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Adolescent development and risk for the onset of social-emotional disorders: A review and conceptual model☆

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ABSTRACT

The adolescent developmental stage appears to be a sensitive period for the onset of several particular forms of mental disorder that are characterised by heightened emotionality and social sensitivity and are more common in females than males. We refer to these disorders (social anxiety disorder, generalised anxiety disorder, eating disorders, major depression) collectively as the social-emotional disorders. The aim of this paper is to address an important question in the understanding of social-emotional disorders – why do these disorders commonly begin during adolescence? We present a conceptual model that describes some of the key changes that occur during adolescence and that addresses some hypothesised ways in which these changes might increase risk for the development of social-emotional disorders. An overview of the extant empirical literature and some possible directions for future research are suggested. The model points to interesting links between psycho-social risk factors that should highlight potentially fruitful directions for both psychopathology research and early intervention programs.

The adolescent developmental period encompasses some of the largest and most dramatic psychosocial changes within the human lifespan. The four to six years that Western societies refer to as adolescence (usually 13–18 years) includes a broad range of physical, cognitive, emotional, and social development. While the majority of adolescents emerge from this period with a positive sense of self, strong relationships, and a good quality of life, this is also a period when many forms of psychopathology first begin to manifest or markedly increase in prevalence. In fact, many authors have pointed to this period as one of heightened risk for psychopathology in general (e.g., E. V. Kelly et al., 2015; Powers & Casey, 2015; Ullsperger & Nikolas, 2017; Zahn-Waxler, Shirtcliff, & Marceau, 2008). However, as we will argue in the following section, rather than providing broad, non-specific risk for psychopathology, the adolescent period appears to confer risk for the onset of a relatively specific set of mental disorders. These disorders, which we refer to as "social-emotional disorders" are characterised by heightened negative affectivity, impaired social functioning, and female preponderance.

The elevated incidence of social-emotional disorders during adolescence, suggests that key developmental changes that occur during these critical years may be partly responsible for the onset of these disorders. As we argue in this conceptual review, possible candidates for these developmental processes include: hormonal and morphological changes linked to puberty; alterations in sleep patterns; difficulties with emotion regulation; changes in the importance and nature of relationships with peers; and altered self-concept. Hence, a plausible developmental story is that known risks for mental disorder during the childhood years (including inherited and temperamental susceptibilities, family risk factors, and social and cognitive vulnerability) may interact with processes underlying normal adolescent development to trigger the emergence of the social-emotional disorders. What those processes are, and how they interact with pre-existing vulnerabilities, has received limited attention in current research and will form the topic of this paper. Before beginning, we will first consider evidence for the incidence of mental disorders during the adolescent period.

1. Adolescence as a risk period for social-emotional disorders

While it is often claimed that the adolescent period is a phase of heightened risk for the onset of mental disorder, referring to psychopathology as a unitary construct in this context risks missing possible variability between disorders. As an example, many externalising

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disorders (e.g., Oppositional Defiant Disorder, Conduct Disorder, Attention Deficit Hyperactivity Disorder) begin prior to adolescence. Attention deficit hyperactivity disorder (ADHD) has a median onset around age 8 years, with 80% of this population having onset between ages 4 and 11 (Kessler et al., 2007), as partly stipulated in the diagnostic criteria. As a result, the prevalence of ADHD actually decreases slightly between childhood and adolescence (Ford, Goodman, & Meltzer, 2003; Lawrence et al., 2015). Similarly, the majority of oppositional defiant disorders and conduct disorders will begin between 5 and 15 years (Kessler et al., 2007). Therefore, median onset across these disorders occurs prior to adolescence at around age 11 (Kessler, Berglund, Demler, Jin, & Walters, 2005).

Substance use stands out from the other externalising disorders due to its later age of onset. However, this is complicated by legal restrictions in most countries that limit access to regulated substances until early adulthood (if at all). Although problematic use of substances does typically onset during adolescence (Dodge et al., 2009) the clinical syndrome, substance use disorder, is a disorder of early adulthood. The inter-quartile range for onset of substance use disorder is 18–27 years with a median onset around 20 years (Kessler et al., 2005). Socio-cultural and legal factors likely complicate its onset and its aetiology clearly differs from the internalising disorders that we describe below. Therefore we did not include it under the disorders reflected in this review. The issue is briefly revisited at the end of the paper under limitations.

In contrast, the anxiety disorders as a group show median onset around adolescence (Bonnewyn, Bruffaerts, Vilagut, Almansa, & Demyttenaere, 2007; Cía et al., 2018; Kessler et al., 2005). However, this figure in itself masks a wide variability between particular disorders (Copeland, Angold, Shanahan, & Costello, 2014; Lijster et al., 2017). Like the externalising disorders, specific phobias and separation anxiety disorder begin quite early in life, with age of onset typically prior to 10 years (Bonnewyn et al., 2007; Copeland et al., 2014; Kessler et al., 2007). In contrast, panic disorder and PTSD have later onset, mostly beginning in adulthood (> 20 years; Bonnewyn et al., 2007; Cía et al., 2018; Copeland et al., 2014; Kessler et al., 2005). The observation that the adolescent developmental period confers risk for the onset of psychopathology, is largely the result of a subset of mental disorders – namely, social anxiety disorder, eating disorders, depression and generalised anxiety disorder. Social anxiety disorder is the prototypical adolescent disorder. There are a wealth of studies describing both clinical samples and population-representative samples reporting mean and median age of onset in the early to mid-teen years (Lijster et al., 2017). Based on population studies, median onset has been reported at age 13–14 years and the majority of cases begin between ages 8 and 18 years (Bonnewyn et al., 2007; Cía et al., 2018; Kessler et al., 2005).

Eating disorders have similarly been linked with the adolescent period, with peak onset between 15 and 19 years (Herpertz-Dahlmann, 2008; Klump, 2013). Some studies have also provided detailed cumulative lifetime prevalence curves, which reflect changes in the rate of onset of disorder. According to these curves, eating disorders are rare in childhood and show a dramatic increased incidence from early to middle adolescence, followed by a levelling off of new cases from around age 19 (Nagl et al., 2016; Preti et al., 2009; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011).

Aside from these two groups of disorders, depression is also commonly conceptualised as a disorder with onset in adolescence. However, the picture here is more complex. There is little doubt that major depressive disorder is rare during childhood and its prevalence increases markedly in adolescence (Costello, Copeland, & Angold, 2011; Ford et al., 2003; Lawrence et al., 2015). Cumulative onset data show a dramatic increase in rates of depression around the period of 14–16 years, especially among girls (Hankin, Abramson, Silva, McGee, & Moffitt, 1998; Roza, Hofstra, van der Ende, & Verhulst, 2003; Wittchen, Kessler, Pfister, & Lieb, 2000). Interestingly, it is harder to determine when this increase in the incidence of depression ends. Some studies show a decreasing incidence by the end of adolescence (Hankin et al., 1998), while others show a decreased incidence toward the end of the 20's (Roza et al., 2003; Wittchen et al., 2000). On the other hand, studies that have reported age of onset retrospectively from adult population samples suggest a very late median age of onset (around 30 years) with 50% of new cases emerging between 18 and 45 years of age (Cía et al., 2018; Kessler et al., 2005). Reconciling these seemingly disparate data is difficult and may reflect greater bias in retrospective reporting for depression relative to other disorders, given its episodic nature and the impact of mood on memory (Gaddy & Ingram, 2014). Nonetheless, the data from most sources consistently demonstrate a sharp increase in depression, especially among females, around mid-adolescence (Merikangas, Nakamura, & Kessler, 2009) pointing to this developmental period as a significant risk for this disorder.

