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Sexual Selection and Sex Differences in the Prevalence of Childhood Externalizing and Adolescent Internalizing Disorders

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Despite the well-established sex difference in prevalence of many childhood and adolescent psychopathological conditions, no integrative metatheory of sex differences in psychopathology exists. This review attempts to provide a metatheoretical framework to guide empirical examination of sex differences in prevalence of childhood-onset "externalizing" and adolescent-onset "internalizing" disorders, based on sexual selection evolutionary theory. Sexual selection theory suggests important between-sex differences in markers, mechanisms, etiology, and developmental timing of risk and resilience relevant to psychopathology. Namely, sexual selection theory hypothesizes that disinhibition and sensation-seeking may be important proximate risk markers for childhood-onset externalizing disorders in males. The theory suggests that these male-biased markers may be a product of their higher exposure to prenatal testosterone, which makes them more susceptible to prenatal stressors with downstream effects on dopaminergic neurotransmission, especially for those with genetic alleles associated with lower dopaminergic function. In contrast, sexual selection theory hypothesizes that negative emotionality, empathy, and cognitive rumination may be important proximate risk markers for adolescent-onset internalizing disorders in females. The theory suggests that these markers are propagated by rapidly rising levels of estradiol at puberty that interact with cortisol and oxytocin. These hormones exert downstream effects on the serotonergic system in such a way as to increase females' sensitivity to interpersonal stressors particularly at puberty and especially for those with lower functional serotonergic activity. Such a metatheory can help integrate prior ideas about sex differences and can also generate new predictions of sex differences in markers, etiology, mechanisms, and developmental timing of common forms of psychopathology.

Keywords: sex differences, psychopathology, evolutionary theory, sexual selection

Sex, or gender, differences in prevalence are perhaps one of the most obvious, but poorly understood, characteristics of many types of psychopathology (as reviewed by Grant & Weissman, 2007; Hartung & Widiger, 1998; Rutter, Caspi, & Moffitt, 2003; Zahn-Waxler, Crick, Shirtcliff, & Woods, 2006; Widiger, 2007). A dramatic sex/gender difference in the relative prevalence of childhood psychopathologies has been extensively documented in the literature, and estimates are that, in general, males are approximately three times more likely than females to be diagnosed with many early-onset disorders, such as autism spectrum disorders, and perhaps particularly "externalizing disorders," including attention-deficit/hyperactivity disorder (ADHD), oppositional-defiant disorder (ODD), and conduct disorder (CD; Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009; Copeland, Shanahan, Costello, & Angold, 2011; Egger & Angold, 2006; Merikangas et al., 2010; Volkmar, Lord, Bailey, Schultz, & Klin, 2004). In contrast, many disorders that have a peak onset during adolescence (as opposed to childhood), particularly most mood and anxiety disorders, as well as eating disorders and borderline personality disorder, are more common in females than in males at a ratio of at least two to one (Costello, Mustillo, Erkanli, Gordon, & Angold, 2003; Culbertson, 1997; Ge, Lorenz, Conger, Elder, & Simons, 1994; Hankin et al., 1998; Hayward & Sanborn, 2002; Nolen-Hoeksema & Girgus, 1994; Pawluck & Gorey, 1998; Silverman, La Greca, & Wasserstein, 1995). Further, females with almost any type of clinical disorder appear to demonstrate greater symptom-related impairment, higher rates of comorbidity, increased continuity of problems over time, and more social and academic impairment (Biederman et al., 2010; Chronis-Tuscano et al., 2010; Costello et al., 2003; Hinshaw & Kranz, 2009; Hinshaw, Owens, Sami, & Fargeon, 2006; Keenan, Loeber, & Green, 1999; Lahey et al., 2007).

Despite the consistency of this pattern of results and general acceptance regarding sex/gender differences in the prevalence of many types of psychopathology and related impairment, to date, the basis for these sex/gender differences is scarcely understood. Although there are a number of theories of sex/gender differences in psychopathology, both at the level of individual disorders (e.g., autism: Baron-Cohen, 2002; depression: Cambron, Acitellil, & Pettit, 2009; depression: Nolen-Hoeksema & Girgus, 1994) and across broader classes of disorders (e.g., across "internalizing" and

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"externalizing" disorders: Hinshaw & Kranz, 2009; Keenan & Shaw, 1997; Zahn-Waxler, Shirtcliff, & Marceau, 2008; personality disorders: Beauchaine et al., 2009; Lynam & Widiger, 2007), as detailed in Table 1, a metatheory that can integrate across biological, psychological, and social factors has been lacking. Although extant theories have suggested that males and females may be differentially exposed to biological risk factors (e.g., prenatal testosterone exposure; Baron-Cohen, 2002), differentially susceptible to environmental factors (e.g., Hinshaw & Kranz, 2009), or exhibit distinct psychological risk factors (e.g., Nolen-Hoeksema & Girgus, 1994), there is no theory that is able to cogently integrate across these different, and yet potentially complementary, accounts.

The goal of the current article is to present a metatheory that can be used to (a) organize what is currently known about sex/gender differences in biological, psychological, and social factors associated with the male-biased prevalence of childhood-onset "externalizing" disorders and the female-biased prevalence of adolescent-onset "internalizing" disorders and (b) generate new, testable hypotheses about sex/gender differences in etiology, mechanisms, markers, and developmental timing of onset of psychopathology. This information could then inform understanding of individual and group differences in risk and resilience for disorder with implications for causal theory and prevention and intervention science. Provision of this kind of framework is timely. Several recent initiatives are geared toward advancing understanding of women's health so a viable integrative framework could have broad-reaching impact on subdisciplines within psychology, as well as across the medical field (Cahill, 2010; National Institutes of Health, Office of Research on Women's Health [ORWH], 2010; "Putting Gender on the Agenda," 2010; Wisner & Dolan-Sewell, 2007).

First, the terms sex and gender must be defined. While the terms are often used interchangeably, their definitions carry somewhat distinctive meanings that have important implications for this type of work. While sex refers to the biological classification of an organism based on sex chromosomes, reproductive organs, and hormones, the term gender refers to an individual's self-representation as male or female and/or to socialization based on others' interpretation of sex (Institute of Medicine, 2001). Sex and gender are dynamically interrelated and difficult to separate from one another. Further complicating matters, male and female (and masculine and feminine) are not always dichotomous and are not even necessarily on opposite sides of a continuum (Baron-Cohen, 2006; Pinel, 2011). For example, an individual can be genetically female, but exhibit a more male-typical profile of sex hormones, suggesting that individuals can exhibit masculine and feminine characteristics simultaneously (e.g., congenital adrenal hyperplasia, androgen insensitivity syndrome; Cohen-Bendahan, van de Beek, & Berenbaum, 2005). Thus, the terms sex and gender are much more complex than common usage suggests. For simplicity, this article utilizes the term sex rather than gender due to the use of ultimate concepts to guide a broad-based review of biological, psychological, and social factors relevant to psychopathology.

Second, psychopathology must be defined. Wakefield (1992, 1997, 2007) provides an evolutionarily informed definition of mental disorder, or psychopathology, in which he describes it as a "harmful dysfunction" where harm is judged according to social values, and dysfunction refers to the failure of an internal mechanism to perform the function for which it was selected. Yet, because of the lack of consensus regarding what constitutes dysfunction, current definitions of psychopathology rely primarily on expert consensus definitions of behavioral or psychological syndromes that are conceptualized as disturbances in cognition, emotion, or behavior that cause clinically significant distress or impairment (American Psychiatric Association [APA], 2000; Stein et al., 2010; www.dsm5.org). Empirical work suggests that, at a descriptive level, such-defined psychopathology is hierarchically organized such that, at the most abstract level, there is a single general psychopathology category, or factor, whereas—at more specific levels—two, three, and more categories can be specified (Lahey et al., 2004; Lahey, Rathouz, et al., 2008; Markon, 2010).

Historically and with substantial empirical support, psychopathology has been broadly divided into two heuristic categories: "externalizing" disorders and "internalizing" disorders (Crijnen, Achenbach, & Verhulst, 1997; Kramer, Krueger, & Hicks, 2008; Krueger, Chentsova-Dutton, Markon, Goldberg, & Ormel, 2003; Trosper, Whitton, Brown, & Pincus, 2012; Verhulst & Achenbach, 1995). These two categories are correlated and exhibit substantial comorbidity (Krueger & Markon, 2006; Lahey, Rathouz, et al.,

Table 1
Major Theories of Sex Differences in Psychopathology

Disorder(s)	Sex difference explanation	Citation
Disorders of childhood and adolescence	Sex differences in biological, physical, cognitive, and social-emotional factors	Zahn-Waxler et al., 2008
Childhood problem behavior	Sex differences in socialization and rate of biological, cognitive, and social-emotional development	Keenan & Shaw, 1997
Personality disorders	Sex differences in five-factor model of personality	Lynam & Widiger, 2007
Antisocial and borderline personality disorders	Sex-moderated genetic vulnerabilities (i.e., Gene × Sex × Environment)	Beauchaine et al., 2009
Depression	Sex differences in rumination	Nolen-Hoeksema & Girgus, 1994
Depression	Sex differences in interpersonal contingent self-esteem in the face of interpersonal stress	Cambron et al., 2009
Autism	Sex differences in systemizing and empathizing due to sex differences in prenatal testosterone exposure	Baron-Cohen, 2002

2008; Markon, 2010), as well as shared risk factors (Lahey, 2009), and yet are also at least somewhat distinct, perhaps most particularly in regard to sex differences in prevalence (N. R. Eaton et al., 2012; Hartung & Widiger, 1998; Rutter et al., 2003; Zahn-Waxler et al., 2006). Thus, despite some limitations, for present purposes, the two-factor structure of externalizing-internalizing is relied on as a heuristic for organizing discussion of sex differences in many common forms of psychopathology based on historical precedent, substantial empirical support, and practicality (Crijnen et al., 1997; Kramer et al., 2008; Krueger et al., 2003; Trosper et al., 2012; Verhulst & Achenbach, 1995). Further, the article will emphasize clinical disorders versus symptom dimensions, based on past precedent and simplicity, although it is increasingly agreed that these are complementary approaches (Markon, Chmielewski, & Miller, 2011; www.dsm5.org).

Childhood-onset "externalizing" disorders, also referred to as disruptive behavior disorders, have historically included oppositional-defiant disorder (ODD), conduct disorder (CD), and attention-deficit/hyperactivity disorder (ADHD; APA, 2000), and these disorders—like many other disorders that have a peak onset during childhood—have a male-biased prevalence rate (Costello, Egger, & Angold, 2005; Egger & Angold, 2006; Egger, Kondo, & Angold, 2006). ODD is characterized by angry/irritable mood, defiant/headstrong behavior, and vindictiveness and affects approximately 10% of children (Keenan et al., 2007; Stringaris & Goodman, 2009). CD is characterized by a persistent behavioral pattern in which the basic rights of others or societal rules or norms are violated and affects approximately 4% of children (Egger & Arnold, 2006; Keenan et al., 2011). ADHD is characterized by behavioral symptoms of inattention, hyperactivity, and impulsivity and affects between three and 5% of children (Nigg, 2006a, 2006b).

These three disorders are observed to frequently co-occur, being comorbid in approximately 30 to 50% of cases (Angold, Costello, & Erkanli, 1999), supporting their conceptualization as externalizing disorders, although ADHD also shares similarities with neurodevelopmental disorders like autism (that also exhibit a male-biased prevalence rate; Baron-Cohen, 2002) and will be so classified in *DSM–5* (Frick & Nigg, 2012; www.dsm5.org). Like many predominately male disorders, all three conditions develop early with a median age of onset of 5 to 7 years (Egger & Angold, 2006; Egger et al., 2006). Rates of all three disorders are substantially higher among males relative to females at a ratio of 3:1 during childhood (Beauchaine et al., 2009; Copeland et al., 2011; Egger & Angold, 2006; Volkmar et al., 2004) and, in many cases, these disorders continue on into adulthood, increasing risk for other disorders like antisocial personality disorders, substance dependence, and associated problems (e.g., assault and homicide; Broidy et al., 2003; Martel, Pierce, et al., 2009; Schaeffer, Petras, Ialongo, Poduska, & Kellam, 2003). Although some have argued that this disparity in diagnosis rates between males and females may be due to problems with measurement (e.g., males are more likely to use easily measured physical aggression [compared to females], whereas females more typically rely on more difficult-to-measure relational aggression [but at rates approximately equal to males]; Archer, 2004; Crick et al., 2006; Ostrov & Crick, 2007), there has been limited evidence to support the contention that this phenomenon significantly influences diagnostic rates (Keenan, Coyne, & Lahey, 2008). Further, sex differences in the behavioral expression of aggression beg the question as to why these differences exist.

In contrast to childhood-onset externalizing disorders, most "internalizing" disorders with a peak onset during adolescence have a female-biased prevalence rate (Costello et al., 2005; Ge et al., 1994; Hankin et al., 1998; Silverman et al., 1995). "Internalizing" disorders have historically included mood disorders and anxiety disorders. Mood disorders are defined by prolonged and marked disturbances in mood and include major depressive disorder, dysthymic disorder, bipolar disorder I and II, and cyclothymic disorder (APA, 2000). Within this category, research has focused mostly on depressive disorders, including major depressive disorder and Dysthymic Disorder. This review will focus on depressive disorders within the mood disorder category. Depressive disorders have a lifetime prevalence rate of approximately 20% in the general population (Kessler et al., 2005). Anxiety disorders, a broad category of disorders unified by the experience of exaggerated agitation, nervousness, or fear, usually in relation to a perceived threat, include panic disorder, specific phobia, social phobia (or social anxiety disorder), generalized anxiety disorder, and historically obsessive-compulsive disorder and posttraumatic stress disorder, although these latter two disorders are likely to be moved to other categories in *DSM–5*; separation anxiety disorder will be moved into the anxiety disorder category in *DSM–5* (APA, 2000; Friedman et al., 2011; www.dsm5.org). Anxiety disorders are relatively common, occurring in approximately 30% of the population at some point during the lifetime (APA, 2000; Kessler et al., 2005; Rapee, Wignall, Hudson, & Schniering, 2000).

Depressive disorders and anxiety disorders are highly comorbid, co-occurring in approximately 50% of people who have one of the disorders (Angold et al., 1999). Depressive disorders have a mean age of onset around age 14 (Carter, Silverman, & Jaccard, 2011; Costello et al., 2005; Costello, Foley, & Angold, 2006), and several anxiety disorders, most particularly social phobia, have peak age of onset around age 13 (Ollendick & Hirschfeld-Becker, 2002; Warren & Sroufe, 2004; Weems & Costa, 2005; Westenberg, Drewes, Goedhart, Siebelink, & Treffers, 2004). As noted, internalizing disorders like depressive and most anxiety disorders are more common in females than in males at a ratio of approximately 2 to 1, beginning in adolescence (Hayward & Sanborn, 2002; Nolen-Hoeksema & Girgus, 1994; Silverman et al., 1995). The focus of the present review will be on common forms of developmental psychopathology that demonstrate striking sex differences in prevalence, namely, childhood-onset externalizing disorders (i.e., ODD, CD, ADHD) that are more common in males and adolescent-onset internalizing disorders (i.e., depression, social phobia) that are more common in females.

The cultural context of psychopathology is important to consider. Extensive literature demonstrates that these sex differences are generally consistent across most modern cultures, exhibiting moderate to large effect sizes (e.g., see Crijnen et al., 1997; Crijnen, Achenbach, & Verhulst, 1999; Del Giudice, 2009; Holtmann, Bolte, & Poustka, 2007; Kramer et al., 2008; Krueger et al., 2003; Verhulst & Achenbach, 1995). Yet, cultural influences affect the expression and mean level of symptoms of psychopathology (as reviewed by Draguns & Tanaka-Matsumi, 2003; López & Guarnaccia, 2000; McLaughlin, Hilt, & Nolen-Hoeksema, 2007; T. W. Miller, Nigg, & Miller, 2009; Weisz, McCarty, Eastman, Chaiyasit, & Suwanlert, 1997). For example, a few cultures (i.e.,

some Mediterranean countries [Mavrea, Beis, Mouyias, Rigoni, & Lyketsos, 1986; Vázquez-Barquero et al., 1987], Old Order Amish [Egeland & Hostetter, 1983], and orthodox Jews in the London area [Loewenthal et al., 1995]) exhibit no or minimal sex differences in rates of depression (as reviewed by Piccinelli & Wilkinson, 2000). Further, cultural and ethnic differences are apparent in the mean level and developmental timing of the appearance of fears (Ollendick, Yang, King, Dong, & Akande, 1996), in the target of fears (e.g., *taijin kyofusho*, a common Japanese disorder characterized by fear of offending others with inappropriate behavior or appearance; Kirmayer, 1991), and in the mean level of ADHD symptoms (T. W. Miller et al., 2009). Thus, any theory that tries to explain sex differences in psychopathology will need to take environmental influences such as contextual cultural and ethnic influences into account because they will necessarily influence the expression of psychopathology.

