al., 2019; Waller et al., 2015; Willoughby et al., 2013). Instead of static interactionism, a form of dynamic interactionism is required to capture the contingent and dynamic ways in

which these resources interact throughout development. In §3, I propose that the theoretical framework of developmental systems theory supplies such a dynamic interactionism which can facilitate a better understanding of how psychopathy is caused.

3. Developmental systems theory

Developmental systems theory is a theoretical framework in the philosophy of biology developed by Susan Oyama, Paul Griffiths, and Russell Gray (Griffiths & Gray, 1994; Oyama, 2000; Oyama et al., 2001). Inspired by the biological insights of Conrad Waddington (1941), Daniel Lehrman (1953), Patrick Bateson (1983), and Gilbert Gottlieb (2003), it emphasizes the way in which development depends on the contingent and dynamic interactions of diverse kinds of resource. No single kind of resource is privileged as the determining cause of a trait or as the fundamental unit of selection. Rather, a pluralistic perspective that acknowledges the causal roles of the various kinds of resource in the developmental process is endorsed. Oyama, Griffiths, and Gray list six themes that characterize developmental systems theory (Oyama et al., 2001, p. 2). In what is to follow, I will elaborate on these six themes and will show how they capture key features of the causation of psychopathy.

3.1 Joint determination by multiple causes

It is nowadays widely accepted that the development of a trait involves interactions between different kinds of resource. However, as noted in §2, conventional interactionism assumes that the correct way to group these resources is into genetic and environmental factors. According to developmental systems theory, there are no objective grounds for favoring one sort of grouping over another, or for privileging one kind of causal factor over another. Rather, there are various ways of grouping the influences on development together and how we choose to do so depends on our explanatory interests. The distinction between genetic and environmental factors is one sort of grouping, but other groupings may be instrumentally useful for addressing other questions.

This is linked to the causal parity thesis, which is the claim that the empirical facts about how different resources contribute to development do not justify the causal privileging of one kind of resource over another (Oyama, 2000). As noted in §2, the development of any given trait is contingent on the dynamic interactions between multiple resources, such that no single kind of resource is sufficient for the development of the trait. If one kind of resource is removed, then the phenotypic outcome will be different. If another kind of resource is removed, then the

phenotypic outcome will also be different. In light of this joint determination, it is dubious to label some resources as the determining causes of the trait and others as mere enabling conditions. This is not to say that there are no differences between the particular *details* of the causal roles of genetic and environmental factors in development. Rather, it is to say that these details do not justify the causal privileging of the former over the latter.

I have already detailed, in §2, some of the diverse range of causal factors that jointly contribute to the development of psychopathy. A child's early social environment is a pervasive influence on personality development and certain social environmental factors in infancy have been shown to causally contribute significantly to the development of psychopathy, including childhood adversity and poor caregiving (Gao et al., 2010; Waller et al., 2015; Willoughby et al., 2013). However, only some children who suffer such adverse environments go on to develop psychopathic traits, and so genetic variation accounts for some of the variation in how people are affected by these adverse environments (Beaver et al., 2011; Loney et al., 2003; Nikitopoulos et al., 2014; Sadeh et al., 2010; Tuvblad et al., 2017). There are also broader cultural factors that influence personality development. For example, a network analysis found that there are striking differences between people from the United States and people from the Netherlands who meet the diagnostic criteria for psychopathy, with callousness being the more prominent feature in the former population and irresponsibility being the more prominent feature in the latter population (Verschuere et al., 2018).

Importantly, no single factor from the above is on its own sufficient to produce psychopathy, and so no single factor can be privileged as the determining cause. Rather, the development of psychopathy is a contingent outcome of various combinations of these factors interacting in complex ways. Furthermore, as noted in §2, these factors are difference makers in the expression of the phenotype, but they are not stable and additive causes of the sorts that satisfy the principle of the composition of causes. Instead, they are interdependent causes whose interactions are dynamic and reciprocal.

3.2. Context sensitivity and contingency

Having acknowledged that the development of a trait is jointly determined by multiple causes, the next step is to note that the ways in which these causes interact are context sensitive and contingent. A given developmental factor, whether it be a specific genetic variant or a specific environmental resource, may have a particular causal role in one context and a different causal role in another context. That is to say, the causal role of a given

resource is not fixed, but is variable and contingent on the state of the rest of the system.

