ABSTRACT—Few periods of the life span are as dynamic, tumultuous, and emotionally salient as puberty. The combination of biological and social change during this transition contributes to sweeping shifts in the prevalence and nature of psychopathology. In this article, I highlight the role of puberty in psychological well-being, reviewing both individual and population-wide trends in psychological symptoms and disorders. Emphasis is on three domains associated with maturation: typicality of symptoms, epidemiological shifts, and individual differences.

KEYWORDS—puberty; psychopathology; internalizing; externalizing

INCREASES IN CLINICAL SYMPTOMATOLOGY ARE ENDEMIC AT PUBERTY

Puberty is typically associated with emotional distress. In both longitudinal and cross-sectional studies, children’s clinical symptomatology increases steeply when they reach puberty (e.g., Ge, Brody, Conger, & Simons, 2006; Hemphill et al., 2010; Oldehinkel, Verhulst, & Ormel, 2011). For some, this is the first onset of psychological symptoms, while for others the increase in symptoms represents an exacerbation or relapse of childhood difficulties. While not all of these emotional changes reach a diagnostic or clinically severe threshold, the ubiquity of the pattern is striking.

Increases in symptoms have been observed across a broad spectrum of disorders and across both genders. For example, progressing through puberty is associated with increases in depressive symptoms, social uncertainty, and anxiety in girls (e.g., Mendle, Harden, Brooks-Gunn, & Graber, 2010; Oldehinkel et al., 2011), and increased aggression, rule breaking, conduct problems, irritability, self-injurious behavior, and substance use in both genders (Hemphill et al., 2010; Oldehinkel et al., 2011; Patton et al., 2004; Patton et al., 2007). Prior to puberty, girls are more satisfied with their bodies, less likely to consider themselves fat, and less likely to diet or restrict eating (O’Dea & Abraham, 1999); later in adolescence, nearly half of girls report dieting and 10–14% report other signs of disordered eating, such as binging or purging (Neumark-Sztainer, Wall, Larson, Eisenberg, & Loth, 2011). These increases in symptoms occur independent of chronological age, implicating the changes that occur at puberty as a primary mechanism of risk. They have also been documented in individuals from a variety of backgrounds. For example, symptoms of major depressive disorder, social anxiety, generalized anxiety, oppositional defiant disorder, and conduct disorder increased at puberty among a group of African American adolescents (Ge et al., 2006). Likewise, among a group of Mexican adolescents, postmenarcheal girls reported difficulties with body image, lower self-esteem, more symptoms of depression, and greater externalizing behavior than premenarcheal girls; in fact, an alarming 31% reached a clinically severe level of depression in the six months following their first menstrual cycle (Benjet & Hernández-Guzmán, 2002).
Explanations for the increase in clinical symptomatology speak to the integrated nature of the pubertal transition. Hypotheses tend to consider biological and social changes, as well as the interplay between them. Physical changes at puberty can be attributed to increases in a number of hormones, most prominently estradiol and testosterone, but also adrenal hormones such as dehydroepiandrosterone (DHEA) and its sulfate, DHEAS. While associations between hormones and psychopathology are still not clearly understood, increases in testosterone during puberty have been linked with the rise in externalizing behavior in boys (Vermeersch, T’Sjoen, Kaufman, & Vincke, 2006) and changes in levels of estradiol have been associated with mood and disordered eating in girls (e.g., Angold, Costello, Erkanli, & Worthman, 1999; Klump, Keel, Sisk, & Burt, 2010).

These shifts in hormones both coincide and interact with ongoing neural maturation and prominent changes in social roles and relationships. Current perspectives on adolescent brain development emphasize inconsistent maturation across systems involved in cognitive control (e.g., prefrontal cortex) and those involved in emotion, arousal, and reactivity (e.g., subcortical limbic regions, including—but not limited to—the ventral striatum, amygdala, and hippocampus). Early in puberty, adolescents begin to show heightened responsivity in subcortical regions in response to socioemotional cues while regions governing cognitive control seem to develop independent of puberty. Greater levels of DHEA, estradiol, and testosterone—but not age—have been linked with processing social and emotional information, and testosterone levels predict activation of the ventral striatum in both boys and girls (reviewed in Peper & Dahl, 2013). Ironically, at the same time these neural and hormonal changes increase sensitivity to social influences, puberty creates a bewildering array of new social norms and expectations that may be perceived as alternately difficult, confusing, or exhilarating. These range from increased contact with peers and potential romantic partners to friendship changes, heightened parent-child conflict, intensified sex-role socialization, and the emergence of psychological distress and aggression in other adolescents in a social network. Without consistent and solid cognitive control, the imbalance across neural systems is believed to contribute to emotion dysregulation and risk for psychopathology in the face of these challenges.