Overall, despite the well-established nature of sex differences in many common forms of psychopathology and the number of theories that have been proposed to account for sex differences in etiological factors, risk factors, and mechanisms related to psychopathology (see Table 1), an integrative metatheory of sex differences in psychopathology has been lacking. The current review seeks to provide such a metatheory by working from the readily available and extensively researched integrative framework of evolutionary psychology and, in particular, sexual selection. Although many other paradigms within psychology might be able to be employed as such a framework (e.g., social learning, socialization, role theory, cultural, feminist and gender oppression, hormonal, etc.), the current review utilizes an evolutionary psychological framework because it is able to encompass and organize these other paradigms and currently available extant theories of sex differences due to its focus on both ultimate (i.e., why) and proximate (i.e., how) mechanisms (Ariew, 2003; Alcock & Sherman, 1994; Belsky, Stenberg, & Draper, 1991; Mayr, 1961; Tinbergen, 1963). An integrative framework of sex differences in psychopathology can and should draw from multidisciplinary research in the areas of clinical psychology, behavioral neuroscience (or biopsychology), cognitive psychology, social and personality psychology, developmental (or child) psychology, and cross-cultural psychology to test its predictions and guide more proximate explanations. Although evolutionary explanations of behavior are sometimes thought to be at odds, or in competition, with environmental, cultural, and social learning explanations of behavior, evolutionary processes and evolution-shaped traits are—in fact—quite sensitive to environmental, learning, and cultural contexts (B. J. Ellis et al., 2012; Geary, 2002a, 2002b, 2010; West-Eberhard, 2003). An integrative evolutionary theory must take these perspectives into account in order to do justice to the complexity of human behavior.

Provision of this kind of an integrative theory is timely due to recent initiatives geared toward increasing understanding of women's health in conjunction with recent research advances in the areas of psychopathology, genomics, neurobiology and neurotransmission, cognition, personality, social interactive processes, and developmental trajectories (Cahill, 2010; ORWH, 2010; "Putting Gender on the Agenda," 2010). Together, these advances have provided a more complex and nuanced picture of human development and disorder than ever before with implications for understanding of individual differences, including both between-sex mean differences and variability within and across sex. An integrative framework for study of sex differences in psychopathology is critical for advancing work in this area because it would allow for the generation of more specific and testable hypotheses of sex differences in the development of common forms of psychopathology and their underlying markers, etiology (i.e., causes), and mechanisms (i.e., Insel & Cuthbert, 2009; Insel et al., 2010), moving work on sex differences in psychopathology from largely exploratory to theory-driven (Cahill, 2010; Geary, 2006; ORWH, 2010; "Putting Gender on the Agenda," 2010). This advance is needed in order to allow for targeted work that can shed light on a fundamental characteristic of many forms of psychopathology: sex differences.

Plan for the Review

Herein, I apply the evolutionary theory of sexual selection to psychopathology by focusing specifically on childhood-onset externalizing disorders (or problems) and adolescent-onset internalizing disorders (or problems, focusing on depression and adolescent-onset anxiety). First, evolutionary theory and the theory of sexual selection, as well as a related evolutionary developmental theory, life history theory, are explicated. Next, the viability of sexual selection for organizing the field's current understanding of sex differences in (a) markers, (b) etiology (or causes), and (c) mechanisms of developmental timing of common forms of psychopathology is examined. Then, using that background, a model of sex differences in common forms of externalizing and internalizing psychopathology is proposed, and specific, a priori hypotheses are provided with attention to the high comorbidity between externalizing and internalizing problems and linkage with prior work and theory. Finally, I conclude with a summary and discussion of applied directions for future work.

Evolutionary Theory of Sexual Selection as a Viable Theoretical Framework for Understanding Sex Differences in Psychopathology

Evolutionary theory can provide a useful organizing framework for many domains of psychological science, including those addressing sex differences in psychopathology, because it seeks to address both ultimate (i.e., why) and proximate (i.e., how) processes, as well as individual differences and developmental expression (Bjorklund & Ellis, 2005; A. H. Buss, 2012; D. M. Buss, 1995; Darwin, 1871; Ketelaar & Ellis, 2000). As background, evolutionary theory, or the theory of natural selection, has two main tenets: (a) there is substantial variability in traits between individuals, and (b) heritable traits that confer a survival advantage are more likely to be passed on to subsequent generations and spread in the population (Darwin, 1859; Geary, 2010). The theory of sexual selection applies these ideas specifically to inter- (i.e., between) and intra- (i.e., within) sex variability by positing that (a) there are substantial intrasex differences, or variability, in traits and (b) heritable traits that confer a reproductive advantage by influencing intrasex competition for mates and/or intersex selection of mates are more likely to be passed on to subsequent generations (Alcock & Crawford, 2008; Darwin, 1871; Geary, 2010). Traits that are passed on in this manner exhibit fairly substantial intersex (parental investment and sexual selection the-

ory; Trivers, 1972) and intrasex (good genes sexual selection theory; G. Miller, 2000) variability.

Based on sexual selection theory, individual differences result in variation in competitive abilities and attractiveness to potential mates and thus contribute to the reproductive dynamics of sexual selection (Darwin, 1871). The dimensions of reproduction that are relevant to sexual selection processes are (a) intrasexual (i.e., male-male and female-female) competition for access to mates or for control of the resources mates need to reproduce (e.g., energetic, financial resources) and (b) intersexual choice (i.e., female choice of male, male choice of female) of mating partner. Although male-male competition and female choice are typically viewed as the most prominent forms of sexual selection within most species, in species where males are involved in parenting (like the human species), male choice and female-female competition are also important.

Sexually selected traits are those traits that provide reproductive advantage and, over evolutionary time, result in the emergence of intersex differences, as well as intrasex variation, in these traits (e.g., physical stature, natural weaponry, ornamentation; Alcock & Crawford, 2008; Andersson, 1994; Geary, 2010; Pomiankowski & Moller, 1995; Rowe & Houle, 1996). Both inter- and intrasex variability in sexually selected traits are presumed to be adaptive in regard to reproduction because they provide ways for the individual and group to (a) cope with circumstances that have tended to reoccur throughout evolutionary time for males and females and (b) adapt to changing circumstances across development (Bjorklund & Ellis, 2005). At an ultimate ("why") level, these traits confer a reproductive advantage within sex. For example, males benefitted from having a larger, more muscular physical stature (e.g., when hunting, fighting other males) in hunter-gatherer societies in the past, and the substantial average intersex differences in physical stature in humans reflect that. Yet, at a proximate ("how") level, sexually selected traits come with a suite of cost-benefit trade-offs and are heavily influenced by environmental conditions and health, resulting in interindividual variability and constraining these traits from becoming too extreme. For example, because of the associated costs of large physical stature (e.g., need for increased caloric intake) and variations in environmental circumstances, there is still substantial intrasex variability in physical stature among males, and males do not grow as large under impoverished conditions (G. C. Williams, 1957). In fact, the theory of sexual selection suggests that sexually selected traits are often more sensitive to environmental context than other traits due to the evolutionary trade-offs inherent in these traits (B. J. Ellis et al., 2012; Geary, 2002a, 2002b, 2010; West-Eberhard, 2003).

In humans, there are not only substantial intersex differences in these types of physical traits, there are also meaningful intersex differences in psychological domains, including cognitive, emotional, and behavioral traits, presumably because these domains were (and are) highly relevant to mating competition and choice in the human species (Andersson, 1994; Bjorklund & Ellis, 2005; Geary, 2006, 2010). Yet, not all domains are subject to sexual selection pressures or exhibit sex differences. Sexual selection theory suggests prominent intersex differences and intrasex variability in traits that facilitate intrasex competition and intersex choice with minimal or nonexistent sex differences in other domains (Darwin, 1871; Geary, 2010).

Geary (2002a, 2010; Geary & Huffman, 2002) divides psychological domains subject to sexual selection processes into three main categories: psychological/social (e.g., nonverbal and verbal skills, cognition), psychological/biological (e.g., knowledge of plants and animals), and psychological/physical (e.g., movement, representation, and engineering or tool use). That is, Geary (2002a, 2010) suggests that there will be substantial intersex differences in traits that fall into these three categories because individual variability in these domains presumably facilitated intrasex competition and/or access to resources, as well as intersex choice in the past. Although all three domains are theorized to be influenced by sexual selection processes, the psychological/social domain is the focus of the present article because it accommodates self-awareness and self-consciousness necessary for competitive and cooperative social dynamics critical for human experiences, as well as encompassing behaviors and traits, or markers, that are considered key to definitions and theories of psychopathology, particularly externalizing and internalizing disorders (Geary, 2010).

For human males in hunter-gatherer societies, psychological/social traits that facilitated male-male competition and access to resources through increases in *social dominance* are theorized to have been particularly important (Geary, 2010). For human females in hunter-gatherer societies, psychological traits that facilitated female choice and offspring caregiving (and perhaps, secondarily, female-female competition for mates) through increases in *interpersonal competence and dominance* are theorized to have been particularly important. Although these types of traits were presumably adaptive in the past and can be adaptive today, depending on context, because they can facilitate intrasex competition and intersex choice, they can also become disturbances that cause significant distress or impairment. For example, extreme manifestation of these traits, limited plasticity of these traits in response to environmental clues, and/or expression of these traits in environments that are a poor match for the traits, as detailed below, may increase risk for psychopathology in a subset of individuals (Wakefield, 2007; i.e., conditional maladaptation in B. J. Ellis, De Giudice, & Shirtcliff, 2013). However, it should be noted that, even in these cases, the traits may still be evolutionarily adaptive because they facilitate reproductive opportunity in particular environmental circumstances.

Take sensation-seeking (i.e., the tendency to seek out experiences that are varied, novel, and intense) as an example. Sensation-seeking exhibits substantial intersex variability; males are typically higher in sensation-seeking than females. This may be because sensation-seeking allowed males to engage in risky behaviors that were advantageous for facilitating exploration and/or establishing dominance in order to acquire resources and/or win mates in hunter-gatherer societies (B. J. Ellis et al., 2012; Zuckerman, Eysenck, & Eysenck, 1978). However, variability in sensation-seeking among males is also present in order to offset the increased mortality risk of excessive sensation-seeking, as well as to allow for population-level flexible adaptation to changing environmental circumstances. In the past and today, although high sensation-seeking may be advantageous for reproduction in certain environments (e.g., in depleted environments, in high-risk neighborhoods), low

sensation-seeking may be more advantageous for reproduction in other environments (e.g., in environments with more resources, in an affluent family). Importantly, when a trait like sensation-seeking is expressed either (a) in extreme form (e.g., excessive sensation-seeking behaviors that are engaged in to the detriment of survival or reproduction goals), (b) with limited plasticity in relation to the environment (e.g., when the individual is unable to adopt a more moderate level of sensation-seeking in response to environmental cues), and/or (c) in environments that provide a poor match to the trait (e.g., in this case, stable, affluent environments), then it can cause distress or impairment that far outweighs its advantages for that individual, thus increasing the likelihood that it will be labeled as psychopathology (B. J. Ellis et al., 2012; Frankenhuis & Del Giudice, 2012). However, when expressed at moderate levels, when expressed flexibly, and/or when expressed under select (e.g., in harsh and/or unpredictable; B. J. Ellis, Figuerdeo, Brumbach, & Schlomer, 2009) circumstances, the same trait can be advantageous and is less likely to be labeled as psychopathology (APA, 2000; Stein et al., 2010; Wakefield, 2007; www.dsm5.org). Thus, the social and cultural environment is critical for determining whether a given trait standing proves to be adaptive and/or is distressing or impairing, thus influencing who is diagnosed with psychopathology.

However, evolutionary theory and the theory of sexual selection are not without limitations. One might wonder about the relevance of evolutionary theory in an age in which cultural adaptations and change have rapidly outpaced evolutionary change. Yet, evolutionary theory and principles can provide some explanation of current evolutionary-influenced human adaptation to these changes, and evolution-influenced traits are highly responsive to context, including cultural change. Critics of evolutionary theory as an explanatory theory of human behavior often object that the theory can be post hoc ("just so") and circular in its reasoning (Halpern, 1997). Indeed, some assertions of evolutionary theory remain very difficult to test and run the risk of being post hoc in nature. However, evolutionary principles have been explored and extensively validated in many species, including those with social structures similar to humans, suggesting the theory's viability as a metatheory of biology and psychology (e.g., Darwin, 1871; B. J. Ellis & Ketelaar, 2000; Geary, 2002a; Ketelaar & Ellis, 2000). Indeed, many of the sex differences examined in the current article appear to be universal, or evident across cultures and across studies (L. Ellis, 2011a, 2011b), exhibiting medium to large effect sizes (Del Giudice, 2009; Holtmann et al., 2007). However, it should be noted that not all traits exhibit (or would be expected to exhibit, based on sexual selection theory) such prominent sex differences (e.g., Hyde, 2005). Although evolutionary theory can be exploited to generate post hoc explanations of human behavior, a strength of the theory is that it can be utilized to organize and generate a priori, refutable, theory-driven hypotheses of sex differences in psychopathology at ultimate (i.e., why) and, perhaps more importantly for clinical science, proximate (i.e., how) levels (e.g., Belsky & Pluess, 2009; Boyce & Ellis, 2005; B. J. Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van Ijkzendoorn, 2011). That potential is of primary interest here.

Life History Theory as a Viable Theoretical Framework for Understanding Sex Differences in the Developmental Timing of the Expression of Psychopathology

The developmental timing of the manifestation of evolutionarily influenced traits, or markers, has been discussed in a corollary evolutionary developmental theory: life history theory. Life history theory addresses the critical question of the timing of relevant trait expression over the lifespan, based on the scientific understanding of evolved strategies for distributing metabolic resources between growth, maintenance, and reproduction (Chisholm, Burbank, Coall, & Gemmiti, 2005; B. J. Ellis, 2004; B. J. Ellis et al., 2012). Life history theory applied to humans describes offspring growth as slow, resulting in an extended prejuvenile period characterized by increased parental investment, child trait plasticity, and practice of reproductively relevant traits (e.g., hunting and aggression in males; mothering in females; B. J. Ellis et al., 2012; Geary, 2002b), compared to other animals, resulting in fewer, but larger, more malleable, and more competitive offspring, particularly in more stable contexts (Geary, 2010). This extended developmental period provides an important function for humans by allowing them to flexibly adapt to context and condition (Bjorklund, 1997). Further, this extended developmental period implies the potential for important influences on reproductively relevant traits at different developmental stages, perhaps particularly during reproductively relevant periods such as the prenatal period (when the organizational framework of the nervous system is developing) and during puberty (when reproductive behaviors are becoming manifest; Bjorklund, Ellis, & Rosenberg, 2007; B. J. Ellis, 2004; B. J. Ellis et al., 2013; Geary, 2002a).

Sexual selection theory likewise suggests that traits influenced by sexual selection are often complex, variable, and particularly susceptible to environmental conditions during specific developmental periods (Brumbach et al., 2009; B. J. Ellis et al., 2012; Geary, 2002a, 2002b, 2010; Glover, 2011; Talge, Neal, Glover, & the Early Stress, Translational Research and Prevention Science Network: Fetal and Neonatal Experience on Child and Adolescent Mental Health, 2007; West-Eberhard, 2003). The timing of sensitivity to environmental conditions and the types of environmental conditions to which individuals are sensitive may differ for males and females due to the different types of sexual selection pressures operating on them (B. J. Ellis, 2004; B. J. Ellis et al., 2009; Geary, 2010). Specifically, because males typically grow larger and stronger than females presumably so they would have been able to more effectively compete for social status during childhood and mates during adolescence in hunter-gatherer societies, they may grow slower during the prenatal period to allow for maximization of fitness through a longer period of sensitivity to informative early environmental conditions. Males may be particularly sensitive to environmental stressors denoting broad ecological conditions during these early periods because these conditions would provide the context for the practice and refinement of reproductive and competitive strategies during childhood (B. J. Ellis et al., 2009; Geary, 2010; Geschwind & Galaburda, 1985; Martel, Klump, et al., 2009; Morris, Jordan, & Breedlove, 2004; Stinson, 1985). For males, then, the prenatal period may be an especially critical time for the development of the structural organization that will set the stage for early childhood social status competitions via rough-and-

tumble play and aggression (Geschwind & Galaburda, 1985; Jašarević et al., 2011). Therefore, the presence of prenatal environmental stressors signaling broader ecological conditions (e.g., a harsh, unpredictable environment) may signal to the developing male fetus that it will be born into a high risk environment, thus suggesting the need for strategies that will facilitate social dominance and expedite reproduction (B. J. Ellis et al., 2012, 2009; Geary, 2010).

