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Andrea Glenn – University of Pennsylvania
Robert Kurzban – University of Pennsylvania
Adrian Raine – University of Pennsylvania

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Evolutionary Theory and Psychopathy

Andrea L. Glenn[1], Robert Kurzban[1], Adrian Raine[2]

[1]Department of Psychology, University of Pennsylvania
[2]Departments of Criminology, Psychiatry, and Psychology, University of Pennsylvania

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Abstract

Psychopathy represents a unique set of personality traits including deceitfulness, lack of empathy and guilt, impulsiveness, and antisocial behavior. Most often in the literature, psychopathy is described as pathology – a disorder that has been linked to a variety of biological deficits and environmental risk factors. However, from an evolutionary perspective, psychopathy, while it could be a disorder, has been construed in the context of an adaptive strategy. In this article we will examine the strengths and weaknesses of two models suggesting that psychopathy is an adaptive strategy, and one model suggesting that it is a form of pathology resulting from accumulated mutations. Overall, we do not find that there is strong enough evidence to draw firm conclusions about one theory over another, but we highlight some areas where future research may be able to shed light on the issue.

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1. Introduction

In this paper, we review research and theory on the origins of psychopathy. We begin by describing psychopathy and suggest that, despite appearances, it might be an adaptation rather than pathology. In this context, two overarching theories have been proposed – balancing selection and contingent shifts. Balancing selection suggests that psychopathic traits have been selected for because they offer a fitness advantage in specific environments. The contingent shifts model suggests that psychopathic traits are an adaptive response to specific environmental conditions. Next we turn to an

alternative hypothesis, that psychopathy is a result of deleterious mutations. In light of growing interest in the application of evolutionary models to individual differences and psychological disorders (Buss, 2009), the goal of the present paper is to provide an up-to-date perspective on how these models may apply to psychopathy.

1.1. Description of Psychopathy

Psychopathy is a personality type describing individuals who demonstrate a pronounced lack of guilt, remorse, and empathic concern for others. Psychopaths appear to lack emotional distress and are impervious to distress in others. In addition, they are superficially charming, manipulative, egocentric, and grandiose. They tend to be impulsive, risk-taking, and fail to plan for the future. They also demonstrate antisocial behavior and poor behavioral control. Individuals with psychopathy are unique in that they demonstrate an increased risk for both instrumental (i.e., predatory, goal-driven) and reactive aggression (Cornell et al., 1996). In this paper, we will focus on research on psychopathic traits specifically, rather than studies that examine antisocial behavior more generally.

As with other individual differences (Bouchard & Loehlin, 2001), psychopathy has a substantial heritable component of about 50% (Blonigen, Carlson, Krueger, & Patrick, 2003; Larsson, Andershed, & Lichtenstein, 2006) and is relatively stable over the course of a lifetime (Loney, Taylor, Butler, & Iacono, 2007). Although psychopathy is often described as a "constellation" of traits, there is growing evidence that psychopathy represents a unified disorder. Behavioral genetics research has shown that genetic influences contribute to the different features of psychopathy (Larsson et al., 2006; Taylor, Loney, Bobadilla, Iacono, & McGue, 2003). These studies found sizable genetic correlations between different factors of psychopathy (Taylor et al.: $r = .74$ between callous/unemotional and impulsive/antisocial factors; Larsson et al.: correlations ranging from .59 to .78 for the grandiose/manipulative, callous/unemotional, and impulsive/irresponsible factors). Furthermore, Larsson et al. (2006) found that the different facets of psychopathy covary with a latent overall psychopathy factor, which is also substantially influenced by genes (Larsson et al., 2006). Although these studies also found that genetic effects uniquely influence different facets of psychopathy, these findings indicate that these facets are linked to a highly heritable psychopathic personality factor, suggesting that psychopathy is a unified construct.

1.2. Dimension versus category

Although there continues to be debate, research to date suggests that psychopathic traits exist on a continuum in the population as opposed to representing a distinct subtype of individuals. This appears to be true regardless of the measure used to assess psychopathy, or whether the sample is incarcerated or not (Edens, Marcus, Lilienfeld, & Poythress, 2006; Guay, Ruscio, Knight, & Hare, 2007; Hare, 2003; Walters, Brinkley, Magaletta, & Diamond, 2008). Thus, a "psychopath" as we think of him/her likely represents the extreme end of the continuum of symptom severity.

An arbitrary threshold has been set by researchers to classify individuals at the extreme end of this dimension who demonstrate characteristics of the prototypical psychopath. The proportion of individuals in the general population who meet this threshold is about one percent (Hare, 2003). However, this threshold has been established for practical purposes and does not indicate that individuals above the threshold are qualitatively different.

1.3. Is psychopathy harmful to the individual?

Might the features of psychopathy coherently be understood as a social strategy that, in ancestral environments, contributed to, rather than detracted from, reproductive success, and was selected for in virtue of the benefits from implementing the strategy (Murphy & Stich, 2000)? Consider, for instance, the historical view of postpartum depression as a disorder. While we take no particular position on whether it is or not, Hagen (1999) suggested that postpartum depression might be a functional response to inadequate social support from fathers and/or other family members, viewing it as a strategy to elicit additional support from those with a stake in the newborn. This distinction reflects a similar approach to pathology outside the psychological realm; fever, for example, while often viewed as a pathology, seems to be functional in some cases, part of the evolved pathogen-defense system (Nesse & Williams, 1994).

So, whereas some mental disorders such as schizophrenia are clearer cases of pathology rather than adaptation (Cosmides & Tooby, 2000), and result in clear reductions in fertility in modern environments (Haukka, Suvisaari, & Lonnqvist, 2003; MacCabe, Koupil, & Leon, 2009), this issue is not clear with respect to psychopathy insofar as many of the traits of psychopathy have both costs and benefits associated with them. Although we tend to think of traits such as a lack of guilt as a deficit, these qualities may not reflect pathology. Instead, a lack of guilt may – under, we stress, the proper circumstances – make it easier for an individual to obtain resources from others, with little deterrence from his or her own emotional state. In laboratory settings, for instance, guilt has been shown to cause people to choose altruistic actions that lead to worse financial outcomes for the self (Ketelaar & Au, 2003).

To further complicate the issue of whether psychopathy is harmful to the individual, the categorization of psychopathy is not entirely homogenous. A variety of distinctions have been made between different types of

psychopaths, one of which is a distinction between "successful" and "unsuccessful" psychopaths. As the titles imply, some individuals with psychopathic traits may be repeatedly incarcerated for a variety of crimes and extremely risk-taking – generally "unsuccessful" in a variety of areas. Other individuals with psychopathic traits may be able to con their way to the top of a company, gaining status and resources with little effort – generally "successful" (Babiak & Hare, 2006). In the "unsuccessful" case, the individual may be less able to gain status, social alliances, and resources if the individual serves long prison sentences (e.g., is exiled from the group) or if he dies at an early age from personal injury due to excessive risk-taking. In the "successful" case, however, the individual may be able to gain these things with little actual investment of his own resources. Thus, in some cases psychopathic traits could be viewed as debilitating and harmful to the individual, whereas in other cases these traits may actually benefit the individual. This makes it plausible that psychopathy might be a strategy rather than pathology (Murphy & Stich, 2000).

2. Theories based on adaptationist analysis

In this section, we review theories of individual differences from an evolutionary perspective, with particular attention to how these theories might apply in the context of psychopathy. Generally, it has been suggested that individual differences are important to the vast majority of social adaptive problems (Buss, 2009). Buss (2009) provides the example of selecting a mate, where only differences between individuals matter in the selection process (e.g., attractiveness, intelligence, dependability, health, agreeableness, ambition, empathy, etc.).

First, in order to understand theories based on adaptationist analysis related to psychopathy, we review the concept of life history strategies. Life History (LH) theory describes tradeoffs that must be made due to limited time and energy budgets. Effort allocated to solving one adaptive problem, for the most part, precludes effort allocated to other adaptive problems. The major predicted LH tradeoffs are: (1) *somatic effort* (resources devoted to continued survival) versus *reproductive effort* (resources devoted to producing offspring), (2) *parental effort* versus *mating effort*, (3) *quality* versus *quantity* of offspring, and (4) *future* versus *present* reproduction (Kaplan & Gangestad, 2005). For example, parental versus mating effort involves tradeoffs in which energy can be allocated toward the process of attracting and retaining a mate long enough for successful conception, or toward parenting and other forms of kin investment. Although previously used to account for differences among species, LH theory has proven useful in understanding individual differences in human behavior as well (Buss, 2009).