Finally, generalised anxiety disorder (GAD), which shares characteristics of both anxiety and mood disorders, shows a pattern of onset with similarities to both of these groups of disorder. As with depression, retrospective reports from adult samples suggest that onset before adolescence is relatively rare (less than 10% before age 13: Kessler et al., 2005) and begins to increase from early adolescence, perhaps suggesting some additional risk during adolescence. Most adults with a history of GAD report onset between 20 and 50 years, with median onset in the 30's (Bonnewyn et al., 2007; Cía et al., 2018; Kessler et al., 2005). However, prospective cumulative age of onset curves suggest an increase in incidence from early to middle adolescence. For example, data from the Early Developmental Stages of Psychopathology study from Germany show a clear increasing incidence from around the age of 11 years, followed by a steeply increasing onset across the adolescent and early adult years, with some indication that incidence decreases from the mid 20's (Beesdo, Pine, Lieb, & Wittchen, 2010). One interesting suggestion is that populations meeting DSM criteria for GAD may comprise two subtypes: an early onset type and a late onset type. Using admixture analyses to model age of onset distributions, Rhebergen et al. (2017) identified a bimodal distribution for age of onset in GAD with a cut-off at 24 years. The early-onset group (43% of the sample) had a mean age of onset of 18.7 years with a narrow distribution (s.d. = 3.5 yr). In contrast, the late onset group showed characteristics more consistent with retrospective studies; a mean age of onset of 30.2 years with broad variability (s.d. = 13.6 yr). When the two groups were compared on descriptive features, the early-onset group showed characteristics concomitant with other anxious populations – a higher proportion of females, higher neuroticism, and a greater likelihood of familial history of anxiety and depression. The later-onset group showed a stronger comorbidity with physical health disorders, perhaps indicating a subgroup with more realistic worries based around ill-health. Hence, despite variability in onset data, there is some evidence that GAD commonly emerges within the adolescent window, especially when onset is tracked prospectively. Like depression, it appears that adolescence is a period of elevated risk for the onset of GAD that is characterised by more typical anxiety features, and that additional developmental factors later in life are also likely to be relevant.

In summary, rather than seeing adolescence as a critical risk period for psychopathology in general, it appears that this developmental stage generates risk for a relatively specific set of mental disorders. Foremost among the “disorders of adolescence” are social anxiety disorder and eating disorders, which clearly show peak onset during this period. Major depressive disorder and GAD also appear to have elevated incidence during this period, although new cases continue to emerge well into adulthood. Although problematic substance and alcohol use also increase during adolescence, there are sufficient differences between this and the above disorders that we will not include it in this discussion (see limitations below).

While the disorders associated with onset during adolescence have a number of distinguishing characteristics, they also share several core features: They are all characterised by heightened negative affectivity and by concomitant dysregulation of mood. They are also characterised

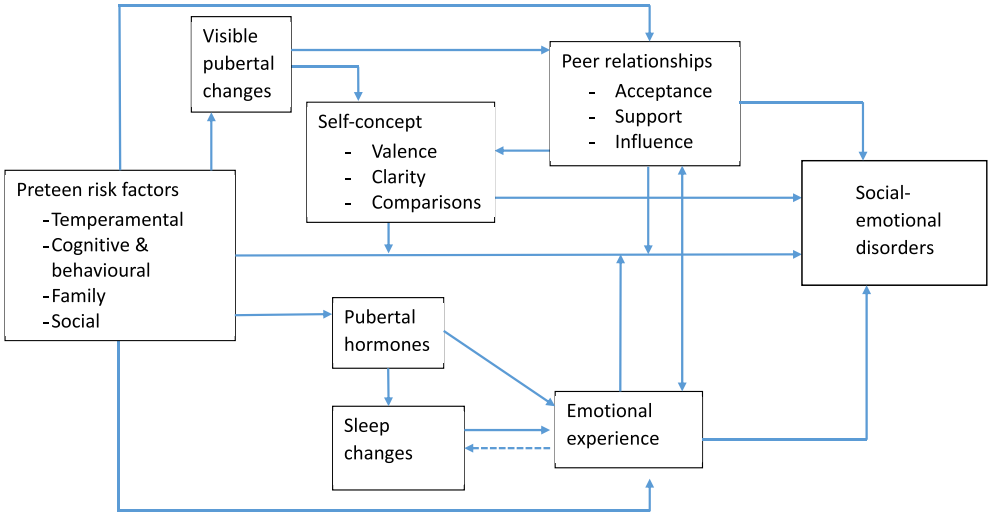

Fig. 1. A conceptual model of the influence of adolescent developmental characteristics on development of social-emotional disorders

by a strong interpersonal focus, reflecting both distress about, and difficulties with, relationships as well as a broader concern about the opinions or observations of others. They are also all particularly prevalent in girls and women. As a group, these disorders all appear to reflect social-emotional difficulties and will be collectively referred to as *social-emotional disorders* in this paper. The key question to address is: How might the characteristics that emerge during the adolescent period confer increased risk for the development of these social-emotional disorders?

2. Characteristics of adolescent development and risk for social-emotional disorders

Having argued that the adolescent period confers risk for the onset of a particular subset of clinical disorders, largely characterised by disturbances in social interactions and emotional regulation, the following sections outline some of the major developmental features of adolescence and their potential links with these disorders. Fig. 1 illustrates a schematic representation of key adolescent developmental processes and ways in which they might inter-relate to increase risk for the onset of social-emotional disorders. In brief, the model predicts that risk factors that exist prior to onset of adolescence will both lead to and interact with typical developmental changes that occur during adolescence to trigger onset of the social-emotional disorders. The following sections will briefly describe these developmental changes and provide an overview of some of the extant literature supporting their relationships with those disorders.

2.1. Pubertal changes

Puberty includes a complex constellation of hormonal and physiological changes that span a large proportion of the child and adolescent developmental periods. The two major phases comprising puberty include adrenarche (incorporating activation of adrenal androgens) and gonadarche (characterised by activation of gonadotrophic hormones) (Patton & Viner, 2007; Vijayakumar, Op de Macks, Shirtcliff, & Pfeifer, 2018). Adrenarche typically begins during middle childhood, although overt indicators of its effects occur sometime later. Gonadarche begins toward the end of childhood and, on average, begins later in boys than girls. Ultimately, many of the major physiological and overt morphological changes associated with puberty (such as genital development; menstruation and ejaculation; breast and hair development; and changes in body fat, skin, and voice tone) overlap with early to middle adolescence (Patton & Viner, 2007; Vijayakumar et al., 2018).

Puberty has been indicated as a risk factor for the onset of social-emotional disorders. The two facets of puberty that have typically been examined in relation to mental health outcomes are pubertal status (the amount or extent of pubertal maturation) and pubertal timing (the extent of pubertal development relative to one's peers). Increases in depression and anxiety have been associated with onset of menarche (Patton et al., 1996) and pubertal status accounts for greater variance in the onset of depression among girls than does chronological age (Angold, Costello, Erkanli, & Worthman, 1999). Similar data have consistently demonstrated associations between pubertal status and eating disorders among girls, but not among boys (Klump, 2013). At a process level, pubertal status is linked with decreases in mood, body satisfaction, social uncertainty, and heightened emotionality (Guyer, Silk, & Nelson, 2016; Klump, 2013; Mendle, 2014).