This context sensitivity underscores the importance of considering development when understanding how various causal factors relate to one another. The interactions of genetic and environmental factors do not unidirectionally direct the developmental process, but the developmental process also influences the ways in which these genetic and environmental factors interact with one another. This is because development involves changes in the internal and external environments of the organism. For example, a particular individual as an infant is physiologically different from that same individual as an adult. Also, that particular individual as an infant interacts with different social and material resources from that same individual as an adult. Given these changes in the environmental context that take place during the course of development, a particular set of factors may interact with the rest of the system in a different way at one developmental stage from how it interacts with the rest of the system at another developmental stage.

I have already alluded to the relevance of context sensitivity to psychopathy in §2. As we have seen, heritability measures of psychopathic traits differ between populations of children and populations of adults, thus suggesting that the influences of different factors on variations in the expressions of psychopathic traits vary across different developmental stages (Tuvblad et al., 2017). Also, the longitudinal studies on the effects childhood adversity and poor caregiving have shown that the roles of these factors in causing psychopathy are contingent on the developmental context, as are the mitigating influences of certain parenting interventions (Hyde et al., 2016; Wagner et al., 2019; Willoughby et al., 2013). Further insights from theoretical models of psychopathy development will be discussed in §3.4.

The aforementioned the influences of genetic and environmental factors in the expression of psychopathy cannot be treated as stable components that can be predetermined without consideration of the developmental context. Accordingly, the advantage of developmental systems theory over conventional interactionism is that the former, insofar as it emphasizes context sensitivity, accommodates the changing influences of these causal factors across development in a way that the latter does not. To be clear, while developmental systems theory characterizes the causal roles of developmental resources as being context sensitive, this does not amount to what has been called "blank slate" cultural determinism, which is the view that psychological traits are completely formed by cultural conditions (Pinker, 2002). As noted above, developmental systems theory fully concedes that genetic variation is an important factor which influences the phenotypic outcome. However, it notes that the particular influence of such genetic variation is contingent on the roles of the other factors in the

developmental system. Indeed, this is supported by the previously mentioned molecular studies of psychopathy, which highlight some of the different ways in which the influences of specific genetic variants depend on the environmental conditions during development (Nikitopoulos et al., 2014; Sadeh et al., 2010).

3.3 Extended inheritance

An argument that has been suggested for the causal privileging of genetic over environmental factors is that genes have high copying fidelity, which enables such genetic factors to be inherited across generations from parent to offspring (Dawkins, 1976; Williams, 1966). A given gene consists of a particular sequence of nucleotides. During replication of the gene in meiosis, this nucleotide sequence is replicated with relatively high fidelity, thus allowing a high chance of this nucleotide sequence remaining stable across generations. In light of this copying fidelity, it has been claimed that the answer to the question of why children resemble their parents is simply that they share many of the same genetic factors (Plomin, 2018).

According to developmental systems theory, genes are not special in their high copying fidelity. This is not to deny that genes have high copying fidelity, but to note that other kinds of resource that contribute to development also have high copying fidelity and are inherited across generations from parent to offspring. Some of these are biological resources internal to the organism, such as cellular membrane proteins, intracellular organelles, and the epigenetic modifications of chromosomal regions. Some are material resources from the external environment, such as the intrauterine conditions during gestation, nutrient availability, and exposures to certain habitats. Finally, some are social resources, such as parental behaviors, linguistic conventions, cultural norms, social institutions, and financial wealth. Many of these diverse resources have distinctive roles in development and tend to be conserved across generations with relatively high degrees of stability. Therefore, according to developmental systems theory, the answer to the question of why children resemble their parents is not merely that they share many of the same genetic factors, but also that they share many of the same material and social resources.

Extended inheritance can help us understand why psychopathic behaviors tend to cluster in families across generations. Plausibly, shared genetic factors may partly influence this (Nikitopoulos et al., 2014; Sadeh et al., 2010). However, other kinds of developmental resource are also inherited across generations and partly account for this clustering. For example, I have already mentioned that childhood trauma, harsh caregiving, and poor parental bonding causally contribute to the development of psychopathy (Gao et al., 2010; Waller et al., 2015; Willoughby et al., 2013). Aina

Sundt Gullhaugen and Jim Aage Nøttestad note that these "dysfunctional early relations seem to replicate themselves in the psychopath's current instable and intense relationships" (Gullhaugen & Nøttestad, 2011, p. 364). When experienced early in infancy, these interactive patterns form schemas for future relationships, which result in them being recreated in the early social environment of the child of the psychopath. Given that the psychopathic individual's pattern of behavior is typically characterized by aggression, callousness, and impulsivity, he or she is more likely than a non-psychopath to interact with his or her child in harsh, neglectful, and inconsistent ways. Therefore, the kinds of childhood social adversity that had contributed to the development of psychopathy in the parent may be reconstructed in the next generation, such that they contribute to the development of psychopathy in the offspring.