Individuals have been found to vary on the relative amount of resource investment along each of the four dimensions. For the most part, these dimensions are related to (i.e., correlated with) each other, so that each individual has a coordinated overall "life history strategy" (Figueredo et al., 2006). In theory, all individuals could be placed along a single continuum of LH strategies, often referred to as the "slow" to "fast" continuum (Gladden, Welch, Figueredo, & Jacobs, 2009b). Organisms that have early sexual maturation and reproduction, produce a large number of offspring, and invest little in parental care are said to have a "fast" life history strategy. Organisms that develop late, delay reproduction, have fewer offspring, and invest heavily in parental care are said to have a "slow" life history strategy.

LH theory predicts that personality traits that facilitate a coordinated fast or slow life history strategy tend to be selected together and, therefore, co-occur (Figueredo et al., 2006; Gladden, Figueredo, & Jacobs, 2009a). In general, a slow life history strategy is associated with secure attachments, supportive communication, support and contact with family and friends, a psychological disposition for long-term planning, and long-term mating effort. In contrast, a fast life history strategy is associated with less focus on planning for the future, short-term mating effort, increased risk-taking, reduced self-control, and a selfish disposition.

Several authors have argued that many of the features of psychopathy represent characteristics of a "fast" life-history strategy (Barr & Quinsey, 2004; Mealey, 1995). Jonason et al. (2010a) found that psychopathic traits were correlated with scores on the Mini-K, a 20-item measure of items thought to be associated with life history strategy (Figueredo et al., 2006), including risk-taking, planning, relationship closeness, social contact, and religiosity. In this study, psychopathy was also positively correlated with a number of risk-taking behaviors, including number of sexual partners, illegal drug use, cigarettes smoked per day, and alcohol consumption. Men scoring higher on psychopathic traits have also been found to engage in more short-term mating behaviors, interest in seeking a short-term mate, sociosexual attitudes, and have a higher number of sexual partners (Jonason, Li, Webster, & Schmitt, 2009).

It has also been suggested that the observed sex differences in psychopathy (more common in males) may reflect sex differences in LH strategies. Because the costs and benefits associated with LH tradeoffs are not the same for males and females, life history theory predicts that there will be sex differences in LH strategies; the sex required to invest more bioenergetic and material resources in offspring will have a relatively slower life history strategy (Figueredo et al., 2006). Because women are biologically obligated to invest more in their offspring than men are, this would suggest that men would be more likely to have a fast LH strategy than women, a prediction for which there is partial support (Jonason et al., 2010a). The large sex differences observed in psychopathy may therefore correspond to differences between men and women in LH strategies.

However, in a factor analysis of items from measures of life-history strategy, psychopathic traits, risk-taking, and mating effort, Gladden et al. (2009a) found that life-history strategy items loaded on one factor, and that psychopathy, risk-taking, and mating effort loaded on a separate factor; the correlation between these two factors was relatively small (-.14). Thus, although psychopathy was associated with short-term mating and risk-taking, it did not appear to be related to other characteristics of a fast LH strategy.

The authors discuss possible domains where psychopathy may be distinguishable from a fast LH strategy. One potential area where psychopathy might not correspond to what is predicted by a fast LH strategy is moral emotions. LH theory suggests that slow LH strategies are favored in stable and predictable environments that signal that investment in somatic effort, parental effort, and the future is likely to result in fitness payoffs. In the social environment, slow LH individuals would require more social stability and social order than fast LH individuals for their strategy to be optimal. Thus, Rushton (1985) predicted that slow LH individuals would exhibit increased group altruism, social organization, and social and moral rule following. Further, in order to promote and maintain social stability and order, slow LH individuals would need to encourage others to follow social and moral rules (Weeden, Cohen, & Kenrick, 2008). Strong moral intuitions (i.e., moral emotions such as anger, contempt, and disgust) could serve to encourage others to follow social rules (Rozin, Lowery, Imada, & Haidt, 1999; Wheatley & Haidt, 2005). Moral intuitions would encourage individuals to punish or deter free-riders that violated social rules, thus enhancing long-term survival prospects and facilitating cooperation in groups.

Recently, Gladden et al. (2009b) demonstrated that a slow LH strategy is indeed associated stronger moral intuitions in the domains of anger, contempt, and disgust; fast LH strategies were associated with weaker moral intuitions. However, individuals with psychopathic traits have not been found to have weaker moral intuitions in these domains. Although psychopathic individuals have deficits in empathy, an emotion that motivates prosocial behavior, there is not much evidence to suggest that they have deficits in the domain of moralistic emotions such as anger, contempt, and disgust (i.e., responding to others' moral infractions). A recent examination of the relationship between psychopathy and five domains of morality suggested that psychopathy is associated with reduced empathic concern for others, affecting concerns about harming others and fairness (i.e., prosocial emotions); however, psychopathy was slightly positively associated with concerns about loyalty or betraying ingroup members and purity, and was not associated with respect for authority or disgust (Glenn, Iyer, Graham, Koleva, & Haidt, 2009). Although more research needs to be done to determine the relationships between psychopathy and these types of moral intuitions, evidence thus far appears to be weak or mixed (Blair, Colledge, Murray, & Mitchell, 2001; Kosson, Suchy, Mayer, & Libby, 2002). Thus, moral emotions beyond empathy may indeed be an area where psychopathy may be distinct from a fast LH strategy (Gladden et al., 2009a).

Overall, there does appear to be significant overlap between many aspects of psychopathy and the predicted characteristics of a fast life history strategy, although there may also be some differences. In the following sections, we review two theories based on adaptationist analysis that suggest that psychopathy represents an alternative strategy (a fast LH strategy) that can be beneficial in some contexts.

2.1. Balancing Selection

One theory that has been frequently suggested to be applicable to psychopathy is balancing selection. Balancing selection occurs when genetic variation is maintained by selection, such that different levels on a trait dimension are favored, or are adaptive, in different environmental conditions (Buss, 2009). The two most relevant forms of balancing selection for personality are environmental heterogeneity in fitness optima and frequency dependent selection.

2.1.1. Environmental heterogeneity in Fitness Optima

This type of balancing selection suggests that since selection pressures vary over time and space, then selection can favor different levels of a personality trait in different environments (Buss, 2009). For example, in some environments, the expected value of the benefits of psychopathic traits may outweigh the costs. However, in other environments, the benefits of psychopathic traits may be much less than the costs. Evidence suggesting that psychopathic traits are more prevalent in specific environments would provide some support for the idea that psychopathy is an adaptive strategy that may be beneficial in some environments.

Unfortunately, cross-cultural studies in psychopathy (i.e., examining psychopathic traits in different environments) are still in their infancy and have primarily been conducted in Western, developed cultures. Several studies have indicated somewhat lower rates of psychopathy in European compared to North American samples (Cooke & Michie, 1999; Dahle, 2006). However, the origin of these differences remain unclear (Wernke & Huss, 2008). Observed differences across cultures may indicate that environmental cues influence the expression of traits. For example, one possibility is that features of contemporary modern environments, such as relative anonymity, evoke psychopathic traits. In modern large-scale societies, many interactions may be one-time encounters, such that individuals may implement strategies of deceit and manipulation with little risk of developing a reputation as a cheater; it is also considerably easier

for individuals to move to other locations where individuals do not know them. Further ethnographic research on psychopathy may shed light on whether psychopathic traits are less beneficial in small-scale societies where individuals have more repeated interactions.

In the future, molecular genetics studies may also be able to shed light on whether psychopathic traits have been favored in particular environments. Studies demonstrating that genes associated with personality traits such as psychopathy may be more advantageous in particular environments (Buss, 2009) would suggest that psychopathic traits can be adaptive. For example, researchers have found that an allele of the DRD4 gene, which has been associated with novelty seeking and extraversion (Ebstein, 2006), is more prevalent in migratory populations than sedentary populations (Chen, Burton, Greenberger, & Dmitrieva, 1999). Evidence suggest that this allele of the DRD4 gene may be more advantageous to nomadic populations (Eisenberg, Campbell, Gray, & Sorenson, 2008) and therefore has a higher prevalence. Although only a few molecular genetics studies have been conducted in psychopathy (e.g., Sadeh et al., 2010), we may find that some of the genes that are associated with it are more prevalent in environments where it confers more of a fitness advantage. This would provide support for the idea that psychopathy represents an adaptive strategy rather than a result of random mutations.

