In Cold Blood: The Evolution of Psychopathy

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"Fool me once, shame on you. Fool me twice, shame on me."

- old (probably Chinese) proverb.

Introduction

Of all the interesting topics in the field of forensic psychology, psychopathy probably generates the most fascination. In university courses covering psychopathy, students wake from their slumber and knock on professors' doors to ask how they can get involved in research on psychopaths. In crime fiction and historical biographies, psychopathic characters are imbued with iconic qualities. It seems that our minds are attuned to psychopathic characteristics in others, and probably for good reason: If psychopaths have been a constant feature of the ancestral social environment of *Homo sapiens*, they would have exerted significant selection pressure. Researchers have not been immune to this fascination. Despite the fact that psychopaths represent a small proportion of criminal offenders, psychological research on psychopathy seems to dominate the forensic literature.

The most important reason for the popularity of psychopathy among forensic researchers is probably the empirical fact that measures of psychopathy are reliable and robust predictors of future criminal behavior in both forensic and non-forensic populations (reviewed in Harris, Skilling, & Rice, 2001; Leistico, Salekin, DeCoster, & Rogers, 2007; Porter & Woodworth, 2006). In fact, one measure of psychopathy, the Psychopathy Checklist-Revised (Hare, 2003), might be the single best psychological predictor of criminal recidivism. In actuarial assessments of dangerousness, scores on measures of psychopathy have very large and often the largest predictive weights (e.g., Hilton, Harris, Rice, Houghton, & Eke, 2007; Quinsey, Harris, Rice, & Cormier, 2006).

Perhaps even more interesting, scores on measures of psychopathy reveal intriguing interactions in other research with offenders. Psychopathy and measures of sexual deviance (or paraphilia) have been found to exhibit a multiplicative relationship such that sex offenders who are both sexually deviant (e.g., sadistic or pedophilic) and psychopathic are much more likely to engage in sexually violent recidivism than all other group combinations (Rice & Harris, 1997; Seto, Harris, Rice, & Barbaree, 2004). Psychotherapy effective in reducing the risk of violence among non-psychopaths has been reported to have the opposite effect on psychopaths, increasing their risk of violence (Hare, Clarke, Grann, & Thornton, 2000; Harris, Rice, & Cormier, 1994; Rice, Harris, & Cormier, 1992). Alcohol abuse is a good predictor of criminal recidivism among schizophrenic offenders, but not among psychopaths, even though psychopathic offenders are more likely to abuse alcohol than schizophrenic offenders (Rice & Harris, 1995). Even more intriguing are empirical reports that psychopaths rated by therapists as having benefited from treatment are subsequently more dangerous than psychopaths rated as not having benefited (Looman, Abracen, Serin, & Marquis, 2005; Seto & Barbaree, 1999; but see Langton, Barbaree, Harkins, & Peacock, 2007).

Greatly facilitating this burgeoning research activity is a valid and reliable measure of male psychopathy, the Psychopathy Checklist, now revised (PCL-R; Hare, 2003). Researchers have also subsequently developed similar psychopathy measures for non-forensic populations, teenagers, and even children. The PCL-R has provided researchers with a common definition of psychopathy, greatly aiding communication and integration of results in the field. Other terms have sometimes (and mistakenly) been used to mean the same thing as psychopathy, such as sociopathy, antisocial personality disorder, and Machiavellianism. Psychopathy now typically refers to “a lifelong persistent condition characterized, in males at least, by aggression beginning

in early childhood, impulsivity, resistance to punishment, general lack of emotional attachment or concern for others, dishonesty and selfishness in social interaction, and high levels of promiscuous and uncommitted sexual behavior" (Harris et al., 2001, p. 197-198). Psychopathy is more restrictive than Antisocial Personality Disorder as defined in the Diagnostic and Statistical Manual of Mental Disorders because the customary diagnostic cut-off for psychopathy is more stringent, but in fact, the indicators of psychopathy and antisocial personality disorder are highly correlated and identify essentially the same individuals (e.g., Skilling, Harris, Rice, & Quinsey, 2002)—contrary to the commonly accepted view (e.g., Livesley, 1998). Psychopathy is mostly a male phenomenon, and in this chapter we focus on male psychopathy.

In sum, psychopathy is (perhaps naturally) fascinating, can be measured reliably, and is an important social phenomenon with significant practical implications. It is thus not surprising that it has generated a large amount of theoretical interest. Where does psychopathy come from? Can evolutionary psychology help us generate new hypotheses about the origins and causes of psychopathy? Before we address these questions, let us examine more closely the construct of psychopathy.

The Construct of Psychopathy

Psychopathy as a Clinical Condition. A century and a half ago, the modern concept of psychopathy originated in the observation that a small minority of people seemed to engage in antisocial, irresponsible, extremely selfish (and even apparently self-destructive) behavior without also displaying any obvious signs of mental derangement. Beginning about 70 years ago, Cleckley (1941) applied the term psychopathy and added clinical descriptions of other, more affective, aspects of this condition; superficial charm and good intelligence, absence of nervousness, dishonesty, lack of remorse, incapacity for love, and shallow emotional responses,

as examples. For the last four decades, Hare (1970; 1998; 2003) has elaborated on Cleckley's clinical observations and brought the study of psychopathy into the realm of scientific investigation. As mentioned above, one of Hare's several contributions has been the development and validation of an effective way to measure the phenomenon. The PCL-R (Hare, 2003) comprises 20 psychopathic characteristics to be assessed primarily based on an individual's life-long pattern of conduct as documented in official records and institutional files, but the scoring of some traits (e.g., grandiose sense of self-worth, lack of remorse, lack of realistic long-term goals, failure to accept responsibility for actions) may also be inferred from a semi-structured interview.