Some evidence for extended inheritance in psychopathy comes from research on the correlation between parental criminality and criminality in offspring. Notably, a multivariate regression analysis by Robert Sampson and John Laub indicates that this correlation is not wholly attributable to any direct between parental criminality and criminality in offspring, but rather that "structural background factors (e.g. parental criminality) influence delinquency largely through their effects on mediating dimensions of family process" (Sampson & Laub, 1993, p. 93). In other words, parents who commit criminal behaviors tend to provide criminogenic family environments to their children, thus contributing to criminal behaviors being committed by these children.

In addition to extended inheritance through the reconstruction of the social environment, another possible means of extended inheritance is via the epigenetic effects of environmental stimuli on chromosomal regions. At present, epigenetic research on psychopathy is very limited. Nonetheless, it has been hypothesized that social stressors may trigger chemical modifications of chromosomal regions, which may also be inherited across generations (Tamatea, 2015). The upshot, then, is that there are a range of inherited resources that influence development, including genetic factors, epigenetic modifications, caregiving styles, and social circumstances. Insofar as developmental systems theory recognizes that there are many developmental resources apart from genes which have high copying fidelity, its advantage over conventional interactionism is that it can accommodate the causal roles that these developmental resources have in the clustering of psychopathy in family members across generations.

3.4 Development as construction

The traditional preformationist account of development holds that the information that determines how an organism develops is programmed in

its genes. By contrast, developmental systems theory holds that a phenotype cannot be said to be programmed in genes, because the same genes may yield a different phenotype if a different set of developmental resources are present. Rather, it endorses an epigenetic account of development, according to which the phenotype is the contingent outcome of interactions between multiple resources through various dynamical states. Importantly, any given dynamical state of the system is the contingent result of the changes to a previous dynamical state. Hence, trait development can be characterized as a process of construction, wherein the fulfillment of each stage is dependent on the prior fulfillment of a previous stage.

The notion of development as construction is supported by the previously mentioned longitudinal studies, which show the influences of childhood adversity and poor caregiving on the subsequent expression of psychopathy to be contingent on the developmental system being in the appropriate dynamical state (Wagner et al., 2019; Willoughby et al., 2013). Theoretical models have also been proposed to account for this developmental complexity of psychopathy. For example, Andrea Glenn (2019) explores how the responsiveness of an individual to the social environment changes across development due to the effects of stressors at different stages. She notes that an individual who is responsive to the social environment early in infancy "may shift toward becoming less responsive in middle childhood following chronic, severe stress", which in turn may bring about the conditions for developing callous-unemotional behavior (Glenn, 2019, p. 48). Hence, according to this view, psychopathy development is contingent on environmental adversity affecting a dynamical state marked by high responsiveness to result in a subsequent dynamical state marked by low responsiveness.

Gullhaugen and Nøttestad (2011) sketch a theory of psychopathy based on object relations theory, which emphasizes how early interactions with caregivers serve as foundations for future relationships with others. The influence of harsh caregiving on the subsequent development of psychopathy is contingent on it occurring at an early stage in infancy, when the schemas for future relationships are still being internalized and before the sense of object constancy has been acquired. Empirical support for this comes from studies on the conditions required for empathy development in infancy. For example, a longitudinal study of infants and their mothers by Grazyna Kochanska (1997) showed that in dyads where the mothers engaged with their children in empathetic ways, the relationships developed to become more mutually binding and the children internalized the maternal values to greater degrees. Also, as noted in §2, parental mental state which demonstrates attunement to the child's subjective experience has been shown to protect against psychopathy via its influences on empathy development (Centifanti et al., 2016; Wagner et al., 2019). Again, what is

suggested here is that the trajectory leading to psychopathy is contingent on how the developmental system is constructed through various dynamical states via interactions with the environment. The advantage of developmental systems theory over conventional interactionism is that the former can account for this dynamic complexity of psychopathy development in a way that the latter cannot.

3.5 Distributed control

A key assumption of the aforementioned preformationist account of development is that the genome contains the information that directs development. The sequence of nucleotides is suggested as having semantic content that can be "read off" by the intracellular components to produce the intended phenotypic outcome. This notion of genetic information has been used to defend the causal privileging of genetic over environmental factors. Genes are considered to be the "blueprints" for development, while environmental factors are considered mere background conditions that enable the genes to be expressed (Plomin, 2018).