2.1.2. Frequency-dependent Selection

This type of balancing selection occurs when two or more strategies are maintained within a population at a particular frequency relative to each other. In some cases, an allele's fitness effects may increase as it becomes rarer. For example, in some species of swordtail fish, there are genetically-influenced alternative male phenotypes. Most of the fish develop large body sizes and actively court females and defend their territories. A subset of the fish are smaller, meaning that they are less successful in male-male competition and are less attractive to the females; however, these males are able to obtain matings through sneak copulations, and can be successful with this strategy as long as they remain at a low frequency in the population (Ryan & Causey, 1989).

This is one of the types of selection that several have hypothesized to explain psychopathy (Barr & Quinsey, 2004; Mealey, 1995; Murphy & Stich, 2000; Raine, 1993). In an environment in which the majority of people adopt a strategy of cooperation, a small number of individuals may be able to maintain an exploitative, socially parasitic strategy. The strategy can bring high fitness benefits when rare, but becomes less rewarding at higher frequencies because of anti-cheater vigilance in the population and because of the increased probability that a cheater will encounter another cheater. Although psychopathic traits are thought to exist on a continuum, approximately 1% of the general population is thought to be highly psychopathic, suggesting that at this low frequency it may be advantageous. Frequency-dependent selection may be a more likely model for psychopathy than for other mental disorders because there are plausible explanations for why the fitness of the alleles associated with psychopathy would increase as their frequency decreases.

One of the challenges of making the case for frequency dependent selection as an explanation for psychopathy is to demonstrate that the costs of psychopathic traits are sufficiently balanced by significant beneficial effects given plausible assumptions about the range of ancestral environments. We have attempted to define some of the cost and benefits that may be associated with the various traits of psychopathy in Table 1.

More direct testing of the costs and benefits of psychopathic traits has been done in the field of mating psychology. Jonason et al. (2010b) found that psychopathy was related to a higher overall incidence and success rates for poaching the mates of others – a likely benefit. It was also associated with higher rates of being poached by others, which may be a benefit in terms of mating opportunity, but may also be costly if it means less support for existing offspring. Finally, the study found that psychopathy was associated with increased risk of having one's own mates poached; this included in short-term relationships, long-term affairs, and long-term relationships. Thus, although psychopathy may facilitate some components of reproductive success such as access to a variety of partners, it also carries costs such as losing mates previously acquired.

2.2 Contingent Shifts

2.2.1 Contingent shifts in Response to the Environment

Psychological mechanisms are designed to respond flexibly to changes in the environment or to one's own characteristics (Buss, 2009), a property sometimes referred to as *contingent shifts* or *conditional adaptation*. Flexible responding is a property of large numbers of physiological and psychological mechanisms. The deployment of the immune system against disease is one example.

TABLE 1. Hypothesized Benefits and Costs of Specific Psychopathic Traits

Trait	Benefits	Costs
Promiscuous sexual behavior / many short-term marital relationships	Mating success	Lack of family stability; poor investment in offspring
Deception / feigned emotions	Ability to gain resources; ability to attract mates, at least short-term, via deception about ability to acquire resources or long-term parenting intentions; hierarchy negotiation	Exile from group (formal incarceration or socially sanctioned); Lack of access to shared resources; lack of harmonious interpersonal relationships; punishment
Coercion	Ability to gain resources; access to mates (e.g., rape); ability to poach mates of others	Exile from group (formal incarceration or socially sanctioned); Lack of access to shared resources; punishment
Glibness, superficial charm	Social allies; attractiveness to mates; hierarchy negotiation; initiation of short-term relationships; ability to poach mates of others	Reduced ability to establish long-term relationships
Impulsivity	Ability to take advantage of immediate opportunities	Poor long-term planning and decision-making
Fearless	Exploration of environment; resilience to stress and depression (and related health consequences)	Physical risks; reduced life expectancy
Unempathic / shallow emotions	Resilience to stress and depression (and related health consequences); unrestrained ability to take advantage of others; lack of neuroticism and anxiety, which may facilitate the pursuit of one's goals through adverse conditions; ability to abandon parental responsibility	Lack of long-term alliances; potential exile from group; poor nurturing of offspring (leading to reduced survival)
Short-term jobs/relationships	Ability to acquire resources and escape detection	Loss of gains from long term associations
Reactive aggression	Punishment of individuals who stand in their way	Lack of harmonious interpersonal relationships; punishment; physical risks; reduced life expectancy
Instrumental (goal-driven) aggression	Ability to gain resources from others	Lack of harmonious interpersonal relationships; punishment; physical risks; reduced life expectancy

Such changes might occur at very high levels of abstraction. Examples of contingent shifts in response to the environment in the context of evolved psychological adaptations could be a shift to a more risk-averse strategy after becoming a father, or more risk-taking during times of famine, when more dangerous action may be required to obtain food (Buss, 2009; Stephens & John, 1986). Contingent shifts may also occur at early stages in life, such that an organism modifies its developmental trajectory (and resulting phenotype) to fit the local conditions of the social and physical environment (Del Giudice, Ellis, & Shirtcliff, in press; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2011). Like balancing selection, according to this model, individual variation in psychological mechanisms is primarily viewed as the result of adaptive mechanisms, rather than the outcome of pathological or dysfunctional processes. However, unlike balancing selection, contingent shifts suggest that individuals are evolved to survive and reproduce in a variety of contexts, and therefore systems develop to be responsive to environmental conditions. The tailoring of systems in response to the environment results in individual differences in life history strategies.

Evidence for these arguments rely on identifying systematic relationships between environmental features and the phenotypic response. For example, Gao et al. (2010) found that poor parental bonding (lack of maternal care and low paternal overprotection) and childhood physical abuse (measured retrospectively via self-report) were associated with increased psychopathy scores in adulthood. Furthermore, a small sample of children who were separated from their parents in the first three years of life were significantly more psychopathic at age 28 than children who were not separated

from their parents (Gao et al., 2010). In other studies, childhood abuse and neglect has consistently been found to be associated with psychopathy in psychopathic prisoners compared to non-psychopathic prisoners (Marshall & Cooke, 1999), in substance abusing adolescents (O'Neill, Lidz, & Heilbrun, 2003) and adults (Bernstein, Stein, & Handelsman, 1998), in delinquent boys (Krischer & Sevecke, 2008), and in a community sample (Lang, af Klinteberg, & Alm, 2002). Prospectively, victims of childhood abuse and neglect have been found to exhibit significantly higher psychopathy scores in adulthood than controls who have not been abused (Weiler & Widom, 1996).

However, studies of early environmental influences also have several limitations. Retrospective studies are faced with the problem that psychopathic traits in adulthood may affect self-reports of childhood experiences. Both prospective and retrospective studies of the effects of childhood abuse are also potentially confounded by genetic factors (i.e., that parents with a predisposition for abuse may pass on more antisocial "risk" genes). Future studies will be necessary to clarify the degree to which early environmental influences affect the development of psychopathic traits. It appears that early trauma is one environmental factor that an individual may respond to, and it is plausible that some individuals may adapt by shifting toward a faster life history strategy.

However, it is also worth noting that findings of environmental influences on the development of psychopathy are also compatible with the idea of psychopathy as dysfunction or pathology (i.e., exposure to stress and adversity derails normal development resulting in altered biology and behavior; see Section 3) and are not direct evidence of conditional adaption. The conditional adaptation model suggests that stressful environments do not disturb normal development, but direct it toward strategies that are adaptive under stressful conditions (Del Giudice et al., in press).

In determining whether the conditional adaptation model may apply to psychopathy, it is important to consider whether there is a plausible mechanism that could facilitate a shift toward a different life history strategy. One mechanism by which this shift may occur is via alterations in the stress response system. A recent model of conditional adaptation has been proposed to explain individual differences in stress responsivity. The Adaptive Calibration Model suggests that one of the roles of the stress response system (in addition to responding to immediate challenges) is to use information from the environment to modify an individual's developmental trajectory (i.e., life history strategy) to match the local conditions of the social and physical environment. Information about resource availability, extrinsic morbidity-mortality, and unpredictability in the environment is detected by the stress response system, which then acts as an integrative mechanism, facilitating the development of alternative LH strategies that are adaptive in different environmental conditions (Del Giudice et al., in press).

