## Sociopathy and sociobiology: Biological units and behavioral units

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**Abstract:** Behavioral biologists have long sought to link behavioral units (e.g., aggression, depression, sociopathy) with biological units (e.g., genes, neurotransmitters, hormones, neuroanatomical loci). These units, originally contrived for descriptive purposes, often lead to misunderstandings when they are reified for purposes of causal analysis. This genetic and biochemical explanation for sociopathy reflects such problems.

The ill-defined field of sociobiology has been a mixed blessing. At its best it has been a heuristic force, shaping new questions and providing new insights into sex ratios, altruism, and a variety of behavioral phenomena. At its worst it has fostered a simplistic set of inferences in which *utility* means *adaptation* eans *evolution* means *natural selection* means *heritability* means *genes*. Mealey's analysis reflects both the strengths and the weaknesses of sociobiology's contribution.

The research on infanticide provides a useful model from which to examine Mealey's views on sociopathic behavior. Infanticide, in both animals and humans, has often been dismissed as maladaptive, pathological behavior; however, sociobiologists, encouraged by a renewed interest in sexual selection, have argued that infanticide might serve biological fitness. Twenty years of research has strengthened that supposition. Mealey suggests that sociopathy, like infanticide, has utility for those individuals who exhibit it, and she notes that it is important for society to recognize this fact in coming to grips with the behavior. Such perspectives are welcome and deserve encouragement, but the comparison to infanticide also illuminates some of the limitations of her argument. Sociobiological interpretations work best when they relate to very specific behavior patterns having a direct bearing on reproductive success. Even then they can be dismissed as "just so" stories unless they carry enough detail and eliminate enough alternatives to make the adaptationist argument inescapable. For example, Vom Saal (1985) finds that the frequency of infanticide among male mice increases dramatically following ejaculation, and it remains high until just before their own young are born. It then drops to a low level until their young disperse, at which time it rises again. These patterns are so complex, so challenging to alternative explanations, and yet so consistent with an adaptationist viewpoint that it is difficult to escape the sociobiological conclusion. Moreover, when males kill the infants of other males, there is an obvious impact on inclusive fitness. An evolutionary explanation is therefore tenable, if not compelling. On the other hand, with the possible exception of rape, sociopathic behavior has no obvious connection to inclusive fitness. To be sure, a competitive edge in the most general sense is implied when it occurs at low frequency in the larger population, but how this advantage translates into a gain in inclusive fitness (a necessity for the evolutionary argument) remains unclear.

Mealey suggests (sect. 3.1.1) that sociopathic behavior actually takes two forms: (1) a primary, "inborn" pattern and (2) a secondary, "environmentally contingent" form. Although only the first of these is explicitly presented as a "genetically *determined* strategy," both are presented as "genetically based." Such preformationistic positions have drawn a barrage of criticism over the past twenty years (cf. Lewontin et al. 1984), and even the most traditional ethologists and sociobiologists now concede that although genes make a difference, they do not govern any behavior in such deterministic fashion. Increasingly, developmental biologists and psychologists recognize that the developing organism is in a sense "self-organizing," and that there is no "blueprint" for behavior in the genome. Even if one accepts Mealey's categories of primary and secondary sociopathy (which I am reluctant to do), there still would be no logical reason to assume that genes are any more involved in one than the other. Moreover, even if the patterns vary in the frequency-dependent way Mealey describes (and there appears to be little evidence for this), it would be virtually impossible to distinguish the secondary sociopathy of an evolutionary adaptation from the coping behavior of a remarkably flexible human cognitive process. Brain mechanisms for the latter have evolved, to be sure, but the specificity of their role is very different from those that sociobiologists envision.