According to developmental systems theory, this notion of genetic information is misleading. As noted in the discussion of context sensitivity in §3.2, a given genotype may yield different phenotypic outcomes under different environmental conditions. Hence, the information provided by the nucleotide sequence is insufficient for determining which phenotypic outcome will result. Further information is also required for this, namely information concerning the set of environmental conditions that are present. Another way to put this is that the sequence of nucleotides can only be considered to constitute information for the development of a given phenotypic outcome if it is assumed that all other developmental resources are held constant. If instead the genes are held constant, then the environmental conditions can be considered to contain the developmental information that is "read off" by the genes to yield the phenotypic outcome.

This further emphasizes the causal parity thesis which I mentioned in §3.1. No single kind of resource is privileged as holding the information that directs the development of psychopathy, or any other phenotype. Again, this view is not an endorsement of "blank slate" cultural determinism, as it accepts that the ways in which developmental factors interact are influenced by genetic factors. Rather, it is the recognition that genes are not the key determining causes of psychopathy and environmental factors are not mere enabling conditions. Developmental information is distributed among the diverse resources that interact to produce psychopathy, as shown by the interdependent relations between genetic variation, interpersonal processes, and the broader social environment at various developmental stages

(Nikitopoulos et al., 2014; Sadeh et al., 2010; Wagner et al., 2019; Willoughby et al., 2013).

3.6 Evolution as construction

An advantage of developmental systems theory over conventional interactionism is that it bridges the gap between development and evolution. Rather than viewing genes or organisms to be the fundamental units of selection, developmental systems theory takes entire developmental systems, consisting of organisms and their environmental niches, to be the unit of selections that are replicated across generations. Organisms are not merely shaped to fit their environments, but actively construct, maintain, and modify their environmental niches. In turn, these environmental niches influence and support the ways the organisms develop. Hence, organisms and their environmental niches co-evolve.

It has been suggested that there are certain social environmental niches in which people who exhibit psychopathic traits tend to thrive and which may be conducive to the development or reinforcement of psychopathy. For example, people diagnosed with psychopathy are especially adaptable in the criminal population and tend to engage in a more diverse range of criminal activities than non-psychopaths (Kiehl & Hoffman, 2011). Outside the criminal world, people with psychopathic personalities have been suggested to be overrepresented in the corporate world and the political world (Boddy, 2011).

What developmental systems theory captures but conventional interactionism does not is that the interactions between people with psychopathic personalities and these niches are dynamic and reciprocal. People with psychopathic personalities do not merely cluster in certain niches because these niches support expressions of their psychopathic dispositions, but these niches are themselves, to certain extents, shaped and maintained by the activities of people with psychopathic personalities. For example, in the corporate world, psychopathic behaviors by senior staff members, including bullying, manipulation, and selfishness, can have ripple effects throughout the organization, thus promoting a corporate culture that rewards such psychopathic behaviors (Boddy, 2011). Similarly, in the criminal world, it is plausible that the violent dispositions of people with psychopathic personalities not only allow these people to thrive, but also contribute to establishing and maintaining such a criminal culture that advantages people who display psychopathic dispositions (Häkkänen-Nyholm & Nyholm, 2012).

To sum up this section, I have drawn on the themes of developmental systems theory described by Oyama et al. (2001) to show how an approach that emphasizes dynamic interactionism can accommodate many of the empirical findings regarding how psychopathy is caused. The discussion

highlights the epistemic advantages of developmental systems theory over conventional interactionism as a theoretical framework for psychopathy development. The former, but not the latter, is able to capture the dynamic and reciprocal ways in which different developmental resources interact, the ways in which the effects of these resources are contingent on the stage of development, the ways in which resources apart from genes can be inherited across generations, and the ways in which the psychopath's behavior has the effect of maintaining features of his or her broader developmental system. Complementing the explanatory comprehensiveness of developmental systems theory are practical implications for the management of psychopathy, which are discussed in §4.