As mentioned at the beginning of Section 2, individual variation in the different life history dimensions tend to be correlated (Figueredo et al., 2006). The Adaptive Calibration Model argues that the stress response system serves as a mechanism for coordinating life-history relevant traits and behaviors. The stress response system has been found to contribute to a wide range of LH-related traits – from sexual maturation and fertility to risk-taking and parenting styles. Stress response system indices have been associated with individual differences in competitive risk-taking, learning, self-regulation, attachment, affiliation, and reproductive functioning (Del Giudice et al., in press). Thus, this system can dictate many of the components of the individual's life history strategy.

Low stress responsivity has consistently been identified in the psychopathy literature, making it a plausible mechanism for the conditional adaptation model. In adults, Cima et al. (2008) reported that psychopathic offenders showed lower levels of the stress hormone cortisol than nonpsychopathic offenders. In undergraduates, O'Leary et al (2007) found that males scoring higher in psychopathy showed less cortisol reactivity to a social stressor than lower-scoring individuals. In general, studies have shown that adult psychopathic, relative to nonpsychopathic, offenders tend to be electrodermally less responsive both when anticipating and reacting to aversive stimuli (Arnett, 1997; Lorber, 2004; Raine, 1993). Another line of research has focused on an abnormal startle reflex response in the context of emotional stimuli in psychopaths. In control subjects, presentation of pleasant stimuli is found to attenuate and unpleasant stimuli to potentiate the startle response, compared with presentation of neutral stimuli (Vrana, Spence, & Lang, 1988). Psychopathic individuals fail to show potentiation of the startle blink when presented with unpleasant (for example, fearful) stimuli (Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002; Patrick, 1994; Patrick, Bradley, & Lang, 1993), indicating reduced responsivity to stress in psychopaths. Thus, it is plausible that early environmental factors may act via the stress response system to shift some individuals toward a faster life history strategy, facilitating the emergence of psychopathic traits.

Another important factor for the conditional adaptation model is demonstrating that the biological and behavioral changes that occur in response to the environment provide benefits to the individual. This type of evidence would distinguish conditional adaptation from the idea of psychopathy as pathology.

With respect to stress responsivity, Del Giudice et al. (in press) highlight the extensive work of Michael Meaney and colleagues, who have shown that relatively low quality maternal care in the rat alters pups' stress physiology and brain morphology, including higher stress hormone levels, shorter dendritic branch lengths, and lower spine density in hippocampal neurons. Although these changes seem disadvantageous, they actually enhance learning and memory

processes under stressful conditions (Bagot et al., 2009). Furthermore, the physiological changes mediate the effects of maternal behavior on defensive and reproductive strategies (Cameron et al., 2008). Thus, it appears that alterations to stress physiology can be strategic ways of developing under different rearing conditions.

In addition to being adaptive in stressful contexts, low stress responsivity may help individuals to maintain calm and vigilance during aggressive encounters. Del Guidice et al. (in press) argue that a calm demeanor can also work as a signaling handicap; by showing that one is not preparing for immediate physical response, an individual sends opponents a credible signal that he/she is not scared, and is ready to withstand an attack. Other possible advantages associated with low stress responsivity may be that the individual is more likely to take risks, which may be advantageous in stressful environments. Low stress responsivity may also mean that the individual is more resilient to negative physical and mental health outcomes in stressful environments. Del Giudice et al. (in press) also argue that insensitivity to social feedback and the social context may enable the individual to adopt and exploitative interpersonal style by shielding the individual from social rejection, disapproval, and feelings of shame. Thus, it is possible that low stress responsivity could be fitness-maximizing in high-risk environments, although it is worth noting that this is a tradeoff that also has significant costs associated with it.

One final aspect of the contingent shifts model is the issue of sex differences. Continent shift models acknowledge that there are sex differences in life history related dimensions, with females employing a slower LH strategy. It is argued that the extent of sex differences in LH-related behavior is not fixed, but depends on environmental factors. In stable environments, both sexes may benefit from engaging in high parental investment, resulting in relatively slow LH strategies for men. As the environment becomes more stressful, males may benefit by shifting to faster LH strategies, whereas females do not have the same flexibility to do so, as high parental investment is required. Thus, the contingent shifts model suggests that sex differences in stress responsivitity (i.e., low stress responsivity being a male-biased phenomenon) result from environmental factors that primarily affect the developmental trajectory of males. These changes in the trajectory may be facilitated by sex-specific hormones such as androgens (Del Giudice et al., in press).

2.2.2 Contingent Shifts in Response to Phenotypic Characteristics

Another type of contingent shift is in response to one's own heritable phenotypic characteristics, which has been referred to as "reactive heritability" (Tooby & Cosmides, 1990). In terms of life history strategy, the optimal tradeoff between different allocations of time and energy are likely to depend on variables such as one's own qualities, both physical and psychological (Buss, 2009). For example, males with larger body size at age 3 have been found to be less agreeable and more aggressive at age 11 (Ishikawa, Raine, Lencz, Bihrle, & Lacasse, 2001). Buss (2009) hypothesized that those with a larger physical size adopt an aggressive strategy because it is easier for them to pursue this strategy effectively compared to smaller children. With respect to psychopathic traits, one hypothesis is that some of the characteristics develop in response to the heritable phenotypic characteristic of reduced emotional responsivity. In a recent 25-year longitudinal study, individuals with a greater degree of psychopathic traits at age 28 were found to demonstrate less fearfulness at age 3 (Glenn, Raine, Venables, & Mednick, 2007). Thus, it may be that children who are fearless find that they can more easily adopt strategies that involve deception, rule-breaking, or risk-taking, as they have less fear of being detected, punished, or of negative consequences. It is important to note that since the phenotypic characteristics such as body size or temperament are heritable, then the traits that condition on them, such as aggression, will appear to be as well.

In essence, contingent shift model recognizes the fact that some amount of individual variation in life history strategies is attributable to genetic predispositions. The model suggests that the individual responds to both environmental influences and one's own genetic predispositions in determining a life history strategy.

2.3. Balancing Selection versus Contingent Shifts

Balancing selection, which suggests that psychopathic traits may be beneficial at a low frequency in the population, has been the evolutionary theory most commonly applied to psychopathy. However, the feasibility of this explanation for psychiatric disorders has been challenged (Keller & Miller, 2006b). The main reason is that the process of natural selection tends to minimize fitness relevant genetic variation rather than maintaining it, as proposed by balancing selection; alleles with even small negative effects on fitness tend to go to a frequency of zero fairly quickly (*fundamental theorem of natural selection*; Fisher, 1930/1999). In order for genetic variation in psychological traits to be maintained, the variant would need to be either adaptively neutral, such that it does not affect fitness in any way, or adaptively maintained by balancing selection, as argued by Mealey (1995). This would require that the alternative alleles underlying a trait's heritable variation have net fitness effects that are exactly equal to each other when averaged across evolutionary time and ancestral environments – a fairly stringent requirement (Keller & Miller, 2006b). However, it may be difficult to determine how common or uncommon it is for alternative alleles to be maintained.

Another argument against balancing selection as a model applicable to psychopathy is that evolutionary theory and research has come to acknowledge that evolution rarely produces one or two fixed strategies for survival and reproduction. It is argued that both stressful and supportive environments have been part of the human experience throughout evolutionary history, and thus our developmental systems have been shaped by natural selection to respond more flexibly to changes in the environment (Del Giudice et al., in press). With respect to life history strategies, balancing selection tends to view life history traits and strategies as more static and genetically determined, whereas contingent shifts models suggest that life history strategies evolve to show developmental plasticity (West-Eberhard, 2003).
The idea that there are two distinct, competing life history strategies may be less likely than the idea of universal systems that can flexibly respond. Indeed, many of the traits of psychopathy reflect capacities that are actually *universal* features of human nature (e.g., deception, risk-taking, aggression), that are affected by universal suites of genes and may be triggered by adverse situations (Keller & Miller, 2006b). For example, a significant amount of research in social psychology has been dedicated to exploring the situations in which "normal" individuals can be driven to aggressive, greedy, deceitful, and generally psychopathic-like behavior (e.g., Hanley, Banks, & Zimbardo, 1973; Milgram, 1963).