Twenty years ago, Hare (Harpur, Hackstian, & Hare, 1988) reported that scores on the PCL-R consisted of two highly related (correlations greater than .50) but conceptually and empirically distinct aspects. The first, usually called Factor 1, comprised *interpersonal and affective* characteristics (e.g., conning and manipulative, callousness and lack of empathy), while Factor 2 described a *deviant, antisocial lifestyle* (e.g., proneness to boredom, poor behavioral controls, early behavior problems, impulsivity, juvenile delinquency, parasitic lifestyle). A few characteristics (sexual promiscuity, many short-term marital relationships, and criminal versatility) did not appear to load on either factor. The names given to the factors did not strictly capture their content, of course—boredom is an affective response; poor behavior controls are about irritable, angry, hostile, violent emotional responses; conning and manipulation are about overt antisocial conduct; and criminal versatility is certainly about an antisocial lifestyle. Nevertheless, at the empirical level, this two-aspect nature of psychopathy has generally held up ever since (Benning, Patrick, Hicks, Blonigen, & Kreuger, 2003; Blackburn, 2007; Loney, Taylor, Butler, & Iacono, 2007; Patrick, Edens, Poythress, Lilienfeld, & Benning, 2006; Skeem,

Johansson, Andershed, Kerr, & Louden, 2007).

It is evident that those who receive maximal scores on a measure of psychopathy such as the PCL-R would, by definition, exhibit both aspects. Also, the well-established empirical association between the two factors means that those who score highly on one aspect have a high probability of also exhibiting the other (Skilling et al., 2002). Nevertheless, some people who receive high scores on such a measure do so via a maximal score on one aspect and perhaps only a moderate score on the other. Indeed, these two aspects appear to be related in opposite directions to such emotions as anxiety and depression (Hicks & Patrick, 2006). As well, many empirical findings about psychopathic responding in the laboratory or in the natural environment seem particularly characteristic of but one of the two aspects (e.g., Carlson, McLarnon, & Iacono, 2007; Hare et al., 2000; Maccoon & Newman, 2006; Molto, Poy, Segarra, Pastor, & Montanes, 2007). Most relevant for forensic application, the second aspect is more predictive of criminal recidivism, violent recidivism, substance abuse, and suicidal behavior (Harris et al., 2001; Leistico et al., 2007; Ousey & Wilcox, 2007; Salekin, Rogers, & Sewell, 2001).

Thus, it appears that some violent offenders have such traits as remorselessness, grandiosity, and insincerity, and are presumably deliberately and planfully violent out of emotional detachment and indifference to others' interests. Another group of violent offenders seem to be impulsive and experience considerable anger, anxiety, and distress and are violent due to such negative emotions. This distinction[1] has long been noted in the psychopathy

[1] Readers might wonder how someone (with a maximal score on the PCL-R, for example) could simultaneously possess the traits of shallow affect and strong negative emotionality. In addition to the possibility of deliberate deception, the answer no doubt lies in the circumstances. Prototypical psychopaths are emotionally indifferent to the unhappiness and suffering of others (but not because they have any trouble perceiving it). They are, however, easily angered and upset by threats to their own interests. When institutionalized for example, they worry about their fate (Cleckley, 1941), angrily guard their rights, are prone to regard themselves as "victims of the system", their own misdeeds notwithstanding (Hare, 1998).

literature, and is often termed primary and secondary psychopathy, respectively. It is now evident that these two aspects of the phenomenon are, at least partly, due to quite distinct underlying basic processes. It has been assumed that the primary, affectively cold-hearted version is that which reflects psychopathy's "core personality" is more constitutional and "biologically" based. On the other hand, the secondary, "behavioral" version has been seen as acquired and contextually caused (Mealey, 1995; Skeem et al., 2007). Current evidence appears, however, to make these etiological assumptions untenable.

Psychopathy in the Context of Development. The first relevant source of data comes from laboratory studies of adults. It is clear that experimenters can arrange test conditions such that psychopaths obtain poorer scores than other groups (e.g., Blair et al., 2006). But it is just as clear that some experimental conditions lead to equivalent or even better performance by psychopathic participants (Budhani, Richell, & Blair, 2006; Book, Holden, Starzyk, Wasilkiw, & Edwards, 2006). Indeed, the core affective psychopathic personality traits appear to be so subtle that it is unclear how they can be characterized (Munro et al., 2007); for example, psychopaths do not seem to exhibit deficits in detecting emotion in others (Glass & Newman, 2006) and might even be better at it (Book, Quinsey, & Langford, 2007).

Conversely, the more behavioral, antisocial lifestyle aspects of psychopathy exhibit profound, inescapable (and utterly unsubtle) findings. As mentioned above, these are the psychopathic traits most predictive of forensically relevant outcomes. These so-called externalizing traits have been reported to exhibit a distinct, natural class (Harris, Rice, & Quinsey, 1994; Harris, Rice, Hilton, Lalumière, & Quinsey, 2007; also see Swogger & Kosson, 2007; but also Edens, Marcus, Lilienfeld, & Poythress, 2006), even in juveniles (Skilling, Quinsey, & Craig, 2001; Vasey, Kotov, Frick, & Loney, 2005). Such externalizing traits,

