Commentary on **Linda Mealey (1995). The sociobiology of sociopathy: An integrated evolutionary model. BBS 18:523–599.**

Abstract of the original article: Sociopaths are "outstanding" members of society in two senses: politically, they draw our attention because of the inordinate amount of crime they commit, and psychologically, they hold our fascination because most of us cannot fathom the cold, detached way they repeatedly harm and manipulate others. Proximate explanations from behavior genetics, child development, personality theory, learning theory, and social psychology describe a complex interaction of genetic and physiological risk factors with demographic and micro environmental variables that predispose a portion of the population to chronic antisocial behavior. More recent, evolutionary and game theoretic models have tried to present an ultimate explanation of sociopathy as the expression of a frequency-dependent life strategy which is selected, in dynamic equilibrium, in response to certain varying environmental circumstances. This paper tries to integrate the proximate, developmental models with the ultimate, evolutionary ones, suggesting that two developmentally different etiologies of sociopathy emerge from two different evolutionary mechanisms. Social strategies for minimizing the incidence of sociopathic behavior in modern society should consider the two different etiologies and the factors that contribute to them.

Heritability estimates provide a crumbling foundation

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Abstract: When Genotype × Environment (G × E) interactions are present, heritability estimates are not interpretable. Mealey cites abundant evidence for G × E interactions in the etiology of sociopathy, thereby completely undermining estimates of the heritability of sociopathy which form the foundation of her model. Without proper evidence for a genetic basis of sociopathy, Mealey's sociobiological model collapses under its own great weight.

In his cogent commentary on Linda Mealey's (1995t) target article, Crusio (1995) states that "The only reasonably solid evidence provided for any genetic basis for either form of sociopathy is heritability estimates" (p. 552). Whereas it is correct that solid evidence for a genetic basis of sociopathic behavior in humans is lacking, it is, however, incorrect to claim that evidence in the form of heritability estimates is "reasonably solid." Mealey cites several studies that show at least one of the key assumptions necessary for making heritability estimates is violated, calling into question a fundamental element of her model: that sociopathy is genetically determined. Without solid evidence for a genetic basis of sociopathy the foundation of her model is significantly weakened.

It is important to state some of the critical assumptions whose violation renders heritability estimates uninterpretable. Graciously, in her note 7 (p. 540), Mealey provides references that the interested reader can consult for further discussions regarding assumptions for estimating heritability that are often violated. Emde et al. (1992) provide a particularly useful discussion of the limitations of twin studies for estimating heritability, which I summarize here.

To estimate heritability using twin studies one must assume that: (1) MZ and DZ twins experience equal environments, (2) it is possible to generalize the results of twin studies to nontwin populations, (3) all genetic effects are additive, (4) parents mate at random with respect to the trait, and (5) there are no important correlations and/or interactions between genotype and environment.

Violating any one, or some combination, of these assumptions, makes it quite difficult (if not impossible) to interpret a resulting heritability estimate. In fact, it is questionable whether heritability estimates can provide meaningful information when experimental control (e.g., of matings) is not possible (Kempthorne 1978; McGuire & Hirsch 1977; Wahlsten 1979; 1994).

I will address only the assumption that there are no important interactions between genotype and environment, not because the other assumptions have not been violated, but because Mealey cites abundant evidence for a plethora of G × E interactions from adoption studies of the heritability of sociopathy and criminality. Mealey cites six studies (Baker et al. 1989; Cadoret et al. 1983; Crowe 1972; 1974; Mednick & Finello 1983; Mednick et al. 1984) that report significant G × E interactions in the development of criminality and three (Cadoret & Cain 1980; 1981; Cadoret et al. 1990) that report such interactions in the development of sociopathy. We can assume that such studies are only the tip of the iceberg with respect to detecting significant G × E interactions, since it is likely that others lack the statistical power to detect them (Wahlsten 1990). Three of the studies cited above (Baker et al. 1989; Cadoret & Cain 1980; 1981) detected interactions that are based on sex; an indication of the complexity that can underlie phenotypes such as sociopathy and criminality.

Unfortunately, the failure to critically assess potential violations of assumptions necessary to estimate heritability for human psychological traits is not uncommon. Alternatives to estimating heritability do exist, however, and are widely available to those interested in studying heredity-behavior relations in humans. Wahlsten (1994) advocates the use of "generic" (e.g., multiple regression analysis) rather than "genetic" (e.g., heritability) statistics to analyze data from twin and adoption studies. In doing so, the researcher avoids making the assumptions so often violated when estimating heritability and also avoids using the term "heritability" which is easily confused with the term "heredity" (Stoltenberg, in press).

To advance our understanding of heredity–behavior relations it is essential that we appreciate the theoretical basis of the statistical techniques we employ and avoid those techniques whose assumptions are rarely, if ever, met. Without such an appreciation, efforts to unravel the complexities of heredity-behavior relations are doomed to fail. The superstructure of Mealey's model is based on a foundation of questionable evidence and therefore adds little to our understanding of the etiology of sociopathy.