This is not to say that there is not also genetic variation that contributes to individual differences in these traits. However, some suggest it is more likely that natural selection would favor behavioral flexibility rather than fixed strategies, such that different behavioral strategies could be pursued by the same individual across different situations. This flexibility would minimize the costs of pursuing a fixed strategy in an environment which is not conducive to that strategy (Keller & Miller, 2006b). A challenge for the contingent shifts model, however, is the idea that some individuals appear to develop psychopathic traits without any indicators of environmental risk factors (e.g., Raine, Stoddard, Bihrle, & Buchsbaum, 1998). More extensive research on environmental factors may be able to determine whether such individuals exist, although this would be difficult to determine definitively. Overall, data suggest that there is heritable variation that contributes to psychopathic traits, but also that environmental factors can have systematic effects, so it may be difficult to determine whether the balancing selection or contingent shift model is more likely. Another hypothesis, discussed next, is that these two models may be compatible.

2.4 Compatibility of Balancing Selection and Contingent Shifts

Conditional adaptation models, such as the Adaptive Calibration Model (ACM) of stress responsivity, can be compatible with balancing selection. The ACM postulates that individual differences in stress responsivity are "largely (though not exclusively) the result of conditional adaptation" and acknowledges that widespread allelic variation exists in many genes that can affect the functioning of the stress response system (Del Giudice et al., in press). This suggests that *within* an individual, both genes and adaptive responding to the environment influence the functioning of the system. The authors also suggest that different developmental pathways may lead to low stress responsivity. In one pathway, an individual with normal stress responsivity may shift toward unresponsivity following chronic, severe stress (conditional adaptation). In the other pathway, unresponsivity may develop even in stable environments because of a strong genetic predisposition. Thus, the conditional adaptation model may apply to some individuals but not others (Del Giudice et al., in press). The ACM does not speculate about the source of this genetic variation (i.e., whether it results from mutation or something like frequency dependent selection).

The idea that different individuals may develop low stress responsivity as a result of different developmental pathways is similar to the model proposed by Mealey (1995), which appears to combine balancing selection and contingent shifts into a model of psychopathy. Mealey suggests that there are two basic categories of psychopaths. The first are "designed" for the successful execution of social deception and are the product of evolutionary pressures; that is, independent of the environmental factors that influence them throughout development, these individuals predominantly pursue a life strategy of manipulative and predatory social interactions. This relatively small percentage of individuals at the extreme end of the continuum of psychopathic traits is fixed. She describes this group as the outcome of frequency-dependent selection. On the other hand, the second group reflects a variable percentage of individuals who are less extreme on the continuum, and who sometimes, in response to environmental conditions during their early development, pursue a psychopathic life strategy. Genetically-based individual differences determine individuals' response to the environment resulting in the pursuit of either cooperative of deceptive social strategies. Thus, this second group proposed by Mealey appears to be largely predicated on the concept of conditional adaptation.

Both Mealey (1995) and DelGiudice et al. (in press) suggest that two distinct groups exist – one in which the trait develops largely independent of the environment, and another in which the trait develops largely depending on environmental conditions. It may be difficult to determine whether such a distinction exists or not. Evidence from one brain imaging study may shed some light on the issue, although the study focused on violence rather than psychopathic traits. In a sample of violent individuals, Raine et al. (1998) found that deficits in the frontal lobe of the brain were particularly pronounced in individuals who had not been exposed to significant social stressors. Murderers from nondeprived home backgrounds showed a 14.2% reduction in functioning in the right orbitofrontal cortex relative to

murderers from deprived home backgrounds characterized by abuse, neglect, and marital violence (Raine et al., 1998). This suggests that some individuals may become violent in the absence of any apparent environmental factors; violent behavior in these individuals may result primarily from genetically driven differences in brain functioning. Other individuals may become violent primarily as a result of environmental factors rather than genetically-based factors such as brain functioning. Additional research will be needed to determine whether this pattern of findings is observed in individuals with psychopathic traits, and if there is reason to distinguish between two groups of psychopathic individuals based on etiological factors.

In sum, adaptationist models suggest that psychopathy represents an alternative life history strategy – one which focuses on mating versus parental effort and an emphasis on reaping short term rather than long term benefits. Frequency dependent selection suggests that this type of social strategy is adaptive in at a low frequency in the population, and that evolution has therefore maintained the alleles associated with it. The contingent shift model suggests that individuals flexibly respond to the environment and that psychopathic traits (a fast LH strategy) may be beneficial in some environments. The stress response system is one mechanism that has been shown to be sensitive to environmental conditions and may facilitate such changes in life history strategy. It is not clear from the current research available that one model is more likely than the other.

3. Mutation load / polygenic mutation-selection balance

An alternative hypothesis to adaptionist models is that psychopathy represents dysfunction and is a result of mutations. All humans carry mutations, some of which are new, but most of which are inherited from ancestors and may be maintained through many generations (Keller & Miller, 2006b). Although mutations with highly harmful effects will be removed quickly from the gene pool, those with only mildly harmful effects can take many generations to be removed. This results in an accumulation of old mutations and is a source of genetic variation between individuals. Individuals vary in the number and type of mutations they carry (mutation load).This source of individual differences is hypothesized to contribute to individual differences in psychological traits and disorders (Buss, 2009). Because personality traits are the product of a large number of genes, there are many opportunities for disruption by random mutations. Traits that are universally and highly valued in a mate, such as emotional stability, kindness, conscientiousness, and intelligence may be disrupted by these mutations. Given that psychopathy likely represents the extreme along a continuum of symptom severity, it is likely influenced by the cumulative effect of many minor dysfunctions at the micro-level of genes and brain development (Keller & Miller, 2006b).

3.1 Evidence supporting mutation load

One factor that suggests that psychopathy may be more likely a result of mutation load than balancing selection is the fact that environmental insults tend to produce patterns of behavior that resemble psychopathy, and neurodevelopmental abnormalities tend to increase rather than decrease the risk of psychopathy. For example, neurological research on individuals who were once normal but who then suffered brain lesions have convincingly demonstrated that damage to the ventral regions of the prefrontal cortex results in poor decision-making, autonomic deficits, and sociopathic behavior (Damasio, 2000). A quasi-experimental group study on head injuries in soldiers revealed that individuals with ventromedial lesions showed greater aggressive, violent, and/or antisocial behavior than individuals with nonfrontal lesions, or non-lesion controls (Grafman, Schwab, Warden, Pridgen, & Brown, 1996). Studies of children with lesions to the prefrontal cortex early in life lend further support to the view that head (and therefore brain) trauma can directly lead to antisocial and aggressive behavior. Anderson et al. (1999) found that individuals who suffered selective lesions to the prefrontal cortex in the first 16 months of life showed early antisocial behavior that progressed into delinquency in adolescence and criminal behavior in adulthood. However, it should be noted that although brain injuries have been found to result in some of the symptoms of psychopathy, none have been found to entirely replicate the disorder entirely; in particular, lesions typically do not lead to increases in instrumental aggression, as is observed in psychopathy (Blair, 2005, 2007). To some degree it is expected that a difference would exist between the resulting symptoms of a developmental condition and an acquired one, since the developmental condition alters the course of development from an earlier point in time. Overall, the inferences one can draw from these lesion data are limited.

A recent study by Raine et al. (2010) reported that individuals with a brain marker for abnormal brain development in the fetus exhibited higher levels of antisocial personality, psychopathy, arrests, and convictions compared to those without the brain marker. This marker is called cavum septum pellucidum and indicates a space near the limbic system of the brain that forms during gestation but then fails to fuse back together upon development of the limbic brain structures such as the hippocampus, amygdala, corpus callosum, and other midline structures. Lack of such limbic development interrupts the closure of the cavum, resulting in the preservation of the cavum septum pellucidum into adulthood. The authors argued that the finding of higher levels of psychopathy and antisocial personality in individuals

with cavum septum pellucidum in adulthood provides initial evidence for a neurodevelopmental abnormality in antisocial individuals.

This type of evidence poses a serious challenge to balancing selection models which posit that psychopathy is an alternative, complex adaptation maintained by selection. Given that adaptations require the complex coordination of many factors, it would be expected that traumas would disrupt this complex adaptation rather than lead to it (Keller & Miller, 2006b). However, the idea that environmental factors can increase the incidence of psychopathy is also compatible with the contingent shifts model. The contingent shifts model suggests that rather than assuming that childhood exposures to stress and adversity result in dysfunction of biological and behavioral functioning, it may be that the developmental systems are responding in ways that are adaptive in stressful environments (Del Giudice et al., in press).