4. Therapeutic implications

A developmental systems theory approach to the causation of psychopathy has potential implications for therapeutic interventions. At a general level, it can serve a cautionary role by warning against therapeutic pessimism. Randall Salekin (2002) notes that there is a widely held assumption in clinical practice that psychopathic individuals are untreatable, which is reflected in the claims of some notable theorists (Cleckley, 1941; Hare, 1991). It is not difficult to see how such pessimism might be encouraged by a preformationist account of development, which considers the phenotypic outcome to be the fulfillment of the developmental information contained in the genome. If psychopathic behavior is assumed to be the inevitable expression of this genetic information in an enabling environment, then this could weaken support for any proposed attempt to change this behavior. Given that developmental systems theory rejects this preformationist account in favor of an epigenetic account of development, it may mitigate against such pessimism. By recognizing psychopathy to be not the immutable product of one's genome, but an outcome that is contingent on dynamic interactions between multiple resources at appropriate developmental stages, a developmental systems theory approach could motivate us to explore ways of intervening on these resources and developmental stages.

Empirical data on the effects of such interventions have shown the assumption of therapeutic pessimism to be somewhat unfounded. As we have seen, interventions which modify the caregiving environments of children with psychopathic traits, including bonding with adoptive parents and specific parenting therapies, have been shown to be effective at reducing and offsetting these traits (Hyde et al., 2016; McDonald et al., 2011). A particular role that developmental systems theory could have in advancing these therapeutic approaches is to emphasize the influence of the developmental context on the effect of any intervention. As noted earlier, parenting interventions appear to be most beneficial when implemented in

early childhood, which could be explained by their effects being dependent on the developmental system's being at a stage when the schemas for future relationships are still being internalized (Gullhaugen & Nøttestad, 2011). By highlighting the context sensitivity of the developmental process, developmental systems theory can inspire more longitudinal therapeutic approaches which focus on how the developmental trajectories leading to psychopathy may be offset by interventions that make changes at particular developmental stages.

These implications are not restricted to interventions in children, but also apply to interventions in adults. Under developmental systems theory, a phenotype is not taken to be a fixed outcome, but a dynamic state that is constructed and maintained through continual interactions between the individual and the environment. Again, this could warn against the assumption that psychopathic behavior is immutable and instead encourage us to explore potential interventions that modify the interactions which maintain the psychopathic behavior. Some empirical evidence in favor of these interventions is provided in a review by Salekin (2002), who suggests that psychopathic traits can be reduced in adults by intensive psychological therapy augmented by group therapy and family therapy. This is of significance for the management of psychopathy in the prison population, as it indicates the possibility of achieving behavioral improvements in adult offenders with psychopathic traits. And so, the idea that the maintenance of psychopathic behavior is a dynamic process involving potentially modifiable interactions between the individual and his or her environment can provide justificatory support for investing in therapies which aim to modify these interactions.

As well as characterizing the developmental system as a *dynamic* system, developmental systems theory characterizes it as an *extended* system comprising of the individual, his or her environment, and the interactions between them. These interactions not only contribute to the construction of the trait in the individual, but also to changes in the environment which in turn contribute to both the maintenance of the trait in the individual and the reconstruction of the trait in the following generation. Developmental systems theory's focus on the extended system is of potential significance for the management of psychopathy, because it suggests that the target of intervention is not just the individual, but the wider developmental system. This strengthens support for therapeutic approaches that aim to modify the wider developmental system, rather than just the individual. Indeed, as we have seen, the effective interventions in infants are those which modify the interactions in dyads of parents and their children (Hyde et al., 2016; Kochanska, 1997; McDonald et al., 2o11). There is also evidence that multisystemic therapy, which focuses on examining and modifying the interactions in the immediate family environments and broader social

environments of individuals, can achieve behavioral improvements in adolescents (Van Der Stouwe et al., 2014). These therapeutic findings sit well with developmental systems theory, insofar as they underscore how the reciprocal interactions between individuals and their social environmental niches influence the developmental trajectories of these individuals.

We have seen, then, that in addition to the epistemic advantages of developmental systems theory, there are ways in which developmental systems theory can influence therapeutic approaches. The characterization of the developmental system as a contingent, dynamic, and extended system lends support to approaches which aim to offset the developmental trajectory leading to psychopathy by modifying processes in the wider developmental system at appropriate developmental stages. Of course, whether or not these therapeutic approaches will turn out to be effective will only become apparent through empirical research. Nonetheless, developmental systems theory can provide a philosophical framework which can support this empirical research and facilitate explanations of its findings, in order to attain a more comprehensive understanding of how different causal processes affect the development of psychopathy at different stages.