In contrast, adolescence (and particularly puberty) may be a particularly critical period for females because this is the time when they first become capable of reproducing with its attendant responsibility of childrearing (B. J. Ellis, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1999; Geary, 2010). Females mature relatively more quickly than males (B. J. Ellis, 2004; Grumbach & Styne, 2003) and quickly become more sensitive to interpersonal aspects of the social environment (e.g., parental responsiveness, warmth, threat; Hankin, Mermelstein, & Roesch, 2007; Keenan & Hipwell, 2005; Kochanska, 1997; Taylor, 2006). Females also reach physical, reproductive maturity more quickly than males (B. J. Ellis, 2004; Grumbach & Styne, 2003), the timing of which is influenced by psychosocial influences (e.g., paternal availability and parent–child relationships; B. J. Ellis et al., 1999; B. J. Ellis, Shirtcliff, et al., 2011). This increased interpersonal sensitivity may function, in part, to increase females' interpersonal competence and dominance and is likely to prepare females for their later prominent role in reproduction and parenting which rely on a strong social support network, intimate interpersonal relationships, and sensitive detection of threat (Colle & Del Giudice, 2011; Geary, 2010; Taylor et al., 2000). Thus, females may be more sensitive to interpersonal aspects of the social environment beginning early on, perhaps with particularly strong effects during adolescence (specifically at puberty) when reproduction and parenting is first possible with attendant facilitation of interpersonal competence and dominance.

However, this is not to say that prenatal and/or more ecological stressors are unimportant for females or pubertal and/or interpersonal stressors unimportant for males. While all types and timing of stressors undoubtedly have some influence on the development of males and females across the lifespan, sexual selection theory suggests that the relative magnitude of the effects of early-occurring, ecological versus later-occurring, interpersonal stressors might differ between the sexes. Namely, sexual selection theory suggests that males (vs. females) might be relatively more sensitive to early-occurring, ecological stressors, and females (vs. males) may be relatively more sensitive to interpersonal stressors occurring later during adolescence (B. J. Ellis, 2004; B. J. Ellis et al., 2009; Geary, 2010).

Provision of General Evolutionarily Informed Hypotheses of Common Forms of Psychopathology

Thus, sexual selection theory and life history theory suggest a number of evolutionarily informed, a priori, general hypotheses regarding sex differences in childhood-onset externalizing and adolescent-onset internalizing disorders. A strength of such an evolutionary framework is that it allows for integration of biological, psychological, and social/cultural accounts of psychopathology, as detailed below. These general hypotheses are stated here, and then are further elaborated and made more specific later in the article after I review work relevant to sexual selection processes operating on childhood-onset externalizing disorders and adolescent-onset internalizing disorders.

General Hypotheses Based on a Sexual Selection Theory of Childhood-Onset Externalizing Disorders and Adolescent-Onset Internalizing Disorders

The most important, or core, hypotheses based on this evolutionary-informed theory of sex differences in psychopathology are covered in the first set of hypotheses presented below with elaboration of developmental and environmental mechanisms detailed in the second and third sets of hypotheses.

1. If sexual selection pressures in the evolutionary past are relevant to sex differences in the prevalence rates of common forms of psychopathology today, then there will be sex differences in traits that function to increase intrasex reproductive advantage, and they will help explain sex differences in the prevalence rates of common forms of psychopathology. Specifically:
   a. There will be sex differences in traits related to social dominance favoring males because establishment of social dominance is important for intrasex competition in males.
   b. There will be sex differences in traits related to interpersonal competence favoring females because interpersonal competence is important for intrasex competition, mate choice, and reproduction in females.
   c. These sexually selected traits will be associated with common forms of psychopathology and will explain sex differences in prevalence rates of common forms of psychopathology.
   d. Sexually selected traits will mediate, or explain, associations between etiological factors and common forms of psychopathology.
2. If sexual selection processes impacted life history strategies differentially in males and females in the evolutionary past, then there will be sex differences in the timing of environmental sensitivity (i.e., sex by developmental timing interaction) associated with common forms of psychopathology and associated sexually selected traits, or markers. Specifically:
   a. If males' achievement of social dominance during the reproductive years requires a long period of practice and refinement of associated physical and social competencies that facilitate intrasex competition, then males will be more sensitive to early environmental stressors with effects on early-emerging sexually selected traits and common forms of psychopathology.
   b. If females' interpersonal competence becomes important for intrasex competition and mate choice during puberty, then females will be more sensitive to interpersonal stressors occurring at puberty and during adolescence with effects on sexually selected traits and common forms of psychopathology.
   c. Sex differences in the timing of particular hormonal surges will mediate, or explain, sex differences in timing of sensitivity to environmental influences.
   d. These hormonal effects will be mediated, or explained, by effects on neurotransmission and neural circuitry with particularly prominent effects for those with genetic alleles that alter relevant neurotransmission systems.

3. If males' and females' reproductive capacities were dependent on different types of environmental constraints in the evolutionary past, then there will be sex differences in the type of environmental cues to which males and females are differentially susceptible (i.e., sex by environment interaction) in relation to common forms of psychopathology and associated sexually selected traits, or markers. Specifically:

a. Males will be differentially susceptible to cues related to the broader ecological environment and available resources with effects on early-emerging sexually selected traits and common forms of psychopathology.
b. Females will be differentially susceptible to aspects of the environment that provide information about interpersonal relationships important for reproduction with effects on sexually selected traits and common forms of psychopathology.
c. These environmental factors will be heavily shaped by culture with effects on sexually selected traits and common forms of psychopathology.

These hypotheses will be used to organize the forthcoming review of what is currently known about sex differences in traits (or markers), biological and environmental etiological factors, and hormonal mechanisms related to the developmental timing and onset of common forms of psychopathology with specific predictions provided for childhood-onset externalizing disorders and adolescent-onset internalizing disorders at the end of the review.

Sexual Selection and Sex Differences in Markers of Psychopathology: Dispositional Traits and Cognition

Evolutionary theory suggests that individual differences in traits broadly defined have evolved in order to facilitate adaptation to particular reoccurring environments in animals and humans (A. H. Buss, 2012; Darwin, 1872) and that the complex trait systems necessary for self-consciousness and social interactions, particularly psychological/social traits, are especially important in our species (Andersson, 1994; Bjorklund & Ellis, 2005; Geary, 2006, 2010). Dispositional psychological/social traits related to emotion and cognition and exhibiting sex differences are possible targets of evolutionary sexual selection processes, and they may help to explain sex differences in the prevalence of psychopathology by serving as potential within-child markers of disorders (A. H. Buss, 2012; Geary, 2010; Nigg, 2006a, 2006b). Dispositional trait structure is fairly universal across cultures (McCrae & Costa, 1997; McCrae, Terracciano, & 78 members of the Personality Profiles of Cultures Project, 2005), and dispositional traits exhibit striking individual differences, particularly sex differences, again across cultures (Else-Quest, Hyde, Goldsmith, & Van Hulle, 2006; Gartstein & Rothbart, 2003). Further, dispositional traits have been linked to the later development of many, if not all, forms of psychopathology (e.g., A. H. Buss, 2012; Kotov, Gamez, Schmidt, & Watson, 2010; Krueger, Markon, Patrick, Benning, & Kramer, 2007; Nigg, 2006a, 2006b; Watson, Gamez, & Simms, 2005). They are arguably the earliest-emerging of currently known risk factors related to psychopathology.

What I refer to herein as dispositional traits are typically referred to in the literature as temperament traits or personality traits. Temperament traits have been defined as constitutionally based, individual differences in reactivity and self-regulation (Rothbart & Posner, 2006). Personality traits have been defined similarly with the major difference that they are believed to be more heavily influenced by socialization (vs. biological influences; McCrae et al., 2000). Yet, recent reviews have clarified that, at the level of behavioral measurement, temperament and personality traits are both influenced by both constitutional and environmental influences to a similar degree and are substantially related to one another (McCrae et al., 2000; Shiner & Caspi, 2003). For this reason, they can be studied within the same general framework (Nigg, 2006a, 2006b; Rothbart, Ahadi, & Evans, 2000; Shiner, 1998). Thus, in this article, temperament and personality traits will be referred to interchangeably under the general terminology of dispositional traits, regardless of developmental period.

Dispositional traits can be measured as early as infancy, and there are a number of models available (e.g., Eisenberg et al., 1996; McCrae & Costa, 1987; Rothbart & Bates, 1998). Empirically, dispositional traits appear to cluster into three different dimensions, at least by early childhood (see Figure 1). These three factors can be labeled as approach (or extraversion; composed of surgency/sociability, exuberance, positive emotionality; related to sensation-seeking), negative emotionality (or neuroticism; composed of sadness, fear, anger), and control (or conscientiousness; including activity level, effortful control, persistence; Markon, Krueger, & Watson, 2005; Mervielde, De Clercq, De Fruyt, & Van Leeuwen, 2005; Nigg, 2006a, 2006b). This broad conceptual model can be used as a framework to organize other models of dispositional traits, as well as being generally able to be applied to animal models, the study of neural systems, and cross-cultural research (A. H. Buss, 2012; McCrae et al., 2005).

Although dispositional traits exhibit striking individual differences, they are moderately stable across development, even when

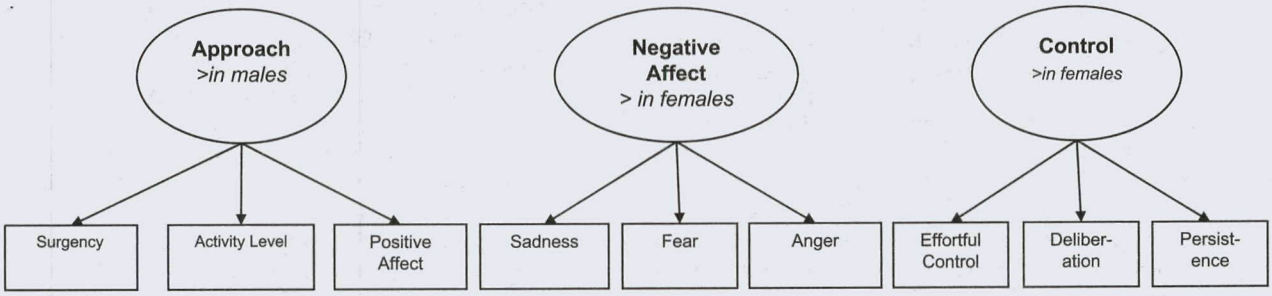

Figure 1. Three-factor dispositional trait model.

measured as early as infancy (Caspi, Roberts, & Shiner, 2005; De Fruyt et al., 2006). Dispositional traits demonstrate well-validated sex differences (Else-Quest et al., 2006) across cultures (Costa, Terracciano, & McCrae, 2001; Feingold, 1994; Schmitt, Realo, Voracek, & Allik, 2008; but see Hyde, 2005). Males are typically rated as exhibiting higher approach than females (i.e., activity and high intensity pleasure; Gartstein & Rothbart, 2003; Martin, Wisenbaker, Baker, & Huttunen, 1997), which is related to sensation-seeking (B. J. Ellis et al., 2012; L. Ellis, 2011b; Zuckerman, 2005). These sex differences become apparent during infancy with male infants being rated as more active than female infants, as measured via laboratory instrument and observer ratings (Campbell & Eaton, 1999; W. O. Eaton & Enns, 1986). In contrast, sex differences favoring females emerge in effortful forms of control a little later during toddlerhood (Bjorklund & Kipp, 1996; Else-Quest et al., 2006; Kochanska, Murray, & Coy, 1997). Further, sex differences in negative emotionality, or neuroticism particularly fear and distress to novelty, appear during adolescence and continue into adulthood with females exhibiting higher levels of these traits compared to males (Costa et al., 2001; Jorm, 1987; Schmitt et al., 2008; Soto, John, Gosling, & Potter, 2011).

Thus, by early childhood, males exhibit higher levels of approach (and later, sensation-seeking) and lower effortful control (or disinhibition) which might be expected to facilitate childhood rough-and-tumble play (Bjorklund, 1997). Rough-and-tumble play during childhood appears to be part of a long process establishing status and dominance among peers, many of whom would be peers for life in a traditional society in the evolutionary past. In addition, rough-and-tumble play may facilitate the practice of behaviors (e.g., aggression, hunting) that would enhance male-male competition during adolescence through achievement of social dominance (Bjorklund, 1997; Pellegrini & Archer, 2005). The intensity, risk of associated injury, and escalation of such childhood play may be exacerbated when environmental resources are more limited due to increased competition for these fewer resources (B. J. Ellis et al., 2012; L. Ellis, 2011a). In contrast, in childhood and adolescence, females exhibit higher levels of negative emotionality and effortful control. High negative emotionality might be exhibited to facilitate the development of empathy and interpersonal sensitivity which in turn might allow for improvement in caregiving abilities, due to increased sensitivity and responsivity to offspring, as well as increased sensitivity to threat (Bjorklund & Kipp, 1996; Pellegrini & Archer, 2005). Further, high effortful control might facilitate increased manipulation of interpersonal contexts, advantageous for female mate choice and female-female competition for mates, in line with sexual selection theory (Bjorklund & Kipp, 1996; Pellegrini & Archer, 2005). Thus, sex differences in traits appear to map on to what one would expect to see, based on sexual selection theory; males exhibit higher mean levels of traits that might be expected to facilitate social dominance and male-male competition (B. J. Ellis et al., 2009; Geary, 2002a, 2010; Pellegrini & Archer, 2005), and females exhibit higher mean levels of traits that might be expected to increase interpersonal competence and dominance and facilitate female choice and female-female competition (Colle & Del Giudice, 2011; Geary, 2002a, 2010).

These individual differences in dispositional traits have been related to risk for psychopathology in children and adults (Krueger & Tackett, 2003; B. P. O'Connor, 2002; Widiger & Trull, 2007), even after controlling for shared, or overlapping, items used to define the constructs (Lemery, Essex, & Smider, 2002; Lengua, West, & Sandler, 1998). A number of different trait-psychopathology models have been proposed (see reviews by Klein, Kotov, & Bufferd, 2011; Tackett, 2006; Watson, Kotov, & Gamez, 2006). For example, dispositional traits have been suggested to predispose individuals to psychopathology, share a common cause with psychopathology, lie on the same continuum as psychopathology, and be exacerbated by or exacerbate psychopathology with the general consensus that extreme temperament traits seem to at least increase risk for (i.e., the probability of) concurrent or subsequent psychopathology (Aksan & Kochanska, 2004; De Pauw & Mervielde, 2011; Eisenberg et al., 2001; Van Leeuwen, Mervielde, De Clercq, & De Fruyt, 2007). Supporting this notion, dispositional traits have been linked longitudinally, in particular combinations, to virtually all forms of psychopathology (Eisenberg et al., 1996; Kotov et al., 2010; Krueger et al., 2002; Lahey, Van Hulle, et al., 2008; Watson et al., 2005), even controlling for item overlap (Lemery et al., 2002; Lengua et al., 1998), although it remains unclear whether traits are—in fact—predisposing markers of psychopathology that predict over and above premorbid psychopathology or are merely co-occurring risk factors or characteristics. Traits may directly and indirectly increase risk for psychopathology (Belsky, Hsieh, & Crnic, 1998; Gallagher, 2002; Lengua, Wolchik, Sandler, & West, 2000; Prinzie et al., 2003; van Zeijl et al., 2007) and are likely to interact with situational context (Mischel & Shoda, 1995; Zakriski, Wright, & Underwood, 2005).