Another factor that suggests that psychopathy may be a result of mutation load is the fact that, at least in youth, it has been found to be highly comorbid with attention deficit hyperactivity disorder (ADHD). In one sample, the percentage of psychopathic youth who also received a diagnosis of ADHD was over 75 percent (Colledge & Blair, 2001). It has been hypothesized that this comorbidity is a result of reduced functioning in a region of the brain, the ventrolateral prefrontal cortex, which is involved in controlling one's responses. Impairment in this brain region, which has been implicated in psychopathy, may be associated with difficulties in behavioral regulation and give rise to the hyperactivity observed in children with ADHD (Blair, Mitchell, & Blair, 2005). The fact that the majority of children with psychopathy demonstrate additional behavioral impairments seems to suggest a deficit (possibly due to increased mutation load) rather than an adaptive mechanism at work.

A final factor that suggests that psychopathy may be a result of mutation load is the type of aggression displayed by psychopaths. Psychopaths are unique in that, unlike individuals with other psychological disorders, they demonstrate instrumental aggression (planned, predatory, goal-driven), which may be beneficial in extracting resources from others. However, they also demonstrate elevated levels of reactive aggression (also referred to as impulsive aggression), which is initiated in response to a frustrating or threatening event and does not aim to achieve an obvious goal. For example, psychopathic individuals may react aggressively to someone who has accidentally bumped into them on the street or made a provocative, yet ambiguous, comment. Psychopathic individuals have been found to be more likely to attribute hostile intent to others' behaviors (hostile attribution bias) (Vitale, Newman, Serin, & Bolt, 2005). Elevated levels of reactive aggression suggest deficits in the executive systems of the brain that help to control behavior.

This trait might still be understood in the context of the idea that psychopathy represents a strategy. Abnormal development of the limbic system might facilitate the reduced empathy and callousness that allow psychopathic individuals to take advantage of others. In turn, reactive aggression might be a consequence of this configuration of the limbic system. Another possibility is that these deficits are a result of pleiotropic effects and are retained because the psychopathic traits they are associated with promote reproductive success.

3.2 Evidence against mutation load

Although there is strong support for the mutation load hypothesis with respect to other psychiatric disorders, one of the problems with mutation load as an explanation for psychopathy is that psychopathic individuals have traits that do appear to enhance their ability to exploit others. The psychopathic personality type is described by a number of influential skills, including superficial charm, manipulation, deceit, and a presentation style often viewed as attractive (high self-esteem and confidence). It has been suggested that some of these traits may serve as valuable personal assets in some environments (Lykken, 1995). A counter-argument to this problem for mutation load is that in the presence of deficits, other systems may attempt to compensate in some way. Another possibility is that some of these features are more like a byproduct of a deficit. For example, features such as charm that appear to be positive may actually stem from an emotional deficit. Without fear of social embarrassment, individuals may appear more relaxed and less inhibited in their social interactions, and thus may come across as charming. Similarly, the ability to deceive and exploit others may also be enhanced by the reduced emotional responsiveness and lack of feelings of guilt and remorse in psychopathic individuals.

Psychopathic traits may be most beneficial in individuals who are able to better regulate their behavior. The concept of the "successful" psychopath has been defined differently in different studies, but is generally thought of as one who refrains from serious antisocial behavior but who embodies the essential personality characteristics of psychopathy. These individuals may achieve personal or professional success at the expense of family, friends, and coworkers. They may use strategies such as flattery, manipulation, and deception to gain social status and resources. Although the use of these tactics may have potentially serious negative social consequences, some individuals may be better able to escape detection than others. One study by Ishikawa et al. (2001) found that psychopathic individuals who did not have a criminal conviction ("successful" psychopaths) demonstrated better autonomic functioning and better executive functioning than both unsuccessful psychopaths and controls, suggesting that successful psychopaths appear to have abilities that are superior to non-psychopathic individuals. Other studies have found that on some biological and behavior factors, successful psychopaths do not demonstrate the same deficits as unsuccessful psychopaths (Raine et al., 2004;

Widom & Newman, 1985; Yang et al., 2005), but on some factors they do demonstrate similar deficits (Belmore & Quinsey, 1994; Benning, Patrick, & Iacono, 2005; Justus & Finn, 2007). Despite the fact that superior abilities are not consistently observed, the evidence that any one system may be functioning better provides a challenge for the mutation load theory, since mutations ought not produce phenotypic improvements.

Future research exploring whether psychopathic individuals demonstrate superior abilities in some domains, despite deficits in others, may help to clarify whether psychopathy is likely a product of mutation load. An example of one such domain may be intelligence. Most studies of adults have found that there is no correlation between psychopathy and intelligence in adults (Blair et al., 2005) or children (Loney, Frick, Ellis, & McCoy, 1998); this is, notably, in contrast to findings in antisocial individuals more generally which tend to find intelligence deficits (e.g., Loney et al., 1998). However, another study in youth found that although total psychopathy scores were not significantly associated with intelligence, scores on the dimension of psychopathy representing a superficial and deceitful interpersonal style were positively associated with verbal intellectual skills as well as creativity, practicality, and analytical thinking (Salekin, Neumann, Leistico, & Zalot, 2004). This suggests that children with psychopathic-like traits, particularly those with the interpersonal features of psychopathy, may demonstrate average to above-average levels of intelligence. Given that intelligence is thought to be affected by a large number of genes, with respect to the mutation load model, it is surprising that deficits in intelligence are not observed in psychopathy.

Another potential problem with the mutation load theory is that it does not, by itself, explain the sex differences in psychopathy or other mental disorders. However, it could be argued that the effects of mutation load are overlaid on the background of normal sexual differentiation of brain development and behavior, and that mutations that disrupt sex-specific mechanisms will have sex specific effects (Keller & Miller, 2006a).

4. Conclusions

Many of these theories have the potential to explain individual differences in psychopathic personality traits. Based on our review, it seems difficult to narrow down a single theory by which psychopathy may be characterized. It may be that psychopathy can be conceptualized as an alternative strategy for solving recurrent adaptive problems, as listed in Table 1. In this sense, psychopathy may not actually be a disorder at all from an evolutionary perspective if it does not reflect genuine maladaptive dysfunctions. However, it is also possible that psychopathy is a result of an accumulation of mutations that are passed on through generations because they do not significantly disrupt the individual's reproductive fitness. Future research on the etiology of psychopathy, with particular focus on genetic and environmental factors, may help to clarify the issue.

References

Anderson, S. W., Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1999). Impairment of social and moral behavior related to early damage in human prefrontal cortex. *Nature Neuroscience, 2*, 1031-1037.

Arnett, P. A. (1997). Autonomic responsivity in psychopaths: a critical review and theoretical proposal. *Clinical Psychology Review, 17*, 903-936.

Babiak, P., & Hare, R. D. (2006). *Snakes in Suits: When Psychopaths Go to Work*. New York: HarperCollins.

Bagot, R. C., van Hasselt, F. N., Champagne, D. L., Meaney, M. J., Krugers, H. J., & Joels, M. (2009). Maternal care determines rapid effects of stress mediators on synaptic plasticity in adult rat hippocampal dentate gyrus. *Neurobiology of Learning and Memory, 92*, 292-300.

Barr, K. N., & Quinsey, V. L. (2004). Is psychopathy a pathology or a life strategy? Implications for social policy. In C. Crawford & C. Salmon (Eds.), *Evolutionary psychology, public policy, and personal decisions* (pp. 293-317). Hillsdale, NJ: Erlbaum.

Belmore, M. F., & Quinsey, V. L. (1994). Correlates of psychopathy in a noninstitutionalized sample. *Journal of Interpersonal Violence, 9*, 339-349.

Benning, S. D., Patrick, C. J., & Iacono, W. G. (2005). Fearlessness and underarousal in psychopathy: Startle blink modulation and electrodermal reactivity in a young adult male community sample. *Psychophysiology, 42*, 753-762.

Bernstein, D. P., Stein, J. A., & Handelsman, L. (1998). Predicting personality pathology among adult patients with substance use disorders: effects of childhood maltreatment. *Addictive Behaviors, 23*, 855-868.

Blair, R. J. (2005). Applying a cognitive neuroscience perspective to the disorder of psychopathy. *Development and Psychopathology, 17*(3), 865-891.

Blair, R. J. (2007). Dysfunctions of Medial and Lateral Orbitofrontal Cortex in Psychopathy. *Annals of the New York Academy of Sciences, 1121*, 461-479.

