Genetic issues in "the sociobiology of sociopathy"

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Abstract: A consideration of the genetics of sociopathy suggests the following. The author's Evolutionary Stable Strategy (ESS) types 2 to 4 are more likely than types 1 and 5 in crimes of violence, and there may not be an ESS for crimes of property or for sociopathy. Correlations between sociopathy and crimes of property are also more likely due to environmental than to genetic variants, and correlations between sociopathy and crimes of property are due more to environmental than genetic variants.

Mealey proposes that there are two types of sociopaths, primary and secondary, with differences in their genetics and life histories. Some aspects of the formal, developmental, population, and quantitative genetics of this proposal will be considered and evaluated here.

First, I will attempt to describe the formal genetics of the two types of sociopathy. As I understand the author, primary and secondary sociopathy involve the same set of many polymorphic genes (sect. 2.1.3, para. 1 and 2). Primary sociopaths have genotypes at the most extreme end of the binomial distribution of genotypes and are thus most likely homozygous for many of the polymorphic genes. It is not known, however, whether there are dominance or epistatic effects for these variant genes. Secondary sociopaths have genotypes elsewhere in the distribution, and these do not overlap those of primary sociopaths. Also, if all genotypes in this set were to experience the same environmental conditions, the risk of secondary sociopathy would be a function of the number of sociopathy facilitator alleles. Again, it is not known whether there is dominance or epistatic effects for these variant genes.

With regard to developmental genetics, it is incorrect to say that primary sociopathy is more genetically determined than secondary sociopathy and that secondary sociopathy depends more on environmental factors than primary sociopathy (sect. 2.3.1, para. 2 and 3; sect. 2.4.2, para. 3). Rather, each phenotype is a response of each genotype to environmental inputs. It may be, as Mealey suggests, that the cheating strategy or sociopathy of individuals with the genotypes from the most extreme end of the binomial distribution of genotypes is not influenced by advantages or disadvantages in social competition during their life history. But this does not mean that the development of primary sociopathy in individuals with these genotypes is independent of other aspects of environment and life history. In fact, Mealey suggests that such individuals would display less sociopathic behavior if the cost of their detected cheating were increased (sect. 3.2.1). Similarly, it may be, as Mealey suggests that the cheating strategy or sociopathy of individuals with genotypes from the rest of the binomial distribution of genotypes depends on advantages or disadvantages in social competition during their life history. However, Mealey recognizes a genotype-environment interaction for this effect (sect. 2.2.1, para. 4; sect. 2.3; sect. 3.1.2, para. 1). Some genotypes are at a greater risk than others for developing secondary sociopathy at a given level of disadvantage in social competition.

With regard to genetic evolution, Mealey suggests that the occurrence of primary sociopathy in a population is due to frequency-dependent selection for ESSs (evolutionarily stable strategies) and that this frequency-dependent selection keeps primary sociopathy at low levels in every society. In other words, the fitness (reproductive advantage) of sociopathy depends on its frequency in the population, high fitness and low frequency being associated. However, Maynard Smith (1989) proposes that there are extreme constraints on frequency-dependent selection of ESSs in sexually reproductive species. These are (1) a pure or mixed ESS can be specified by a genetic homozygote; these would be represented by Mealey's ESSs of types 2, 3, and 4 rather than 1 and 5 for sociopathy; (2) also, a mixed ESS can be specified when there are only two pure strategies in the ESS and when the genetic system can generate the required phenotypic distribution. The existence of secondary sociopathy with continuous degrees of expression seems inconsistent with the requirement for two pure strategies. In addition, very little appears to be known about the genetic system underlying primary sociopathy, to say whether a distribution with less than 3%–4% male and 1% female primary cheaters is an ESS.