The age of onset of puberty and its accompanying overt bodily changes can vary markedly between individuals. The most extensive research into the impact of pubertal development and psychopathology has evaluated the effects of pubertal timing. There is now consistent evidence that differences in the timing of puberty are associated with a variety of mental disorders, although there is some inconsistency in the exact patterns (Graber, 2013; Mendle & Ferrero, 2012; Mendle, Turkheimer, & Emery, 2007). Earlier onset of puberty relative to peers has been consistently associated with increased risk for psychopathology among girls. The evidence for a relationship between early pubertal timing and emotional functioning among boys has been somewhat less consistent. Similarly, evidence for the impact of late pubertal development in comparison to peers on psychopathology has also shown inconsistent effects among both boys and girls. In a comprehensive review and meta-analysis, Ullsperger and Nikolas (2017) found small, significant effects (d = .21) of early pubertal timing on psychopathology across 101 studies. Somewhat surprisingly this analysis did not indicate major differences in effects between males and females, nor between internalising and externalising psychopathology. However, there was a suggestion that among studies examining clinical populations, the largest effects were shown for internalising disorders in females (d = .36). The relationship between late pubertal timing and psychopathology was negligible for both girls and boys (d = .03) (Ullsperger & Nikolas, 2017). Hence, earlier onset of puberty relative to one's peers, especially among girls, appears to increase risk for clinical internalising disorders (including the social-emotional disorders) moreso than other disorders. At least some evidence suggests that the impact of early pubertal timing on internalising disorders might be moderated by pre-morbid temperamental characteristics, such as emotional reactivity and self-regulation (Crockett, Carlo, Wolff, &

Hope, 2013). Pubertal onset can also be affected by a range of genetic and environmental factors that themselves confer early risk for psychopathology, including family variables and low socioeconomic status (Ellis, 2004; Guyer et al., 2016). Hence certain pre-adolescent vulnerabilities may increase the likelihood for early pubertal timing, which in turn increases risk for social-emotional disorders.

A critical question is to ask in what ways puberty confers risk for mental disorder. Changes associated with puberty broadly comprise two components that likely hold unique relevance to mechanisms underlying psychopathology: direct hormonal changes and overt morphological changes. It is highly likely that these components will represent complementary risk pathways in the development of social-emotional disorders (see Fig. 1). Hormonal factors directly impact brain development, particularly linked to emotional reactivity and social-reward pathways. At the same time, the observable changes affiliated with puberty almost certainly trigger alterations in self-concept, social reactions, and the links between them. Each will be described in turn.

2.1.1. Hormonal aspects of puberty

The hormonal changes underpinning puberty impact on the development and maturation of a variety of brain regions, including those that underpin social-emotional expression, such as the amygdala, hypothalamus, striatum, and medial pre-frontal cortex (Guyer et al., 2016; Somerville, 2013; Vijayakumar et al., 2018). For example, the amygdala, which plays a critical role in emotion regulation, is one of the few brain structures that contain sex hormone receptors and its increasing volume of grey matter from childhood through to adolescence appears to be directly related to pubertal maturation (Hu, Pruessner, Coupé, & Collins, 2013; Scherf, Smyth, & Delgado, 2013).

Evidence supports links between changes in the levels of pubertal hormones and social-emotional disorders, especially among girls. In a review of 14 cross-sectional and longitudinal studies, higher levels of estradiol (a form of estrogen) were shown to be associated with depression among girls (Balzer, Duke, Hawke, & Steinbeck, 2015). There is some suggestion that estradiol level more strongly predicts depression than does overt pubertal development (Angold et al., 1999). Genetic factors associated with estrogen activation at puberty may also be linked to the emergence of eating disorders among girls (Klump, 2013; Klump, Keel, Sisk, & Burt, 2010). Direct evaluation of the relationship between hormonal changes and anxiety is less extensive, but some evidence has begun to show associations between gonadal and adrenal hormones and anxiety, although current data suggest a more consistent effect among boys (Reardon, Leen-Feldner, & Hayward, 2009). In contrast to most pubertal hormones, testosterone appears to show little relationship with depression (Duke, Balzer, & Steinbeck, 2014) and might even protect against its development (McHenry, Carrier, Hull, & Kabbaj, 2014). These associations are mostly consistent with the female-preponderance of social-emotional disorders and especially with the dramatic increase in depression among female adolescents.

Centrally, estrogen homeostasis appears to directly impact monoamine levels, especially serotonin (affiliated with both anxiety and depression) (Halbreich & Kahn, 2001; Rubinow, Schmidt, & Roca, 1998). At the state level, the hormonal changes of puberty may be at least partly responsible for the increased negative affectivity that characterises adolescence (see below). For example, pubertal changes in sex hormones increase emotional reactivity (Balzer et al., 2015; Guyer et al., 2016). In turn, the greater emotional reactivity associated with puberty may increase risk for emotional disorders, especially among youth with pre-existing vulnerabilities (Hayward et al., 1997; Spear, 2000) (see next section). In addition, many of the hormones linked to puberty have an impact on sleep (Mong et al., 2011) and pubertal changes in estrogen and testosterone may delay circadian rhythms and the sleep/wake cycle (Hagenauer & Lee, 2012). Adolescent sleep phase shifts are likely linked to risk for social-emotional disorders (see below).

2.1.2. Morphological aspects of puberty

In addition to its direct hormonal effects, pubertal changes are overtly reflected in morphological development that can impact both self-concept and the responses of others to the young person. In turn, these factors are likely to have profound effects on social-emotional mental health. Among girls, the physical changes associated with pubertal development have been shown to alter self-concept (Koff, Rierdan, & Silverstone, 1978), increase self-consciousness (Hyde, Mezulis, & Abramson, 2008), and predict more extensive peer adversity (Petersen & Hyde, 2009). It has been suggested that the physical changes of puberty place girls more at odds with the societally prescribed ideal of beauty characterised by thinness (Blyth, Simmons, & Zakin, 1985; Hyde et al., 2008). Weight gain associated with puberty is linked to increased perception of being overweight, increasing risk for depression, eating disorders, body image disturbance, and low self-esteem (Ge, Elder Jr, Regnerus, & Cox, 2001; Hyde et al., 2008; Klump, 2013).

The relatively rapid development of overt pubertal characteristics also brings young adolescents quickly toward a more adult-like appearance. In turn, family, peers, and society at large will react to them in different ways. As secondary sex characteristics develop, there is an increase in sexualisation and objectification, especially for girls (Hayward & Sanborn, 2002; Koff et al., 1978; Mendle, 2014). Early developing boys on the other hand might be seen as more self-sufficient and less in need of support (Mendle & Ferrero, 2012). For both girls and boys, earlier pubertal development is likely to increase social expectations of maturity and potentially confuse self-concept (Mendle & Ferrero, 2012). Cultural norms might also influence the meaning and subsequent affective valence of pubertal development (Hayward & Sanborn, 2002), opening the possibility for cultural differences in the development of social-emotional disorders.

2.2. Emotional experience

One of the hallmarks of adolescence is heightened emotionality. Relative to both children and adults, adolescents show more frequent, intense, and unstable expression of emotions that is most pronounced early in adolescence (Bailen, Green, & Thompson, 2018; Guyer et al., 2016). Heightened emotional experience has been demonstrated most consistently in response to social cues (Crone & Dahl, 2012; Guyer et al., 2016; Silvers et al., 2012; Somerville, 2013) and tends to be stronger in girls than boys (Bailen et al., 2018).

Emotional experience involves a discrepancy between: 1) emotional reactivity and 2) emotional regulation (inhibition). Theoretical models have proposed that emotional reactivity increases rapidly in early adolescence (underpinned largely by hormonal changes as described above) and this is followed by a slower and more gradual increase in response inhibition (characterised by maturation of cortical brain regions and improved coping and regulation skills as described below) that continues into early adulthood (Somerville, Jones, & Casey, 2010; Steinberg, 2008). Hence the discrepancy between reactivity and inhibition (referred to here as emotional experience) peaks early in adolescence and gradually decreases across the adolescent period. In turn, we expect that pre-adolescent vulnerabilities to negative affectivity will increase the discrepancy between adolescent emotional reactivity and inhibition, thereby magnifying emotional experience. The heightened experience of negative emotion during adolescence provides obvious links with the emotional disorders characteristic of this period – anxiety, negative body image, and mood disorders.