Speaking more generally I might add that hormones, neurotransmitters, brain nuclei, and so on also make a difference in behavior, but again, they do not control behavior in the strong sense implied by Mealey's characterization. Testosterone's action is a case in point. Mealey's exposition would lead one to believe that higher levels of this hormone make male adolescents bigger, stronger, and more aggressive. The research literature remains unclear on testosterone's relationship to aggression, but it is quite clear on its relationship to body size. Testosterone terminates the growth of the long bones. Young boys who take anabolic steroids are short, and the castrati of 18th- and 19th-century Italy were excessively tall. Mealey suggests that aggression and testosterone are mutually stimulating and generate a positive feedback loop, but the relationship is far more complex than she suggests. True, subnormal levels of testosterone may indeed correlate with subnormal levels of behavior, but once serum testosterone reaches normal levels or above, the relationship breaks down. A panoply of internal and external contextual conditions modulate testosterone's influence. Liver activity (which may reflect drug and alcohol use), carrier proteins, receptor numbers, and past experience are among the many factors that complicate the relationship between blood testosterone level and aggressive or sexual behavior. Moreover, many of these factors have rate-limiting effects, preventing excessive testosterone from having excessive effects on behavior. In short, neither testosterone nor any other behaviorally relevant chemical plays a *determining* role in any behavior. Generally speaking, biological units (genes, hormones, neurotransmitters, or chunks of brain) do not have a one-to-one causal relationship with specific behavior patterns.

Mealey musters an impressive volume of literature in her analysis of sociopathy. Much of it contributes to a better understanding of this intriguing behavior, but the sociobiological perspectives are largely untestable and add little. Moreover, by suggesting that the behavioral variants are genetically determined, this formulation encourages the unfortunate conclusion that developmental studies are irrelevant when, in fact, they are critical.

## Psychopathology: Type or trait?

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**Abstract:** Mealey proposes two categorical classes of sociopath, primary and secondary. I criticize this distinction on the basis that "type" constructs of this kind have proved unrealistic in personality taxonomy and that *dimensional* systems capture reality much more successfully. I suggest how such a system could work in this particular context.

Mealey's discussion of the sociobiology of sociopathy is remarkably complete and succinct; it covers much ground and establishes its primary contentions admirably. I have little to criticize, other than the main point of the paper, namely, establishing the two categorical classes of sociopaths – primary and secondary (with criminals who are not sociopathic as a third type, presumably).

I have always argued that the psychiatric commitment to a categorical sphere of diagnosis is fundamentally wrong and has to be replaced by a dimensional system, if we are to be governed by factual evidence (Eysenck 1960; 1970). The low reliability of categorical diagnoses, resulting from overlapping classifications, is notorious, and the claim that the intersituation has been remedied by successive editions of the APA Diagnostic and Statistical Manual has not been found to be justified (Kirk & Kutchins 1992). Even here, sanity is breaking in; DSM-IV agrees that a dimensional approach is scientifically preferable, although refusing to use it in actuality because of old, established habits of medical diagnosis.

Mealey suggests an absolute contrast between primary and secondary sociopaths but reports no evidence that these could be separated diagnostically with any acceptable reliability. From experience, I would be surprised if agreement between professional observers would be greater than 0.3 or thereabouts; clearly insufficient for scientific or practical purposes. I believe a much better picture would be one in which individuals were represented by points in a hollow, three-dimensional globe, the three diameters of which would represent Extraversion, Neuroticism, and Psychoticism (Eysenck & Gudjonsson 1989); the group of primary and secondary sociopaths would then appear as clusters of points in the $E + N + P +$ octant, but certainly not as two quite separate and distinct clusters. The differences Mealey notes would locate primary sociopaths further out toward the periphery, and possibly closer to $P$ than secondary sociopaths, who might be closer to the centre, and nearer to $E$ and $N$. But all differences would be dimensional, with no absolute demarcations. And, clearly, a system of diagnosis referring each point in this globular universe to the three dimensions, as a three-digit number, would be more reliable and more valid than a verbal type of construct, as suggested by Mealey.

It would be possible, of course, to rotate these three reference axes to accommodate the theories of Gray and Cloninger, as Mealey suggests. I believe that both have made important contributions to the psychophysiological interpretation and understanding of personality-related behaviour, but I am not convinced that the evidence presented by them is adequate to suggest the rotation of the three primary axes. The latest study of the Gray system (Carver & White 1994) suggests to me that the activity of the BAS system aligns it with extraversion, that of the BIS system with neuroticism. But however that may be, and even if future research should force some degree of rotation, the principle of dimensional diagnosis would remain unaffected. If nature has not in fact produced some 300 separate psychiatric illnesses, as DSM suggests it has, then no human effort to force diagnoses into this Procrustean bed is likely to be successful. Nor will efforts to force behaviours into a two-type system be any more successful. Primary and secondary psychopaths may occupy different positions in our globe, but there are innumerable gradations observable in the points lying between pure examples of these points. If the notion of categorical disease entities breaks down completely when such time-honoured groups as schizophrenics and manic-depressives are concerned (Kendell & Brockington 1980), what hope is there for primary and secondary sociopaths?