With regard to quantitative genetics, the heritability of criminality and sociopathy is relevant to the developmental and ESS aspects of cheating. My comments are based on critical reviews of this material by Carey (1994) and by Raine (1993). First, neither twin nor adoption studies reveal a nonzero heritability for violent crimes. This suggests that there may be no genetic correlation between crimes of violence and either type of sociopathy. However, a single homozygous genotype for our species could, as discussed above, result in an ESS for crimes of violence. This would be Mealey's ESS types 2 to 4 rather than ESS types 1 and 5. Second, both twin and adoption studies show a nonzero heritability for crimes of property. As discussed above, this genotypic polymorphism may not be consistent with there being an ESS for crimes of property. Also, there is a substantial effect of the environment on variation in crimes of property. Thus, correlations between sociopathy and crimes of property are probably due to both genetic and environmental effects. Third, there have been some attempts to assess the heritability of antisocial personality disorder, which may be related more directly to sociopathy than crime is (Baker 1986; Grove et al. 1990). In these studies, the heritability of antisocial personality is low (about 0.27). To the extent that this is an index of sociopathy, these studies suggest that its heritability is lower than that for crimes of property (about 0.45), and that genotypic correlations for crime and sociopathy will make a modest contribution to their phenotypic correlations. In addition, the genetic polymorphism for antisocial personality may not be consistent with there being an ESS for this trait and perhaps for sociopathy.

Diathesis stress model or "Just So" story?

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Abstract: Mealey's sociopathy model is an exemplar of popular diathesis-stress models. Although such models, when presented in descriptive language, offer the illusion of integrative explanation, their actual scientific value is very limited because they fail to make specific, quantitative, falsifiable predictions. Conceptual and quantitative weaknesses of such diathesis-stress models are discussed and the requirements for useful models are outlined.

Rube Goldberg is famous for his ingenious designs of elaborate contraptions that perform simple tasks in unnecessarily complicated ways. Some psychologists have shown a similar design approach, proposing theoretical models that are far more complicated than they need be. Of course, Goldberg's designs were amusing precisely because they were so absurd; analogous designs offered by psychologists in the name of science, however, tend to be treated seriously. Mealey's proposed model for sociopathy provides a good case example. To her credit, Mealey

has gone farther than most other sociopathy theorists to make her model explicit. Upon careful examination, however, her model seems Goldbergian. If competing models, based on similar concepts, were to be specified in comparable detail, we suspect that they would fare no better.

Mealey proposes a diathesis-stress model of sociopathy in which two distinct and distinguishable subtypes of sociopathy are produced from the independent and interacting contributions of a continuously distributed genetic diathesis and a kitchen-sink collection of specific environmental stressors. This rendering of the diathesis-stress model is not unique. Mealey goes to great lengths to provide a scholarly integration of research literature from a variety of fields in an effort to demonstrate the power of her model. However, as is typical of such reviews, her integration is entirely post hoc; if the research had yielded different or even conflicting results, she might have subsumed those findings just as easily. This illustrates one of the problems with "diathesis-stress" models, namely, they are capable of explaining virtually any outcome; yet they really are not explaining anything at all, except after the fact. A truly powerful model must make risk predictions that go beyond the original data that gave rise to the model in the first place (Feynman 1985; Popper 1962). For diathesis-stress theories to be scientifically useful, they must specify a testable underlying quantitative model that explains the existing data while making clear predictions that go beyond known data.

Model testing can be an extremely difficult task, even when dealing with diametrically opposed theories in relatively simple psychological domains. An instance of this difficulty is found in determining simply whether people scan short-term memory in a parallel or serial fashion (Sternberg 1966). It has been found that in common experimental paradigms, models of either variety often can mimic exactly the behavioral predictions of the other (Townsend 1974). Precise mathematical knowledge of the models and testing situation, however, leads to testable predictions (Townsend 1990a).

