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Pubertal development and the emergence of the gender gap in mood disorders: A developmental and evolutionary synthesis

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This book addresses mood and anxiety disorders in women. It takes a broad developmental approach, aimed at understanding and offering appropriate treatment for women with such disorders. The primary aim of this first chapter is to examine the emergence of the gender gap in depressive disorders at puberty, and to compare alternative theories as to the factors that underpin gender differentiation in depression at this developmental stage. These models are synthesised using an evolutionary perspective on gender differences to integrate the insights provided by socialisation, life stress, and biological models of pubertal development. Anxiety and anxiety disorders are addressed only peripherally, given the relative paucity of research in this area.

The emergence of gender differences in affective disorders during adolescence

Perhaps the most robust finding in psychiatric epidemiology is that the rate of unipolar depression is higher among females than it is among males (Weissman et al., 1996). Females are nearly twice as likely as males to experience case-level depression diagnoses (Kessler et al., 1994; McGrath et al., 1990; Nolen-Hoeksema, 1990; Weissman & Klerman, 1977), and this finding holds true across a variety of cultures and racial/ethnic groups (Gater et al., 1998). Furthermore, studies of non-clinical depressed mood states have shown that females experience more symptoms during episodes of depressed mood than do males (Wilhelm et al., 1998). Although studies have generally found that there are not gender differences in depression in prepubescent children, by 15 years of age females are twice as likely as males to have experienced a major depressive episode (e.g., Hankin et al., 1998). This places the gender disparity in depressive disorders firmly within the domain of developmental psychopathology, and specifically those developmental processes

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associated with early adolescence. An understanding of the developmental processes that underlie the emergence of this gender difference may have implications for understanding vulnerability to depression throughout the life cycle.

While anxiety disorders have also been shown to be more common in females (Yonkers & Gurguis, 1995; and see Chapter 4), there is comparatively less research on the causes and developmental pattern of gender differences in anxiety. Lewinsohn et al. (1998) found that retrospective reports of anxiety disorders indicated that the gender gap in such disorders emerges much earlier in life than it does in depression; by age six, females are twice as likely as males to have experienced an anxiety disorder. Given that the early emergence of anxiety disorders is thought to be a marker of risk for later depressive disorder (Parker et al., 1999), the gender gap in anxiety may be a developmental precedent for the gender disparity in depressive disorder that emerges in adolescence. In what follows, we concentrate explicitly on depression and depressive disorders.

The gender gap: fact or artefact?

Before examining theories to account for the gender difference in the prevalence of depression, it is important to establish that the female preponderance is not artefactual. Two major reporting artefacts have been cited as potentially contributing to the reported gender gap in depression; a help-seeking artefact, and a recall artefact. The *help-seeking artefact* refers to a perceived reticence in males to seek treatment or advice for depressive symptoms, which could explain the preponderance of females reporting for treatment. However, in a comparison of data from two worldwide multicentre studies conducted by the Cross National Collaborative Group and the World Health Organization, Kuehner (2003) found that the rates of depression identified in community samples were in accord with those reported from primary care settings. The *recall artefact* postulates that women's recall is biased in favour of past negative affective states, and thus women report a higher rate of lifetime depression. Kuehner (1999) conducted a controlled test of this by comparing men and women's reports of depressive symptomatology during a depressive episode, and their recall of these symptoms 6 months later. He found no recall artefact: there was no disparity in the reported severity of symptoms at times one and two, between males and females.

A third suggested artefact is that the higher rate of sexual abuse and rape experienced by girls and young women accounts for subsequent depression rates, although it is worth noting that this does not constitute an artefact per se, but rather a potential aetiological explanation for the gender difference in mood disorders. Kessler (2000) did find that after rape and sexual trauma were controlled for in a population database, the gender difference in a first episode of depression was halved. However, when traumatic experiences more likely to be experienced by

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men were also controlled for, the female preponderance was restored. Findings such as these suggest that the increased prevalence of depression in females is not due to artefact.

Theories of the emergence of the gender gap during early adolescence

The gender intensification hypothesis

The *gender intensification hypothesis* (Hill & Lynch, 1983; Wichstrom, 1999) suggests that gender role orientations become more differentiated between the sexes over the adolescent years, as a result of exacerbated gender socialisation pressures during this time. For women, these pressures, both direct and in the form of social learning, are primarily thought to occur through observation of their parents' marital relations, which emphasise lesser public power and greater responsibility for the domestic sphere and care as part of the female gender role (Obeidallah et al., 1996) and through the socialising effects of parenting behaviours (Sheeber et al., 2002). The hypothesised effect of these socialisation experiences is the promotion of assumptions that emphasise collectivity, and a lower sense of self-esteem amongst females. These tendencies, in turn, contribute to the increase in depressive symptoms in women. The consequence of this intensified gender typing, according to these theorists, is that deficits in efficacy and instrumentality, reflected in low levels of traditionally masculine personality characteristics, may place young adolescent girls at higher risk for depression through greater exposure to experiences that promote learned helplessness (Obeidallah et al., 1996).