High negative emotionality has been linked to both externalizing and internalizing disorders and may be a global marker of psychopathology (Burke, Hipwell, & Loeber, 2010; Dougherty, 2006; Kotov et al., 2010; Lahey, 2009; McBurnett et al., 2005; Stringaris & Goodman, 2009; Watson et al., 2005; Wright et al., 2012), although some have suggested it is particularly relevant for internalizing disorders (Compas et al., 2004; Trosper et al., 2012; Watson et al., 2005; Wright et al., 2012). Low effortful control may be somewhat more specifically associated with externalizing disorders (Eisenberg et al., 2001; Krueger et al., 2002, 2007; Martel & Nigg, 2006; although it has also been linked to internalizing disorders; e.g., Kotov et al., 2010), and low positive emotionality may be somewhat more specifically associated with internalizing disorders such as depression (Clark & Watson, 1991; Kotov et al., 2010; Watson et al., 2005; Wright et al., 2012). Thus, these sex differences in dispositional traits may be related to sex differences in the prevalence rates of externalizing and internalizing psychopathology such that males exhibit lower levels of effortful control which is associated to a somewhat greater extent with externalizing disorders, and females exhibit higher levels of negative emotionality and lower levels of approach which are associated to a somewhat greater extent with internalizing disorders and possibly higher levels of comorbidity.

More specifically, it may be the typical, or average, sex differences in the relative constellations of traits that may confer risk for externalizing and internalizing disorders. That is, because males—at the group level—are more likely to exhibit lower negative emotionality, higher approach, and lower effortful control, compared to females, there may be a larger subgroup of males (vs. females) who are at risk for externalizing disorders. Since females, as a group, are more likely to exhibit higher negative emotionality,

lower approach, and higher effortful control, there may be a larger subgroup of females (vs. males) who are at risk for internalizing disorders. Further, males exhibiting a more female-typical profile of traits might then be at risk for internalizing disorders, and females with a more male-typical profile of traits might then be at risk for externalizing disorders.

As early as age two, related sex differences begin to emerge in social behavior and cognition. These sex differences may be due to sexual selection processes, may relate to sex differences in dispositional traits, and also appear to be related to psychopathology. Sex differences in behaviors relevant to externalizing disorders, such as physical aggression, can be seen as early as 17 months of age, with males more likely to exhibit physical aggression compared to females. At 17 months, 5% of males, but only 1% of females, manifest physical aggression on a frequent basis (Baillargeon et al., 2007), possibly related to sex differences in approach (Harmon-Jones, 2003; Smits & Kuppens, 2005). Further, sex differences in the forms (appearance; i.e., physical/overt and relational) and functions (purpose; i.e., proactive/instrumental and reactive) of aggression are manifest as early as the second year of life with males more likely to engage in physical forms of aggression than females (Alink et al., 2006; Card, Stucky, Sawalani, & Little, 2008). By early childhood, or 4 years of age, males exhibit more physical, or overt, aggression which may be important for the later facilitation of social dominance and male-male competition (Geary, 2002b, 2010). Also by early childhood, females who engage in aggression are more likely to utilize relational (vs. physical) aggression, exhibiting rates of relational aggression comparable to males (which makes relational aggression females' preferred method of aggression; Crick & Grotpeter, 1995; Ostrov & Crick, 2007). Relational aggression may facilitate female-female competition through establishment of interpersonal dominance (Geary, 2002b, 2010). Such sex differences emerging early on may allow for early practice and preparation of reproductively relevant behaviors that will facilitate intrasex competition later during puberty (Bjorklund, 1997; Geary, 2002b, 2010).

Sex differences in cognition that may be due to sexual selection processes, may relate to dispositional traits, and that are relevant to psychopathology also emerge fairly early during childhood. As early as 14 months of age, females begin to exhibit higher levels of guilt and empathy, compared to males (Kochanska, Barry, Jimenez, Hollatz, & Woodard, 2009; Kochanska, Koenig, Barry, Kim, & Eun Yoon, 2010), and—by early adolescence—females exhibit lower levels of callous-unemotional traits (Loney, Butler, Lima, Counts, & Eckel, 2006; Marsee, Silverthorn, & Frick, 2005). High guilt and empathy, related to negative emotionality, appear to facilitate interpersonal relationships and caregiving (de Wied, Branje, & Meeus, 2007; Landry, Smith, & Swank, 2006; Kochanska & Aksan, 2004; Kochanska & Murray, 2000; Paleari, Regalia, & Fincham, 2005), as well as provide relative protection from the development of externalizing disorders (Kochanska, Barry, et al., 2009). At age two, females begin to outperform males in general and specific language performance (Bornstein, Hahn, & Haynes, 2004), which may be underpinned by their higher levels of effortful control (C. Blair & Razza, 2007). Increased language abilities may make females more responsive to socialization, facilitate development of interpersonal relationships and caregiving, and also provide protection against childhood externalizing problems (Durkin & Conti-Ramsden, 2007; Vygotsky, 1962). Likewise, by childhood, females outperform males on verbal tasks, whereas males outperform females on spatial tasks (Halpern, 2004; Hines, 2010; Kimura & Hampson, 1994). However, higher levels of guilt and empathy may be a double-edged sword for females, ultimately predisposing them to the later development of depression and some kinds of anxiety in response to stressful interpersonal experiences (Keenan & Hipwell, 2005; Zahn-Waxler, 2000; Zahn-Waxler, Cole, & Barrett, 1991).

By early adolescence, there is a sex difference in cognitive rumination such that females are more likely to ruminate than males (Broderick, 1998), seemingly related to sex differences in negative emotionality (Mor & Winquist, 2002). Sex differences in corumination seem to facilitate the development of friendships, romantic relationships, and parental relationships (Rose, 2002; Rose, Carlson, & Waller, 2007; Starr & Davila, 2009; Tompkins, Hockett, Abraibest, & Witt, 2011; Waller & Rose, 2010), presumably through increased sensitivity to conflict and threat. However, rumination also appears to at least partly explain the sex difference in the prevalence of depression that begins in early adolescence (Nolen-Hoeksema, 2000, 2012; Nolen-Hoeksema & Jackson, 2001; Nolen-Hoeksema, Larson, & Grayson, 1999; Verstraeten, Vasey, Raes, & Bijttebier, 2009). Thus, all of these sex differences may increase female interpersonal competence and facilitate female choice and female-female competition by aiding in the establishment of social support networks, as well as by allowing for the manipulation of interpersonal contexts related to mate choice and retention. However, when experienced at extremely high levels, when utilized inflexibly, or when expressed in particularly stressful environments (described in more detail later), they may predispose to psychopathology such as internalizing disorders.

In line with sexual selection theory, these sex differences in dispositional traits, behavior, and cognition have important influences on socialization, social development, and social interactions (Pellegrini & Archer, 2005). Of course, these influences are probably bidirectional with socialization also exerting effects on traits, cognition, and behavior (McCarthy & Arnold, 2011). The theory of sexual selection suggests that sexually selected traits are highly vulnerable to environmental influences, including cultural influences (Bjorklund & Ellis, 2005; Geary, 2002b), and there is substantial empirical support for this idea because temperament and parenting interact with one another (e.g., Bates, Pettit, Dodge, & Ridge, 1998; Kochanska, Aksan, & Joy, 2007; Lengua & Kovacs, 2005). During toddlerhood and preschool, females exhibit higher levels of responsiveness to parents, compared to males (Kochanska, 1997). Also by toddlerhood, females exhibit higher levels of committed compliance (i.e., willing, eager compliance with parental commands) compared to males (Kochanska, Coy, & Murray, 2001; Kochanska, Tjebkes, & Forman, 1998), possibly as a result of their higher levels of negative emotionality, effortful control, better verbal skills including language comprehension and expression, and higher guilt and empathy (Kochanska et al., 2010, 1997; Kochanska, Murray, Jacques, Koenig, & Vandegeest, 1996; Murray & Kochanska, 2002; Vygotsky, 1962).

Overall, then, sexual selection may exert sex specific effects on psychopathology indirectly by operating on sex differences in dispositional traits, cognition, and behaviors that influence social interactions (e.g., same-sex play and competition, responsiveness to parenting, parenting ability) and which—when expressed in extreme form, inflexibly, or in mismatched environments—can

increase the probability of the development of psychopathology. Based on this review, the most probable candidates for sexually selected trait markers of male-biased childhood externalizing disorders are high approach (or sensation-seeking) and low effortful control (or disinhibition) which are sexually selected due to their facilitation of male-male competition through facilitation of social dominance. In contrast, females may be relatively protected from externalizing disorders by their relatively higher levels of effortful control, better verbal abilities, and increased guilt and susceptibility to socialization influences. However, some of these same protective factors, particularly high negative emotionality, empathy, and rumination, may increase risk for adolescent-onset internalizing disorders in females, even though they are sexually selected due to their facilitation of female choice, female-female competition, and caregiving through increases in interpersonal competence. In contrast, males may be relatively protected from internalizing disorders by their high approach (or positive emotionality) and decreased negative emotionality, guilt, and empathy. Yet, the subset of males exhibiting more female-typical patterns of traits may be vulnerable to internalizing disorders, and the subset of females exhibiting more male-typical patterns of traits may be vulnerable to externalizing disorders.

Sexual Selection and Etiology: Serotonergic and Dopaminergic Genes, Neurotransmission, and Neuroanatomy

The theory of sexual selection suggests that individual differences in traits that confer a reproductive advantage within sex by influencing competition for mates and/or mate selection will be more likely to be passed on to subsequent generations (Darwin, 1871; Geary, 2010). In fact, substantial comparative research has indicated that heritable variation in sexually selected traits is typical (Geary, 2002a; Jašarević et al., 2011; McCarthy & Arnold, 2011). However, the expression of sexually selected traits is also heavily dependent on current environmental conditions and developmental health, being influenced by parenting, nutrition, and social conditions (e.g., intensity of competition) and allowing for flexible adaptation to changing conditions (Barker, 1995; Flinn & Ward, 2005; Geary, 2002a; Jašarević et al., 2011; McCarthy & Arnold, 2011). Genetic allelic variability may increase an individual's sensitivity to the environment (called conditional adaptation; Bjorklund & Ellis, 2005; B. J. Ellis, 2004; B. J. Ellis et al., 2009; or differential susceptibility; Belsky & Pluess, 2009; B. J. Ellis, Boyce, et al., 2011), and genes underpinning sexually selected traits may be particularly likely to exhibit these types of effects. Further, although the mechanisms of these effects are not yet fully understood, environmental conditions can influence patterns of gene expression via alterations in levels of sex hormones, as well as through epigenetic changes (e.g., DNA methylation; McCarthy & Arnold, 2011; Meaburn, Schalkwyk, & Mill, 2010; Meaney, 2010). Thus, the evolutionary theory of sexual selection suggests several promising avenues for the study of etiology, or biological and environmental causes, of psychopathology like externalizing and internalizing disorders and underlying traits, or markers.

Genetic allelic variability is perhaps the most extensively studied etiological factor of psychopathology. Work conducted thus far suggests that a series of the same genes are implicated in externalizing and internalizing disorders and associated markers (Schmidt, Fox, Rubin, Hu, & Hamer, 2002), namely, genetic variation that influences functional serotonergic and dopaminergic neurotransmission. These two systems provide probable mechanisms of sensitivity to environmental conditions (Beauchaine et al., 2009; B. J. Ellis, Boyce, et al., 2011). I provide two examples with the serotonin transporter gene (that influences serotonergic neurotransmission) and the dopamine transporter gene (that influences dopaminergic neurotransmission). These two genes were chosen as an illustration of the relevance of individual variation in these key amine systems. Of course, a number of other genes are also probably important for understanding the multifactorial etiology of psychopathology and associated markers, and sexual selection theory suggests some important new directions for work in this area (e.g., dopamine receptor D4 gene and the oxytocin receptor gene, detailed later).

Individual Differences in Serotonergic Neurotransmission and the Serotonin Transporter Gene

The short allele of the serotonin promoter polymorphism of the serotonin transporter gene (5HTTLPR) has been widely studied in psychiatric genetics, exerts functional effects on serotonergic neurotransmission, and has been associated with both externalizing and internalizing disorders and related markers (Clarke, Flint, Attwood, & Munafò, 2010; Lesch et al., 1996; Seeger, Schloss, & Schmidt, 2001; van Goozen, Fairchild, Snoek, & Harold, 2007; Waldman & Gizer, 2006). One or two copies of the short allele of 5HTTLPR (or the recently discovered Lg allelic variant; see Beitchman et al., 2006) results in reduced transcription of the serotonin transporter and lower reuptake activity of serotonin in the synapse (Greenberg et al., 1999; Hariri et al., 2002; Kobiella et al., 2011; Lesch et al., 1996). Further, the short allele of 5HTTLPR has been linked to greater amygdala neuronal activity in response to threatening environmental stimuli like fearful faces (Bertolino et al., 2005; Hariri et al., 2005, 2002; Heinz et al., 2005; Kobiella et al., 2011; Munafò, Brown, & Hariri, 2008), as well as trait anxiety and neuroticism (Munafò, Clark, & Flint, 2005; Pergamin-Hight, Bakersmans-Kranenburg, van IJzendoorn, & Bar-Haim, 2012; Schinka, Busch, & Robichaux-Keene, 2004; Sen, Burmeister, & Ghosh, 2004; but see Terracciano et al., 2009).

Like internalizing disorders, the dispositional trait of negative emotionality exhibits moderate heritability (Yamagata et al., 2005; S. E. Young, Stallings, Corley, Krauter, & Hewitt, 2000). As noted above, negative emotionality and related traits like neuroticism and harm avoidance have been linked to genetic polymorphisms influencing serotoninergic neurotransmission like the short allele of 5HTTLPR (Greenberg et al., 2000; Lesch et al., 1996, 1999; Pezawas et al., 2005; for negative findings see Terracciano et al., 2009). Negative emotionality, such as fear, appears to rely on a neural circuit involving the amygdala, hippocampus, anterior cingulate cortex, and right prefrontal cortex with particular reliance on serotonergic neurotransmission (Depue & Lenzenweger, 2006; Derryberry & Tucker, 2006; Pezawas et al., 2005; Rothbart & Posner, 2006; Whittle, Allen, Lubman, & Yucel, 2006), similar to those circuits implicated in internalizing disorders like depression (Drevets & Raichle, 1992; Nestler et al., 2002; Siegle, Steinhauer, Thase, Stenger, & Carter, 2002). Indeed, low levels of serotonergic neurotransmission have been linked with internalizing disorders

like mood and anxiety disorders (Deakin, 1991; Lucki, 1998; Mann, 1999; Parsey et al., 2006; Selvaraj et al., 2011).

Further, the serotonergic neurotransmission system exhibits prominent sex differences. The effects of 5HTTLPR on serotonergic neurotransmission seem to vary as a function of sex (and ethnicity; R. B. Williams et al., 2003). Further, females have higher serotonin levels, more serotonin receptors and synthesize serotonin slower than males (Cosgrove, Mazure, & Staley, 2007; Duchesne, Dufresne, & Sullivan, 2009). Thus, serotonergic genetic risk and neurotransmission has been associated with internalizing and externalizing disorders, as well as negative emotionality, with important sex differences that may be relevant to sexual selection processes. Further complicating matters, serotonergic neurotransmission can modulate dopamine neurotransmission (Carver, Johnson, & Joormann, 2008; Spoont, 1992).

Individual Differences in Dopaminergic Neurotransmission and the Dopamine Transporter Gene

The 10-repeat allele of the dopamine transporter (*DAT1*) has been widely studied, influences the function of the dopaminergic neurotransmission system, and has been more specifically associated with externalizing disorders, most particularly ADHD, and associated markers (Brookes et al., 2006; Burt & Mikolajewski, 2008; Faraone et al., 2005; Heinz et al., 2000; Volkow, Wang, Fowler, & Ding, 2005; Waldman & Gizer, 2006). Homozygosity for the 10-repeat allele of *DAT1* has been linked to alterations in dopamine transporter binding, particularly in the striatum (Fuke et al., 2001; Heinz et al., 2000; Jacobsen et al., 2000; Van Ness, Owens, & Kilts, 2005). Further, *DAT1* has been linked to individual differences in effortful control and executive attention (Posner, Rothbart, & Sheese, 2007; Rueda, Rothbart, McCandliss, Saccomanno, & Posner, 2005).