The difficulties of comparing exclusively parallel or serial models of processing pale in comparison with the difficulties of comparing diathesis-stress models, each of which generally posits an unspecified mixture of the same etiological factors. These models are unlikely to be testable without much stronger and well-specified assumptions. In the present domain we have an abstract "input" space, say the two-dimensional plane, to represent the environment x genetic predisposition, and some frequency function that specifies the likelihood of a person possessing any particular combination of genetic predisposition and environment. Let us agree to call the resultant plane the predisposition space. On the "out" end of matters, we have a behavioral space, including behaviors subject to measurements defining one's status on the sociopathy construct. But a real theory of sociopathy must do more than posit multiple input factors and summarize what is known about descriptive psychopathology on the output side. The theory should specify a set of functions linking the points in the predisposition space to points in the behavioral space. These intermediary functions must permit the derivation of a frequency function on likelihood of each "behavior" and/or its severity, for each point in the predisposition space. Similarly, the theory should specify functions linking the behaviors comprising each sociopathic syndrome. The more specific the theories' predictions about expected distributions on the output side, the greater our ability to reject one theory in favor of another (Townsend 1990b).

Such a rich set of quantitative predictions may seem very ambitious given our current knowledge of psychopathology, but it is simply the case – and this contention can be made precise (see, e.g., Townsend et al., in preparation) – that without a very tight set of constraints, most models that sound qualitatively distinct in English will not be differentiable on the basis of the type of data referenced in the target article. In the absence of these ambitious predictions, it will not be possible to reject one theory in favor of another. We use Mealey's model as an example, but the problems extend to all diathesis-stress models.

Mealey's model posits discrete thresholds in both the predisposition space (where, at a specific genetic threshold, environmental variation becomes irrelevant) and the behavior space, where primary and secondary sociopathy emerge as differentiable types. But in the absence of hypothesized functions linking the two spaces it is not clear how the theory may be tested. There always will be functions that can map discrete inputs onto continuous outputs, or continuous inputs onto discrete outputs, so there is no particular parsimony in parallel discrete mappings. On the input side, Mealey's genetic threshold model does not appear to make any predictions that can be differentiated from those of a simple linear model in which continuous genetic variation, continuous environmental variation (and perhaps a cross-product) combine to predict sociopathic behavior. On the output side, the theory does not explain why discrete types of sociopathy should emerge, nor does Mealey provide any evidence that primary and secondary sociopathy are discrete types.

What does the theory predict? It predicts that a higher prevalence of sociopathy will be seen in high-risk environments, but all reasonable theories predict this. It predicts that the most severely disturbed individuals (primary types) will be more difficult to treat, but theories positing one type of sociopathy, or even those positing a continuous distribution of sociopathic behavior and no sociopathic type at all, would make such a prediction. The theory predicts that more severely disturbed individuals will be more likely to remain disturbed across situation and time than less disturbed individuals. Alas, there are simply too many ways to arrive at these same predictions, so evaluation of these predictions cannot support any particular theory.

Integrated reviews of the psychopathology literature should be encouraged, but there is little point in casting about for a generic diathesis-stress theory to explain the literature, because diathesis-stress theories are vague enough to explain any existing data. We need theories that predict a specific set of future results and, equally important, forbid another specific set of future results. We believe that this is unlikely to happen until theorists begin to specify falsifiable quantitative models of the functions relating etiological factors and behavioral outcome. Mealey's interesting review may yet inspire a scientifically useful theory of sociopathy, but she has not provided one here.

Adaptive and nonadaptive explanations of sociopathy

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Abstract: We doubt that primary sociopathy is adaptive, for three reasons: First, its prevalence is too low to require an adaptive explanation. Second, a common sequela of damage to the orbito-frontal lobes is "pseudopsychopathy." Any pattern of behavior that can be produced by brain damage is unlikely to be adaptive. Third, we argue that most human social behavior is not under tight genetic control, but is produced by open-ended calculation of fitness-contingencies.

As Darwinians, we applaud Mealey's comprehensive review of the literature on sociopathy in the context of evolutionary