As noted above, increases in emotional reactivity during adolescence are strongly associated with pubertal hormones and concomitant brain development. Adolescents generally react with stronger emotions, especially to social cues, than do adults or children (Bailen et al., 2018; Hare et al., 2008). A related finding is the demonstration across both humans and animals of attenuated fear extinction during adolescence relative to both childhood and adulthood (Baker, Den, Graham, &

Richardson, 2014; Ganella, Drummond, Ganella, Whittle, & Kim, 2018; Pattwell et al., 2012), which appears to be mediated by the changes in brain structure underpinning adolescent emotionality and inhibition (Baker et al., 2014; Ganella et al., 2018). Reduced extinction of fears may be an important mechanism in the onset or at least the maintenance of anxiety disorders. This effect appears to be even stronger in the presence of higher depression (Den, Graham, Newall, & Richardson, 2015), pointing to one specific process that may increase comorbidity across the social-emotional disorders.

Adolescence incorporates a period of development for both improved coping and emotion regulation (Compas et al., 2017; Schäfer, Naumann, Holmes, Tuschen-Caffier, & Samson, 2017; Zimmer-Gembeck & Skinner, 2011). These regulatory processes include both covert (cognitive) and overt (behavioural) strategies, which begin to develop in mid-to-late childhood, but undergo substantial development right across adolescence. Changes in these volitional regulatory strategies are accompanied by maturation and development in brain structures that underpin response inhibition (Guyer et al., 2016; Steinberg, 2008). Clearly individual differences exist in the development of inhibitory abilities and these differences may be linked to onset of emotional disorders.

Research with adolescents has mostly demonstrated general (transdiagnostic) associations between maladaptive emotion control strategies and social-emotional disorders (Compas et al., 2017; Rood, Roelofs, Bögels, Nolen-Hoeksema, & Schouten, 2009; Schäfer et al., 2017). Poor emotional control has been associated with emotional disorders throughout development. However, the associations between some maladaptive coping strategies and internalising disorders appear to be especially strong during the adolescent years (Compas et al., 2017; Rood et al., 2009; Schäfer et al., 2017). For example, reappraisal, rumination, and emotional suppression all appear to have stronger associations with internalising distress during adolescence than earlier in childhood. Adolescent girls use more maladaptive and fewer adaptive coping strategies than boys (Hampel & Petermann, 2005), corresponding with the female preponderance in these disorders.

In summary the heightened emotional experience that characterises adolescence is likely to play a central role in triggering the onset of all of the social-emotional disorders. Heightened emotional expression is likely to increase incidence of these disorders both directly, and through moderation with pre-adolescent emotionality, cognitive biases, and temperament. In addition, as described in the next section, there is evidence for reciprocal relations between heightened emotional expression and sleep deficits that may further magnify these effects.

2.3. Sleep

Pre-to early adolescence signals the onset of major changes in sleep patterns that continue well into the adolescent period. Most notably there is both a shift in the timing of sleep toward later sleep onset and later wakening (often referred to as eveningness) and a general reduction in the total quantity of sleep (Carskadon, 2011). Optimal sleep duration across adolescence is thought to be just over 9 h per day (Gradisar, Gardner, & Dohnt, 2011; Short, Weber, Reynolds, Coussens, & Carskadon, 2018), however, sleep debt among adolescents is widely reported and is considered by some to be a serious public health issue (Adolescent Sleep Working Group, Committee on Adolescence, & Council on School Health, 2014).

These normative changes in adolescent sleep patterns are attributable to a range of biopsychosocial factors. Biological shifts towards later sleep timing are primarily caused by changes to the circadian and sleep homeostatic systems. Endogenous rhythms of core body temperature and melatonin (both of which regulate sleep and alertness across the 24-hr day) become later (Carskadon, Labyak, Acebo, & Seifer, 1999; Carskadon, Wolfson, Acebo, Tzischinsky, & Seifer, 1998) and sleep pressure (i.e., sleepiness) takes longer to accumulate across the day (Jenni, Acherman, & Carskadon, 2005), allowing adolescents to stay awake longer. As noted earlier, some of these shifts are also linked to hormonal changes during puberty. In concert with these circadian shifts, school start times naturally curtail adolescent sleep duration and sleep debt can compound over the school week and school term (Lo et al., 2017). Adolescents often try to "catch up" on sleep on weekends, although this may not be sufficient to make up the overall debt (Lo et al., 2017). Indeed, when allowed to sleep *ad libitum* (during school holidays, weekends), adolescents elect to go to bed and wake later, resulting in increased sleep duration (Bei et al., 2014).

Sleep duration in adolescence is also heavily impacted by a number of other social and environmental factors. For example, although parental control over bedtimes typically decreases as children move into adolescence (Short et al., 2011), research shows that adolescents whose parents continue to set bedtimes report more sleep and daytime wakefulness and less fatigue than those without set bedtimes (Gangwisch et al., 2010; Short et al., 2011). In turn, having parents who set earlier bedtimes is associated with less depression and suicidal ideation (Gangwisch et al., 2010). Similarly, having a positive family environment (potentially a pre-adolescent protective factor) is associated with earlier adolescent bedtimes and longer sleep duration (Bartel, Gradisar, & Williamson, 2015). The adolescent years are also characterised by increasing social demands and activities, as well as the escalating use of technological devices. There is growing evidence that the use of technological devices late at night or in bed, is associated with reduced sleep among teenagers (Bartel et al., 2015; Hale & Guan, 2015).

Chronic sleep restriction has a wide range of negative consequences (Gregory & Sadeh, 2012; Shochat, Cohen-Zion, & Tzischinsky, 2014). The most immediate effects of insufficient sleep include deficits in emotion regulation and increases in general negative affectivity (Baum et al., 2014; Palmer & Alfano, 2016). Following these short-term effects, sleep deficits have been associated with a wide range of psychopathology (Harvey, Murray, Chandler, & Soehner, 2011). Harvey et al. (2011) suggest that sleep difficulties are a transdiagnostic cause of psychopathology, although some empirical literature has shown particularly robust associations between sleep difficulties and internalising disorders among adolescents – particularly anxiety and depression (Alfano & Gamble, 2009; Alfano, Zakem, Costa, Taylor, & Weems, 2009; R. J.; Kelly & El-Sheik, 2014; Lovato & Gradisar, 2014).

Emotional disorders and sleep difficulties are clearly bi-directionally related, although the bulk of evidence appears to indicate that sleep disturbance more strongly predicts adolescent anxiety and depression, rather than the opposite direction (R. J. Kelly & El-Sheik, 2014; Lovato & Gradisar, 2014; Roberts & Duong, 2017). Importantly, the relationship between sleep problems and internalising symptoms in adolescence appears to be stronger following puberty, compared to earlier in childhood (R. J. Kelly & El-Sheik, 2014; McMakin & Alfano, 2015), highlighting sleep disturbance as an adolescent risk for social-emotional disorders.

Considerably fewer studies have investigated the relationship between sleep and eating pathology, although short sleep duration does appear to pose a risk for overweight and obesity (Fatima, Doi, & Mamun, 2015). One large population-based study showed that adolescents with symptoms of insomnia were four times more likely to experience an eating disorder (Blank et al., 2015). A longitudinal study with undergraduate students found disordered eating (bulimic behaviours, social pressure to eat) also predicted later symptoms of insomnia (Bos et al., 2013). However, studies investigating the bidirectionality between sleep disturbance and eating pathology are lacking, particularly in younger adolescent samples.