One important source of socialisation experiences is parenting behaviours throughout childhood and adolescence. Based on reviews of clinical and developmental literature, Hops (Hops et al., 1990; Hops, 1992, 1996) posited two paths by which parents may inadvertently increase their daughters' risk for depressive symptomatology and disorder. First, familial socialisation processes may serve to normalise and encourage girls' expression of depressive-like behaviours (e.g., sadness; self-derogation). Second, differential parental reinforcement of gender-typic behaviours may lead girls to display less instrumental and more relationship-focused behaviours, both of which are related to theoretically derived and empirically supported risk factors for depression. These gender-typic behavioural patterns, learned in early childhood and reinforced across time, are hypothesised to hamper girls' ability to meet the normative challenges of adolescence, thus contributing to the increased prevalence of depressive symptomatology among girls in this developmental stage.

There is some evidence that girls may be differentially socialised to display depressive behaviours during childhood. In a recent review on parental socialisation of emotion, Eisenberg et al. (1998) found that although parents do not typically

report reacting differently to girls' and boys' emotional displays, observational data suggest that there are indeed differences, "albeit perhaps less than one might expect". In particular, a series of studies indicated that parents put more pressure on boys to control their emotions and "unnecessary" crying. Block (1983) reported that parents were quicker to respond to crying in girls than in boys. Parents' meta-messages about the acceptability of emotional expressions are apparently clear to children in that boys expect their parents to disapprove of their expression of sadness more so than girls (Fuchs & Thelen, 1988).

Parents' reactions to children's negative emotions may also provide them with gender-differentiated strategies for regulating negative affect. Although the data are limited, some evidence suggests that boys may be encouraged to use distraction and problem-solving more so than are girls (Eisenberg et al., 1998). In fact, one study indicated that school-age children expected fathers to respond to boys' emotional expressions with problem-solving and mothers to respond to girls' by focusing on feelings (Dino et al., 1984). Similarly, in a review of the origins of ruminative coping styles, Nolen-Hoeksema (1998) indicates that failure to teach girls active coping strategies for dealing with negative affect contributes to girls' greater use of ruminative styles of responding to depressed moods. Further, she suggests that to the extent girls are told that they are naturally emotional, they may have lower expectations that their behaviour can influence their affective experiences.

It has also been proposed that parents socialise girls to be more relationship-oriented and less instrumental than boys, and that this may in turn result in the socialisation not of depressive behaviours themselves, but rather risk factors for depression. Huston (1983) reported that girls receive more encouragement for dependency and affectionate behaviour. They are also reported to receive more support for nurturant play (Ruble et al., 1993). Interestingly, Block (1983) reported that in Baby X studies in which infants are "assigned" a gender (i.e., the same baby is labelled a "boy" and a "girl" in interactions with different participants), adults provided more reinforcement for nurturant play when the baby was said to be a girl; such evidence is compelling in that Baby X studies control for gender differences in children's actual behaviour. On a related theme, evidence suggests that mothers encourage girls more than boys to have concern for others, share, and behave prosocially (see Keenan & Shaw, 1997 for a review). Additionally, they may be less attentive to girls' assertive behaviour (Kerig et al., 1993). Parents may also impede the development of girls' sense of mastery by limiting their activities and freedom. In a 1983 review of the literature, it was reported that mothers were more likely to give unnecessary assistance to girls than to boys, and were more likely to reward frustration with physical comfort (Block, 1983).

There is also modest evidence that girls have lower self-evaluations of their own efficacy and that such evaluations are related to depressive symptomatology

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(Avison & McAlpine, 1992; Ohannessian et al., 1999). In a recent review, Ruble et al. (1993) reported that preadolescent girls report lower expectations for success, more maladaptive attributions for success and failure, and poorer self-esteem, than age-matched boys. Though gender differences did not emerge in all of the studies reviewed by Ruble et al., the direction of effects was consistent when gender differences were observed. However, it is important to remain cognizant of the likelihood that disturbances in perceived self-competence may be sequelae rather than causes of depressive symptomatology. Two recent longitudinal studies by Cole et al. (1998, 1999) suggest that children's underestimates of their own competence emerge as a function of depressive symptomatology and that controlling for depression eliminates the observed gender differences in estimation of their competencies.