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Simulation and the psychology of sociopathy

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Abstract: Mealey's (1995a) psychological explanation of the sociopath's antisocial activity appeals to an incomplete or nonstandard theory of mind. This is not the only possible mechanism of mental state attribution. The simulation theory of mental state ascription offers a better hope of explaining the diverse elements of sociopathy reported by Mealey.

Mealey (1995t) argues that a genotype exists that disposes some human beings toward *primary sociopathy.* Primary sociopaths

(hereafter, PSs) exhibit chronic antisocial behavior marked by lack of empathy and a focus on rational, calculated manipulation of others. Mealey briefly addresses the status of the underlying psychological mechanism characteristic of PSs:

> Because they are not intellectually handicapped, these individuals will progress normally in terms of cognitive development and will acquire a theory of mind. Theirs, however, will be formulated in purely instrumental terms, without access to the empathic understanding that most of us rely on so much of the time. They may become excellent predictors of others' behavior, unhandicapped by the vagaries and "intrusiveness" of emotion, acting . . . solely on nomothetic laws and actuarial data rather than on hunches and feelings (p. 536).

This is explained in terms of an incomplete or defective theory of mind. There has been an explosion of interest in the "folk" ability to explain one another's actions by appeal to inner mental states. Theory-theorists (Astington & Gopnik 1991; Astington et al. 1988; Gopnik 1993; Gopnik & Wellman 1992; Wellman 1990) hold that adults have a tacit theory that describes generalizations mediating behavior or observable characteristics and mental states. Observed behavior plus background knowledge is the input to this theory, while a mental state attribution is the output.

According to Mealey, the PSs theory lacks (1) noninstrumental characterizations of intentional agents (sect. 3.1.1) and (2) an apparatus for empathic projection of oneself onto others (sect. 2.2.3). Neither of these is a plausible explanation of sociopathy.

The first feature – the absence of noninstrumental characterizations of targets – would render the PSs theory of mind hopelessly inadequate. Having an instrumental theory of mind results in PSs "using a pure cost-benefit approach based on immediate personal outcomes, with no 'accounting' for the emotional reactions of the others with whom they are dealing" (p. 536). In one sense of accounting, this could not be correct. Without knowing what the present and future emotional states of others *are,* how could a theory of mind be successful? The PS, in order to achieve his goals, will need to correctly predict whether the person he is interacting with will become enraged or irrationally vengeful. An inability to make this prediction would render sociopathy a risky business, since the sociopath would never know, for example, whether or not a partner in a prisoner's dilemma situation was on the brink of anger. Hence what Mealey must mean is that sociopaths fail to *experience* the emotional state they ascribe to the target, and that this is a fundamental flaw in their underlying theory of mind. This version of the worry collapses the first feature of the sociopath's theory of mind into the second feature.

By Mealey's lights, an intact theory-theory will have both *cognitive* elements and *emotional* elements. Sociopaths lack the latter, making them unable to experience and hence to account for the emotional component. What is mysterious about this proposal is that the theory-theory is not viewed by anyone as providing any empathic information beyond a mental state ascription. A successful theory of mind only yields judgments such as "Mother is angry" or "Smith believes that Brown is in Barcelona." There is no output of a theory of mind that would solve Wittgenstein's Beetle-in-a-Box problem (1958, para. 293). That is, there is nothing about a theory of mind that allows one to know what it is like to have the mental state imputed to the target. And theory-theorists never had any such pretensions. So Mealey has attempted to explain sociopathy by claiming that a part of the theory-theory is missing that no one ever supposed the theory-theory to have.

Fortunately, there is an alternative to the theory-theory in explaining mental state ascription. Recently, a *simulation* account has been offered as a competitor (Goldman 1989; 1992; Gordon 1986; Harris 1989; Heal 1986). In order to ascribe mental states to others, the simulationist claims people use the target's circumstances as make-believe inputs for their own mental state mechanism and generate a mental state off-line. This is the mental state ascribed to the target. The input to the simulation may come from perceiving the target's circumstances, or from a fund of background knowledge about the target, or both. The simulation proposal gets some of its intuitive impetus from the insight that the most efficient and accurate guide to *other people's* mental state to behavior transitions will be the cognitive mechanism that performs this function in one's own case. Of course, simulation has more than just intuitive appeal; a growing research effort is underway to explore it (for an overview, see Davies & Stone 1995).

The simulation theory offers clear explanatory advantages in accounting for the sociopath's behavior, while avoiding the difficulties with Mealey's theory-theory-based explanation. When Mealey alludes to the empathic elements of the theory-theory, she suggests that they manifest themselves as "hunches and feelings" (p. 536). The simulationist, on the other hand, has specific, empirically testable proposals about how empathic projection is achieved, namely, ascribers imagine themselves in the target's circumstances to determine what mental state they would have, were they the target. There has even been some preliminary conceptual work on simulation in explaining human moral judgments (Goldman 1995). Although the simulation proposal does not solve the Beetle-in-the-Box problem either, it does make clear what an empathic projection would be. Unlike theorizing about a target, simulating it can appeal to all the first person mental states (and their phenomenological components) available to the ascriber, including moods, passions, and emotions. Since ascribers generate those mental states in order to simulate, they may properly account for them in the off-line case. If it can be shown that the underlying psychology of sociopaths renders them unable to simulate effectively, we will have the beginnings of an explanation for their antisocial behavior.