Blair, R. J., Colledge, E., Murray, L., & Mitchell, D. G. V. (2001). A selective impairment in the processing of sad and fearful facial expressions in children with psychopathic tendencies. *Journal of Abnormal Child Psychology, 29*, 491-498.

Blair, R. J., Mitchell, D. G., & Blair, K. (2005). *The Psychopath: Emotion and the Brain*. Oxford: Blackwell.

Blonigen, D. M., Carlson, S. R., Krueger, R. F., & Patrick, C. J. (2003). A twin study of self-reported psychopathic personality traits. *Personality & Individual Differences, 35*, 179-197.

Bouchard, T. J., & Loehlin, J. C. (2001). Genes, evolution, and personality. *Behavior Genetics, 31*, 243-273.

Buss, D. M. (2009). How can evolutionary psychology successfully explain personality and individual differences? *Perspectives on Psychological Science, 4*, 359-366.

Cameron, N. M., Shahrokh, D., Del Corpo, A., Dhir, S. K., Szyf, M., Champagne, F. A., et al. (2008). Epigenetic programming of phenotypic variations in reproductive strategies in the rat through maternal care. *Journal of Neuroendocrinology, 20*, 795-801.

Chen, C., Burton, M., Greenberger, E., & Dmitrieva, J. (1999). Population migration and the variation of dopamine D4 receptor (DRD4) allele frequences around the globe. *Evolution and Human Behavior, 20*, 309-324.

Cima, M., Smeets, T., & Jelicic, M. (2008). Self-reported trauma, cortisol levels, and aggression in psychopathic and non-psychoathic prison inmates. *Biological Psychiatry, 78*, 75-86.

Colledge, E., & Blair, R. J. (2001). Relationship between attention-deficit-hyperactivity disorder and psychopathic tendencies in children. *Personality & Individual Differences, 30*, 1175-1187.

Cooke, D. J., & Michie, C. (1999). Psychopathy across cultures: North America and Scotland compared. *Journal of Abnormal Psychology 10*, 58-68.

Cornell, D. G., Warren, J., Hawk, G., Stafford, E., Oram, G., & Pine, D. (1996). Psychopathy in instrumental and reactive violent offenders. *Journal of Consulting and Clinical Psychology, 64*, 783-790.

Cosmides, L., & Tooby, J. (2000). Consider the source: The evolution of adaptations for decoupling and metarepresentation. In D. Sperber (Ed.), *Metarepresentations: A multidisciplinary perspective* (pp. 53-115). New York: Oxford University Press.

Dahle, K. (2006). Strengths and limitations of actuarial prediction of criminal reference in a German prison sample: A comparative study of the LSI-R, HCR-20, and PCL-R. *International Journal of Law and Psychiatry, 29*, 431-442.

Damasio, A. R. (2000). Commentary: A Neural Basis for Sociopathy. *Archives of General Psychiatry, 57*, 128-129.

Del Giudice, M., Ellis, B. J., & Shirtcliff, E. A. (in press). The Adaptive Calibration Model of stess responsivity. *Neuroscience and Biobehavioral Reviews*.

Ebstein, R. P. (2006). The molecular genetic architecture of human personality: Beyond self-report questionnaires. *Molecular Psychiatry, 11*, 427-445.

Edens, J. F., Marcus, D., Lilienfeld, S. O., & Poythress, N. G. (2006). Psychopathic, not psychopath: Taxometric evidence for the dimensional structure of psychopathy. *Journal of Abnormal Psychology, 115*, 131-144.

Eisenberg, D. T. A., Campbell, B., Gray, P. B., & Sorenson, M. D. (2008). Dopamine receptor genetic polymorphisms and body composition in undernourished pastoralists: An exploration of nutrition indices among nomadic and recently settlesd Ariaal men of northern Kenya. *BioMed Central Evolutionary Biology, 8*, 173.

Ellis, B. J., Boyce, W. T., Belsky, J., Bakermans-Kranenburg, M. J., & van Ijzendoorn, M. H. (2011). Differential susceptibility to the environment: An evolutionary-neurodevelopmental theory. *Development and Psychopathology, 23*, 7-28.

Figueredo, A. J., Vasquez, G., Brumbach, B. H., Schneider, S. M. R., Sefcek, J. A., Tal, I. R., et al. (2006). Consilience and life history theory: From genes to brain to reproductive strategy. *Developmental Review, 26*, 243-275.

Flor, H., Birbaumer, N., Hermann, C., Ziegler, S., & Patrick, C. J. (2002). Aversive Pavlovian conditioning in psychopaths: Peripheral and central correlates. *Psychophysiology, 39*, 505-518.

Gao, Y., Raine, A., Chan, F., Venables, P. H., & Mednick, S. A. (2010). Early maternal and paternal bonding, childhood physical abuse and adult psychopathic personality. *Psychological Medicine, 40*, 1007-1016.

Gladden, P. R., Figueredo, A. J., & Jacobs, W. J. (2009a). Life History strategy, Psychopathic Attitudes, personality, and general intelligence. *Personality & Individual Differences, 46*, 270-275.

Gladden, P. R., Welch, J., Figueredo, A. J., & Jacobs, W. J. (2009b). Moral intuitions and religiosity as spuriously correlated life history traits. *Journal of Evolutionary Psychology, 7*, 167-184.

Glenn, A. L., Iyer, R., Graham, J., Koleva, S., & Haidt, J. (2009). Are all types of morality compromised in psychopathy? *Journal of Personality Disorders, 23*, 384-398.

Glenn, A. L., Raine, A., Venables, P. H., & Mednick, S. (2007). Early temperamental and psychophysiological precursors of adult psychopathic personality. *Journal of Abnormal Psychology, 116*(3), 508-518.

Grafman, J., Schwab, K., Warden, D., Pridgen, B. S., & Brown, H. R. (1996). Frontal lobe injuries, violence, and aggression: a report of the Vietnam head injury study. *Neurology, 46*, 1231-1238.