together with some callous and unemotional traits, exhibit a distinct developmental trajectory detectable in individuals as young as age three (Glenn, Raine, Venables, & Mednick, 2007; Moffitt & Caspi, 2001; Shaw, Bell, & Gilliom, 2000; Shaw, Gilliom, Ingoldsby, & Nagin, 2003; Vizard, Hickey, McCrory, 2007). There is also clear evidence that this pattern of externalizing traits represent a stable, life-course phenomenon (Loney et al., 2007; Lynam, Caspi, Moffitt, Loeber, & Stouthamer-Loeber, 2007). These externalizing aspects of psychopathy associated with negative emotions are at least as heritable as the affectively coldhearted traits (Burt, McGue, Carter, & Iacono, 2006; Hicks et al., 2007; Larsson et al., 2007; Larsson, Andershed, & Lichtenstein, 2006; Viding, Frick, & Plomin, 2007). Indeed, an externalizing factor among elementary school-aged children has been reported to exhibit a heritability coefficient of .96 (Baker, Jacobson, Raine, Lozano, & Bezdjian, 2007). These externalizing traits seem to be more closely associated and central to the characteristic cognitive differences associated with psychopathy (Maccoon & Newman, 2006). Finally, externalizing traits, as assessed by the PCL-R, for example, appear to predict violent behavior even among adults who are unlikely to meet any criteria for classification as psychopaths (Harris, Rice, & Camilleri, 2004; Hilton, Harris, Rice, Houghton, & Eke, 2007; Rice & Harris, 1992).

The Two Factors Revisited. Recent empirical research on psychopathy has clarified many features of this forensically important and fascinating condition. That same research, however, has also raised new questions. For example, should the condition of psychopathy be conceptualized as a disorder of personality whose core features of callousness and affective shallowness directly (but not inevitably) cause antisocial conduct (e.g., Cooke, Michie, & Hart, 2006; Cooke, Michie, & Skeem, 2007; Widiger, 2006)? Is psychopathy better conceived as a collection of enduring characteristic behaviors and interpersonal tactics (e.g., Hare & Neumann,

2006; Harris et al., 2007)? Is antisocial and criminal conduct merely the rather obvious and expected consequence of theoretically more interesting core psychopathic personality (e.g., Cooke et al., 2007)? Or are some aggressive and violent behaviors actually so much at the core of psychopathy that such antisocial behaviors are actually most diagnostic (e.g., Harris et al., 2007)? It is likely that not all the phenotypic traits of psychopathy have so far been optimally identified. It also seems possible, even probable, that one aspect of the condition (and its neurophysiological substrates) will ultimately be de-emphasized in favor of the other. Though it appears that the externalizing, behavioral horse has a small lead in this race, is not yet clear which path empirical and theoretical development will take and what the final result will be. In the following, we describe evolutionary accounts of psychopathy, and show that these can generate novel and testable hypotheses about the core features of psychopathy.

Explanations of Psychopathy

Traditional approaches to the study of antisocial behavior assume that the behavior would not occur if appropriate genetic, prenatal, family, socialization, and economic conditions were in place. There is, in fact, some support for these ideas; convincing evidence suggests that antisocial behavior, and especially violent behavior, is sometimes associated with some rare genetic mutations, poor prenatal or perinatal conditions (e.g., maternal malnutrition, birth complications), family instability, poor parental monitoring during adolescence, and low socio-economic status (for a review see Quinsey, Skilling, Lalumière, & Craig, 2004). Because some of these putative and potential causes involve disruptions of otherwise normally functioning systems, it seems that antisocial behavior can sometimes result from pathological causes.

Elsewhere, we have argued that the presence of pathological causes for a given trait or behavior does not necessarily imply that the trait or behavior in question is a pathological

outcome (Lalumière, Harris, Quinsey, & Rice, 2005; Mishra & Lalumière, this volume; Quinsey et al., 2004). It is quite possible that antisocial behavior is part of an adaptive response to specific and difficult conditions. For example, pathological causes having to do with early development (e.g., early malnutrition, the experience of physical abuse) may provide the child with information about the likely features of his future environment. This information may divert the child toward a developmental pathway that facilitates aggression, impulsivity, and high mating effort, tendencies that might better allow him to reach fitness-relevant goals. Alternatively, those early difficult conditions may reduce embodied capital (i.e., intrinsic attributes, such as health, skills, or attractiveness), leading to reduced ability to compete for resources, status, and mates, and forcing the adoption of alternative tactics of social competition. These two scenarios imply that antisocial behavior may be an adaptation (i.e., ancestrally selected) specific to "pathological" circumstances (just like an immune response is an adaptation designed to respond to infections). These ideas are discussed in more detail in Mishra and Lalumière (this volume).

What about psychopathy? Psychopaths are quite different from other offenders, even other life-course persistent violent offenders. They differ, for instance, with regard to some aspects of their criminal behavior (e.g., more violent, more goal-directed), how punishment and rewards affect their behavior in the laboratory, how they process emotional information, their physiological responses to aversive events, as well as cerebral lateralization and cerebral activity while processing verbal information (reviewed in Barr & Quinsey, 2004; Harris et al., 2001; Lalumière et al., 2005; Lalumière, Harris, & Rice, 2001; Quinsey et al., 2004). Also, signs of early neurodevelopmental problems that are associated with persistent violent criminality are not associated (and perhaps even negatively associated) with psychopathy (Harris, Rice, &