Adolescent sleep disturbance is likely to confer risk for social-emotional disorder via a number of underlying mechanisms. Although many suggestions have been made about processes that might mediate the relationships between sleep and psychopathology (Gregory & Sadeh, 2012; McMakin & Alfano, 2015), one of the most likely mediators is emotional experience (Palmer & Alfano, 2016) (Fig. 1). Sleep deprivation studies show that short-term sleep loss increases negative

affectivity and reduces positive affectivity (Lo, Ong, Leong, Gooley, & Chee, 2016; McMakin et al., 2016). At least some of this effect is due to increases in emotional reactivity (Palmer & Alfano, 2016; Reddy, Palmer, Jackson, Farris, & Alfano, 2017). However, there is evidence that sleep deficits also impact on the ability to regulate emotions (Baum et al., 2014; Palmer & Alfano, 2016).

A number of related processes that likely underpin development and maintenance of social-emotional disorders have been associated with sleep disturbances. For example, rumination may be facilitated in the pre-sleep period (Lovato & Gradisar, 2014), which may result in biased consolidation of emotional memories (Blake, Trinder, & Allen, 2018). Similarly, sleep problems may be linked with social-emotional disorders by magnifying interpretation biases (e.g., unhelpful thinking styles), judgement biases and negative attribution styles (e.g., control beliefs) (Blake et al., 2018). A recent population-based evaluation of over 10,000 adolescents aged 13–18 years, showed significant relationships between self-reported sleep problems and increased levels of rumination and suppression, along with poorer problem-solving (Palmer, Oosterhoff, Bower, Kaplow, & Alfano, 2018).

Insufficient sleep in adolescence and associated daytime sleepiness may also lead to social withdrawal and difficulties interacting with peers (Carney, Edinger, Meyer, Lindman, & Istre, 2006; Sarchiapone et al., 2014), which may precipitate or perpetuate symptoms of depression and anxiety (Blake et al., 2018). Conversely, there is evidence suggesting social isolation predicts sleep disturbance and depressive symptoms (Harris, Qualter, & Robinson, 2013). Therefore, the relationship between sleep and social problems in adolescence is likely bidirectional although longitudinal studies are required to determine the temporal order and effects on social-emotional disorders. As discussed in the next section, interpersonal relationships are critical to adolescents and can drive both psycho-social growth and risk.

2.4. Peer relationships

The adolescent period is critical for social development and is marked by both an increasing salience of peer relationships relative to family and greater volatility and complexity of social relationships (Crone & Dahl, 2012; O'Brien & Bierman, 1988; Somerville, 2013). Compared with children, adolescents spend increasingly greater amounts of time with their peers (Meuwese, Cillessen, & Güroğlu, 2017), thereby magnifying the impact of peers on the adolescent's behaviour. Although peer relationships can impact the emergence of a variety of disorders throughout development, the increased importance of peers during adolescence is especially likely to amplify any impact of peer relationships on the incidence of social-emotional disorders during this period. This is particularly true among girls for whom the impact of peer evaluation on self-worth is especially important (O'Brien & Bierman, 1988).

The transition into adolescence often coincides with a move from primary school to upper (middle or high) school. For many young people, this move results in a disruption of existing (often long-standing) friendships and peer networks along with the need to establish new affiliations and hierarchies (Meuwese et al., 2017). As young people make the transition to high school, peer crowd affiliation becomes a new and important aspect of peer relations. Thus, peer status becomes increasingly important, often causing individuals to compete for the attention of those at the top of the social hierarchy, potentially exposing them to greater peer conflict (Meuwese et al., 2017). As the complexities of peer interactions increase, so does the likelihood of experiencing negative peer interactions, if individuals are unable to effectively interpret and communicate in novel social situations (Rudolph, 2002). Hence social competence is a key feature of peer relationships during adolescence and is inherently tied to peer rejection or acceptance (Brown & Larson, 2009). Lack of social competence is also likely to be one vulnerability factor that increases risk for mental disorders prior to adolescence, interacting with the increasing salience of peers during the developmental transition to adolescence. The increasing importance of peer relationships in adolescence is evidenced by research demonstrating that levels of interpersonal stress markedly increase from preadolescence to adolescence for both girls and boys (Sontag & Graber, 2010).

As represented in Fig. 1, the impact of peer relationships on social-emotional disorders is underpinned by individual differences in peer acceptance, peer support, and peer influence. Each of these will be discussed below.

2.4.1. Peer acceptance

Peer acceptance (or rejection) is an especially critical component of peer relationships. This includes both the level of acceptance (vs. rejection or neglect) by the broader peer group as well as the adolescent's affiliation with particular peer types or crowds (e.g. sporty, popular, intellectual). Both, in turn, reflect their status within the social hierarchy. Experimental evidence has demonstrated stronger negative affective reactions to peer rejection and ostracism among adolescents than among either pre-adolescent children or adults (Sebastian, Viding, Williams, & Blakemore, 2010; Silk et al., 2012) and perceived peer status is an important moderator of the extent to which teenagers' behaviour is affected by their peers (Nesi & Prinstein, 2015; Prinstein & La Greca, 2002).

A wealth of evidence has demonstrated associations between social-emotional disorders and low acceptance from peers. Empirical evidence has consistently shown associations between peer acceptance, rejection, and neglect, and both depression and anxiety (e.g., Epkins & Heckler, 2011; Rubin, Chen, McDougall, Bowker, & McKinnon, 1995). Both experimental and longitudinal studies demonstrate that peer rejection can lead to depression among adolescents (Nolan, Flynn, & Garber, 2003; Platt, Kadosh, & Lau, 2013). This relationship is not specific to adolescence, but the greater salience of peers during the adolescent period is likely to strengthen these relationships. For example, inhibited and withdrawn behaviour appears to be reasonably acceptable to peers during the early childhood years, but becomes increasingly less accepted with increasing age (Ladd & Burgess, 1999; Rubin, Bowker, Barstead, & Coplan, 2018), increasing the likelihood that temperamentally withdrawn youth will be rejected when they reach adolescence. Further, the depressogenic impact of peer rejection appears to be stronger among adolescents than among children or adults (Platt et al., 2013). Similarly, social-emotional distress is associated only with severe peer rejection in childhood, but with both peer rejection and peer neglect in adolescence (Epkins & Heckler, 2011; Newcomb, Bukowski, & Pattee, 1993). Further, the greater importance of social relationships for girls compared with boys (Rose & Rudolph, 2006) means that socially-related stressors, such as peer rejection, precipitate greater depression among girls (Hankin, Mermelstein, & Roesch, 2007; Rudolph, 2002), contributing to the sex difference in this disorder.

The most extreme example of peer rejection is peer victimisation, for which there is extensive evidence associating it with anxiety and depression (Casper & Card, 2017; Hawker & Boulton, 2000; Tsaousis, 2016). Moreover, specific teasing about weight is associated with body dissatisfaction and this relationship appears to be mediated by social comparisons (see below) (Schaefer & Blodgett Salafia, 2014). Longitudinal research demonstrates that the relationships with victimisation are bi-directional – peer victimisation predicts social-emotional disorders, which also predict peer victimisation (Forbes, Fitzpatrick, Magson, & Rapee, 2018; Reijntjes, Kamphuis, Prinzie, & Telch, 2010). In contrast, the data for broader peer acceptance and rejection is a little more complex. Both acceptance and rejection appear to be bi-directionally related with internalising symptoms when based on adolescents' self-reported measures (Cole, Martin, Powers, & Truglio, 1996; Nolan et al., 2003). However, when using multi-informant data (parents, teachers, adolescents), unidirectional relationships are indicated, where peer rejection and low acceptance predict later internalising symptoms among adolescents (Cole, Martin, & Powers, 1997). Pre-

existing child characteristics, such as poor social competence, can reduce peer acceptance in adolescence (Brown & Larson, 2009). There is also some indication that the impact of peer rejection can be moderated by pre-existing vulnerabilities such as negative attitudes and emotional symptoms (Braet, Van Vlierberghe, Vandevivere, Theuwis, & Bosmans, 2013; Prinstein & Aikins, 2004). Hence adolescent peer acceptance and rejection may both moderate and mediate the relationships between childhood vulnerabilities and later disorder.