Suppose there is a heritable cluster of deficits in the ability to simulate targets. What would such a defect amount to? One possibility is the inability to take one's own mental state mechanism fully off-line. This would result in ascriptions that are infused with one's own mental states. The influence of one's own mental states is observed in young children. Before age three, children fail the false-belief task (Perner et al. 1987). When a target's beliefs based on ignorance of the situation are false, young children with better information impute their own belief. By age four, this error is no longer made; four-year-olds realize that others might have false beliefs. The simulationist account for this is that the simulation becomes better able to abstract away from the ascriber's mental states.

This pattern of misattribution does *not* seem to be observed in sociopaths. Mealey reports that low Machs – the non-sociopaths – do worse on predictions of what others will answer on Machiavelli tests because they misattribute *their own* mental states to others (p. 534). Low Machs are victim to something akin to an adult version of failure in the false belief task. The low Machs tailor their responses as best they can given the information they have about the target, while the high Machs ignore the information they have. But rather than showing that primary sociopathy cannot be explained by appeal to a simulation deficit, these results are just what simulation would predict. Lacking further information about the target, the low Machs's simulation uses first-person input data, resulting in an ascription close to the mental states of the ascriber. Thus, low Machs "guess[ed] at a level that was more reflective of their own scores than those of the population at large" (p. 534). The error that toddlers make lies at one end of a continuum; low Mach adult judgments make some headway toward a richer simulation.

To explain the performance of high Machs, we may suppose the simulation mechanism completely defective in the ability to go off-line. As high Mach children grow into adolescence and adulthood, the data in favor of a difference between others' mental states and one's own become overwhelming. In light of this, some representation of other people's mental states would be required. Without the ability to simulate, ascription based on actuarial data is the natural fallback. Predicting other people's mental states becomes a species of general prediction. Given the utility of making predictions based on the mean, this nonsimulational strategy would be further reinforced. One might predict that high Machs follow a slower course of development to success on the false belief task.

The simulation proposal explored here is also consistent with the data reported on the role of mood (pp. 535–536). Mealey reminds us that Dodge and Newman (1981) show that, in (not necessarily sociopathic) boys, aggressiveness covaries with an overattribution of aggression to others. According to the simulation view, the aggressiveness of the attributers affects their simulation of targets of mental state ascription; their aggression infuses the simulation, so that the target is judged as more aggressive than he actually is. The theory-theory can at best invoke *ad hoc* ancillary mechanisms to explain these data.

Finally, where Mealey cites biological data correlated with sociopathic behavior, the theory-theory needs some explanation of why serotonin or testosterone levels (p. 534), for example, should affect the theory of mental state ascription adversely while leaving other theoretical domains (folk physics, folk biology) unimpaired. Simulation theory has recourse to a *process deficit*. We would expect to find physiological manifestations of a defective or nonstandard simulation module. An explanation that relied solely on the theory-theory would be hard-pressed to make sense of the physiological data reported in the target article without positing a dedicated theory-of-mind mechanism. Some, but not all, theory-theorists do this (Baron-Cohen 1995; Leslie 1994).

Mealey sometimes casts the underlying psychological mechanism of sociopathy solely within the framework of the theory-theory (see the quotation at the beginning of this commentary as well as pp. 529; 584–85, and sect. R5; Mealey 1995). Elsewhere, she hints at something like a proto-simulation theory: "most children . . . are biologically prepared to learn empathy" (p. 533). There are strong reasons for Mealey to draw more on the simulation theory.

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Sociopathy: Adaptation, abnormality, or both?

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Abstract: Mealey's article on the sociobiology of sociopathy raises questions about the relationship between adaptiveness and abnormality. Although sociopathy may be adaptive, simply defining medical and psychiatric disorders in terms of evolutionary function is problematic. Rather, sociopathy may be characterized as a relatively atypical disorder, an entity to which it may be useful to extend the metaphors of the medical model.

Mealey's (1995t) article on the sociobiology of sociopathy and the ensuing debate in this journal is a welcome contribution to the growing field of evolutionary psychiatry. Given that evolutionary science is concerned with adaptation and that psychiatry is concerned with abnormality, it is perhaps not surprising that this discussion soon turned to the question of the relationship between adaptiveness and abnormality. The phenomenon of psychopathy may be a particularly useful one with which to think about this relationship, as this condition, which exists at the borders of psychiatry, law, and morality, has an unclear and complex categorical status.

There have been attempts to define medical and psychiatric abnormality specifically in terms of dysfunction (Boorse 1975; 1976). However, this is not so easily done. For one thing, although function is now increasingly defined in terms of evolutionary theory (Allen & Bekoff 1995), this logic cannot always be readily extrapolated to disorders. For example, dysfunction might be defined in terms of decreased reproductive success (RS). But medical and psychiatric disorders do not necessarily involve a lack of RS, and a lack of RS is not sufficient to entail the presence of disorder (for example, homosexuality is no longer included in the Diagnostic and Statistical Manual of Mental Disorders).