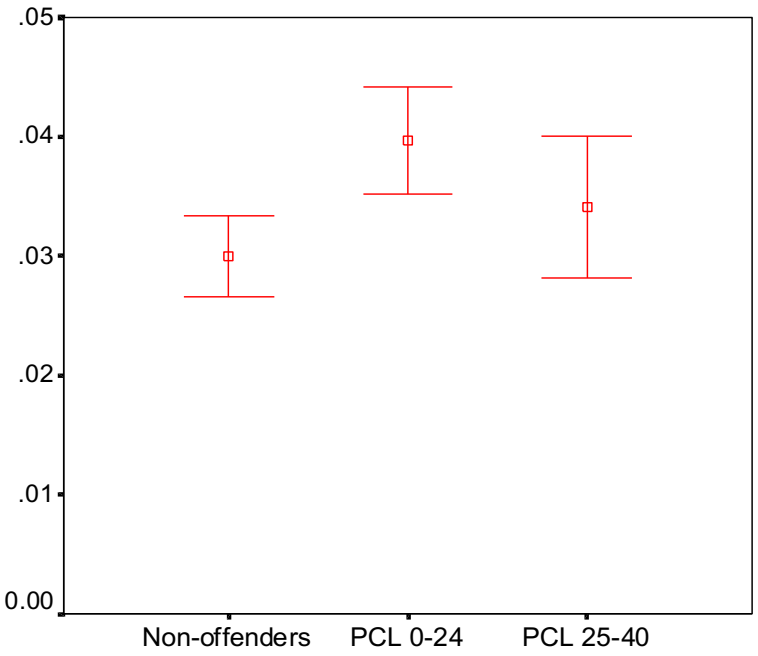
Lalumière, 2001; Lalumière et al., 2001).

Can conditional (facultative) developmental accounts of the sort mentioned above explain the psychopathic phenotype? Such an account would predict that psychopaths have experienced difficult early conditions statistically predictive of an inhospitable future biotic or social environment, or have reduced embodied capital and ability to compete. Evidence so far does not support such an account. As mentioned, psychopathy is unrelated to early signs of neurodevelopmental problems (e.g., obstetrical complications, low IQ, learning problems). Also, psychopaths show lower fluctuating asymmetry—a measure of developmental instability and a possible indicator of low embodied capital—than other violent offenders (Figure 1; Lalumière et al., 2001). In our study of fluctuating asymmetry, the psychopaths with the most extreme PCL-R scores were just as physically symmetrical as the non-offenders (hospital staff), and much more symmetrical than violent offenders with low PCL-R scores.

Psychopathy also seems to be unrelated to social factors generally associated with delinquency and conduct problems. For instance, in a study on the link between the quality of parenting on childhood conduct problems, ineffective parenting was associated with a higher number of conduct problems exhibited by children (as expected), but only among the children who did not display lack of empathy, manipulativeness, lack of guilt, or emotional constriction, all features of psychopathy (Wootton, Frick, Shelton, & Silverthorne, 1997; see also Oxford, Cavell, & Hugues, 2003). Children displaying psychopathic features had more conduct problems than other children, regardless of the quality of parenting. In fact, there was a tendency for *fewer* conduct problems among "psychopathic" children who experienced ineffective parenting compared to "psychopathic" children who had experienced more effective parenting. Although more research is needed, so far there is no evidence that the origin of

psychopathy involves the types of pathological causes implicated for persistent violent offending more generally. This result should be surprising to developmental psychologists because psychopathic children would be expected to at least *elicit* parental behaviors and social responses that often lead to neural, developmental, and social problems (e.g., excessive physical punishment, withdrawal of parental investment, peer rejection). If psychopathy is not the result of pathological processes of the kind already identified for other life-course persistent offenders, what might explain psychopathy?

Figure 1. Fluctuating Asymmetry Values for Non-Offenders (*n* = 31), Non-Psychopathic Offenders (*n* = 25), and Psychopathic Offenders (*n* = 15). Adapted from Lalumière, Harris, & Rice (2001).



Perhaps the most often discussed evolutionary explanation of psychopathy is the frequency-dependent selection account. In the most common version of this account,

psychopaths have evolved to take advantage of the fact that most people are co-operators by defecting in social interactions. Thus, psychopathy represents an alternative strategy (in the genetic sense) that is successful only at a particular low relative frequency in the population. If there are too many cheaters (or defectors), non-psychopaths become very vigilant and cheating opportunities disappear. It is not hard to imagine how the constellation of psychopathic characteristics (e.g., manipulative, charming, lack of empathy, failure to learn from punishment, unresponsive to cues of distress in others) would facilitate such a strategy. By this account, some individuals are born with a propensity for psychopathy, and the phenotype manifests itself early and perhaps without any environmental cues (e.g., Mealey, 1995). This type of obligate strategy has been observed in other species (see Textbox), but it is fairly rare compared to conditional (facultative) strategies.

Frequency-Dependent Selection in the Animal World

In the bluegill sunfish, there appear to be three types of males, distinguishable both behaviorally and morphologically. The largest males, called *parental* types, invest heavily in growth in the first few years of life, delaying reproduction. These males eventually build nests and use their size to defend nesting territories. *Satellite* males mimic females behaviorally and in physical appearance; they attempt to interrupt courting territorial males and intercept females in an attempt to fertilize them. Finally, *sneaker* males tend to stay near the lake bottom and make quick attempts to enter and exit nests, releasing ejaculate quickly. Sneaker males mature in two to three years, investing more in immediate reproductive capabilities than long-term growth. As sneaker males mature, they become satellite female mimics, but never grow to the size of the parental males. The two smaller morphs do not incur the same parental investment costs as the larger parental morphs – building a nest, defending a territory, courting females, and caring for

the eggs, a necessary condition for the hatching and survival of young. Instead, the sneaker and satellite morphs parasitize the larger males by attempting to gain fertilizations covertly.