Like externalizing disorders, traits associated with effortful control including conscientiousness exhibit moderate heritability (Yamagata et al., 2005; S. E. Young et al., 2000) and have been associated with genetic polymorphisms affecting dopaminergic neurotransmission such as *DAT1* (Aarts et al., 2010; Brocki, Clerkin, Guise, Fan, & Fossella, 2009; Colzato, Van Den Wildenberg, Van Der Does, & Hommel, 2010; Holmboe et al., 2010; Rueda et al., 2005). Control processes such as effortful control (Nigg, 2000) rely heavily on the prefrontal cortex, especially the dorsolateral, orbitofrontal, and anterior cingulate cortex (Derryberry & Tucker, 2006; Rothbart & Posner, 2006; Whittle et al., 2006), similar to those circuits involved in externalizing disorders (R. J. R. Blair, Peschardt, Budhani, Mitchell, & Pine, 2006; Crowe & Blair, 2008; Dickstein, Bannon, Castellanos, & Milham, 2006; Krain & Castellanos, 2006; Nigg & Casey, 2005; Raine, Lencz, Bihrle, LaCasse, & Colletti, 2000; Rubia et al., 2008; Seidman, Valera, & Makris, 2005). Personality traits related to incentive-based and effortful control are associated with variations in the neurotransmitter dopamine particularly (Depue & Lenzenweger, 2006; Rothbart & Posner, 2006; Whittle et al., 2006; Zald et al., 2008). Further, low levels of dopamine have been associated with externalizing disorders like ADHD (Carboni & Silvagni, 2004; Volkow et al., 2009) and perhaps particularly with symptoms of hyperactivity and impulsivity (Diamond, 2007; Durston et al., 2005).

The dopamine system exhibits prominent sex differences. Males appear to have lower presynaptic dopamine tone and lower extrastriatal dopamine receptor density and availability compared to females (Cosgrove et al., 2007; Duchesne et al., 2009; Laakso et al., 2002; Mozley, Gur, Mozley, & Gur, 2001). Thus, dopaminergic genetic risk and neurotransmission have been consistently associated with externalizing disorders and effortful forms of control and show consistent sex differences, similar to the serotonergic system.

Gene × Environment × Sex Interactions Across Development

Evolutionary theory suggests that genetic risk does not always operate directly on traits and behavior relevant to psychopathology, but rather appears to frequently exert effects via interactions with the environment (Bjorklund & Ellis, 2005). In fact, some genetic allelic variability, perhaps particularly that underpinning sexually selected traits, may have evolved in order to allow for individuals to adaptively respond to changing and/or unpredictable environments (B. J. Ellis et al., 2009; Flinn & Ward, 2005). Indeed, evolutionary theory suggests that sexually selected traits may be more sensitive to many environmental and cultural conditions than other traits (Geary, 2002a; Jašarević et al., 2011; McCarthy & Arnold, 2011). Based on evolutionary-neurodevelopmental theory, genetic alleles may interact with the environment so as to make traits (and individuals) more susceptible, or sensitive, to a number of negative *and* positive environmental influences, a theory known as differential susceptibility (Belsky & Pluess, 2009; B. J. Ellis, Boyce, et al., 2011; Hankin et al., 2011; Moffitt, 2005; Moffitt, Caspi, & Rutter, 2006; Rutter, 2006; Rutter, Moffitt, & Caspi, 2006).

Substantial work indicates that serotonergic and dopaminergic genes and neurotransmission are probable systems underlying these plasticity effects (reviewed in Bakermans-Kranenburg & van IJzendoorn, 2011; Beauchaine et al., 2009; Belsky & Pluess, 2009; Caspi, Hariri, Holmes, Uher, & Moffitt, 2010; Gatzke-Kopp, 2011; Karg, Burmeister, Shedden, & Sen, 2011). There is evidence of gene by environment interactions involving serotoninergic and dopaminergic function related to internalizing and externalizing disorders and associated markers. Specifically, children with particular genetic alleles (e.g., short allele of 5HTTLPR; 10-repeat allele of *DAT1*) may be more sensitive to negative environmental influences in a way that increases their risk for later internalizing and/or externalizing problems when exposed to stressors (Caspi et al., 2010; Karg et al., 2011; Martel et al., 2011; Nigg, Nikolas, & Burt, 2010; Nikolas, Friderici, Waldman, Jernigan, & Nigg, 2010). However, sexual selection theory suggests that genetic risk factors may interact differentially with environmental stressors occurring across development based on child sex. That is, there may be gene by environment interactions that operate on one sex, but not on the other, based on the developmental timing and the nature of the environmental exposure. Thus, males and females may be differentially susceptible to different types of environmental stressors during different periods of development in such a way as to lead to sex differences in traits and prevalence rates of common forms of developmental psychopathology.

Sexual Selection and Mechanisms of Timing: Organizational and Activational Hormonal Effects

The organizational-activational hypothesis of hormonal effects suggests the mechanisms that might facilitate this sort of developmental timing of sex differences in traits, acting by increasing sensitivity to the environment during select developmental periods (Geary, 2002b; McCarthy & Arnold, 2011). Briefly, the organizational-activational hypothesis suggests that gonadal hormones, including androgens (e.g., testosterone) and estrogens (e.g., estradiol), exert prominent effects on human development at distinct points of development (Phoenix, Goy, Gerall, & Young, 1959). Prominent organizational effects occur via prenatal (and early postnatal) testosterone exposure during early development and exert relatively permanent and long-lasting effects on physical and neural development with effects on sex-typed traits that appear early in development (Breedlove & Hampson, 2002; Phoenix et al., 1959). In contrast, activational hormonal effects occur first during puberty (as well as during other important transitional periods; e.g., across the menstrual cycle, during pregnancy, at menopause) via rising levels of gonadal hormones including estradiol. Activational hormonal effects are believed to activate, or "switch on," the previously organized physical and neural structures in response to somatic and environmental conditions with downstream effects on sex-typed traits (Romeo, 2003; Schulz, Molenda-Figueira, & Sisk, 2009; Sisk & Foster, 2004; Sisk & Zehr, 2005). Historically, these activational effects were thought to be much more transient in nature, compared to organizational hormonal effects, but recent work suggests that activational hormonal effects can be just as long-lived as organizational hormonal effects (Schulz et al., 2009; Sisk, Schulz, & Zehr, 2003; Sisk & Zehr, 2005).

Importantly, hormones instantiate environmental influences on the organism, supplying information about environmental context to the somatic and mental state of the individual (Plant, 2001; Terasawa & Fernandez, 2001). Sex differences in hormones may be a mechanism by which sex differences in the developmental timing of gene by environment interactions are propogated. In fact, evolutionary theory suggests that organizational effects (via testosterone) may be particularly important for males because adequate early, structural development is critical in allowing for the practice of social dominance strategies during childhood (Bjorklund, 1997), whereas activational effects (via estradiol) may be particularly important for females because, during puberty, females can become burdened with reproduction and its attendant responsibilities (B. J. Ellis, 2004; B. J. Ellis et al., 1999). Thus, the organizational-activational hypothesis of hormonal effects may be able to explain sex differences in the timing of sensitivity to relevant aspects of the environment with resulting effects on traits and behavior.

Prenatal Testosterone Effects on Childhood Sex-Typed Traits and Behavior

Prenatal testosterone appears to influence traits and behaviors relevant to psychopathology by slowing the rate of fetal growth and influencing the development of the catecholaminergic neurotransmission system (Hernandez et al., 1994; Kritzer & Creutz, 2008; Kuhn et al., 2010; Martel, Klump, et al., 2009; Morris et al., 2004), although the exact mechanisms of these effects need further study. Substantial empirical evidence supports the idea that prenatal testosterone levels are associated with sex differences in early childhood traits and behaviors relevant to psychopathology. Increased prenatal testosterone exposure has been associated with dispositional traits like low effortful control, high sensation-seeking, and lower empathy (Auyeung et al., 2009; Fink, Neave, Laughton, & Manning, 2006; Hampson, Ellis, & Tenk, 2008; Hines, 2010; Knickmeyer & Baron-Cohen, 2006; Knickmeyer, Baron-Cohen, Raggat, & Taylor, 2005; Martel, 2009; Stenstrom, Saad, Nepomuceno, & Mendenhall, 2011), as well as more masculinized patterns of childhood play (e.g., increased rough-and-tumble play) and childhood aggression (Auyeung et al., 2009; Bailey & Hurd, 2005; Hönekopp & Thierfelder, 2009). There has been some suggestion that prenatal testosterone influences these behaviors indirectly through increasing the motivation for social dominance (Coates, Gurnell, & Rustichini, 2009; Millet, 2011; Schaal, Tremblay, Soussignan, & Susman, 1996). Importantly, prenatal testosterone effects on childhood behavior seem to be stronger in males than females (Bailey & Hurd, 2005; Fink, Manning, Williams, & Podmore-Nappin, 2007; Martel, Gobrogge, Breedlove, & Nigg, 2008).

Children with ODD and/or ADHD appear to be characterized by exposure to higher levels of prenatal testosterone, measured via more masculinized finger-length ratios (Fink et al., 2007; Martel et al., 2008; McFadden, Westhafer, Pasanen, Carlson, & Tucker, 2005). Indeed, children with genetic causes of high androgen exposure during the prenatal period (e.g., Congenital Adrenal Hyperplasia) exhibit increased incidence of ADHD and other externalizing disorders (S. C. Mueller et al., 2010; Pasterski et al., 2007). Thus, the organizational effects of high prenatal testosterone appear to influence early-emerging traits and behaviors that exhibit sex differences relevant to social dominance and male-male competition, including low effortful control and high sensation-seeking, perhaps via modulation of dopaminergic neurotransmission and slowing of growth. Extreme levels of these traits appear to increase risk for childhood-onset externalizing disorders. What remains unclear is whether and how the environment might influence these effects and through what mechanisms.

Circulating Hormonal Effects on Adolescent Sex-Typed Traits and Behavior

Circulating, or activational effects of, hormones appear to have direct and indirect influences on traits and forms of psychopathology that exhibit sex differences, particularly those that exhibit prominent changes at puberty. Very low levels and/or large fluctuations in estradiol are associated with increased depression and negative emotionality, beginning at puberty and throughout the remainder of the lifespan, including postpartum and during and following menopause (Brooks-Gunn & Warren, 1989; C. M. Buchanan, Eccles, & Becker, 1992; Cohen, Soares, Vitonis, Otto, & Harlow, 2006; Paikoff, Brooks-Gunn, & Warren, 1991; Poromaa, Smith, & Guilinello, 2003; reviewed in Altemus, 2006; Martel, Klump, et al., 2009). However, the effect sizes of hormonal (i.e., estradiol) effects on depression and negative emotionality tend to be small and interactive with life events (Brooks-Gunn & Warren, 1989; reviewed by C. M. Buchanan et al., 1992; Nolen-Hoeksema, 2001).

The activational effects of ovarian hormones during adolescence also seem to exert important behavioral effects on traits, or markers, relevant to psychopathology indirectly by influencing neurotransmission, as well as other hormonal and neuropeptide systems. Ovarian hormones like estradiol directly modulate serotonergic neurotransmission (Bertrand et al., 2005; for reviews see C. M. Buchanan et al., 1992; Cameron, 2004; Steiner, Dunn, & Born, 2003) which—as already discussed—exhibits prominent sex differences (Cosgrove et al., 2007; Duchesne et al., 2009). Ovarian hormones also appear to directly influence the hypothalamic-pituitary-adrenal (HPA) axis (McCormick, Linkroum, Sallinen, & Miller, 2002; E. A. Young & Altemus, 2004; E. Young & Korszun, 2010) which is also influenced by serotonergic neurotransmission (Chen, Joorman, Hallmayer, & Gotlib, 2009; Wüst et al., 2009) in a sex-specific manner (Wankerl et al., 2010). The HPA axis is the neuroendocrine axis that regulates stress responses to the environment through the release of stress hormones such as cortisol (Fisher, Stoolmiller, Gunnar, & Burraston, 2007; Kajantie & Phillips, 2006; as reviewed by B. J. Ellis et al., 2013; Romeo, 2010; Romeo & McEwen, 2006; E. A. Young & Altemus, 2004). HPA axis activity, including cortisol levels, exhibit developmental change over time with prominent changes occurring during puberty (Gunnar, Wewerka, Frenn, Long, & Griggs, 2009; McCormick, Mathews, Thomas, & Waters, 2010; Romeo et al., 2006; Romeo & McEwen, 2006). At puberty, HPA function is characterized by prolonged activation and higher cortisol responsivity to stressors compared to childhood or adulthood (McCormick & Mathews, 2007; Netherton, Goodyer, Tamplin, & Herbert, 2004). These effects are particularly evident for females compared to males (McCormick et al., 2002; McCormick & Mathews, 2007; Netherton et al., 2004; Romeo, Lee, & McEwen, 2004; E. Young & Korszun, 2010).

Higher basal cortisol levels have been linked to higher levels of negative emotionality in children (Scher, Hall, Zaidman-Zait, & Weinberg, 2010), and experimental induction of negative emotionality has further been linked to increases in cortisol responsivity in response to stressful situations (T. W. Buchanan, al'Absi, & Lovallo, 1999; Mendonça-de-Souza et al., 2007). Higher cortisol levels and reactivity have been linked to increased rumination, even using experimental paradigms (Denson, Fabiansson, Creswell, & Pedersen, 2009; McCullough, Orsulak, Brandon, & Akers, 2007; Zoccola, Dickerson, & Zaldivar, 2008), and has been posited as a physiological mechanism of empathy (T. W. Buchanan, Bagley, Stansfield, & Preston, 2012). Further, increased cortisol reactivity appears to be more strongly related to internalizing problems, including mood and anxiety problems, in females versus males (Gunnar et al., 2009; Natsuaki et al., 2009). Thus, interactions between rapidly rising ovarian hormones and cortisol in females during puberty may operate to differentially increase susceptibility to the environment in females during this time period (B. J. Ellis, 2004), potentially via negative emotionality and rumination.

The activational effects of ovarian hormones like estradiol also appear to interact with oxytocin, a neuropeptide that has been implicated in human social and emotional behaviors such as parenting (Chibbar, Toma, Mitchell, & Miller, 1990; McCarthy, McDonald, Brooks, & Goldman, 1997; Michopoulos, Checchi, Sharpe, & Wilson, 2011). Preliminary work suggests that the oxytocin system matures substantially during the pubertal period (Chibbar et al., 1990; Tribollet, Charpak, Schmidt, Dubois-Dauphin, & Dreifuss, 1989; as reviewed in Spear, 2010) with important sex differences emerging during this period (Francis, Young, Meaney, & Insel, 2002; Insel & Young, 2000). Further, oxytocin has been linked with empathic responding in humans (Rodrigues, Saslow, Garcia, John, & Keltner, 2009). Experimentally induced increases in oxytocin increase emotional empathy (Bartz et al., 2010; Hurlemann et al., 2010), and empathy toward strangers increases oxytocin release (Barraza & Zak, 2009). These oxytocin effects seem to be mediated by the amygdala and serotonergic neurotransmission (Hurlemann et al., 2010; Jørgensen, Riis, Knigge, Kjaer, & Marberg, 2003; Kirsch et al., 2005). Oxytocin changes in females during adolescence have been interpreted to be consistent with a tend-and-befriend response to stress (Taylor et al., 2000), suggesting that oxytocin may be important for female-typical responses to stress that may increase risk for depression and anxiety.

Thus, the activational effects of ovarian hormones appear to operate directly and indirectly (via serotonergic neurotransmission, cortisol, and oxytocin) on sex differences in traits relevant to adolescent-onset internalizing disorders. Ovarian hormones appear to directly and indirectly increase negative emotionality, rumination, and empathy in females which in turn increases interpersonal competence and can facilitate female choice and/or competition. However, these traits, particularly in interaction with stressful environmental influences, may increase risk for internalizing disorders like depression and anxiety disorders.

Summary on Hormonal Effects on Timing of Environmental Sensitivity

Hormonal changes across the lifespan might propagate sex differences in traits, or markers, of psychopathology by increasing sensitivity to environmental contexts during different developmental time periods for males versus females. The organizational effects of prenatal testosterone that occur early on during the prenatal period appear to enhance traits that will shape the early practicing of behaviors that will then facilitate later male-male mate competition in males. Therefore, higher levels of prenatal testosterone might increase traits that facilitate the early practice and establishment of social dominance, thereby increasing sensation-seeking and disinhibition, perhaps via modulation of the dopaminergic neurotransmission system. Males with extreme levels of these traits, limited plasticity in these traits, and/or who express these traits in mismatched environments may then be at increased risk for externalizing psychopathology during childhood, compared to females.