Recent work in cognitive science on complex categories (such as disorder) demonstrates that invariably these are not definable in terms of necessary and sufficient criteria (Lakoff 1987). Certain typical medical disorders (e.g., pneumonia) entail such features as a clear proximate cause (e.g., a bacterium), that results in specific harmful symptoms (e.g., shortness of breath), which excuses the person from ordinary obligation so that medical treatment (e.g., an antibiotic) can be given. However, there are also atypical medical conditions (e.g., macromastia or large breasts), where there is no clear proximate cause, where the harmfulness is more debatable (a cosmetic surgeon may emphasize the sequelas of back pain in this case), and where the person may not be excused from ordinary obligations in order to receive treatment (e.g., cosmetic surgery may not be considered a medical necessity by many).

Similar concepts might also apply to psychiatry. In certain typical disorders (e.g., post-traumatic stress disorder), there is a clear proximate cause (e.g., being raped), that results in harmful symptoms (e.g., hyperarousal), which excuses the person from ordinary obligations so that medical treatment (e.g., psychotherapy) can be given. However, there are also atypical medical disorders (e.g., psychopathy), where there may be no clear proximate cause, where the harmfulness is more debatable (in some respects psychopathy may be adaptive), and where the person may be more likely to be referred to the courts or to the church for management, given that usual psychiatric treatments have not proven to be particularly useful.

Mealey's analysis might be taken to suggest that primary psychopathy is more of a typical medical/psychiatric condition, whereas secondary psychopathy is more of an atypical medical/psychiatric disorder. Primary psychopathy has a genetic proximate cause, which may be amenable to psychopharmacological treatment. (Moore & Rose, 1995, note that primary psychopathy is similar to pseudopsychopathy after brain damage, and Mealey, 1995, notes that this entity fits the medical model.) Secondary psychopathy on the other hand has a multivariate origin, and intervention is perhaps best at the level of social change. Bailey (1995) suggests that psychopathy is not in fact abnormal, and Mealey (1995) agrees that the medical model does not fit psychopathy.

On the other hand, it may be argued that it is important to extend the metaphors of medicine and psychiatry to psychopathy in order to emphasize the possibility that this condition has underlying biopsychological proximate causes and is amenable to medical intervention (Stein 1994). Given the atypicality of psychopathy, such medical interventions may be unusual (e.g., instead of a psychopharmacological agent, limit-setting may be prescribed; Stein 1996). Nevertheless, these interventions are perhaps better construed as medical than as simply moral or legal. Thus, evolutionary explanations of the adaptiveness of behavioral traits does not in and of itself imply that we should not categorize them as medically abnormal and intervene accordingly.

Author's Response

Heritability, theory of mind, and the nature of normality

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Abstract: It is impossible to discuss the constructs "heritability," "theory of mind," and "normality" in a single coherent essay. The following three rejoinders address each of these exceedingly complex constructs individually, as each relates to the two-path model of sociopathy and psychopathy.

R1. Heritability estimates provide no foundation at all (Reply to Stoltenberg)

Stoltenberg's critique of my model (Mealey 1995) can be presented as a syllogism. He claims that: (1) "estimates of the heritability of sociopathy . . . form the foundation of [my] model" (Abstract), and (2) because "it is questionable whether heritability estimates can provide meaningful information when experimental control (e.g., of matings) is not possible" it follows that (3) "without proper evidence for a genetic basis of sociopathy [the] model collapses" (Abstract). First I will comment on premise (2) of the syllogism, and allow the reader to arrive at an individual judgment about the meaningfulness of heritability estimates of criminality and sociopathy. I will then show that regardless of one's opinion on this controversial topic, the soundness of the model is not based on, or even dependent upon, one's faith or lack of faith in heritability estimates. In attacking heritability estimates, Stoltenberg attacks a straw man.

Stoltenberg lists five supposed assumptions of twin studies that affect the validity and generalizability of the heritability estimates that come from them. He directly addresses only assumption 5 as it relates to the target article, but since he implies that all five are important and that all five were violated in the studies I cited, I will briefly address them all.

The criticism of heritability estimates based on violations of the first two assumptions – that MZ (monozygotic) and DZ (dizygotic) twins experience "equal environments" and that it is possible to generalize from twins to nontwins is fairly trivial. First, *no individuals,* twins or not, experience *identical* environments. That fact is very important in terms of individual development (and is addressed at length in section 2.3.1 of the target article); it is not an important fact, however, in terms of the methodology of obtaining heritability estimates from twins. What *is* important for good heritability estimates is that the means and variances of the relevant aspects of the environments be the same for MZ, DZ, and nontwin individuals. Most twin studies document that this is the case: the means and variances of the standard sociological predictors of crime are similar for all three groups (e.g., Christiansen 1977; Rowe 1983), thus not violating the first assumption. Second, there are few outcomes in life for which being a twin is, per se, a relevant factor (assumption 2). Indeed, in a paper cited in the target article, Rowe (1983) directly addresses this issue as it relates to criminogenesis; Stoltenberg does not refer to Rowe's work.