Sneaker males have a much larger testis to body mass ratio, and also have greater sperm counts in their ejaculates. These characteristics lead to increased success in sperm competition. Fu, Neff, and Gross (2001) found that sneaker males fertilize more eggs than parental males during sperm competition, with satellite males falling between the sneaker and parental types in terms of fertilization success. Genetic analyses suggest that the mean paternity estimate for parental males is 76.9 percent, and for cuckolder males (sneakers and satellites) 23.1 percent (Neff, 2001). Although cuckolder males are more successful in fertilizing females in the context of sperm competition, the higher percentage of paternity in parental males is likely due to increased mating opportunities with females afforded by the defence and maintenance of a stable territory. Previous studies have found that cuckolders comprise approximately 21 percent of the bluegill sunfish population, suggesting that the parental and sneaker/satellite strategies have approximately the same mean fitness outcome. Even though both parental and sneaker/satellite strategies appear to offer the same fitness outcomes, the success of alternative cheater strategies is likely contingent on their frequency in the population: Modeling of bluegill populations suggests that cuckolders become less successful as their numbers increase (Gross, 1991). Bluegill sunfish are but one of several species that appear to have undergone frequency-dependent selection for stable alternative life history strategies. Alternative reproductive phenotypes have also been observed in isopods, swordtails, and ruffs (Gross, 1996).

Harpending and Sobus (1987) noted that an evolutionary explanation of psychopathy based on non-reciprocation requires that the psychopath be not only difficult to detect and highly mobile, but be “especially skilful at persuading females to copulate and at deceiving females

about his control of resources and about the likelihood of his provisioning future offspring." (p. 65S). Using contemporary terminology, psychopaths should invest highly in mating effort (energy and resources devoted to increasing mating access), and should advertise parenting effort without actually engaging in it. High mating effort, however, is a hallmark of general criminal offending, not just psychopathy (reviewed in Lalumière et al., 2005). Interestingly, of the three items that do not load onto one of the PCL-R factors, two involve mating effort (many short-term marital relationships and sexual promiscuity). Could it be that these two items do not capture the type of mating effort that is required by an obligate account of psychopathy?

Harris et al. (2007) hypothesized that, although high mating effort is associated with persistent antisocial behavior, early-onset and coercive mating effort should be particularly associated with psychopathy if it is an early-onset, obligate strategy. Harris et al. suggested that the reason these two items are orphans on the PCL-R is because they are diagnostic of antisocial behavior generally (and perhaps even other male life history strategies), not psychopathy in particular. Under the obligate, frequency-dependent selection explanation of the type discussed by Harpending and Sobus (1987), psychopathy should emerge early, and the aspects of sexuality that are diagnostic of psychopathy should not be general features associated with high adult mating effort, but functional features that develop and are expressed early.

Harris et al. tested the predictions that a factor comprising early onset and coercive sexuality items should positively correlate (in a sample of violent offenders) with the traditional PCL-R factors, should show taxonicity (i.e., evidence that scores on the factor identify types of offenders, psychopaths versus non-psychopaths, as opposed to a dimensional trait), and should also show a pattern of correlation with individual characteristics predicted by the account (e.g., negatively associated with signs of early neurodevelopmental perturbations, positively associated

with number of victims of reproductive value). All of these predictions were confirmed. These results not only clarified the unique sexuality of psychopaths, but provided support for the idea that evolutionarily informed research can improve the conceptualization and measurement of the phenomenon.

At the moment, there is some evidence that psychopathy might be the product of frequency-dependent selection. At the very least, there is evidence that psychopathy is *not* the result of early pathological conditions, such as those associated with general adult criminality. The particular structure of the definite evolutionary model of psychopathy probably remains to be elucidated, but it is clear that studies designed to test such models will continue to lead to further advances in our understanding of psychopathy. In the remainder of the chapter we discuss the relevance of, and some results from, three active lines of research for the study of the evolution of psychopathy.

Computer Simulations and Experimental Games

Models of the evolution of cooperation can help to shed light on the evolution of psychopathy. These models are also germane to the idea of different "types" of individuals (or strategies) interacting in a social environment. The Prisoner's Dilemma, a non-zero sum game, has been used to model the evolution of cooperative behavior in the face of defection (Axelrod & Hamilton, 1981; Axelrod, 1984).

In the Prisoner's Dilemma, two players are in a hypothetical situation in which both are imprisoned and accused of having colluded to commit a crime. If both players cooperate and do not implicate the other (mutual cooperation), they each receive a minimum sentence. There is, however, a greater incentive for each player to implicate the other (defection), thus earning his own complete freedom at the expense of the other's maximum sentence. If both defect and

implicate the other (mutual defection), both remain imprisoned with a long sentence. In this game, there is a small reward for mutual cooperation, a larger reward for the individual who defects (as long as the other co-operates), and costs for mutual defection. If the Prisoner's Dilemma is played only once, the optimum strategy for each player is to defect. When the same two players play repeatedly (the iterated Prisoner's Dilemma, or IPD), mutual cooperation becomes optimal.

Axelrod and Hamilton (1981) invited game theorists to submit a computer program designed to optimize success in a round-robin IPD tournament. The conditions were as follows: two players (i.e., computer program) interacted, simultaneous choices were made consisting of either cooperation or defection, the magnitude of payoffs was fixed beforehand, and the history of choices made by the other players was known to each player in the tournament. One simple strategy triumphed over all others, known as *tit-for-tat*. In this strategy, cooperation is the first move of the game, and the other player's move is copied on all subsequent moves. An ecological simulation comparing various strategies of cooperation also showed that tit-for-tat quickly became the most common or evolutionarily stable strategy in a population.