In contrast, the activational effects of ovarian hormones that occur during puberty in females may directly and indirectly enhance traits that facilitate nurturance and the development of social networks important for female choice, as well as reproduction and parenting. That is, increases in ovarian hormones like estradiol may interact with the HPA axis and stress hormones like cortisol, as well as neuropeptides like oxytocin and serotonergic neurotransmission, to increase negative emotionality, empathy, and rumination. Females with extreme levels of these traits, limited plasticity in these traits, and/or who express these traits in stressful environments may be at increased risk for internalizing psychopathology like depression and some anxiety disorders during adolescence.

Sexual Selection and Prenatal Ecological Environmental Influences on Externalizing Disorders and Markers in Males

Evolutionary theory and the organizational-activational hypothesis suggest that high levels of prenatal testosterone slow down fetal development during the prenatal period in males to allow them an increased opportunity to adjust to environmental conditions, maximize their fitness, and compete more effectively (Geary, 2010; Geschwind & Galaburda, 1985; Glover, 2011; Morris et al., 2004; Stinson, 1985; Talge et al., 2007). In this way, prenatal testosterone exposure may make males more sensitive to cues about ecological environmental conditions during the prenatal period and/or may make males sensitive to environmental conditions for a longer period of time during the prenatal period, compared to females, so social dominance-related traits can accordingly flexibly adjust. These effects may be mediated in part by the dopaminergic neurotransmission system due to its susceptibility to alterations in the prenatal environment and its purported modulation by androgens like testosterone (Hernandez et al., 1994; Kritzer & Creutz, 2008; Kuhn et al., 2010; Martel, Klump, et al., 2009; Morris et al., 2004).

Prenatal environmental influences on childhood externalizing disorders have received extensive attention (Disney, Iacono, McGue, Tully, & Legrand, 2008; Nigg & Breslau, 2007; Wakschlag, Pickett, Cook, Benowitz, & Leventhal, 2002). Work to date suggests that prenatal environmental influences on externalizing disorders such as ADHD operate primarily via their interaction with genes influencing dopaminergic neurotransmission (reviewed by Nigg et al., 2010). Specifically, functional dopaminergic genetic markers like the 10-repeat allele of *DAT1* have been shown to interact with prenatal environmental conditions, particularly in instances where the prenatal environment suggests unpredictable or harsh environmental conditions (e.g., through maternal use of alcohol and nicotine during pregnancy), to increase risk for childhood externalizing problems (Becker, El-Faddagh, Schmidt, Esser, & Laucht, 2008; Brookes et al., 2006; Kahn, Rhoury, Nichols, & Lamphear, 2003; Langley, Holmans, van den Bree, & Thapar, 2007; Langley et al., 2008; Neuman et al., 2007; Pluess, Belsky, & Neuman, 2009). One study found that the interaction between the *DAT1* allele and prenatal smoke exposure influences hyperactive-impulsive ADHD symptoms in males but not females (Becker et al., 2008), although most studies have not examined males and females separately or three-way interactions specifically. Further, interactions between dopaminergic genetic risk (i.e., *DRD2*) and prenatal nicotine use are associated with inattention and executive dysfunction (Wiebe et al., 2009), although effects may be at least partially confounded with other environmental influences (D'Onofrio et al., 2010, 2007).

However, in contrast to work on prenatal substance abuse, prenatal maternal stress appears to increase risk for psychopathology in both males and females (Bergman, Sarkar, Glover, & O'Connor, 2010; Goel & Bale, 2009; Huizink, Robles de Medina, Mulder, Visser, & Buitelaar, 2003; B. R. Mueller & Bale, 2008; T. G. O'Connor, Heron, Golding, Beveridge, & Glover, 2002; reviewed by Harris & Seckl, 2011; Huizink, Mulder, & Buitelaar, 2004) with sex-specific effects on outcomes based on the timing of the exposure. That is, females appear sensitive to maternal stress only early on during pregnancy with effects on the HPA axis and later depression and anxiety, whereas males appear to be more sensitive to maternal stress throughout the duration of pregnancy with effects on the hippocampus and the catecholaminergic system, as well as later attention and cognition (Bowman et al., 2004; Brunton & Russell, 2010; Goel & Bale, 2009; Kapoor, Dunn, Kostaki, Andrews, & Matthews, 2006; T. G. O'Connor et al., 2002; Sarkar, Bergman, O'Connor, & Glover, 2008; Weinstock, 2007).

Thus, overall, sexual selection theory suggests that males may be particularly vulnerable to prenatal environmental influences signaling a harsh and unpredictable ecological environment, such as maternal substance abuse, perhaps via a longer sensitive period during the prenatal period, so that they can sensitively adjust their early development to match the environment to more effectively compete for social status. In line with this idea, limited research to date supports the idea that males may be particularly sensitive to prenatal environmental stressor effects (e.g., maternal substance abuse) that influence effortful control and executive function which can, in turn, increase risk for ADHD and other externalizing disorders. Further, males homozygous for the *DAT1* 10-repeat allele may exhibit particular sensitivity to these prenatal environmental conditions. However, females also appear vulnerable to some types of prenatal stressors, particularly those occurring early during pregnancy and those involving the emotional state of the caregiver.

Sexual Selection and Adolescent Interpersonal Environmental Influences on Internalizing Disorders and Markers in Females

In contrast, evolutionary theory suggests that females' interpersonal competence will be particularly important for successful and supported reproduction, beginning at puberty and through adolescence and adulthood (Colle & Del Giudice, 2011; Geary, 2010). Activational hormonal effects may influence the HPA axis (and cortisol), oxytocin, and serotonergic neurotransmission that impact traits such as negative emotionality, rumination, and empathy that—in turn—increase females' responsivity and vulnerability to a wide variety of proximal interpersonal stressors that become particularly numerous and salient at puberty, including abuse, socialization and peer influences, as well as sensitivity to cultural ideals and pressures (Hinshaw & Kranz, 2009). In line with this idea, empirical work suggests that females appear to be more vulnerable to proximal interpersonal stressors involving family members, peers, and romantic partners, compared to males (Davies & Windle, 1997; Hankin et al., 2007; Keenan & Hipwell, 2005; Rose & Rudolph, 2006; Shih, Eberhart, Hammen, & Brennan, 2006). Further, females are more likely to experience negative emotionality (and depression) following exposure to psychosocial environmental risk factors such as childhood abuse, stressful life events, and marital conflict, compared to males (Bouma, Ormel, Verhulst, & Oldehinkel, 2008; Cambron et al., 2009; Conley, Rudolph, & Bryant, 2012; Davies & Lindsay, 2004; Oldehinkel & Bouma, 2011; Richmond & Stocker, 2007; Rudolph & Hammen, 1999; Thompson, Kingree, & Desai, 2004; Yuan et al., 2009). Females' increased vulnerability to stress and problem behavior following negative interpersonal life events appears to become particularly striking during adolescence (Conley et al., 2012; Davies & Windle, 1997; Hankin & Abramson, 2001; Leadbeater,

Blatt, & Quinlan, 1995), possibly at least partially due to increased interpersonal stressors occurring during this epoch in females compared to males (Hinshaw & Kranz, 2009; Shear, Halmi, Widiger, & Boyce, 2007).

Culture plays a particularly important role in influencing the types, intensity, and chronicity of interpersonal stressors encountered during this period for females and males and, thus, exerts effects on both the rates and presentation of psychopathology. For example, in several cultures (e.g., Mediterranean countries, Amish, orthodox Jews), the female role is highly valued, and there are presumably decreased interpersonal stressors for females; in these cultures, there are no—or minimal—sex differences in rates of depression (as reviewed by Piccinelli & Wilkinson, 2000). In addition, the presentation of disorders can be influenced by the ideology and goals of a culture (e.g., culture-bound syndromes; e.g., *koro, ataque de nervios*; Levine & Gaw, 1995). As but one example, in Japan, a culture characterized by collectivism, taijin kyofusho is a common anxiety disorder characterized by fear of offending others with inappropriate behavior or appearance (Kirmayer, 1991). Further, the cultural context may be very important in modulating the impact of environmental influences on externalizing disorders. For example, the effect of parental physical discipline on child externalizing behaviors appears to depend on the cultural context (Deater-Deckard, Dodge, Bates, & Petit, 1996). Finally, the peer group context may be important for some kinds of psychopathology (Maccoby, 2002).

Functional serotonin genetic markers, like the short allele of 5HTTLPR, interact with family environmental factors like parental conflict, maltreatment, and stressful life events to predict internalizing problems like depression, as well as externalizing problems (Canli & Lesch, 2007; Caspi et al., 2003; Karg et al., 2011; Kochanska, Philibert, & Barry, 2009; Laucht et al., 2009; Li & Lee, 2010; Nikolas et al., 2010; Pluess, Belsky, Way, & Taylor, 2010; Sonuga-Barke et al., 2009; for negative findings, see Gillespie, Whitfield, Williams, Heath, & Martin, 2005; Risch et al., 2009). Females with this genetic liability may be particularly vulnerable to interpersonal stressors. There has been some support for this idea, particularly in the depression literature. Females with a parent with a lifetime depressive episode appear more vulnerable to developing depression after experiencing a stressful life event, compared to males (Bouma et al., 2008). Further, females with the short allele of 5HTTLPR appear more susceptible to the effects of chronic stress, family stress and adversity, and relational aggression with outcomes of increased psychopathological symptoms, particularly depression (Åslund et al., 2009; Barr et al., 2004; Benjet, Thompson, & Gotlib, 2010; Brummett et al., 2008; Eley et al., 2004; Hammen, Brennan, Keenan-Miller, Hazel, & Najman, 2010; Sjöberg et al., 2006).

Limited work also suggests that these kinds of gene by environment interactions are operative in relation to cortisol, amygdala resting activation, and dispositional traits associated with negative emotionality. Individuals with the short allele of 5HTTLPR genotype exhibit elevated cortisol levels in response to a stressor, particularly when they have a history of negative life events (Alexander et al., 2009; Gotlib, Joormann, Minor, & Hallmayer, 2008; Way & Taylor, 2010). Further, 5HTTLPR genotype interacts with life stress to influence amygdala resting activation (Canli et al., 2006). Fox et al. (2005) found an interaction between the short allele of 5HTTLPR and lower levels of social support predicting increased behavioral inhibition in middle childhood. Further, the short allele of 5HTTLPR interacts with negative life events to predict higher levels of neuroticism in healthy young adults (Pluess et al., 2010). Finally, infants with the short allele of 5HTTLPR who were also insecurely attached develop higher levels of negative emotionality (Pauli-Pott, Friedl, Hinney, & Hebebrand, 2009). What remains to be tested is whether these types of interactions are—in turn—moderated by sex such that females are driving these effects.

Thus, females, particularly those with the short allele of 5HTTLPR, may be more sensitive to interpersonal stressors during puberty with specific effects on negative emotionality and amygdala activation patterns, as well as mood and anxiety problems. Although male susceptibility to the environment during puberty has been less frequently studied (Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004), males are certainly susceptible to some childhood and adolescent environmental stressors, perhaps particularly those related to hierarchies (e.g., coercive parenting; Patterson, DeBaryshe, & Ramsey, 1989) and establishment of social dominance in the peer group (e.g., peer deviancy; Dishion, Spracklen, Andrews, & Patterson, 1996; Snyder et al., 2008).

Summary of Posited Gene × Environment × Sex Interactions Based on Developmental Period

Overall, gene by environment interactions may operate differentially based on sex due to hormonal mechanisms that increase sensitivity to certain aspects of the environment in males and females at key, but distinct, development periods. Males may be more vulnerable to prenatal stressors that provide clues about the broad ecological environment (e.g., maternal substance abuse) due to their higher levels of prenatal testosterone with downstream effects on disinhibition, sensation-seeking, and externalizing disorders during childhood, perhaps via dopaminergic neurotransmission. In contrast, females may be more vulnerable to later-occurring, culturally shaped interpersonal environmental stressors due to their higher levels of ovarian hormones, cortisol, and oxytocin which influence serotonergic neurotransmission and amygdala activation. These factors may heighten negative emotionality, rumination, and/or empathy which—in turn—may increase risk for internalizing disorders in females during adolescence.

Sexual Selection Model of Male Vulnerability for Childhood-Onset Externalizing Disorders

A sexually selected pathway to childhood externalizing disorders via facilitation of male-male competition may center on the male-biased traits of sensation-seeking (or approach) and disinhibition (see Figure 2). These traits would be sexually selected because, in the past, they presumably increased social dominance, including males' ability to compete with other males for resources and/or dominance and thus reproductive opportunity. These traits are likely to become important early during childhood as they allow males to practice and refine behaviors (e.g., rough-and-tumble play, aggression) that increase social dominance and the likelihood of having a mate during and following puberty (Bjorklund, 1997). The emergence of sex differences in these traits during childhood would have allowed males in hunter-gatherer

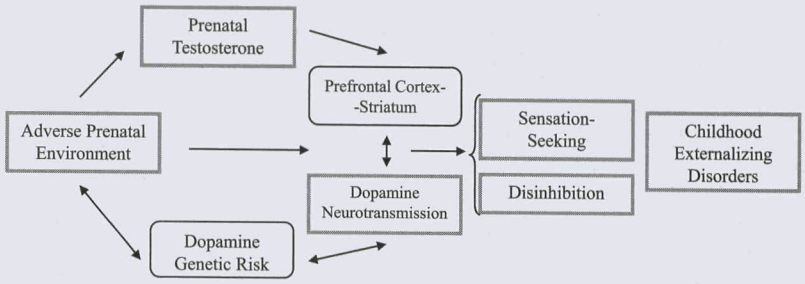

Figure 2. Sexual selection model of childhood externalizing disorders. Prenatal testosterone exposure increases male susceptibility to prenatal environmental stressors, particularly for those with dopaminergic genetic risk, influencing the development of the prefrontal-striatal circuitry and dopaminergic neurotransmission. Individual differences in prefrontal-striatal circuitry and dopaminergic neurotransmission increase sensation-seeking and disinhibition, which increase risk for childhood-onset externalizing disorders.

societies to establish social status early in their communities and/or practice these dominance-related traits before the onset of puberty, when they became more critical.

The neural systems underlying sensation-seeking and disinhibition may be sensitive to environmental conditions for a longer period of time during the prenatal period in males due to the mechanism of higher prenatal testosterone exposure. That is, higher prenatal testosterone exposure may lengthen the sensitive period during which males (vs. females) are susceptible to the effects of prenatal environmental conditions, particularly those signifying ecological information about harsh and/or unpredictable environments (e.g., maternal alcohol/nicotine use), via modulation of the development of the dopaminergic neurotransmission system. This increased prenatal sensitivity can be considered adaptive in relation to reproductive success in that it increases variability in traits conditional on cues about current ecological environmental conditions to facilitate flexible adaptation to the environment. Those with specific dopaminergic genetic alleles may be particularly sensitive to the effects of adverse prenatal environmental exposures on behavioral outcomes because they are already predisposed to low functionality in the dopamine system and associated prefrontal cortex-striatum circuitry which underpin high levels of sensation-seeking and disinhibition.

Thus, males exposed to high levels of prenatal testosterone who have dopaminergic genetic risk alleles and who *also* experience high levels of prenatal stress are likely to exhibit extremely high and/or inflexible levels of sensation-seeking and disinhibition. This, then, may predispose this subset of males to externalizing disorders during childhood, perhaps particularly when these traits are expressed in certain kind of familial and peer environments (e.g., coercive rearing environments: Patterson et al., 1989; when exposed to deviant peers: Dishion et al., 1996; Snyder et al., 2008). Based on this theory, one might expect that a subset of males (and a larger subset of males compared to females) who have higher prenatal testosterone exposure, dopaminergic genetic risk alleles, and who also experience high levels of ecological stressful events early on such as prenatal maternal substance abuse will have higher levels of sensation-seeking and disinhibition during early childhood which will—in turn—predispose them to externalizing disorders.

Specific Hypotheses Based on a Sexual Selection Theory of Childhood-Onset Externalizing Disorders

I now reconsider the hypotheses presented earlier in more detail, providing more specific predictions in relation to childhood-onset externalizing disorders, including identification of independent variables (IV), dependent variables (DV), mediating variables, and moderating variables. Recall that the first set of predictions are the core predictions from the model, being particularly germane to its support/refutation, and the second and third sets of predictions extend these initial predictions through consideration of developmental and environmental mechanisms.