Thus, although these data suggest that parents' early gender-differentiated socialisation of children's emotional and social behaviours may have effect on children's ability and motivation to regulate emotion, large gaps remain in the literature. In particular, the largely cross-sectional studies do not provide evidence that these parental behaviours are predictors and not consequences of children's sex-typed behaviours. For example, if girls display more sadness and boys more anger, it would be reasonable to hypothesise that parents' tendency to discuss sadness with girls and anger with boys emerged consequent to the children's behavioural propensities. Similarly, it is not clear whether parents' tendency to be more emotion focused in response to young girls emotions and problem focused in response to young boys, doesn't reflect girls' earlier verbal and emotional development (Keenan & Shaw, 1997). Additionally, as a caveat it is important to keep in mind that the research discussed herein focuses on parents' responses to children's normative emotional expressions. Though we consider it reasonable to construe depressive symptomatology as being at one end of a continuum of normative affective expression, the connection between early socialisation of depressive-like behaviours and subsequent depressive functioning is, at this point, quite speculative.

Moreover, in a meta-analysis of research dating back to the 1950s, Lytton and Romney (1991) concluded that although there is modest evidence of parental encouragement for sex-typed activities, the evidence does not support overall differences in parental restrictiveness or encouragement of either achievement or dependency as a function of child gender. Hence, it appears that the evidence for the shaping of differential activities for girls and boys is stronger than that for other areas of gender-socialisation.

Limitations of the gender intensification hypothesis

Aside from the limited number of studies that would allow strong causal inferences to be drawn regarding socialisation and the emergence of the gender gap in mood disorders, there is also some literature that is inconsistent with this hypothesis.

Studies documenting the trajectories of children’s own gender role concepts have shown that the rigidity of children’s gender stereotypes tends to *lessen* as adolescence approaches. These studies have also found that boys are likely to labour under more rigid self-imposed sex-types than girls (Banerjee & Lintern, 2000). Also, gender intensification theorists cite the emergence of the gender gap at adolescence as support for their argument; however, as will be discussed below, the onset of the gender difference in depression is not predicted by age as such, but rather by pubertal status of the individual (Angold et al., 1998). If female depression is a consequence of a broad societal pressure applied to girls when they reach a certain age or stage of schooling, this should not be the case.

Another problematic finding with regards to gender intensification theories of adolescent depression is that the rates of female depression have not lessened over the last 50 years (Weissman et al., 1996). While the feminist movement has yet to yield true gender equality, it is clear that the status and opportunities that adolescent girls may expect at present far outstrip those available 50 years ago. Gender intensification theories would logically predict that a rise in the financial and social power of women would be accompanied by a commensurate fall in depression onset at that developmental stage, where the assumption of adult female roles is hypothesised to be paramount to adolescent girls’ sense of self-worth and efficacy. This matter, however, may not be as straightforward as it first appears. If the socialisation of female roles in terms of reduced instrumentality and increased sensitivity to social relationships has not changed fundamentally over time, then the increased opportunity for females may actually increase the gap between their behavioural repertoire and the demands placed on them. Nevertheless, the lack of change in the gender gap in rates of depression over a period of dramatic historical change in the type and range of roles socialised in young women does seem puzzling if gender socialisation is the key process driving this phenomenon.

Another contradiction within these theories is their heavy emphasis on the timing of gender intensification as a corollary to the timing of the emergence of the gender gap in affective disorders. As noted above, social learning and gender typing have been shown to begin in the first few years of life. If identification with a feminine stereotype is as strongly linked to depression as some of these theorists suggest, the finding that the rates of depression in prepubertal children are equal between genders is counter-intuitive (Gelman et al., 2004). A more sophisticated version of the gender intensification hypothesis posits that what is socialised in females during childhood are reductions in instrumentality and increased experiences of helplessness and dependency (see above; Sheeber et al., 2002). These socialisation experiences then constitute a diathesis that interacts with the developmental demands of early adolescence to create greater risk for depression in

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females. However, even this very plausible view of the role of socialisation pressures during adolescence needs to explain what it is about the developmental demands of early adolescence that is specifically associated with the emergence of depressive disorders in vulnerable females (as opposed to, say, anxiety where gender differences are seen much earlier in life (Lewinsohn et al., 1998)).