An evolutionarily stable strategy (ESS) is one that, if adopted, cannot be invaded by alternative strategies. In the context of an IPD, Axelrod and Dion (1988) demonstrated that, if the chance of future interaction is high, no player in a population can do better than to cooperate. Axelrod and Dion also suggested that even in an already established population of defectors, a small cluster of players using cooperation quickly takes over, and an established population of cooperators cannot be easily invaded (replaced) by those using defection. More recent research, however, suggests that there is no true ESS for the IPD (e.g., Marinoff, 1990). The strategy Axelrod and colleagues (1981, 1984, 1988) described as an ESS is also subject to numerous

restrictions, many of which are not ecologically tenable.

Of particular relevance to the study of psychopathy is the requirement of future interaction. The tit-for-tat model of stable cooperation put forth by Axelrod and colleagues specifically requires that players have multiple interactions, and that all interactions are remembered. Harpending and Sobus (1987) modeled a population similarly to Axelrod and colleagues, and found comparable results: If all players in a simulation have perfect memories, cooperators (engaging in tit-for-tat) always do better than individuals who always defect (cheaters). In a population where players are fallible, however, with a 10% probability of any player forgetting all encounters, it was observed that the relative frequencies of cooperators and defectors varied over time. Harpending and Sobus argued that these findings suggest that a small population of defectors could succeed if they were difficult to detect, mobile, and skilled at manipulating others, with males successfully persuading females to mate. Thus, psychopathy is analogous to the strategy of repeated defection in social interactions (perhaps after showing signs of cooperation), and so might persist at a low frequency in a population if an absolute ESS for an "always cooperate" strategy cannot be strictly maintained.

More recent research by Kurzban and Houser (2005) is consistent with the idea that there is not a single ESS for social exchanges, but rather, that there has been selection for frequency-dependent cooperative "types" in humans, exhibiting a more complex equilibrium, where the success of multiple strategies has been equal over time. Three strategies or types were identified: reciprocity contingent on cooperation by others (analogous to tit-for-tat), cooperation regardless of the actions of others, and "free-riding"—consistent defection regardless of the actions of others. Although groups comprised mostly of cooperating or reciprocating types did better than groups that included a free-rider, at the individual level, all three types experienced equivalent

average earnings. These findings are consistent with the notion of a polymorphic equilibrium where payoffs for different types or strategies are equal.

Simulations and experimental games exploring how evolution by natural selection could have given rise to subpopulations of social cheaters can inform accounts of psychopathy. It is certainly plausible that psychopathy evolved as a frequency-dependent life history strategy of defection, whereby psychopathic characteristics (manipulation, charm, dishonesty, callousness, aggression, irresponsibility, promiscuity, and a parasitic lifestyle) comprised a suite of adaptive traits and behaviors that exploited a social environment mostly characterized by cooperation.

The Genetics of Psychopathy

There are two general approaches to studying the genetics of a trait. The first is behavior genetics, the study of the relative contribution of genes and environment in explaining variance in the trait.[2] The second is molecular genetics, the study of the role of particular genes in producing the phenotypic characteristic. These two approaches are often complementary (exceptions arise when a trait shows no variance — e.g., number of fingers—but has a clear genetic basis). Although the construct of psychopathy has a long history, its valid measurement is fairly recent, and therefore there are very few genetic studies of psychopathy proper. In addition, psychopaths are very socially mobile, so it might be difficult to include family members in genetic studies. In the following, we briefly review the few studies available, but first we discuss the relevance of such studies for testing the frequency-dependent selection account of psychopathy.

The frequency-dependent selection explanation discussed above makes the clear

[2] In behavior genetics, *heritability* has a special technical meaning that is often misinterpreted. Heritability is the proportion of phenotypic variance that can be accounted for by genetic variance.

prediction that measures of psychopathy should show high heritability in behavior genetics studies, and that gene variants unique to psychopathy would be identified in molecular genetics studies. Although these predictions are straightforward, there are a few complications to consider. First, almost all psychological traits show moderate to high heritability, but few evolutionary psychologists would suggest that these traits (e.g., major personality dimensions) are the result of frequency-dependent selection. High heritability can also result from weak ancestral selection pressure on the trait in question, high mutation rates, sexual recombination, or a history of host-parasite co-evolution (see Tooby & Cosmides, 1990). At the very least, however, low heritability of psychopathy would seriously question the validity of the frequency-dependent selection account.

Second, gene variants might have low penetrance or require specific environmental triggers for expression, and so would be difficult to detect with simple genetic linkage and association studies that do not examine contextual factors. For example, Caspi et al. (2002) found an interaction between allelic variation in a gene coding for a neurotransmitter enzyme and the experience of childhood maltreatment in predicting adult antisocial tendencies. Although frequency-dependent models imply genetic differences among individuals (or morphs), it is possible that genes associated with a particular trait still require some kind of environmental cue. Perhaps psychopathy remains "dormant" unless the relevant cues are present, increasing the challenge of genetic studies.

Third, how likely is it that just one or a few gene variants are contributing to the development of something as complex as psychopathy? One might think that a multitude of genes have to be involved, making the task of finding relevant genes almost impossible because molecular genetics typically has low statistical power to detect individual genes with

small effects. Recent studies in evolutionary developmental biology, however, suggest that the task might not be as hopeless as it looks. These studies show that some master genes (e.g., hox genes) control the activity of many other genes and thereby the development of complex phenotypic features. Perhaps one or a few "psychopathy" master genes affect the expression of other genes, leading to all characteristics of psychopathy. Psychopaths, after all, look very much like exaggerated young males (except that they display risk taking, high mating effort, antisocial behavior, etc., throughout their lifetime). That is, the human genome may already have the capacity to produce all or most of the characteristic phenotypic psychopathic traits. Perhaps all that evolution required was a master gene that controls the expression of such existing traits (for an accessible and fascinating introduction to the field of evolutionary developmental biology, see Carroll, 2005).