1. If sexual selection pressures in the evolutionary past are relevant to the male-biased prevalence rates of childhood-onset externalizing disorders today, then there will be sex differences in traits that function to increase intrasex reproductive advantage by increasing social dominance, and they will help explain sex differences in the prevalence rates of childhood-onset externalizing disorders. Specifically:
   a. As a group, males will exhibit higher sensation-seeking and disinhibition than females.
   b. Extremely high levels of the sexually selected traits of sensation-seeking and disinhibition (IV) will predict childhood-onset externalizing disorders (DV) and will explain the male-biased prevalence rate of childhood-onset externalizing disorders.
   c. The sexually selected traits of sensation-seeking and disinhibition will mediate, or explain, the etiological effects of prenatal stress exposure (IV) and alterations in dopaminergic neurotransmission (IV) on childhood-onset externalizing disorders (DV).

2. If sexual selection processes impacted life history strategies relevant to childhood-onest externalizing disorders differentially in males and females in the evolutionary past, then there will be sex differences in the timing of environmental sensitivity (i.e., sex by developmental timing interaction) associated with childhood-onset externalizing disorders and associated sexually selected traits, or markers. Specifically:
   a. If males' achievement of social dominance during the reproductive years requires a long period of practice and refinement of associated physical and social competencies that

facilitate intrasex competition, then males will have a longer sensitive period during early development than females; this sensitive period will begin early during the prenatal period and continue into early childhood to allow for play-related refinement of social dominance strategies. That is, the influence of prenatal stressors (IV) on externalizing disorders (DV) and its associated sexually selected traits (i.e., sensation-seeking, disinhibition; DV) will be stronger for males compared to females (i.e., child sex as moderator).

b. High prenatal (and early postnatal) testosterone exposure will mediate, or explain, male sensitivity to the prenatal environment during pregnancy. That is, prenatal testosterone (mediator) will explain the effects of prenatal stressors (IV) on externalizing disorders (DV) and associated sexually selected traits (i.e., sensation-seeking, disinhibition; DV), especially for boys, compared to girls (child sex as moderator; a moderated mediation model).

c. High prenatal (and early postnatal) testosterone exposure effects on childhood-onset externalizing disorders and traits of sensation-seeking and disinhibition will be mediated through the dopaminergic neurotransmission system and prefrontal cortex-striatal circuitry in response to early stress exposure, particularly for those with genetic risk influencing the dopaminergic neurotransmission system. In other words, dopaminergic neurotransmission (mediator) and prefrontal cortex-striatal circuitry (mediator) will explain associations between prenatal testosterone (IV) and externalizing disorders (DV) and associated sexually selected traits (i.e., sensation-seeking, disinhibition; DV), especially for those exposed to prenatal stressors (moderator) and those with genetic risk influencing the dopaminergic neurotransmission system (moderator; a moderated multiple mediation model).

3. If males' and females' reproductive capacities were dependent on different types of environmental constraints during the evolutionary past, then there will be sex differences in the type of environmental cues to which males and females are differentially susceptible (i.e., a sex by environment interaction) in relation to childhood-onset externalizing disorders and associated sexually selected traits, or markers. Specifically:

a. Males will be differentially susceptible to early-occurring stressors providing information about the broader ecological environment and its resources (e.g., maternal substance abuse, nutrition, prenatal care), compared to females, with effects on childhood-onset externalizing disorders because the broader ecological environment is important for the development of strategies (e.g., sensation-seeking and disinhibition) that will facilitate successful competition and reproduction. These types of early-occurring stressors (IV) will predict the sexually selected traits of sensation-seeking and disinhibition (mediators) which will increase risk for childhood-onset externalizing disorders (DV), especially for males versus females (child sex as moderator; moderated mediation).

b. Cultural influences on early ecological stressors will influence the sexually selected traits of sensation-seeking and disinhibition which will affect the prevalence rate of childhood-onset externalizing disorders. For example, coercive cycles with caregivers and teachers (IV) and deviant interactions with peers (IV) will interact with (and increase) child sensation-seeking and disinhibition (moderators) to influence externalizing disorders (DV).

Sexual Selection Model of Female Vulnerability for Adolescent-Onset Internalizing Disorders

A sexually selected pathway to adolescent internalizing disorders via facilitation of female choice and/or competition may center on the female-biased traits of negative emotionality, empathy, and rumination, shown in Figure 3. These traits may have been sexually selected because they were adaptive in regard to facilitating females' interpersonal competence and dominance by increasing sensitivity to interpersonal contexts so as to facilitate females' ability to generate and manipulate the social networks that are important for mate choice, female-female competition,

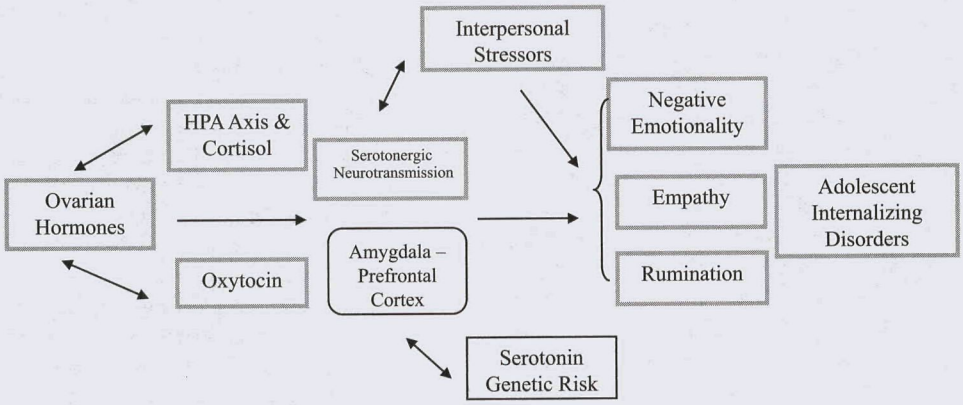

Figure 3. Sexual selection model of adolescent internalizing disorders. Rising levels of ovarian hormones in females during puberty interact with cortisol and oxytocin to influence serotonergic neurotransmission and amygdala-prefrontal cortex circuitry function. Individual differences in serotonergic neurotransmission and amygdala-prefrontal cortex circuitry underpin individual differences in negative emotionality, empathy, and rumination that, in the context of exposure to interpersonal stressors and seroninergic genetic risk, lead to adolescent-onset internalizing disorders. HPA = hypothalamic-pituitary-adrenal.

pregnancy, and childrearing in hunter-gatherer societies (Colle & Del Giudice, 2011; Geary, 2010). Negative emotionality, empathy, and rumination may be preferentially sensitive to interpersonal environmental conditions during childhood and early adolescence due to the importance of puberty and the timing of puberty for the reproductive success of females in the past.

Increases in ovarian hormones during puberty in females have interactive effects on the HPA axis and cortisol, as well as the neuropeptide oxytocin. These hormones influence the amygdala and serotonergic neurotransmission, potentially thereby increasing negative emotionality, empathy, and rumination, particularly at puberty. These traits and cognitive patterns then may increase females' vulnerability to culturally influenced interpersonal stressors, perhaps most particularly for females with certain serotonergic genotypes. Further, when expressed in particularly stressful interpersonal environments, high negative emotionality, empathy, and cognitive rumination may make a subset of females more vulnerable to adolescent-onset internalizing disorders like depression and some anxiety disorders.

Thus, based on this theory, one might expect that a subset of females (and a larger subset of females compared to males) who have large increases in ovarian hormones at puberty in combination with highly stressful interpersonal events occurring during late childhood or early adolescence and serotonergic risk alleles will exhibit extremely high negative emotionality, empathy, and rumination and will be more likely to develop depressive and/or anxiety disorders.

Specific Hypotheses Based on a Sexual Selection Theory of Adolescent-Onset Internalizing Disorders

I now reconsider the hypotheses presented earlier in more detail, providing more specific predictions in relation to adolescent-onset internalizing disorders, including identification of independent variables (IV), dependent variables (DV), mediating variables, and moderating variables. The first set of predictions are the core predictions from the model, being particularly germane to its support/refutation, and the second and third sets of predictions extend these initial predictions by consideration of developmental and environmental mechanisms.

1. If sexual selection pressures in the evolutionary past are relevant to the female-biased prevalence rates of adolescent-onset internalizing disorders today, then there will be sex differences in traits that function to increase intrasex reproductive advantage by increasing interpersonal competence and dominance, and they will explain sex differences in the prevalence rates of adolescent-onset internalizing disorders. Specifically:

a. As a group, females will exhibit higher negative emotionality, empathy, and rumination compared to males.
b. Extremely high levels of the sexually selected traits of high negative emotionality, empathy, and rumination (IV) will predict adolescent-onset internalizing disorders (DV) and will mediate, or explain, sex differences in the prevalence rates of adolescent-onset internalizing disorders.
c. The sexually selected traits of high negative emotionality, empathy, and rumination during adolescence will mediate, or explain, the effects of rapidly rising levels of ovarian hormones (IV) and alterations in serotoninergic neurotransmission (IV) on adolescent-onset internalizing disorders (DV).

2. If sexual selection processes impacted life history strategies relevant to adolescent-onest internalizing disorders differentially in males and females in the evolutionary past, then there will be sex differences in the timing of environmental sensitivity (i.e., sex by developmental timing interaction) associated with common forms of psychopathology and associated sexually selected risk markers, or traits. Specifically:

a. If females' interpersonal competence becomes important for intrasex competition, mate choice, and reproduction at puberty, then females will have a longer sensitive period during early adolescence than males; this sensitive period will begin shortly before or at puberty and will last into adolescence to allow for establishment of traits that will facilitate the development of intimate relationships and social support needed for sustaining pregnancy and child-rearing. Therefore, adolescent stressors (IV) will have a particularly prominent effect on adolescent-onset internalizing disorders (DV) and associated sexually selected traits (i.e., negative emotionality, empathy, rumination; DV) for females compared to males (sex as moderator).
b. Rising levels of ovarian hormones (IV) will interact with the HPA axis and cortisol and oxytocin (moderators) to increase sensitivity to the interpersonal environment during adolescence (DV) via increasing negative emotionality, empathy, and rumination (mediators; mediated moderation), particularly for females (sex as moderator; moderated mediation).
c. Ovarian hormones directly and indirectly via interactions with cortisol and oxytocin will influence amygdala function and serotonergic neurotransmission, with particularly adverse effects on negative emotionality, empathy, rumination, and adolescent-onset internalizing disorders in response to interpersonal stressor exposure, particularly for those with genetic risk influencing the serotonergic neurotransmission system. So, ovarian hormones directly (IV) and in interaction with cortisol and oxytocin (moderators) will influence serotonergic neurotransmission and amygdala function (mediators; mediated moderation model) which will—in turn—influence negative emotionality, empathy, rumination, and adolescent-onset internalizing disorders (DVs), particularly for those exposed to interpersonal stressors (moderator) and for those with serotonergic genetic risk (moderator; moderated mediation model).

3. If males' and females' reproductive capacities were dependent on different types of environmental constraints during the evolutionary past, then there will be sex differences in the type of environmental cues to which males and females are differentially susceptible (i.e., sex by environment interaction) in relation to adolescent-onset internalizing disorders and associated sexually selected traits, or markers. Specifically:

a. Females will be differentially susceptible to stressors that provide information about the cohesion of interpersonal relationships (e.g., social support, intimacy) at puberty, compared to males, because they have a large impact on intrasex competition, mate choice and retention, pregnancy, and child-rearing, and these stressors will impact the sexually selected traits of negative emotionality, empathy, and rumination, traits that can facilitate the development of interpersonal relationships and increase risk for adolescent-onset internalizing disorders. Thus, interpersonal stressors (IV)

will influence negative emotionality, empathy, and rumination (mediators), particularly in females compared to males (sex as a moderator), and these traits will increase risk for adolescent-onset internalizing disorders (DV; mediated moderation model).

b. Culture will play a particularly important role in influencing the types, intensity, and chronicity of interpersonal stressors encountered during puberty and adolescence and will exert strong effects on the sexually selected traits of negative emotionality, empathy, and rumination, as well as prevalence of adolescent-onset internalizing disorders. That is, culture will moderate the effect of interpersonal stressors (IV) on the sexually selected traits of negative emotionality, empathy, and rumination (DV) and adolescent-onset internalizing disorders (DV). For example, in cultures (e.g., Mediterranean countries, Amish, orthodox Jews) in which the female role is highly valued and there are decreased interpersonal stressors for females, there will be no—or minimal—effects of interpersonal stressors on negative emotionality, empathy, rumination, or rates of depression. In cultures in which the female role is less valued and there are increased interpersonal stressors for females, there will be strong effects of interpersonal stressors on negative emotionality, empathy, rumination, and rates of depression. Cultural differences will influence the target and expression of anxiety (e.g., social anxiety will be more common in more collectivist cultures), as well as the developmental trajectories of normative and atypical fears (e.g., separation anxiety will be more common during early childhood, and social anxiety will be more common during adolescence).

Other Considerations: Comorbidity

Aside from the sex-biased prevalence rate of many forms of psychopathology, another common feature of many disorders is their high comorbidity, or high co-occurrence (APA, 2000; Angold et al., 1999). At first, the high comorbidity of externalizing and internalizing disorders, or their co-occurrence in approximately 30% of cases (Angold et al., 1999), might seem to challenge a theory of sex differences in pathways to externalizing and internalizing disorders. The just-described pathways developed using an evolutionary framework may provide some insight into this phenomenon. Namely, negative emotionality may serve as a shared mechanism for externalizing and internalizing psychopathology, predisposing children to both forms of psychopathology (Carver et al., 2008; Lahey, 2009). In fact, comorbidity is higher in females, as might be expected based on this idea (Costello et al., 2003). However, negative emotionality may interact with, or be modulated by, levels of approach and effortful control to differentiate children with "pure" externalizing and "pure" internalizing disorders from children with comorbid externalizing and internalizing disorders (Carver et al., 2008). Since males typically exhibit higher levels of approach and females typically exhibit higher levels of effortful control, these sex-specific profiles of traits may result in males being more likely to exhibit "pure" externalizing disorders and females being more likely to exhibit "pure" internalizing disorders.

As shown in Figure 4, children with "pure" externalizing disorders may be characterized by a more male-typical profile of traits including high levels of disinhibition (or low effortful control), high approach, and high negative emotionality manifested

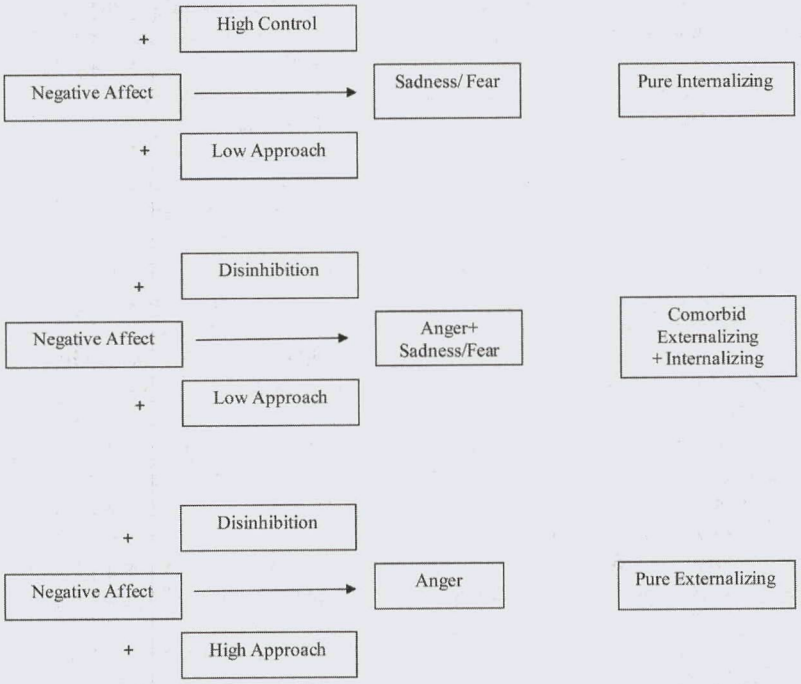

Figure 4. Sexual selection model of comorbidity of internalizing and externalizing disorders. Negative emotionality interacts with different profiles of disinhibition and approach to increase risk for "pure" versus "comorbid" internalizing/externalizing profiles.

primarily as anger. In contrast, children with "pure" internalizing disorders may be characterized by a more female-typical profile of traits including low approach, high effortful control (or low disinhibition) and high negative emotionality which primarily manifests itself as sadness in depression or fear in anxiety disorders. Finally, children with comorbid externalizing and internalizing disorders may follow a pathway that incorporates components of both the male- and female-typical pathways described above, exhibiting high negative emotionality, low effortful control (or high disinhibition), and low approach. Further, they may have multiple differential susceptibility genes (e.g., *DAT1*, 5HTTLPR), they may have more variable hormone patterns during the prenatal period and puberty, and they may be exposed to both early-occurring ecological and later-occurring interpersonal stressors (which, may, in many cases, be confounded; D'Onofrio et al., 2010, 2007). Thus, children with comorbid externalizing and internalizing disorders may experience a number of different etiological and mechanistic risks which then predispose them to all forms of negative emotionality, including fear, sadness, and anger. These children may also experience the most subjective distress associated with their symptomatology because they do not exhibit the buffering effect of high approach evident in children with "pure" externalizing disorders or high effortful control seen in children with "pure" internalizing disorders.