Before I conclude, it is worth considering how developmental systems theory could enrich our approaches to understanding and managing behavioral syndromes other than psychopathy. Insofar as developmental systems theory is a general theoretical framework for understanding how traits develop and evolve, it could plausibly be applied to a number of behavioral dispositions. I suggest that its benefits would be most apparent for behavioral phenotypes whose etiologies are complicated by the sorts of multifactorial causation and developmental complexity that we saw in the case of psychopathy. Cases like these vividly reveal the shortcomings of conventional interactionism and warrant a theoretical framework with greater explanatory comprehensiveness. Here, I shall briefly consider the specific example of pedophilic disorder to illustrate how developmental systems theory might be applicable beyond the case of psychopathy.

Pedophilic disorder is a diagnosis that is assigned to adults who have arousing fantasies about prepubescent children. Specifically, the diagnosis applies to adults who are at least five years older than the children (American Psychiatric Association, 2013). Hence, a mutually consensual sexual relationship between adolescents of similar ages who are close to the legal ages of consent would not qualify as pedophilic and is usually not considered immoral or developmentally abnormal in the way that pedophilia is, as reflected by the close-in-age exemption laws of some countries which prevent sexually active adolescents of similar ages who are close to the legal ages of consent from being prosecuted (Kanbur, 2019). Like psychopathy, pedophilia is associated with significant legal and moral implications. Pedophilic acts are considered deeply immoral and are criminal offenses in

most parts of the world. Also, as with the case of psychopathy, the discussion of pedophilia in popular culture has often been mired in the nature-nurture dichotomy (Doolittle, 2009). Unsurprisingly, empirical research has suggested that neither genetic nor environmental factors by themselves are sufficient to account for the development of pedophilia, but rather that pedophilia is a contingent outcome of diverse interacting factors. For example, suffering sexual abuse in childhood is recognized as being a significant causal factor in the subsequent development of pedophilia, but only a proportion of people who suffer sexual abuse in childhood go on to develop pedophilia (Jespersen et al., 2009). Meanwhile, twin study has yielded a heritability of 0.146 for pedophilia, which suggests that a large degree of the variance is attributable to environmental factors in the studied population (Alanko et al., 2013). Wider cultural factors, such as social environments that fail to inhibit pedophilic desires, have also been hypothesized as contributors to the development of pedophilia (Finkelhor & Araji, 1986).

Again, a major challenge is to understand how these diverse factors relate to one another. The framework of developmental systems theory can facilitate such an understanding in the following ways. First, the notions of context sensitivity and development as construction emphasize how the effects of these interacting factors are contingent on particular developmental stages. For example, the effect of sexual abuse may be dependent on its interacting with the developmental system at a stage where the capacities and schemas for sexual arousal and interest are still being formed. Second, the notion of extended inheritance can facilitate an understanding of the way in which the suffering of sexual abuse in childhood can lead to the individual subsequently reconstructing this abusive behavior in adulthood. This could partly help to explain pedophilic acts that cluster in families across generations, especially where the perpetrators and the victims are members of the same families. Third, the notion of evolution as construction can highlight how certain social environmental niches, such as online networks and pedophilia rings, may be conducive to the development and reinforcement of problematic desires and behaviors that might otherwise have been inhibited in other social environmental settings. Just as with the case of psychopathy, this characterization of the developmental system as contingent, dynamic, and extended could potentially inform therapeutic and preventative approaches that aim to modify processes in the broader developmental system at appropriate stages of development.

5. Conclusion

Is psychopathy born or made? I have argued that there is much wrong with this question. Nobody is born to be a psychopath. Likewise, poor caregiving in infancy and childhood trauma are causal factors in psychopathy development,

but do not inevitably make someone into a psychopath. Rather, whether someone develops a psychopathic personality is a contingent outcome of multiple resources interacting in dynamic ways throughout development, including genetic and epigenetic variations, early social experiences, relationships with caregivers, and wider cultural and material circumstances. These resources are not additive components that contribute to the expression of psychopathy in stable proportions, but are interdependent factors whose joint interactions are contingent on the dynamical state of the developmental system. Moreover, no single kind of resource is privileged over other kinds, either with respect to its high copying fidelity or its being a source of developmental information. Herein, I have proposed that the philosophical framework of developmental systems theory can facilitate an understanding of how psychopathy is caused which captures its developmental complexity in a way that conventional interactionism cannot. I have also argued that it can strengthen support for exploring therapeutic interventions that aim to modify aspects of the developmental system at different developmental stages. Endorsing developmental systems theory, therefore, not only has epistemic benefits for achieving an empirically comprehensive explanation of psychopathy development, but has potential practical benefits for the ways we might think about treating and forestalling antisocial behavior.

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