Primary sociopaths are said to be mainly activated by *genetic* factors, secondary ones by *environmental* ones, but surely no one would argue that the ratio of these two factors is not infinitely variable, and at present incapable of measurement. All the genetic and environmental correlates and possible causes of both conditions (and criminality as well) are continuously variable; they cannot conceivably give rise to two categorically distinct groups. Indeed, it is not even clear just how Mealey would diagnose her primary sociopaths phenotypically. She says that "there will always be a small, cross-culturally similar and unchanging baseline frequency of sociopaths and a certain percentage of sociopaths . . . will always appear in every culture, no matter what the socio-cultural conditions" (sect. 3.1.1, para. 1). But how would we ever test such an hypothesis, in the absence of methods of diagnosing the genetic make-up of a given individual? And is it really suggested that each and every one of these primary sociopaths in fact had the identical genetic make-up? Surely there would always be a more-or-less and a gradual fading into the genetic make-up of the secondary sociopath, with no clear-cut boundary between them. And it is of course quite doubtful whether the proportion of sociopaths would indeed be *identical* from group to group. Rushton (1994) has reported evidence of very marked differences in criminality between racial groups; it is not unlikely that this portends similar differences in the number of primary sociopaths.

Mealey also seems to suggest that treatment, whether preventive or later in life, should be *categorically* different for primary and secondary sociopaths. This again seems to go counter to fact. Unless diagnosis was well above the 0.70 level, any scheme of allocating treatment differentially to primary and secondary sociopaths would be doomed to failure. Considering that the great majority would fall between the two extreme *pure* groups, what should we do with them? Again, a graded, dimensional system would seem much better able to accommodate the facts of human diversity. Fortunately, it should not be difficult to translate the numerous psychological, hormonal, and physiological characteristics of Mealey's two types into a dimensional language, thus transforming an unnatural typological system into a much more natural dimensional one.

## The epigenesis of sociopathy

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**Abstract:** Mealey distinguishes two types of sociopathy: (1) "primary," or obligate, and (2) "secondary," or facultative. Either sociopathy evolved twice, or one form is derived from the other, e.g., through: (1) genetic assimilation generating polymorphism in the relative strength of biases favoring the development of otherwise facultative strategies, or (2) independently heritable but strategically relevant characteristics biasing the optimal selection of facultative strategies.

Mealey's target article represents an important contribution to the study of sociopathy. She is to be congratulated on a theoretically insightful synthesis and creative reinterpretation of a wide-ranging assemblage of scientific findings on the topic. This kind of work is a sure sign that evolutionary psychology is rapidly maturing in its ability to incorporate and accommodate otherwise fragmented and disparate empirical content from the traditional social sciences by providing a meaningful functional context and a powerful interpretive framework. Mealey's major claim is that sociopathy can be productively seen as an evolved adaptive strategy for intraspecific social parasitism, or "cheating." Although this identity remains far from conclusively established, the case for functional equivalence is too compelling to dismiss as mere coincidence. A less persuasive secondary claim, however, is the proposed distinction between "primary" and "secondary" sociopathy in both phenomenology and etiology. It is about this second and more minor claim that I harbor certain reservations.

Although I have never worked explicitly on sociopathy, I have recently been involved in various collaborative research projects dealing with the deviant social and sexual strategies of competitively disadvantaged males. A major and recurring problem in this line of research concerns whether the deviant behavior patterns represent what behavioral biologists distinguish as "alternative" versus "conditional" adaptive strategies (Alcock 1989; cf. Mayr 1974). An "alternative" strategy can be defined as an adaptive strategy that is *obligate* for the individual,