Unlike assumptions 1 and 2, "assumptions" 3, 4, and 5 are nontrivial and are generally acknowledged to be violated in twin studies. For this very reason, however, they are *not,* generally, assumed. Genetic effects are *not* assumed to be purely additive, parents are *not* assumed to mate at random, and gene–environment interactions *are* anticipated. That one cannot afford to assume otherwise is one reason why adoption and twin studies are used in a complementary fashion. This topic is addressed extensively in the target article in footnote 7 (a footnote that is in fact, more than half as long as the section of the target paper that Stoltenberg attacks, and almost as long as his own commentary).

The "assumptions" of purely additive genetic variance and of random mating are particularly relevant for agricultural models where the goal is to select "for" certain traits in a directional and quantitative fashion; they are not useful for those who study humans or other animals with the goal of understanding an already existing situation. (Perhaps it is because they work exclusively with animals that Stoltenberg and Crusio [1995; the only one of the original 42 commentaries that Stoltenberg cites] focus so often on these parameters.) Indeed, I hope to have made clear, if not in the target article, then at least in the first-round Author's Response, that I expect *nonadditive* genetic variance to be *particularly important* in the etiology of what I called "primary sociopathy" or "psychopathy." Likewise, the fifth "assumption" Stoltenberg lists, that of lack of gene-environment interactions is *not* made, and is another reason that adoption studies (which can assess these interactions) are cited together with twin studies (which cannot). In the model, gene-environment interactions are considered to be just as important in the genesis of secondary sociopathy as nonadditive genetic variance is in the etiology of primary sociopathy.

As these elements clearly have not been ignored in the target paper itself, Stoltenberg's criticism is restricted to the claim that since the methodology of some of the *cited* papers ignores these factors, the heritability estimates that came from those studies may not be "meaningful." Taken individually, this is perhaps the case. But when assessed collectively alongside the results of adoption studies, studies of twins reared apart, comorbidity studies, and now, studies of individual genes, the same general conclusion emerges: both genetics and environment contribute significantly to phenotypic variance in criminality and sociopathy. (For a brief update of additional studies since the publication of the target article see Carey & Gottesman 1996.) Convergence toward the same conclusion via methodological triangulation is generally considered to give strength to a model (Mealey 1994), and the triangulation technique of the target article has been singled out by one philosopher of science as a paradigmatic example of how to build an explanatory model in an area which is complex and, in many ways, not amendable to direct experimentation (Holcomb 1995; forthcoming).

No matter how one chooses to weight the heritability estimates cited in the target article, they do not provide the foundation upon which the model is based. The foundation of the model is provided by the theoretical arguments presented in sections 1.1 and 1.2. Heritability estimates provide only a small fraction of the evidence marshalled in support of the model (slightly more than one of the ten pages of sections 2.1–2.5), and the fraction of that coming from twin studies constitutes a mere paragraph. Supporting evidence was drawn from a wide variety of fields utilizing a wide variety of methodologies, and once again, a convergence of evidence (with or without the heritability estimates) leads to a single conclusion: support for the two-path model of sociopathy.

Stoltenberg closes with the imperative that "to advance our understanding of heredity–behavior relations, it is essential that we appreciate the theoretical basis of the statistical techniques we employ." Others appear to view the field and its future course otherwise. To quote David Wasserman, convener of the recent, widely publicized conference "Research on Genetics and Criminal Behavior" (September 1995, Queenstown, Maryland),

> The sun is setting on traditional heritability research on social behavior (as) it has served its purpose. Researchers expect the sunset of heritability to be followed by the bright dawn of

neurogenetic research, which will trace the complex causal pathways through which specific genes influence those behaviors" (1996, p. 108)

This is indeed the next frontier: our goal is to understand the internal and external environmental triggers that activate genetic, physiological, and psychological mechanisms that contribute to complex social behavior.

R2. The psychopath's theory of mind (Reply to Cruz)[1]

Cruz's commentary raises questions about the nature of a psychopath's theory of mind. Although I touched on this issue only briefly in the Mealey (1995t) target article, of all the questions I raised then, this is the one that has since intrigued me the most. In fact, the decision in my original Response (Mealey 1995r) to change from the terms "primary" and "secondary" sociopath to the terms "psychopath" and "sociopath" respectively, was based in part on the historical trend to use the term "psychopathy" to describe a mental type (e.g., "moral insanity"), and the term "sociopathy" to describe a completely different, social phenomenon. It is only the psychopath, not the sociopath, about whom Cruz and I share the belief that there is something qualitatively "different" in terms of theory of mind. This "typology" position fits better with historical notions of "psychopathy" than with the current DSM definition of "anti-social personality," and I hope it will stimulate new and different research initiatives. Given our basic agreement on this point, I do not particularly wish to argue against Cruz's specific notion about the theory of mind of the psychopath, but in the hope that this line of commentary and discussion will continue (in these pages or elsewhere), I will take the opportunity to clarify my position, both where it contacts and where it departs from that of Cruz.