Finally, although the degree of gender gap in depressive disorders is unevenly distributed, such that women in disadvantaged sectors of society (e.g., women of colour, women living in poverty, single mothers, and those with less than a high school education) are disproportionately affected (Everson et al., 2002), the fact that the gender gap is also reliably observed in more privileged social groups also casts doubt on the validity of a theory which posits membership of a devalued social group as the primary causative influence on depression.

Gender roles and individual differences in personality

Investigations into developmental changes in gender identity often use self-report measures of traits traditionally thought to be more characteristic of males than females, or vice versa. In the Personal Attributes Questionnaire, for example, the “masculine” item endorsements include: *independent, active, competitive, making decisions easily, self-confident, not giving up easily*, and *standing up well under pressure* (Spence et al., 1974). The finding that high scores on these traits are protective against depression certainly seem to be robust (e.g., Hoffman et al., 2004), but the assumption that these are indeed inherently masculine traits may need re-evaluation. When these traits are considered in their own right, decoupled from their description as “masculine”, the feminist argument becomes circular, in claiming that, for example, low self-confidence leads to depression, which is in part indexed by low self-confidence (Barrett & White, 2002).

One way to reframe the relationship between gender roles and depressive phenomena is by taking into account the correlation between sex role inventory scores, and broader personality traits. For example, Francis and Wilcox (1998) found that high scores on the Bem sex role inventory (Bem, 1974) masculinity scale were associated with low neuroticism and high extraversion, whereas high scores on femininity were associated with high neuroticism. Neuroticism has previously been identified as a candidate temperament trait that may place some individuals at higher risk for depression and anxiety, and is also reliably found to be higher in females (Fanous et al., 2002). O’Shea (2002, cited in Parker & Brotchie, 2004) found that, though female adolescents did record higher neuroticism scores than males, high neuroticism was a strong predictor of first onset depression, regardless of gender. In fact, adjustment for neuroticism scores greatly attenuated the gender differential in first onset depression in this sample. This suggests that it may be neuroticism, rather than gender role per se, that confers risk for depression, although

this still begs the questions as to why female gender and typical female gender role descriptions are associated with greater levels of neuroticism.

Pubertal development and the diathesis for affective disorder

The relevance of pubertal changes to the developmental examination of the gender gap lies primarily in the potential role of hormonal changes at puberty as catalytic agents for the development of depression in those females placed at risk by temperamental predisposition. Angold et al. (1998) found that the transition to Tanner stage III (an index of body shape change) of puberty predicted the increase in rates of diagnostic and statistical manual of mental disorders (DSM-IV) unipolar depression in girls, exhibiting a much larger effect on rates of depression than chronological age. This supports the view that changes in rates of depression at adolescence are specifically related to the physical changes of puberty, rather than to broad psychosocial factors common to girls at a particular stage of adolescence. However, it does not rule out the possibility that societal pressures are the primary precipitators of depression, but are prompted not by age but by the visible manifestations of puberty. This problem was later clarified by the addition of hormonal variables into the model (Angold et al., 1999), which eliminated the effect of morphological status, strongly implicating the effects of oestrogen in the development of depression in adolescent girls.

At puberty, females' hormonal levels begin to fluctuate cyclically over a broader spectrum than males'. Oestrogen in particular is recognised as playing an important role in mediating females' sensitivity to stress. Oestrogen apparently acts as an anxiolytic, and thus the cyclical withdrawal of oestrogen that occurs shortly prior to menstruation may be analogous to the physiological effects of anxiolytic withdrawal, creating a greater sensitivity in menarcheal and adult females to the anxiogenic and depressogenic effects of negative life events. At the onset of puberty for males, on the other hand, the increase of testosterone has been found to have a protective effect against depression and anxiety, although it tends to increase aggressive and risk-taking behaviours (Seeman, 1997).

It is important to note that the cyclical release and withdrawal of oestrogen alone is not suggested to be the causative factor for the sudden increase in the incidence of depressive episodes in adolescent girls. Rather, this cycle is thought to form a biological kindling which increases the stress reactivity of at-risk individuals to negative life events. In this respect, negative perceptions of gender role expectations or a socialised limitation in the behavioural repertoire of females may indeed come into play as proximal risk factors, but are regarded as precipitants of the development of mood disorder in girls who are, by nature of temperament and biology, already at risk. In other words, the interaction of temperament and hormonal change may create a vulnerability diathesis upon which social factors may act as a catalyst to generate gender difference in rates of depression.