Linkage With Prior Work and Theory on Sex Differences in Psychopathology

Framing psychopathology in the context of sexual selection may also shed light on why prior work on sex differences in psychopathology and related psychological and behavioral phenomenon has been somewhat contentious. That is, although some research has demonstrated that males and females are different from one another (e.g., Cahill, 2005, 2010), other research has demonstrated that males and females are relatively similar to one another (e.g., reviewed in Hyde, 2005), depending on the domain assessed, as well as the magnitude of the effect used to justify claims of difference or similarity. Evolutionary theory sheds light on this phenomenon by suggesting that there will be sex differences in only those domains that contribute to reproductive fitness (e.g., psychological/social, biological, physical; Geary, 2002a, 2010) with minimal or nonexistent sex differences in other domains (e.g., Kling, Hyde, Showers, & Buswell, 1999). Further, sexual selection theory suggests substantial intrasex variability which may, in some instances, trump intersex differences (Darwin, 1871). Finally, sex differences in some traits may only emerge during particular sexual selection-relevant developmental periods (e.g., sex differences in negative emotionality may only emerge during adolescence) and in response to particular environmental influences because sexually selected traits exhibit increased sensitivity to environmental influences (Geary, 2002a, 2002b, 2010; B. J. Ellis et al., 2012; West-Eberhard, 2003). Although such environmental influences can increase risk for psychopathology, they can also be harnessed, through interventions for example, to facilitate resilience to psychopathology (Bakermans-Kranenburg, van IJzendoorn, Mesman, Alink, & Juffer, 2008).

Further complicating matters, research has indicated that there is substantial variability within sex that, in some cases, is larger than variability between sexes (Holden, 2005; Pomiankowski & Moller, 1995; Rowe & Houle, 1996). Evolutionary theory also sheds light on this phenomenon by positing that sexually selected traits exhibit substantial intrasex variability and have evolved to be more sensitive to environmental and cultural conditions than other traits. As a result, sexually selected traits are often more variable than other traits (Jašarević et al., 2011; Rowe & Houle, 1996). In fact, sexual selection theory would predict *both* intersex differences, as well as intrasex variability. That is, even though each sex has its own normative pattern of strengths and weaknesses, there will always be individual variation around that normative pattern. This maximizes the reproductive success of the individual and the species by allowing for more flexible adaptation to changing environmental conditions. In addition, it suggests that the pathway of all males or all females will not be the same, consistent with developmental pathway models of psychopathology (e.g., Frick & Morris, 2004; Moffitt, 1993; Sonuga-Barke, 2005). Thus, although the theory of sexual selection suggests that markers of externalizing and internalizing disorders may be different in females and males, at the group level, ultimately rendering males more vulnerable to childhood externalizing disorders and females more vulnerable to adolescent internalizing disorders, individual differences within sex are also notable and appear to be based on variability in genetic alleles and hormone levels, sensitivity to environmental conditions, and actual and experienced environmental conditions.

Further, sex (and gender) is complex and multidetermined without being deterministic in any sense, meaning that individuals will vary even in their "maleness" or "femaleness" (Baron-Cohen, 2006; Pinel, 2011). For example, although males are typically exposed to higher levels of prenatal testosterone (as only one component of their "maleness"), compared to females, there will be individual differences in the exact amount of fetal testosterone to which a male fetus is exposed (Blanchard, 2001). Thus, although sex differences are an important category of individual differences that may be able to shed light on the development of psychopathology, they are not more important than other kinds of individual differences and will not always be important for understanding every given marker, mechanism, or etiological factor related to psychopathology, or even for understanding every individual.

Sexual selection also provides a provisional framework for the integration of two theories of sex differences in etiology of psychopathology: the polygenic multiple threshold model and the constitutional variability model (Arnold, 1996; Rhee, Waldman, Hay, & Levy, 1999, 2001). The polygenic multiple threshold model states that sex differences in psychopathology are evident because the less frequently affected sex may require a higher liability (i.e., more of the same causal risk factors) than the more commonly affected sex. In contrast, the constitutional variability model suggests that sex differences in psychopathology are due to the involvement of different risk factors for each sex.

Based on the evolutionary theory of sex differences in psychopathology presented here, both theories might be able to explain sex differences in etiology of psychopathology by describing the process by which male- or female-specific markers affect psychopathology at different levels of analysis. That is, males may require a higher liability for adolescent-onset internalizing disorders to exhibit the female-biased markers of negative emotionality, empathy, and rumination, consistent with the polygenic multiple threshold model. However, males—but not females—may be most susceptible to the etiological effects of prenatal substance abuse occurring across the

prenatal period on childhood-onset externalizing disorders due to their higher levels of prenatal testosterone, in line with the constitutional variability model. Likewise, females might require a higher liability for childhood-onset externalizing disorders to exhibit the male-biased markers of disinhibition and sensation-seeking, consistent with the polygenic multiple threshold model. However, females—but not males—may be most susceptible to the etiological effects of interpersonal stressors on adolescent-onset internalizing disorders due to their pubertal hormone levels, in line with the constitutional variability model.

Applied Directions for Future Work

Thus, evolutionary sexual selection theory (and life history theory) provides a way to integrate and consolidate what is currently known about sex differences in common forms of psychopathology such as externalizing and internalizing disorders. However, perhaps more important, these theories, as organized in this review, provide a framework that is able to generate specific, testable, and refutable hypotheses. Since many view evolutionary theory as post hoc and irrefutable, such a priori hypotheses provide an important means by which to test the usefulness of sexual selection theory for organizing work on sex differences in common forms of psychopathology. Thus, the metatheory presented in this article advances work in this area by suggesting a number of specific, a priori hypotheses and predictions at the proximate level that have not yet been empirically tested. In addition, the theory provides a way to organize, synthesize, and extend hypotheses generated by prior theories that have focused more exclusively on one disorder or one level of analysis.

Empirical testing of the hypotheses and predictions generated by the sexual selection theory of childhood-onset externalizing and adolescent-onset internalizing disorders presented in this article can advance the field's understanding of sex differences and their influence on and/or relation to common forms of psychopathology. The use of specific testable hypotheses is deemed to be more advantageous than the post hoc and largely exploratory examination of possible sex differences and sex effects, like is often currently used in the field (Cahill, 2010; Geary, 2006; ORWH, 2010; "Putting Gender on the Agenda," 2010). Challenges to and/or replacement of this theory will only push forward our understanding of the complex topic of sex differences in risk markers, mechanisms, etiology, and timing of onset of common forms of psychopathology.

Although the current review focused on childhood-onset externalizing and adolescent-onset internalizing disorders, this general evolutionary framework may be useful for understanding other disorders with a sex-biased prevalence rate, including substance dependence, eating disorders, and personality disorders like antisocial and borderline personality disorders. Further, this theory suggests some important topic areas that need further empirical attention, perhaps most particularly cultural environmental influences on psychopathology. In addition, it suggests important new targets for work on candidate genes and environmental factor interactions. In regard to childhood-relevant gene by environment interactions, other genes influencing the dopaminergic neurotransmission system (e.g., DRD4) may be important candidate genes to consider, and other prenatal environmental stressors providing cues about the broader ecological environment (e.g., maternal nutrition and prenatal care during pregnancy) may be important environmental factors to consider. In regard to adolescent-relevant gene by environment interactions, genes influencing the oxytocin system (e.g., oxytocin receptor) may be important candidate genes to consider, and environmental influences providing information about the interpersonal environment (e.g., social support) may be interesting new leads in the environmental domain. More generally, this metatheory suggests the importance of integrating across numerous, disparate topic domains (e.g., biology, social) to elucidate a complex topic like sex differences in common forms of psychopathology.

Critical examination of sex differences in psychopathology is urgently needed, as they are essential characteristics of psychopathology with important implications for furthering understanding of typical and atypical individual differences (Widiger & First, 2007). Thus, even though strategic study of sex differences in psychopathology will be difficult due to the problems inherent in collecting these kinds of samples and in obtaining sufficient power to detect sex-specific effects and two-, three-, or four-way interactive effects, it is an important undertaking that merits funding by grant agencies with consideration of the extra time often required to obtain samples with adequate representation of each sex (ORWH, 2010; "Putting Gender on the Agenda," 2010). Further, the possible importance of sex differences in relation to an investigator's research question deserves careful attention as it may dramatically affect the kind of sample, and the relative proportion of females to males, that must be recruited. For example, if an investigator is interested in critically examining sex differences in early pathways to childhood externalizing disorders, then he or she will need to overrecruit both clinical cases and females represented in the sample which may add substantially to the amount of time it takes to recruit the sample. Further, doubtless, special recruitment methods will need to be undertaken.

Following sample recruitment and data collection, an investigator also faces the difficult decision of how to best analyze and report sex differences. To date, this has typically been done in one of two ways. Many times, sex is not the focus of a particular research study/question and the overrecruitment of a particular sex within a clinical sample, especially when the clinical sample is being compared to a control or nonclinical sample, is viewed as a nuisance variable and subsequently covaried. Given that sex differences in prevalence appear to be an important characteristic of many types of psychopathology, this approach should be carefully thought out rather than being used as the default. If markers/etiology/treatment response differ in meaningful ways between males and females with a given disorder, then this approach will ignore that possibility and—perhaps—even lead to misleading results.

Articles that address sex differences in more depth typically examine possible sex differences using a post hoc, somewhat exploratory approach. That is, most research articles on psychopathology typically address a main research question that does not directly target sex differences and then utilize secondary analyses to explore whether main study results hold in both males and females. While this approach might be an improvement to the covarying approach described above when significant sex differences are apparent, it still remains limited because it is post hoc in nature. Thus, it is underpowered due to unequal representation of both sexes in clinical samples, and negative findings are unsur-

prising because examination of sex differences was not guided by a strong theoretical approach.

Of course, neither of the two approaches discussed above is inherently inappropriate. What is of concern is that typically neither of the approaches is firmly grounded in theory. Use of a theory like the one advanced in this article will lead to more specific predictions (like those advanced above) and the use of more focused statistical tests. For example, as mentioned above, gene by environment interactions may themselves be moderated by sex (Beauchaine et al., 2009), and/or markers may mediate the association between etiological factors and psychopathology only for one sex or the other (i.e., moderated mediation; Preacher, Rucker, & Hayes, 2007). It is important that specific and complex hypotheses like these begin to be tested now that research has established between-sex differences and preliminary associations within sex.

Summary and Conclusions

This review advanced an integrative metatheoretical framework of sex differences in childhood-onset externalizing disorders and adolescent-onset internalizing disorders based on the evolutionary theory of sexual selection and life history theory. The provision of such a theory is timely due to recent initiatives geared toward furthering understanding of women's health (Cahill, 2010; ORWH, 2010; "Putting Gender on the Agenda," 2010; Wisner & Dolan-Sewell, 2007). In addition, the theory has implications for many subdisciplines within psychology, particularly those that converge on the study of psychopathology, including clinical psychology, behavioral neuroscience (or biopsychology), cognitive psychology, social and personality psychology, developmental (or child) psychology, and cross-cultural psychology. In a nutshell, this review suggested that male- and female-typical markers of psychopathology differentially increase risk for childhood-onset externalizing and adolescent-onset internalizing disorders, respectively, due to hormonally based influences on biological plasticity that create sex differences in timing of sensitivity to particular types of environmental influences across development.

Males may be at increased risk for childhood-onset externalizing disorders due to high levels of sensation-seeking and disinhibition. High levels of these traits are adaptive from a sexual selection viewpoint because they increase social dominance and can facilitate the practice of male-male competition during childhood. These sexually selected traits may be particularly vulnerable to early ecological environmental influences occurring during the prenatal period. This prenatal environmental sensitivity can also be considered adaptive because it helps the individual prepare for future environmental conditions based on cues such as maternal substance abuse. Prenatal environmental sensitivity may be instantiated by high levels of prenatal testosterone which may influence the development of the dopaminergic neurotransmission system, with perhaps most prominent effects for those with specific dopaminergic genetic alleles. Alterations in dopaminergic neurotransmission and functionality of prefrontal cortex-striatum circuitry may then lead to high sensation-seeking and disinhibition which—in their extreme forms, when expressed inflexibly (e.g., in affluent environments), or when expressed in certain childhood environments (e.g., in coercive environments or when exposed to deviant peers)—predispose a subset of males to childhood-onset externalizing disorders.

In contrast, females may be at increased risk for adolescent-onset internalizing disorders due to high levels of negative emotionality, empathy, and rumination. These traits and cognitive patterns are adaptive from a sexual selection viewpoint because they facilitate female advantage in interpersonal competence and dominance which is important for mate selection and manipulation, as well as female-female competition and the obtainment of social support during and following puberty in preparation for pregnancy and child rearing. Changes in these traits at puberty appear to be instantiated by increases in ovarian hormones which have interactive effects on the HPA axis and cortisol, as well as the neuropeptide oxytocin. These hormones influence amygdala function and serotonergic neurotransmission, potentially thereby increasing negative emotionality, empathy, and rumination, as well as females' sensitivity to the interpersonal environment and associated stressors, perhaps particularly for those with specific serotonergic alleles. This increased sensitivity to interpersonal stressors at puberty is adaptive because it facilitates females' ability to responsively adapt to environmental conditions that are likely to impact their reproductive success. However, extremely high and/or inflexible levels of negative emotionality, empathy, and rumination, particularly when experienced in the context of intense and/or chronic interpersonal stress, may make a subset of females more vulnerable to adolescent-onset internalizing disorders. Thus, there may be notable sex differences in risk markers, etiology, mechanisms, timing, and pathways of common forms of psychopathology such as externalizing and internalizing disorders.

Implications

Work on sex differences in psychopathology has greater implications than just furthering understanding of psychopathology, a laudable goal in and of itself. Work on sex differences has implications for advancing personalized treatment approaches and for furthering understanding of resilience (ORWH, 2010). If males and females are vulnerable to psychopathology via different markers and mechanisms, then more efficacious and effective treatments may be able to be generated focusing on specific markers and mechanisms based on child sex (Sonuga-Barke & Halperin, 2010). Further, work on sex differences in psychopathology can shed light on factors that can protect against the development of psychopathology (i.e., resilience). For example, elucidation of the markers that protect females from childhood-onset externalizing disorders and males from adolescent-onset internalizing disorders can pave the way for development of prevention approaches that facilitate these protective factors, furthering understanding of individual differences in resilience (Luthar & Cicchetti, 2000). Sexual selection theory, in fact, suggests that sexually selected traits are particularly sensitive to environmental influences and, thus, they may be important, malleable targets for prevention and intervention efforts (Bakermans-Kranenburg et al., 2008; Geary, 2002a, 2002b, 2010; B. J. Ellis et al., 2012; West-Eberhard, 2003).

Only through critical, empirical examination of sex differences in psychopathology via specific, theory-driven hypotheses can clarity be reached regarding the true extent of these individual differences. The evolutionary theory of sexual selection advanced here may be able to explain sex differences in common forms of psychopathology via male- and female-typical markers of psychopathology that facilitate male-male competition and female choice

and/or competition, respectively, and yet simultaneously—in their extreme forms, in certain environments, and for a subset of individuals—increase risk for childhood-onset externalizing and adolescent-onset internalizing disorders. Sex differences in hormonally based influences on biological plasticity may affect the timing of sensitivity to environmental influences across development differentially for males and females with downstream effects on sexually selected traits and behaviors relevant to psychopathology. However, as has been noted, sex is not deterministic, but rather multiply determined, complex and only probabilistically related to psychopathology. Thus, information on sex differences in markers of psychopathology can shed light on important population-level intergroup differences between males and females, but does not preclude important intrasex individual differences in risk and resilience. Overall, this theoretical framework has important implications for multidisciplinary work on psychopathology, resilience, and personalized intervention/prevention.

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