Some interesting research has connected the impact of pubertal timing on social-emotional disorders with peer stress (a broad term incorporating a range of peer relationship difficulties such as few friendships, peer disagreements, and victimisation). As noted above, early pubertal maturation may provide risk for the development of internalising disorders, partly through its social impacts. Young people (and especially girls) who visibly mature earlier than their peers are likely to be treated differently to their peers, including experiencing higher amounts of peer-related stress. In a longitudinal study, peer stress was shown to both moderate and mediate the relationship between early pubertal timing and depression for girls but not boys (Conley & Rudolph, 2009; Conley, Rudolph, & Bryant, 2012). Girls who perceived themselves as more developed than their peers and who also experienced heightened peer stress, reported more depression than other girls, 12 months later. Further, early pubertal timing among girls predicted greater peer stress over the following year which, in turn, predicted more depression 12 months later. Hence, the morphological changes associated with early puberty increase risk for peer relationship difficulties, ultimately increasing risk for social-emotional disorders.

2.4.2. Peer support

During the transition from childhood to early adolescence, peers become increasingly important providers of social support (Furman & Buhrmester, 1992; Laible, 2007; Zimmer-Gembeck & Skinner, 2011), which is a protective factor for social-emotional disorders right across the lifespan (Gariépy, Honkaniemi, & Quesnel-Vallée, 2016). This increasing focus on support from peers continues throughout adolescence and is eventually subsumed by a romantic partner in late adolescence (Collins & Laursen, 2004; Furman & Buhrmester, 1992). Interestingly, however, having a close and supportive specific friendship may be less important for mental health than acceptance from the general peer group (Rueger, Malecki, Pyun, Aycock, & Coyle, 2016).

The increasing salience of peers can also have a negative impact on the nature and quality of non-peer relationships, especially family relationships, across adolescence. Adolescents tend to disengage from parental control and assert more autonomous control over their decision making, emotions and behaviours (Choudhury, Blakemore, & Charman, 2006; Hostinar, Johnson, & Gunnar, 2015). This often contributes to increasing parent/child conflict, which peaks around mid-adolescence. During this time, dependencies are transferred from parents to peers, with early adolescents seeking out peers as their primary source of intimacy and social support (Laible, 2007) until later adolescence, after which perceived parental support once again increases (Furman & Buhrmester, 1992). Yet despite the increasing conflict between adolescents and their parents, families remain important providers of support right across development (van Harmelen et al., 2016).

Social support from friends and peers can provide protection from social-emotional distress (van Droogenbroeck, Spruyt, & Keppens, 2018). For example, in a longitudinal Norwegian sample, social skills at age 12 predicted symptoms of depression at age 16 (for girls but not boys) and this effect was mediated by peer support (but not by support from teachers or parents) in the interim period (Nilsen, Karevold, Røysamb, Gustavson, & Mathiesen, 2013). In a comprehensive review of the literature associating social support with depression in adolescents, small to moderate relationships were demonstrated both cross-sectionally and longitudinally (Rueger et al., 2016). Among studies that utilised psychometrically sound instruments and a longitudinal design, functional (perceived quality) support from the general peer group provided considerably stronger protection from depression (r = .31), than support from close friends (r = .08) and somewhat stronger effects than support from teachers or family (r = .16 and r = .21, respectively).

2.4.3. Peer influence

Peer influence refers to the many ways in which friends and the broader peer group affects the adolescent's attitudes, behaviours, and functioning. Although peers can be influential across development, the increased importance of peers during the adolescent period makes this a developmental phase that is especially vulnerable to these effects (Gardner & Steinberg, 2005; van Hoorn, Fuligni, Crone, & Galvan, 2016), particularly during the middle adolescent years (Steinberg & Morris, 2001). Peer influence (including what is known as peer contagion) has been demonstrated on both negative characteristics (such as aggression and negative affectivity) and positive behaviours (such as learning and prosocial behaviour) (Choukas-Bradley, Giletta, Cohen, & Prinstein, 2015; Dishion & Tipsord, 2011; van Hoorn et al., 2016; Van Hoorn, van Dijk, Meuwese, Rieffe, & Crone, 2014).

Considerably more research has been conducted into peer influence with respect to externalising disorders than internalising disorders (Dishion & Tipsord, 2011). Nonetheless, there are sufficient examples in the literature to suggest that peer influence is an important process in the development of social-emotional disorders. For example, suicidal adolescents are more likely to associate with peers who are high in internalising and suicidal ideation (Fulginiti, Rice, Hsu, Rhoades, & Winstrobe, 2016; Ho, Leung, Hung, Lee, & Tang, 2000). More broadly, peer contagion has been shown to impact the development of depression among adolescents. In one longitudinal study, Stevens and Prinstein (2005) showed that depressive symptoms and depressogenic attributional styles reported by adolescents' best friends were prospectively related to their own levels of depression. Other research has indicated that the effects of peer contagion can be moderated by existing vulnerability (Cohen & Prinstein, 2006). Using an experimental manipulation, the authors demonstrated that peer influence was stronger when adolescents who were low in social anxiety were exposed to high status peers, but adolescents high in social anxiety were similarly influenced by peers regardless of social status. In a subsequent study, Prinstein (2007) demonstrated that the effects of peer contagion on depressive symptoms were strongest among girls who were high in social anxiety, thereby indicating that pre-morbid characteristics can moderate the influence of peers on development of social-emotional disorder.

Additional evidence has demonstrated a peer influence effect with respect to eating disorders and dieting behaviour (Paxton, Schutz, Wertheim, & Muir, 1999; Schaefer & Blodgett Salafia, 2014). Adolescent girls have been shown to associate with girls who share similar attitudes as themselves toward dieting, weight loss, and purging (Hutchinson & Rapee, 2007; Paxton et al., 1999; Rayner, Schniering, Rapee, Taylor, & Hutchinson, 2013). These associations may partly represent contagion effects since some research has indicated that individual dieting attitudes and behaviours are preceded by similar attitudes among friendship groups (Field et al., 2008; Paxton, Eisenberg, & Neumark-Sztainer, 2006).

Mechanisms through which peers may influence each other's internalising symptoms have not been extensively studied. However, one process that has received some evaluation is co-rumination. Co-rumination refers to the repeated discussion and rehashing of problems, emotional issues, and negative affect between friends and is comprised of components of both peer support (positive) and shared rumination (negative) (Rose, 2002). In her initial evaluation, Rose (2002) demonstrated a small but significant association (r = .2) between co-rumination and internalising symptoms. Co-rumination was higher among girls and provided a significant indirect path between gender and internalising symptoms. Further, the difference in co-rumination between

girls and boys was greater among adolescents than among pre-adolescents. Several studies have now demonstrated positive associations between co-rumination and depression (Hankin, Stone, & Wright, 2010; Rose, Glick, Smith, Schwartz-Mette, & Borowski, 2017; Schwartz-Mette & Rose, 2012). Importantly, this work predicts cascading relationships such that a tendency to engage in co-rumination increases depressogenic processes over time, thereby increasing depression (Rose et al., 2017).