Cruz summarizes my portrayal of the psychopath's theory of mind with two paraphrases: (1) the psychopath "lacks noninstrumental characterizations of intentional agents"; and (2) the psychopath "lacks an apparatus for empathic projection of oneself onto others." The first paraphrase is quite accurate; the second is close but for one critical word which, as I will show, has important implications. Cruz cannot be blamed for this misattribution, since the target article addresses theory of mind only in passing and makes no distinction between the various models. I will therefore summarize my overall position in the theory of mind debate, then relate that view to the question of the theory of mind of the psychopath.

As an evolutionary psychologist, I am partial to the modular, domain-specific view of mental processes according to which there is a theory of mind module (ToMM) which, as a consequence of its domain-specificity, operates with limited input and is constrained to limited output: the input consists of knowledge derived from first-hand, phenomenological experience, as well as from the observation of contingencies related to behavior of the self and others; the output consists of inferences regarding the emotional/motivational state and behavioral propensities of others, given certain inputs. The ToMM is not a general-purpose reasoning device and is not responsible for the generation of any theory other than theory of mind.

In my model of psychopathy, the inability to experience social emotions figures prominently. This emotional deficit limits the phenomenological, first-hand input to the psychopath's ToMM, leaving him with only primary emotions and observations of behavioral contingencies as inputs. The resulting inferences (output) of the psychopath's ToMM are accordingly limited – but not because his ToMM is damaged or absent. The psychopath's ToMM is normal, but is operating with less information than is normal; this is what results in the "instrumental," non-empathic nature of the psychopath's theory of mind.

In his commentary, Cruz first claims that a psychopath cannot have a purely instrumental theory of mind, because such a mechanism could not be "successful" at predicting others' behavior and would "render [primary] sociopathy a risky business." Well, primary sociopathy (psychopathy) *is* a risky business and many psychopaths are utter failures; indeed, the psychopath's lack of ability to learn from experience is a recurring theme in much of the clinical literature (see sect. 2.4.1 of the target article and commentaries by Barresi 1995, Kosson & Neuman 1995, Moore & Rose 1995, and Raine 1995). An instrumental, non-empathic theory of mind can only be as successful as the intelligence of its owner allows, and it is this that accounts for the fact that the intelligent psychopath is more likely than the unintelligent one to find a way to integrate successfully into modern society. In section 3.1.1 of the target article I stated that in the absence of intellectual handicap, psychopaths may become excellent predictors of others' behavior – not that all will.

Cruz next claims that my argument regarding the "instrumental" nature of the psychopath's theory of mind "collapses" into the argument that the psychopath has no "apparatus for empathic projection." I have no argument with the "interpretation" that the first half of my argument "collapses" into the second half, in that I believe that the instrumental nature of the psychopath's attributions results as a consequence of his lack of emotional competence. I do, however, take issue with Cruz's use of the term "apparatus" in his attempt at paraphrase, as I do not postulate that the ToM "apparatus" (module) of the psychopath is deficient; it is only the inputs to it that are deficient.

In his next step, Cruz suggests that a "successful" theory of mind (according to "theory-theorists") is one which yields ascriptions of mental states to others, but not knowledge about what it is like to experience that state. [See also Gopnik: How We Know Our Minds. *BBS* 16(1):1–14.] Using this description of theory theory, Cruz points out that my explanation of psychopathy relies on the psychopath's "missing" an attribute of theory of mind that is not actually posited to exist. I agree that any model which stops at ascriptions of beliefs without addressing the all-important ability to interpret an attribution in terms of another's probable motivational state lacks a significant element. To the extent that a psychopath cannot predict another's behavior using a purely instrumental approach, it is precisely because he does not make the (emotional) connection between the ascription of the other's mental state and the other's motivational state. But Cruz's suggestion that theory theory makes no attempt to account for feelings does not jibe with my reading of the literature. Cruz, like Harris (1989, whom he cites) makes a straw man of theory theory by asserting that it ignores input from personal experience. Wellman (1990) objects to this characterization, saying "It is misleading to oppose theorizing and imaginative projections . . . The use of imaginative projections is complementary with, indeed typically encompassed" by theory theory (p. 199).

Like Wellman (and Cruz), I believe that something akin to simulation (imaginative projection) must be occurring in those with a "normal" theory of mind. On the other hand, I do not believe that this is a conscious process (as Cruz explains: "ascribers imagine themselves in the target's circumstances"); nor do I find myself in agreement when Cruz attempts to apply the simulation model to the psychopath. Cruz suggests that a psychopath may not be able to simulate others' states well because the "simulation mechanism [is] completely defective in the ability to go off-line." When Cruz attempted to paraphrase me saying that the psychopath "lacks . . . an apparatus for empathic projection of oneself onto others," he was really voicing his own model. In my model, the psychopath's deficit is not in terms of a faulty "apparatus." The psychopath does not have a problem going "offline" to "simulate" another; it is that when he does, he has an impoverished pool of emotional experiences and background knowledge from which to draw in order to create his "simulation."