Guay, J. P., Ruscio, J., Knight, R. A., & Hare, R. D. (2007). A taxometric analysis of the latent structure of psychopathy: Evidence for dimensionality. *Journal of Abnormal Psychology 116*, 701-716.
Hagen, E. H. (1999). The functions of postpartum depression. *Evolution and Human Behavior, 20*, 325-359.
Hanley, C., Banks, W. C., & Zimbardo, P. G. (1973). Interpersonal dynamics in a simulated prison. *International Journal of Criminology and Penology, 1*, 69-97.
Hare, R. D. (2003). *Hare Psychopathy Checklist-Revised (PCL-R): 2nd Edition*. Toronto: Multi-Health Systems, Inc.
Haukka, J., Suvisaari, J., & Lonnqvist, J. (2003). Fertility of patients with schizophrenia, their siblings, and the general population: a cohort study from 1950-1959 in Finland. *American Journal of Psychiatry, 160*(460-463).
Ishikawa, S. S., Raine, A., Lencz, T., Bihrle, S., & Lacasse, L. (2001). Autonomic stress reactivity and executive functions in successful and unsuccessful criminal psychopaths from the community. *Journal of Abnormal Psychology, 110*(3), 423-432.
Jonason, P. K., Koenig, B. L., & Tost, J. (2010a). Living a fast life: The Dark Triad and life history theory. *Human Nature, 21*, 428-442.
Jonason, P. K., Li, N. P., & Buss, D. M. (2010b). The costs and benefits of the Dark Triad: Implications for mate poaching and mate retention tactics. *Personality & Individual Differences, 48*, 373-378.
Jonason, P. K., Li, N. P., Webster, G. D., & Schmitt, D. P. (2009). The Dark Triad: Facilitating a short-term mating strategy in men. *European Journal of Personality, 23*, 5-18.
Justus, A. N., & Finn, P. R. (2007). Startle modulation in non-incarcerated men and women with psychopathic traits. *Personality & Individual Differences, 43*, 2057-2071.
Kaplan, H. S., & Gangestad, S. W. (2005). Life history theory and evolutionary psychology. In D. M. Buss (Ed.), *The Handbook of Evolutionary Psychology* (pp. 68-95). Hoboken, NJ: John Wiley & Sons, Inc.
Keller, M. C., & Miller, G. (2006a). An evolutionary framework for mental disorders: Integrating adaptionist and evolutionary genetic models. *Behavioral and Brain Sciences, 29*, 429-441.
Keller, M. C., & Miller, G. (2006b). Resolving the paradox of common, harmful, heritable mental disorders: Which evolutionary genetic models work best? *Behavioral and Brain Sciences, 29*, 385-452.
Ketelaar, T., & Au, W. T. (2003). The effects of guilty feelings on the behavior of uncooperative individuals in repeated social bargaining games: An Affect-as-information interpretation of the role of emotion in social interaction. *Cognition & Emotion, 17*, 429-453.
Kosson, D. S., Suchy, Y., Mayer, A. R., & Libby, J. (2002). Facial affect recognition in criminal psychopaths. *Emotion, 2*(4), 398-411.
Krischer, M. K., & Sevecke, K. (2008). Early traumatization and psychopathy in female and male juvenile offenders. *International Journal of Law and Psychiatry, 31*, 253-262.
Lang, S., af Klinteberg, B., & Alm, P. O. (2002). Adult psychopathy and violent behavior in males with early neglect and abuse. *Acta Psychiatrica Scandinavica, 106*, 93-100.
Larsson, H., Andershed, H., & Lichtenstein, P. (2006). A genetic factor explains most of the variation in psychopathic personality. *Journal of Abnormal Psychology, 115*, 221-230.
Loney, B. R., Frick, P. J., Ellis, M., & McCoy, M. G. (1998). Intelligence, callous-unemotional traits, and antisocial behavior. *Journal of Psychopathology and Behavioral Assessment, 20*, 231-247.
Loney, B. R., Taylor, J., Butler, M. A., & Iacono, W. G. (2007). Adolescent psychopathy features: 6-year temporal stability and the prediction of externalizaing symptoms during the transition to adulthood. *Aggressive Behavior, 33*, 242-252.
Lorber, M. F. (2004). Psychophysiology of Aggression, Psychopathy, and Conduct Problems: A Meta-Analysis. *Pychological Bulletin, 130*(4), 531-552.
Lykken, D. (1995). *The antisocial personalities*. Hillsdale, NJ: Erlbaum.
MacCabe, J. H., Koupil, I., & Leon, D. A. (2009). Lifetime reproductive output over two generations in patients with psychosis and their unaffected siblings: the Uppsala 1915-1929 Birth Cohort Multigenerational Study. *Psychological Medicine, 39*, 1667-1676.
Marshall, L. A., & Cooke, D. J. (1999). The childhood experiences of psychopaths: a retrospective study of familial and societal factors. *Journal of Personality Disorders, 13*, 211-225.
Mealey, L. (1995). The sociobiology of sociopathy: an integrated evolutionary model. *The Behavioral and Brain Sciences, 18*, 523-599.
Milgram, S. (1963). Behavioral study of obedience. *Journal of Abnormal and Social Psychology, 67*, 371-378.
Murphy, D., & Stich, S. (2000). Darwin in the madhouse: evolutionary psychology and the classification of mental disorders. In P. Carruthers & A. Chamberlain (Eds.), *Evolution and the human mind: Modularity, language and meta-cognition* (pp. 62-92). Cambridge, UK: Cambridge University Press.

Nesse, R. M., & Williams, G. C. (1994). *Why we get sick: The new science of Darwinian medicine*. New York: Times Books.
O'Leary, M. M., Loney, B. R., & Eckel, L. A. (2007). Gender differences in the association between psychopathic personality traits and cortisol response to induced stress. *Psychoneuroendocrinology, 32*(2), 183-191.
O'Neill, M. L., Lidz, V., & Heilbrun, K. (2003). Predictors and correlates of psychopathic characteristics in substance abusing adolescents. *International Journal of Forensic Mental Health, 2*, 35-45.
Patrick, C. J. (1994). Emotion and psychopathy: Startling new insights. *Psychophysiology, 31*, 319-330.
Patrick, C. J., Bradley, M. M., & Lang, P. J. (1993). Emotion in the criminal psychopath: Startle reflex modulation. *Journal of Abnormal Psychology, 102*, 82-92.
Raine, A. (1993). *The psychopathology of crime: Criminal behavior as a clinical disorder*. San Diego, CA: Academic Press.
Raine, A., Ishikawa, S. S., Arce, E., Lencz, T., Knuth, K. H., Bihrle, S., et al. (2004). Hippocampal structural asymmetry in unsuccessful psychopaths. *Biological Psychiatry, 55*, 185-191.
Raine, A., Lee, L., Yang, Y., & Colletti, P. (2010). Neurodevelopmental marker for limbic maldevelopment in antisocial personality disorder and psychopathy. *British Journal of Psychiatry, 197*, 186-192.
Raine, A., Stoddard, J., Bihrle, S., & Buchsbaum, M. S. (1998). Prefrontal glucose deficits in murderers lacking psychosocial deprivation. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 11*, 1-7.
Rozin, P., Lowery, L., Imada, S., & Haidt, J. (1999). The CAD triad hypothesis: A mapping between three moral emotions (contempt, anger, disgust) and three moral codes (community, autonomy, divinity). *Journal of Personality and Social Psychology, 76*, 574-586.
Rushton, J. P. (1985). Differential K theory: The sociobiology of individual and group differences. *Personality & Individual Differences, 6*, 441-452.
Ryan, M. J., & Causey, B. A. (1989). Alternative mating-behavior in the swordtails *Xiphophorus nigrensis* and *Xiphophorus pygmaeus* (Pices, Poeciliidae). *Behavioral Ecology and Sociobiology, 24*, 341-348.
Sadeh, N., Javdani, S., Jackson, J. J., Reynolds, E. K., Potenza, M. N., Gelernter, J., et al. (2010). Serotonin transporter gene associations with psychopathic traits in youth vary as a function of socioeconomic resources. *Journal of Abnormal Psychology 119*, 604-609.
Salekin, R. T., Neumann, C. S., Leistico, A. M., & Zalot, A. A. (2004). Psychopathy in youth and intelligence: An investigation of Cleckley's hypothesis. *Journal of Clinical Child and Adolescent Psychology, 33*, 731-742.
Stephens, D. W., & John, R. K. (1986). *Foraging theory*. Princeton, NJ: Princeton University Press.
Taylor, J., Loney, B. R., Bobadilla, L., Iacono, W. G., & McGue, M. (2003). Genetic and environmental influences on psychopathy trait dimensions in a community sample of male twins. *Journal of Abnormal Child Psychology, 31*, 633-645.
Tooby, J., & Cosmides, L. (1990). On the universality of human nature and the uniqueness of the individual: The role of genetics and adaptation. *Journal of Personality, 58*, 17-68.
Vitale, J., Newman, J. P., Serin, R. C., & Bolt, D. M. (2005). Hostile attributions in incarcerated adult male offenders: An exploration of diverse pathways. *Aggressive Behavior, 31*, 99-115.
Vrana, S. R., Spence, E. L., & Lang, P. J. (1988). The startle probe response: a new measure of emotion? *Journal of Abnormal Psychology 97*, 487-491.
Walters, G. D., Brinkley, C. A., Magaletta, P. R., & Diamond, P. M. (2008). Taxometric analysis of the Levenson Self-Report Psychopathy scale. *Journal of Personality Assessment, 90*, 491-498.
Weeden, J., Cohen, A. B., & Kenrick, D. T. (2008). Religious attendance as reproductive support. *Evolution and Human Behavior, 29*, 327-334.
Weiler, B., & Widom, C. S. (1996). Psychopathy and violent behavior in abused and neglected young adults. *Criminal Behavior and Mental Health, 6*, 253-271.
Wernke, M. R., & Huss, M. T. (2008). An alternative explanation for cross-cultural differences in the expression of psychopathy. *Aggression and Violent Behavior, 13*, 229-236.
West-Eberhard, M. J. (2003). *Developmental plasticity and evolution*. New York: Oxford University Press.
Wheatley, T., & Haidt, J. (2005). Hypnotically induced disgust makes moral judgments more severe. *Psychological Science, 16*, 780-784.
Widom, C. S., & Newman, J. P. (1985). Characteristics of non-institutionalized psychopaths. In D. P. Farrington & J. Gunn (Eds.), *Aggression and dangerousness* (pp. 57-80). New York: Wiley.
Yang, Y., Raine, A., Lencz, T., Bihrle, S., Lacasse, L., & Colletti, P. (2005). Volume reduction in prefrontal gray matter in unsuccessful criminal psychopaths. *Biological Psychiatry, 15*(57), 1103-1108.