With these considerations in mind, we examine the few genetic studies of psychopathy. It should be noted that there have been dozens of studies of antisocial tendencies, conduct problems, delinquency, and criminality, but these studies have not distinguished between psychopathy and general antisociality. These studies have typically obtained fairly high heritability estimates (reviewed in Blonigen, Carlson, Krueger, & Patrick, 2003; Lykken, 1995; Quinsey et al., 2004) but it is unclear whether, and by how much, these estimates were influenced by psychopathy. The frequency-dependent selection account would suggest that heritability estimates in these studies would be positively affected by the number of psychopaths in the samples studied.

A study of children has revealed substantial heritability for callousness and unemotionality, traits that are strongly associated with psychopathy. Viding, Blair, Moffitt, and Plomin (2005) examined a large sample of seven-year olds rated by teachers as extreme on a

callous-unemotional scale. The monozygotic co-twins of these probands scored much more similarly to the probands on the same scale than dizygotic co-twins, with an estimated heritability value of .67 and no effect of the shared environment. The heritability of a measure of antisocial conduct was also found to be high, but only if the probands scored high on the measure of callous-unemotionality. In a subsequent analysis, Viding et al. (2007) reported a substantial genetic influence overlap for callous-unemotional traits and antisocial conduct.

In a twin study of older male children (10-12) and adolescents (16-18), Taylor, Loney, Bobadilla, Iacono, and McGue (2003) examined the heritability of a self-reported psychopathy measure comprised of two related factors, antisocial and impulsive behavior, and callousness and emotional detachment. The heritability estimates varied between .36 and .54 in the older age group, and between .50 and .52 for the younger age group (our calculations). The univariate biometric analysis revealed a significant additive genetic effect and an unshared environment effect for both psychopathy factors.[3] The bivariate model suggested that the correlation between the two psychopathy factors could be attributed to additive genetic and unshared environmental effects.

In another large study of pre-adolescent twin boys and girls (9 to 10 years old), Baker et al. (2007) examined the heritability of a common factor underlying psychopathic traits, aggression, and conduct problems. Most of the variation was accounted for additive genetic effects (.96) and the remainder by unshared environment effects (.04), in both sexes.

Blonigen et al. (2003) examined the heritability of a self-report measure of psychopathic personality in a cohort of young adult male twins. Twin correlations suggested perfect

[3] Additive gene effects refer to the simple combination of gene effects at different loci (as opposed to non-additive effects, which refer to the interaction between different genes and gene dominance, among other things). Unshared environmental effects refer to environmental factors that operate to make siblings different from one another.

heritability, but the correlation for dizygotic twins was non-significantly different from zero. Biometric modelling suggested moderate non-additive genetic effects due to epistasis (interactions between genes at different loci) and a moderate effect of unshared environment. Finally, in a large study of twins aged 16-17 using a self-report questionnaire assessing three correlated psychopathic characteristics, heritability estimates were .36 to .54 for boys and .52 to .70 for girls (our calculations). All remaining variance could be attributed to unshared environmental effects (Larsson et al., 2006). Additive genetic factors accounted for 63% of the variance in the latent psychopathy factor, with the remainder attributable to unshared environmental effects (see also Larsson et al., 2007).

In sum, these five studies of psychopathic traits revealed moderate to high heritability estimates and no effect of the shared environment (see also Waldman & Rhee, 2006). These results are consistent with the frequency-dependent selection account of psychopathy and not with facultative (conditional) explanations. If any aspect of the expression of psychopathy is conditional on environmental cues, those cues remain unidentified. More work remains to be done to elucidate the gene-environmental interactions that influence psychopathy. Although these studies have used different measures specifically designed to assess psychopathy, three relied on self-report only, all used different measures, and none used samples likely to contain a high proportion of psychopaths (except perhaps for Viding et al., 2005). No study has yet examined the heritability of psychopathy using the PCL-R. We were unable to locate any research on the molecular genetics of psychopathy.

The Brains of Psychopaths

Although differential brain functioning in psychopaths is not surprising—after all, differences in behavior must be caused by some brain differences—studies that have investigated

differences between "normal" brains and psychopathic brains are useful for understanding the mechanisms underlying psychopathy. It is a common mistake in studies of psychopathy to assume evidence of brain differences (especially between clinically identified subjects and unaffected controls) to be evidence of damage, dysfunction, or pathology.

Kiehl (2006) reviewed several studies noting that damage to various components of the paralimbic system result in some symptoms and cognitive impairment associated with psychopathy. Changes in behavior associated with paralimbic damage, however, are also associated with impaired decision-making and greater reactive (but not instrumental) aggression, both of which are strongly indicative of generalized criminal behavior, but not psychopathy. There is little evidence to suggest that the very specific constellation of behaviors associated with psychopathy can be induced by any specific brain injury. The frequency-dependent selection explanation would suggest that the brains of psychopaths are necessarily different from the brains of non-psychopaths, but that this difference is not due to pathological causes. Although brain imaging studies cannot infer much about pathological causes, they can still shed light on patterns of brain functioning unique to psychopathy.