EPIDEMIOLOGICAL PATTERNS OF PSYCHOPATHOLOGY CHANGE AT PUBERTY

In addition to intraindividual changes in symptomatology, puberty marks a period of notable population shifts in the prevalence of disorders. The most well-known example of this is the emergence of gender differences in depression: While rates of depression across gender are roughly comparable during childhood (with some studies even suggesting slightly higher rates of depression in boys), women are approximately twice as likely as men to be depressed. This disparity begins in early puberty; by midpoint in the transition, girls are significantly more likely to be depressed than boys, and this discrepancy persists through the rest of the life span. Conversely, externalizing behavior is more common in males than females. Conduct problems and aggression rise for both genders beginning at puberty and continue through adolescence, but the increase is more pronounced for girls than for boys, resulting in a narrower gender gap in externalizing behavior than at any other point in development. In some studies, levels of aggression and delinquency are actually comparable across genders by late puberty (e.g., Najman et al., 2009).

The overall frequency of certain disorders also shifts at puberty. For example, both panic attacks and panic disorder are rare in childhood (Craske et al., 2010), but they increase exponentially by the middle of puberty. Among a sample of middle school girls, none of the girls who were prepubertal or in the early stages of puberty experienced a panic attack compared to 8% of postpubertal girls of the same age (Hayward et al., 1992). The pattern for eating disorders is similar, with eating disorders relatively uncommon in prepubertal children of both genders. The prevalence of eating disorders expands rapidly for girls through puberty and adolescence, culminating in a 10:1 female-to-male ratio. Incidence of less common disorders—such as trichotillomania or excoriation disorder (picking at the skin)—also rise at puberty, with the common onset of picking for both disorders occurring shortly after the start of puberty (American Psychiatric Association, 2013).

Finally, puberty is a time of changes in prevalence of particular symptoms within disorders. In other words, once a child reaches puberty, a disorder may be characterized by different symptoms. For example, in bipolar spectrum disorders, manic/hypomanic symptoms tend to predominate in childhood while depressive symptoms predominate during puberty and adolescence (Schaufnagel, Brumback, Harper, & Weinberg, 2001). Depression during and after puberty seems to be characterized by more cognitive symptoms (e.g., hopelessness, helplessness, suicidal ideation) and vegetative symptoms (e.g., hypersomnia, lethargy) than depression before puberty, which is marked by greater irritability and somatic complaints (e.g., Oldehinkel et al., 2011; Yorbik, Birmaher, Axelson, Williamson, & Ryan, 2004). These changes in clinical presentation are likely a byproduct of the other changes, both social and biological, that occur during puberty—such as increased stress, rises in gonadal hormones, intensification of sex-role socialization, reactions to weight gain, and changes in circadian rhythms and sleep/wake cycles.

INDIVIDUAL DIFFERENCES AT PUBERTY ARE PSYCHOLOGICALLY INFORMATIVE

Associations of puberty with psychopathology have been explored most frequently through the lens of individual differences. There are substantial fluctuations in how puberty unfolds...
from person to person. To date, the most commonly discussed individual difference at puberty has been pubertal timing—
when adolescents reach specific physical milestones relative to peers of the same age and gender. In females, earlier timing of puberty predicts depression, suicidality, panic attacks, body dissatisfaction, disordered eating, delinquency, substance use and abuse, and academic difficulties. It is also associated with an increased risk for experiences that make adolescents more vulnerable, such as sexual victimization and intimate partner violence (e.g., Foster, Hagan, & Brooks-Gunn, 2008; Mendle, Turkheimer, & Emery, 2007). In males, earlier timing of puberty historically has been viewed as advantageous because it conveys physical advantages for athletic activities. However, more recent research suggests that boys who mature early are generally similar to girls who mature early with regard to internalizing and externalizing behaviors (reviewed in Mendle & Ferrero, 2012).

This consistency has typically been attributed to the natural difficulty many younger individuals may have navigating the pressures and changes of puberty, especially when compared to peers who reach the same developmental milestones at a later chronological age. A few additional studies indicate that very late maturation might be problematic for boys, though these findings are inconsistent. As (Rudolph, 2014) astutely notes, studies that report late puberty as difficult for boys generally used cross-sectional designs and measured puberty through subjective perceptions, whereas studies that report early puberty as difficult used longitudinal designs and more precise measures of development.

Adolescents also differ in the pace at which they mature, known as pubertal tempo. While the process of puberty typically spans about 4 years, variability in tempo means that puberty can range from 1 to 7 or more years. Although research on pubertal tempo is still emerging, a more rapid tempo of development seems more strongly associated with problem outcomes (e.g., depression, substance use, peer conflict) than a slower tempo (Castellanos-Ryan, Parent, Vitano, Tremblay, & Séguin, 2013; Marceau, Ram, Houts, Grimm, & Susman, 2011; Mendle et al., 2010, 2012). This is perhaps unsurprising, given that sudden change—regardless of when in the life span it occurs—can be more challenging than slower or more gradual change.