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In support of the putative interaction between pubertal oestrogen changes and negative life events, Brooks-Gunn and Warren (1989) found that while pubertal oestrogen level rise accounted for 4% of variance in increased negative affect reported by adolescent girls, the joint contributions of oestrogen rise and negative life events accounted for 17% of the variance. It would appear that the onset of puberty sensitises young women to the stress of negative life events, and possibly desensitises young men, via the protective influence of testosterone. Prepubertal boys and girls tend to show similar correlations between number of life stressors and depressive affect. However, after the onset of puberty, the relationship between stress and negative affect strengthens for young women, and declines almost to elimination in young men (Angold et al., 1999).

Pubertal change and interpersonal stressors

Given the hypothesis that the relation between pubertal hormone changes and depression in females is mediated by negative life events, it is notable that the majority of stressors preceding the onset of adolescent depression are of an interpersonal quality (Cyranowski et al., 2000). Consistent with this observation, it has been proposed that the relationship between the quality of family relationships and depressive symptoms may be stronger for females than males (Kavanagh & Hops, 1994). During adolescence females tend to gain independence more slowly from their families (Huston & Alvarez, 1990) and have relationships that are both more disclosing and more conflictual with their parents than do males (Montemayor, 1983; Noller, 1994). Many studies using self-report measures have in fact found a stronger negative correlation between cohesive and supportive family relationships and depressive symptoms amongst girls than boys (Avison & McAlpine, 1992; Rubin et al., 1992; Slavin & Rainer, 1990; Windle, 1992), although a study by Sheeber et al. (1997), which included observational as well as self-report measures of family functioning, found that the relationship between family functioning and depression was equivalent for males and females. This suggests that the stronger link between family processes and depression amongst females may be primarily determined by the way depressed states affect female *perceptions* of the family environment rather than objectively observable features.

The correspondence between the findings of a specific association between interpersonal stress and risk for depression on the one hand, and the evidence of increased affiliative proclivities amongst females, (both as compared to males, and after menarche) on the other, led Cyranowski et al. (2000) to examine potential biological substrates for female affiliative behaviour. Non-human mammal research has strongly implicated the hypothalamic neuropeptide oxytocin in affiliative and care-giving behaviours (Depue & Lenzenweger, 2001). Oxytocin transmission is thought to be regulated by oestrogen and progesterone levels, giving rise to the idea

of a hormonally driven pubertal increase in affiliative proclivity for females (Cyranowski et al., 2000). This potential connection between pubertal development and an increase in biologically controlled sensitivity to social stressors allows for a synthesis of psychosocial, biological, and stress-response precipitants to adolescent onset depression, with the important caveat that the role of oxytocin in human female affiliative behaviour is yet to be fully understood. Although the formation of affiliative networks is in itself a protective factor against depression, the increased desire and need for these relationships, as well as the burden of increased care-taking within relationships, may mean that affiliative failures form potent stressors for girls who are temperamentally at risk, as has been emphasised by recent evolutionary models of depression.

An evolutionary synthesis of pubertal influences on vulnerability to depressive disorders

Many theorists have argued that depressed states are most essentially related to reductions of positive affect (anhedonia being a key defining feature), and that the regulators of positive affect are embedded in *social* cognition and behaviour (e.g., Allen & Badcock, 2003; Joiner & Coyne, 1999; Gilbert, 1989, 1992; Gotlib & Hammen, 1992; Watson, 2000). Critical to a functional or evolutionary view of depression is the proposition that there are various biological processes that guide individuals to enact certain social or interpersonal roles, including gender roles. There are many clues in the research literature to suggest that social processes (both in terms of social cognition and interpersonal behaviour) play a critical role in the aetiology and maintenance of depressed states in both genders. Critical empirical observations here include findings demonstrating that depression is often precipitated by interpersonal events (as noted above), and that interpersonal processes often mediate the exacerbation or resolution of depressive episodes (Joiner et al., 1999). Stressful interpersonal contexts are amongst the most reliable antecedents of depressed states (e.g., Kendler et al., 2003; Monroe et al., 1999) and certain interpersonal behaviours, such as excessive reassurance seeking, are strong and specific predictors of risk for depression (Joiner & Metalsky, 2001).

A recently proposed model of the function of depressed states seeks to explain why there is such a close link between social cognition, social behaviour, and depressive phenomena. The *social-risk* hypothesis of depression (Allen & Badcock, 2003) suggests that depressed mood (i.e., down regulation of positive affect and confident engagement in the world) evolved to facilitate a risk-averse approach to social interaction in situations where individuals perceive their social resources (e.g., status, affection, friendship, power) to be at critically low levels. Thus, whereas positive affect encourages engagement in a range of activities, most especially social ones,