Imaging studies utilizing positron emission tomography (PET) and magnetic resonance imaging (MRI) test whether the unique pattern of behavior observed in psychopathy is due to differential anatomy or activation in the brain, as compared to "normal" populations. Several imaging studies have been conducted on violent offenders and those diagnosed with antisocial personality disorder (APD). The problem with these studies, however, is that these samples contained probable life-course persistent offenders, only some of whom were psychopaths. As we mentioned already, an evolutionary view of psychopathy would expect important differences between psychopathic and non-psychopathic life-course persistent offenders. Unless

psychopathy is diagnosed using a validated measure, such as the PCL-R, it is difficult to know whether these imaging studies reveal brain structures or functions unique to psychopathy, or structures or functions typical of violent offenders or those with APD more generally. In this section, we report on the few imaging studies conducted on psychopaths.

The paralimbic system has been identified as an important activating circuit possibly relevant to psychopathy. Damage to subcomponents of this system has been associated with an increase in some behaviors also symptomatic of psychopathy (e.g., extreme aggression, lack of empathy, general callousness), a phenomenon termed "pseudopsychopathy" (Kiehl, 2006). Behaviors similar to these have been observed in studies manipulating animal brains, as well as case studies in humans where damage has occurred as a result of head injury (for a comprehensive review, see Kiehl, 2006). Although these studies provide evidence that some clinical features that resemble psychopathic traits can be due to brain damage, it is far from clear that the kind of damage studied so far can produce the full spectrum of psychopathic traits.

In the few studies that have investigated brain functioning in true psychopathic samples, little convergent evidence has emerged to indicate whether particular brain areas or systems are implicated in psychopathy. Two studies have found differences in amygdala functioning, and one found a difference in the frontal cortex. The amygdala plays a role in aversive conditioning, instrumental learning, and general processing of emotion and fear (LeDoux, 2003; Blair, et al., 2003). Thus, differences in amygdala function could give rise to many of the characteristics of psychopathy, such as low empathy, little response to aversive stimuli, and general absence of emotional responding. Using volumetric MRI techniques, Tiihonen et al. (2000) found that high levels of psychopathy, as scored using the PCL-R, were associated with lower amygdala volume. In another study, Kiehl et al. (2001) used an emotional memory task, where participants

processed words of neutral and negative valence. Participants who scored high on the PCL-R showed reduced MRI-measured amygdala response compared to lower scoring individuals. These results are suggestive of potential differential amygdala function in psychopathy.

The frontal cortex, encompassing both the orbitofrontal and prefrontal cortices, is associated with conscious decision-making and executive control. Thus, differences in the functioning of this region could also result in some behaviors typical of psychopaths, including social behavioral problems and high aggression (Blair, 2003). Damage to the frontal cortex, however, is inconsistent with other typical psychopathic behaviors, especially instrumental violence, and thus, planning ability. Raine et al. (2000) assessed individuals who scored highly on the PCL-R using MRI, and found that there was reduced prefrontal grey matter volume, but not white matter volume. Another MRI study by Laakso et al. (2002) found no difference between psychopathic and non-psychopathic populations in total prefrontal or prefrontal white and cortical volumes. Together, these findings provide little evidence for generalized frontal cortical dysfunction in psychopaths. Blair (2003) suggested, however, that differences in one particular area of the frontal cortex—the orbitofrontal cortex (OFC)—might be consistent with lower amygdala activity in psychopathy, in that the OFC shares several neural projections to and from the amygdala. Blair also points out that parts of the OFC are associated with instrumental learning and response reversal, functions said to be different in psychopaths. Nevertheless, little empirical data exist to support the idea of *impaired* OFC functioning, and it remains unclear whether any differences in frontal cortical structure are associated with psychopathy.

Summary and Conclusions

Further understanding of psychopathy will benefit from studies aimed at refining its conceptualization and measurement. Much of the current debate focuses on the distinction

between core personality features and more easily observable antisocial and externalizing traits, and on whether psychopathy is an aggregation of multiple and distinct factors or one general constellation of traits. It is also possible that some psychopathic features are missing or incorrectly conceptualized. One version of the frequency dependent selection account of psychopathy suggests that early-onset and coercive mating effort might be a key feature of psychopathy, and this feature is not properly captured in the current version of the PCL-R, the gold standard assessment tool for offender samples. Most of the theoretical work on psychopathy is traditionally bound to medical and pathological notions and has suffered from evolutionary neglect (the neglect of consideration of ultimate causes). So far, non-pathological (adaptationist) accounts have survived empirical disconfirmations, and research in the area of experimental games of cooperation, genetics, and brain imaging are likely to provide further insights.

Many people, scientists included, find the contemplation of psychopathy especially fascinating. The idea that a minority of male criminals is engaged in a life history strategy distinct from that of most people can be hard to accept at first, however. That is, most of us would like to believe that people are all generally alike and that the worst antisocial conduct would disappear if everyone had free and equal access to the best care, opportunities, and resources. Some selectionist accounts of psychopathy suggest, however, that these beliefs might not be completely correct – perhaps most people are alike and generally interested in mutual co-operation, and this has permitted a niche for a minority, alternative, non-pathological life history strategy characterized by social defection, emotional indifference to others, interpersonal exploitation, aggression, and coercive mating effort. If so, free and equal access to resources and opportunities (and providing psychotherapy) might have little effect on the prevalence of psychopathy, which might actually be altered only by the vigilance and contingencies non-

psychopaths can bring to bear. It is somewhat curious in this context that few have noticed that phenotypes that include special skills (e.g., glibness and charm, manipulation, parasitic lifestyle) are rarely the result of pathology.

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