Puberty is not an isolated process, but part of a larger developmental continuum. Whether a child matures earlier or later (or more quickly or slowly) is neither coincidental nor random. Genes play an important role, with twin and family studies yielding heritability estimates for age at menarche in the .4 to .8 range (e.g., Anderson, Duffy, Martin, & Visscher, 2007). Similarly, genes accounted for 83% of the variance in many pubertal indicators, such as the timing of changes in skin or body shape, for both boys and girls (Mustanski, Viken, Kaprio, Pullkinen, & Rose, 2004). With regard to social contexts, earlier onset of puberty occurs disproportionately among adolescents whose environments are marked by adversities such as child maltreatment, poverty, unstable family structure, harsh or unsupportive parenting, and exposure to intimate partner violence (reviewed in Ellis, 2004). Likewise, low levels of parental warmth have been associated with faster tempo and more advanced development relative to peers among adolescents who displayed high levels of physiological reactivity to stress in early childhood (Ellis, Shirtcliff, Boyce, Deardorff, & Essex, 2011).

These correlations present a familiar conundrum for developmental psychopathologists: Because many of the antecedents of earlier (or more rapid) puberty also predict psychopathology, it can be difficult to discern if early (or more rapid) puberty represents an independent risk or is simply part of a larger nexus of adversity. One way to disentangle causation and reduce third-variable confounds is behavioral genetic designs, which account for differences in both genes and family environments among participants. Such studies tend to implicate consistent effects of earlier pubertal timing for a variety of outcomes, including disordered eating, depression, and delinquency (e.g., Harden & Mendle, 2012; Harden, Mendle, & Kretsch, 2012). Moreover, studies of twins and family members verify that social context explains pubertal timing matters. For example, environmental influences on behavior problems were greater for early developers than later-maturing peers (Burt, McGue, De Marte, Krueger, & Iacono, 2006). These results are consistent with various cross-cultural comparisons indicating weaker associations between pubertal timing and externalizing behavior in conservative cultures with limited contact between peers and members of the opposite sex during adolescence (Skoog, Stattin, Ruisevola, & Özdemir, 2013).

**IS WHAT HAPPENS AT PUBERTY IMPORTANT FOR LATER LIFE?**

A recurrent and pragmatic question is the extent to which clinical symptoms at puberty persist into adulthood. Little research addresses this question definitively, since most studies—even longitudinal ones—do not include assessments past middle to late adolescence. Answering this question requires three distinctions: between genders, between types of psychopathology, and between adolescents who mature normatively and those whose puberty is early, rapid, or otherwise not typical.

For girls, most studies suggest a relatively enduring association of high levels of internalizing symptoms during puberty with high levels of internalizing symptoms throughout adolescence. This seems particularly true for early matures, who continue to show elevated rates of depression and suicidality compared to peers into early adulthood (e.g., Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004; Natsuaki, Biehl, & Ge, 2009). The longest prospective study followed Swedish girls born in 1955 from ages 10 to 43. At age 43, early matures were less healthy physically and lacked educational/career achievement, but psychological differences between early developers and their peers had largely attenuated by this point (Johansson & Ritzén, 2005). For boys,
the limited longitudinal findings are mixed. For example, Natsuaki et al. (2009) reported that early-maturing boys had higher levels of depression in early adulthood relative to other male participants (though not relative to female participants), whereas Graber et al. (2004) did not. Studies of animals suggest that mice subjected to high levels of stress during puberty display greater levels of depressive symptoms when administered estrogen in adulthood than mice who were stressed before or after puberty. Although preliminary, one logical implication is that puberty is a sensitive period, with experiences at this time carrying enduring effects for estradiol-related behaviors and emotions (Blaustein & Ismail, 2013). This might explain the tendency toward relapses into depression at other points in life known for hormonal change, such as postpartum or menopause.

Externalizing behavior in general tends to decline as adolescents become adults: The typical pattern is for externalizing symptoms to begin to rise at puberty, persist through midadolescence, and decline in late adolescence and early adulthood. Teenagers who matured early or rapidly showed higher rates of violent and nonviolent delinquency and substance use continuing through adolescence (Castellanos-Ryan et al., 2013; Harden & Mendle, 2012). Such risk may affect functioning in adulthood: Women who reported depression in early adulthood were most likely to have been early maturers with high levels of externalizing as adolescents (Copeland et al., 2010). This may be a natural reflection of intermediary events and life circumstances, given the overlap between earlier puberty and stressors relevant for psychological functioning—including rape, intimate partner violence, teenage pregnancy, and career/economic hardship.

To date, many psychologists, educators, policy makers, and parents have operated under the premise that puberty is a normative transition, albeit one that carries some “growing pains.” Although the available longitudinal data limit a straightforward conclusion, for most individuals, distress during puberty is likely to be transient. Yet the real hazard of puberty is not whether psychological difficulties endure through middle age, but rather that they are maintained through the pivotal period of adolescence and early adulthood. The symptoms and correlates of pubertal development are hardly trivial to the adolescents who experience them and may place them on a path from which it is difficult to deviate. What happens during the span of reproductive maturation contributes to subsequent levels of physical and psychological health, educational attainment, and financial security not just for individuals, but for their offspring and families, the peers and romantic partners with whom they interact, and their broader neighborhoods and communities. For adolescents to thrive and our interventions to be effective, we must remember the importance of this transition. Unlike most other psychological risks, puberty itself is not preventable, but fortunately for prevention, intervention, and research, problems at this time are predictable.

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