Both Cruz's "simulation deficit" model and my "emotional deficit" model are tenable. The "emotional deficit" model, however, is more parsimonious than the "simulation deficit" model, and fits better with what we already know about psychopaths. Consider, for example, Cruz's convoluted explanation of the relatively poor performance of low Mach individuals compared to high Mach individuals, in assessing the Mach levels of others. The "simulation deficit" model first requires that low Machs (normal prosocials) "are victim to something akin to an adult version of failure in the false belief task," resulting in a partial ascription of their own mental state to others; second, it requires that high Machs have a complete deficit in going "off-line," but rather than to err by fully ascribing their own state to others, they learn, in reaction to some kind of dissonance, to use better actuarial predictors. The simulation-deficit model thus requires not only that low Machs have a partial simulation deficit to start with, but that compared to high Machs they also have a learning deficit! This conclusion is in direct contradiction to known facts. My explanation of the Mach assessment results, on the other hand, is simply that both groups rely on a comparable "simulation apparatus" but, because of their differential pools of background experiences and emotions, they create different "projections"; the differential pool of emotions from which the two groups draw is, in essence, the affective equivalent of two groups having different cognitive availability biases (Tversky & Kahneman 1982).

The "emotional deficit" model is also more parsimonious in its better fit with known physiological differences between psychopaths and normals. In his closing remarks, Cruz asks why, if there is no deficit of process (in the psychopath), "serotonin or testosterone levels, for example, should affect the theory of mental state ascription adversely while leaving other theoretical domains (folk physics, folk biology) unimpaired"? The answer is that the known physiological anomalies of the psychopath are completely consistent with the idea of a deficit in the ability to generate a broad spectrum of emotions; there is no need to postulate an additional deficit in the ToMM.

Whether Cruz's notion, or mine, or neither is correct, will only be revealed by future research: the empirical investigation of theory of mind is only in its early stages. Cruz's conjectures about the possible application of these emerging concepts to the topic of psychopathy raise an intellectual challenge which should stimulate a new generation of exciting questions. I look forward to seeing the answers.

R3. The nature of normality (Reply to Stein)

Stein is absolutely right that different definitions and uses of the term "normal" (and its complement, "abnormal") have different implications. Typically, the term "abnormal" connotes undesirability and, consequently, encourages efforts at "improvement" via prevention or intervention. Stein suggests that evolutionary adaptiveness/maladaptiveness does not map directly onto medical functionality/dysfunctionality, and that we must not, therefore, rely solely on adaptiveness as a criterion for making medical or social policy decisions. I fully agree, but would like to point out that the medical model is equally limited, nor should it be relied on exclusively to set the criteria for social policy decisions.

I would like to present two schemes for classifying abnormalities – not to argue for or against either, but to highlight the difficulties that any classification scheme will entail. The first scheme, which I will address only briefly, consists of a set of five different definitions of "normality" that are all commonly used, but by no means convergent. The second scheme is one that I have derived based on an evolutionary perspective.

The common approaches to distinguishing between normality and abnormality include statistical normality, the medical model, psychological normalcy, socially prescribed norms, and legal norms. Each of these definitions has a value within a particular framework: the statistical approach is most useful in the context of empirical description; the medial approach is most useful when the object, trait, or attribute of inquiry has a single, known function, the achievement of which is obvious and easily measurable (a rare circumstance, I would argue); the psychological approach is most useful in a personal counseling or mental health setting; the social approach is the most useful in terms of the tremendous insight it gives us into human nature; and the legal approach is useful as an applied social tool (to be discussed a bit later). Each approach also has severe limitations: the statistical approach is fraught with arbitrariness; the medical approach is predicated on knowledge that we simply do not have; the psychological approach fails us when, for a variety of possible reasons, individuals do not "feel" the way we expect or want them to "feel"; the social approach results in significant temporal and geographical differences in categories and judgments; and the legal approach is limited by human conflicts of interest and issues of practical application.

Each of these approaches was addressed to some extent in my first round Response (Mealey 1995r), and although I have no space to go into more detail here (see Mills & Mealey, forthcoming, for more), it is clear that they do not converge on similar "solutions." Stein's example of homosexuality is an excellent one to revisit, in that homosexuality is a trait/behavior that is common or uncommon, functional or dysfunctional, normal or abnormal, prescribed or proscribed, legal or illegal – depending on where you happen to be standing and who you happen to ask!

Besides the problem of incongruity, our difficulty in finding criteria for deciding whether to try to promote, prevent, or intervene in the development of various traits/outcomes is based on the fact that all of the above

definitions tell us something, but none of them tells us anything about ethics.

My evolutionary classification scheme considers (not surprisingly) adaptive value, but it also considers ethics. Like psychiatrist Nesse and evolutionary biologist Williams (Nesse & Williams 1991; 1994), I consider that in a multiparty interaction (be it a host–parasite interaction, two humans interacting with one another, or one human interacting in a social network), what is adaptive for one party may not be adaptive for another. Thus, what is "functional" or what is "normal" or what is a "desirable outcome" is always relative to the perspective taken by the different parties in the interaction. I present this scheme here simply because I find it useful; it is not intended to stand alone.