2.5. Self-concept

Adolescence is a key period for the development of self-concept, as the adolescent builds a stronger sense of the self, distinct from their family. An adolescent's sense of self is influenced by multiple factors, but is broadly affected by a combination of personal experience and social feedback (Sebastian, Burnett, & Blakemore, 2008). Along with the increased importance of peers during adolescence comes greater concern about social evaluation, including increased self-consciousness (Steinberg, 2005; Westenberg, Gullone, Bokhorst, Heyne, & King, 2007). In fact, the perceived expectations from significant others are often more important than self-expectations for development of the adolescent self-concept (O'Brien & Bierman, 1988; Prinstein, Rancourt, Guerry, Browne, & D'Onofrio, 2009). As adolescents age, they gradually develop a more integrated and consistent (or clear) sense of themselves (self-concept clarity) and ratings of self-concept have been shown to be more stable later in adolescence than earlier (Crocetti, Rubini, Branje, Koot, & Meeus, 2015). Similarly, although the valence of self-concept (self-esteem) might decrease in early adolescence, it appears to gradually increase again from middle adolescence (Kort-Butler & Hagewen, 2011). As indicated in Fig. 1, individual differences in both the clarity and valence of self-concept are likely to be related to development of social-emotional disorders, both directly, and in interaction with pre-adolescent risk factors.

The social-emotional disorders are all characterised by negative representations of the self across the lifespan. A wide variety of negative beliefs along with negatively biased perceptions of appearance, performance, and global self-descriptors have been commonly associated with all of the anxiety, mood, and eating disorders (Kyrios et al., 2016). In fact, negative perceptions of the self are so intertwined with the social-emotional disorders as to be considered a hallmark of these conditions. Low self-esteem predicts future depression, anxiety, and body dissatisfaction among adolescents (Lee & Hankin, 2009; Paxton et al., 2006), although this relationship is not specific to adolescence and appears bi-directional (Sowislo & Orth, 2013). Consistent with the female preponderance in these disorders, boys generally report more coherent self-concept and positive self-esteem than girls (Crocetti et al., 2015).

In addition to the importance of the valence of self-concept, a smaller body of research has pointed to a link between the clarity of self-concept (the extent to which an individual feels certain about who they are and describes themselves in consistent ways) and social-emotional disorders. Evidence has shown associations between poorer self-concept clarity and anxiety, eating disorders, and depression among adolescents (G. Liu et al., 2017; Schwartz et al., 2011; van Dijk et al., 2014; Vartanian, Hayward, Smyth, Paxton, & Touyz, 2018). At least some of this work has demonstrated prospective relationships in which lower clarity of self-concept predicts future anxiety and depression (Schwartz et al., 2011; van Dijk et al., 2014).

The increased fluidity and weaker integration of self-concept in early adolescence (Crocetti et al., 2015) provides a window of opportunity for self-concept to change in a way that may result in more adaptive emotional functioning, or more negative social-emotional outcomes. Many factors are likely to influence the clarity and valence of self-concept. Among them, existing vulnerabilities from childhood (such as pre-existing self-esteem) are likely to provide the most powerful influence. However, concurrent influences including peer factors, negative life events, and family support may also affect the adolescent's emerging self-concept, with the interaction of these factors having the potential to influence emotional outcomes.

Social comparisons (i.e., the extent and nature of the ways in which adolescents compare themselves against their peer group) are an especially powerful way by which adolescents inform their sense of self, and the frequency of comparisons increases during adolescence (Martin & Kennedy, 1993; Schutz, Paxton, & Wertheim, 2002). Adolescents repeatedly compare themselves to their peers to establish what is normal and to determine where they fit. Upward social comparisons (comparing to someone perceived to be “better” than you on an attribute) can variously increase aspiration for self-improvement or promote negative self-evaluation, depending on the perceived relevance of the attribute (Tesser, Millar, & Moore, 1988; Wood, 1989). Given the importance of appearance and social status among adolescents, upward comparisons may be especially harmful during this period (Hargreaves & Tiggemann, 2004). Although peers are the most salient comparator during adolescence, they are not the only source of information – exposure to idealised images from broader media and even to siblings produces similar effects (Jensen, Pond, & Padilla-Walker, 2015; Mulgrew, Volcevski-Kostas, & Rendell, 2014; Tiggemann & Miller, 2010).

An extensive body of research has demonstrated the importance of social comparisons to the development of body image concerns and low mood. Adolescents who engage in a greater frequency of upward social comparisons report poorer body image, higher social anxiety, and more depression (Cunha, Soares, & Pinto-Gouveia, 2008; Durkin & Paxton, 2002; Irons & Gilbert, 2005; Webb, Zimmer-Gembeck, & Donovan, 2014) and this relationship appears stronger among adolescents than young adults (Groesz, Levine, & Murnen, 2002; Myers & Crowther, 2009). Experimental work has demonstrated a causal relationship, at least at a state level. Specifically, exposure to images of socially idealised figures among adolescent girls leads to state reductions in body satisfaction and mood (Groesz et al., 2002). Although moderation of this effect has been demonstrated in response to pre-existing body-relevant factors (Durkin & Paxton, 2002; Groesz et al., 2002), moderation by broad, childhood vulnerability variables has not been evaluated. On the other hand, social comparison has been shown in at least one study to mediate the relationship between childhood vulnerability (childhood inhibition reported retrospectively) and adolescent social anxiety (Cunha et al., 2008). Finally, evidence suggests that young adolescents who enter puberty early engage in more appearance comparisons than later-developing youth (Mitchell, Petrie, Greenleaf, & Martin, 2012; Petrie, Greenleaf, & Martin, 2010), suggesting that social comparisons might mediate the relationship between early pubertal timing and social-emotional disorder.

In the twenty-first century, online comparisons through social media have become particularly important to adolescent development. Social media allows teenagers unprecedented access to their peers along with extensive opportunity for social comparison through the image-based focus of the most popular current platforms. Greater time spent using social media appears to be negatively related to self-esteem (D. Liu & Baumeister, 2016). Although the direction of this effect is not yet known, one recent study demonstrated that low self-esteem predicts greater subsequent use of social media sites (Valkenberg, Koutamanis, & Vossen, 2017). However, it is the nature of the activity on social media that is likely to be of greatest importance. Most youth present themselves on social media using overly positive representations, which is likely to lead to greater engagement in upward social comparisons among viewers. Hence viewing and comparing to peers on social media may be especially likely to trigger social-emotional distress. Adolescent girls frequently use social media to engage in social comparison (Burnette, Kwitowski, & Mazzeo, 2017), and those who do have poorer body image and lower mood than those who use it for other purposes (Rousseau, Eggermont, & Frison, 2017; Weinstein, 2017). In one of the few longitudinal studies in this field, Nesi and Prinstein (2015)

demonstrated that among adolescents aged 12–16 years, higher engagement in social comparison and feedback-seeking over the internet predicted greater depression one year later. This effect was moderated by gender and popularity – girls who were low in peer popularity and who engaged in higher levels of social comparison online were most likely to be depressed a year later.

3. A conceptual model of the impact of adolescent characteristics on the development of social-emotional disorders

As outlined in the previous sections, a variety of the characteristic features of adolescent development are likely to confer increased risk for the onset of the mental disorders that we refer to as "social-emotional". Following from the relationships and evidence described above, we propose a conceptual model that is depicted in Fig. 1. It is important to point out that we are not arguing that adolescent characteristics account for the majority of risk for onset of these disorders, much of which will come from pre-adolescent (childhood) risk factors. Rather, within a context of a range of risk factors relevant to each disorder, there exist a subset of risk factors (characteristic of adolescent development) that increase the likelihood that the disorder will manifest at that time. We also do not claim that all aspects of the model will relate to each of these disorders equally. While the social-emotional disorders described here all share considerable comorbidity and therefore are likely to share risk, each also has a number of unique characteristics and risk factors.