First in my evolutionary scheme are the "true pathologies." These are abnormalities or dysfunctions from the perspective of the single, individual human who is directly affected. This category would include, for example, effects of toxins, infectious disease, and injury. Abnormalities in this category are likely to reduce fitness, but lowered fitness per se cannot be used as a criterion for identifying them. Like some supposed medical and psychiatric "disorders," they may indeed have an unknown function or they may be the best available option of a set of alternative strategies – what Dawkins (1980) calls "making the best of a bad job." True pathologies can only be recognized as such in that they elicit a combative (healing) response from the individual. Toxins, infectious disease, and injury for example, all elicit complex, coordinated, obviously evolved, adaptive responses. These responses are evidence of selection pressures in the past, demonstrating that the insult indeed has a history of causing harm. We have no ethical dilemma in deciding whether to try to prevent or intervene in such cases; dilemmas in such cases are instead more likely to involve practical questions such as how to allocate scarce resources.

Second in my scheme are what I call the "modern pathologies." These are also pathologies or abnormalities from the perspective of the individual who is affected, and, like true pathologies, they are likely, on average, to lead to a reduction in fitness. Modern pathologies are to be discriminated from true pathologies in that there is no identifiable coordinated counter-response from the affected individual – indeed, the source of the "problem" may seem to be internal. Modern pathologies are likely to represent adaptations gone awry. They may have a complicated genesis that consists of a variety of coordinated changes in the state of the organism but these coordinated changes reduce, rather than enhance, adaptive function. This set of circumstances would suggest that an evolved mechanism has been triggered, but that its deployment is no longer appropriate in the modern human environment. Examples include obesity, myopia, anorexia, and endogenous depression. (See Anderson et al. 1992; Nesse & Williams 1994; Price et al. 1994; Surbey 1987; and Wallman 1994 for possible proximate explanations of these mechanisms-gone-awry.) In the case of modern pathologies we are confronted with an ethical dilemma, in that the afflictions of some individuals are, in essence, costs of "social progress." As with illnesses caused by pollutants that never existed before in our evolutionary history (asbestos, radioactive waste), the prevention of such pathologies may require that we, as a society, give up some modern conveniences and innovations (both technological and sociological); there are likely to be significant disagreements amongst people on this point.

Last, and most important for this discussion, are what I call the "ethical pathologies." Ethical pathologies are traits or behaviors that may be functional and adaptive for one individual in a social interaction, but which have dysfunctional, maladaptive consequences for one or more other participants in the interaction. Ethical pathologies would include rape, theft, adultery, and warfare. On average, such traits or behaviors presumably increase one party's fitness to the detriment of another, but the only way one can identify them is by looking for complex, coordinated response systems. Ethical pathologies will be identified by finding corresponding, coevolved, complementary response sets amongst the different parties to the interaction. Various deception strategies, for example, will be countered by deception-detection strategies (e.g., Alexander 1987; Cosmides 1989; Mealey et al. 1996); rape attempts will be countered by rape-avoidance strategies; theft will be countered by protective measures and so on. Since ethical pathologies may involve large numbers of interactors, we can expect to see complex social strategies evolve out of this type of "arms race." I see elements of government and various service providers (counselors, lawyers, maybe even political activists) as part of the "extended phenotype" (Dawkins 1982) that individuals, acting as potential victims, have evolved in order to counteract the strategies of the potential perpetrators and social parasites among us.

Despite their different proposed etiologies, psychopaths and sociopaths both fall in the category of social parasites, and their attributes/behavior fall therefore, in my way of thinking, into the category of ethical pathology. When it comes to ethical pathologies, we are all potentially victims and we are all potentially perpetrators. How we want to deal with that fact as a society is a totally separate question.

NOTE

1. I would like to thank Virginia Slaughter for her helpful feedback on an earlier version of this rejoinder.

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Commentary on **Mark L. Latash and J. Greg Anson (1996). What are "normal movements" in atypical populations? BBS 19:55–106.**

Abstract of the original article: Redundancy of the motor control system is an important feature that gives the central control structures options for solving everyday motor problems. The choice of particular control patterns is based on priorities (coordinative rules) that are presently unknown. Motor patterns observed in unimpaired young adults reflect these priorities. We hypothesize that under certain atypical conditions, which may include disorders in perception of the environment and in decision making, structural or biochemical changes within the central nervous system (CNS), and/or structural changes of the effectors, the central nervous system may reconsider its priorities. A new set of priorities will reflect the current state of the system and may lead to different patterns of voluntary movement. Under such conditions, changed motor patterns should be considered not pathological but rather adaptive to a primary disorder and may even be viewed as optimal for a given state of the system of movement production. Therapeutic approaches should not be directed toward restoring the motor patterns to as close to "normal" as possible but rather toward resolving the original underlying problem. We illustrate this approach using, as examples, movements in amputees, in patients with Parkinson's disease, in patients with dystonia, and in persons with Down syndrome.