At the heart of the model is the assumption that most cases of social-emotional disorders will have their origins in vulnerability that begins well before the adolescent period. Widely researched risk from birth and through childhood includes genetic vulnerability; temperament; parent psychopathology; parenting style; cognitive styles; and social skills and interactions (Hankin, 2012; Rapee, Schniering, & Hudson, 2009; Yap & Jorm, 2015). We argue that normal characteristics of adolescent development subsequently interact with these pre-adolescent risks to trigger onset of disorder. Of course, in some cases, extremes of normal adolescent characteristics could act as main effects, eliciting onset of social-emotional disorder despite a lack of pre-adolescent risk, but in most cases we predict that adolescent development will interact with earlier risk. Pre-adolescent risk factors can also predict extremes of normal adolescent developmental characteristics. Thus, adolescent developmental characteristics can also mediate the relationship between childhood risk and social-emotional disorder, essentially reflecting developmental cascades (Masten & Cicchetti, 2010). For example, a child whose temperament is characterised by heightened emotionality may be more likely to be rejected by peers during adolescence, thereby increasing risk for depression. In summary, adolescent developmental characteristics can both moderate and mediate pre-adolescent risk factors to trigger onset of social-emotional disorders.

According to the model, pubertal changes early in adolescence will have internal, emotional, impact as well as external, observable effects. Internally, the hormonal changes during puberty will trigger a combination of sleep shifts and increased emotionality that are both amplified by pre-existing risk and moderate that risk to elicit social-emotional disorder. Heightened negative emotional reactivity and diminished emotion regulation capacity that characterises adolescence will likely initiate cascading effects that lead to the onset of disorder. Emotional volatility commonly impacts relationships and activities, leading to vicious cycles between negative emotion and negative (non-independent) life events (Hammen, 2005). The four key disorders that increase during adolescence, social anxiety disorder, generalised anxiety disorder, eating disorders, and major depression, all have negative affectivity at their core.

The observable aspects of puberty will lead to a range of social interaction effects that provide heightened risk for social-emotional disorders. Young people often find the observable changes associated with puberty to be difficult, especially when these changes occur earlier than the majority of their peers. Young people with pre-existing weight concerns or heightened self-consciousness are likely to be especially impacted by these changes. According to the model, the observable changes associated with puberty, especially when they occur earlier than the immediate peer group, will negatively impact self-concept. In turn, this will increase the level of self-consciousness and the frequency of social comparisons, thereby increasing the likelihood of social-emotional disorders, especially among those with existing vulnerability.

As noted, increased importance of peer relationships is one of the primary aspects associated with adolescence, especially in the earlier years. This increased salience means that negative peer experiences, such as peer rejection and lack of support, have a particularly strong impact. Behavioural influence from peers is also heightened during this period. As for other aspects of the model, both mediation and moderation are predicted: these characteristics are likely to be partly a result of pre-existing vulnerabilities (e.g., poor social skills) and also intensify the impact of these vulnerabilities on the development of social-emotional disorders.

A characteristic feature of all of the social-emotional disorders is that they are more common among girls and women than boys and men. As noted throughout the preceding review, many of the features of adolescence described here that provide risk for social-emotional disorders are of greater significance for girls than for boys. Most importantly, the social risks, including the overall salience of social relationships, engagement in social comparisons, and the importance of peer support, hold greater relevance for girls than for boys. In this way, the eventual sex differences in social-emotional disorders are woven into the predictions from this model.

4. Limitations

The model and literature review have focused on Western culture, since that is where most of the extant literature exists. The model itself is somewhat agnostic with respect to cultural impact, although it inevitably has a Western bias. The entire concept of adolescence is culturally defined and different cultures have somewhat different views of the timing and meaning of adolescence. In that sense various aspects of the model may differ in non-Western cultures. Cross-cultural comparisons will be needed to determine the ways in which post-childhood developmental characteristics influence the development of social-emotional disorders across different cultures.

Naturally, the model is limited by the nature of our diagnostic systems. Comorbidity between disorders is common and there have been many attempts to delineate alternative models of psychopathology that account for these patterns (e.g., Krueger et al., 2018). Re-conceptualising the classification of mental disorders may alter the definitions of the constituent 'social-emotional disorders' as well as change patterns of incidence and comorbidity among them. However, the proposed model is inherently transdiagnostic, with a focus on shared characteristics and risk factors that span the putatively distinct disorders, and may represent a valuable contribution to developmental perspectives of these models.

While problems with substance use often increase during adolescence, we did not include them in the proposed model for two reasons: First, as reviewed earlier, onset of the diagnostic entity, substance use disorder, commonly falls within early adulthood. Second, substance use disorders are conceptually different to the social-emotional disorders discussed here and are consistently associated with externalising forms of psychopathology (Carragher et al., 2015), whereas the other disorders included in this model all fall within internalising. There is little doubt that aspects of adolescent development do confer risk for the onset of substance use disorders, but this is likely to be strongly linked with adolescent increases in risk taking (Carragher et al., 2015; Steinberg, 2008), which is different to the processes discussed in this model.

Finally, within the model we refer to broad adolescent processes (e.g., development of self-concept). However, under each lies several more specific processes (e.g., social comparison, self-esteem, self-consciousness). While we expect transdiagnostic relationships, specificity may emerge in how the constructs in the model relate to each other and to particular types of psychopathology. These relationships will become clearer as empirical tests of the hypothesised relationships in the model are conducted.

5. Implications

Increasing our understanding of the development of psychopathology requires theories that address each of the unique characteristics of mental disorders. One of the intriguing distinguishing features of many mental disorders is their different associations with particular life stages. For example, panic disorder has long been seen as a disorder that is rarely seen in childhood and several early theories tried to explain this specificity (Nelles & Barlow, 1988). Yet the same consideration has been rare among other forms of psychopathology. We have provided a unique psycho-social model that considers why it is that a specific subset of social-emotional mental disorders largely manifests in adolescence. While a few comprehensive examinations of this issue have been written they generally focused on hormonal and brain characteristics (Mendle, 2014; Paus, Keshavan, & Giedd, 2008; Powers & Casey, 2015). Therefore, the current model provides a unique psychosocial perspective that should generate novel research and add to our understanding of the development of these important disorders.

The key value in theories of psychopathology is the predictions they make toward improved management of the relevant disorders. Developmental theories have their primary implications for prevention. Current prevention of internalising disorders in adolescence focus primarily on reduction of symptoms (Werner-Seidler, Perry, Calear, Newby, & Christensen, 2017). Following from the current model, high schools might begin to focus on selective intervention (intervention aimed at risk factors). For example, programs might be targeted toward early adolescents with a history of pre-adolescent risk. Given the critical role of peer relationships in adolescence, greater incorporation of efforts to boost positive relationships should be especially important. Similarly, developing methods to reduce the tendency toward negative peer contagion and upward social comparisons may show important effects. Building emotion regulation skills, especially in early to middle adolescence, should be fruitful (Forbes, Rapee, & Krueger, 2019). The model also suggests critical developmental windows for maximum impact. For example, increasing knowledge about and acceptance of early morphological changes associated with puberty will be important during pre-adolescence. Evidence that self-concept shows greater fluidity and variability early in adolescence suggests that this would be a prime period during which to promote positive self-esteem and an increasing sense of self.

The adolescent developmental period has one of the most significant influences on an individual's functioning across the lifespan. It is a time of great opportunity and in most cases sets up a young person to maximize their opportunities across their life. However, a significant proportion of youth develop social-emotional mental disorders that will follow them well into their adult years. Identifying risk for the development of these disorders is the best way to establish effective prevention (Rapee, 2008). We have presented a model that we hope will guide new research into the processes that occur during adolescence that can increase the likelihood that a young person will develop social-emotional disorders. In turn, the model and subsequent research will help to guide more refined interventions for management and prevention of these disorders to give all young people the best chance to fulfil their maximum potential.

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Declaration of competing interest

The authors declare no conflict of interest.

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