

Descriptive Review: Hormonal Influences on Risk for Eating Disorder Symptoms During Puberty and Adolescence

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ABSTRACT

Objective: Puberty is an important period of risk for the onset of eating pathology in adolescent females. This review focuses on changes in reproductive hormones during puberty as one specific psychopathogenic mechanism.

Method: Studies of puberty and eating disorder-related phenotypes were identified using search databases and the reference sections of previous literature.

Results: Correlational studies of adult women and experimental studies of animals provide evidence for the effects of reproductive hormones on eating disorder symptoms. Very few studies of puberty, however, have directly measured or tested the effects of hormonal change in samples of human adolescents. Commonly used measures of pubertal development, such as menarche

or self-reported pubertal status, are relatively poor indicators of individual differences in hormones. The extent to which puberty-related hormonal change accounts for elevated risk for disordered eating remains unclear.

Discussion: Future research is necessary to elucidate the specific relations between hormonal change during puberty and risk for disordered eating. In particular, there is a need for longitudinal studies with multivariate measurement of pubertal development, including direct measures of change in reproductive hormones. © 2014 Wiley Periodicals, Inc.

Keywords: puberty; adolescence; hormones

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Hormonal Influences on Risk for Disordered Eating During Puberty and Adolescence

Eating disorder (ED) diagnoses in prepubertal children are extremely rare, but rapidly increase in prevalence over the course of puberty and adolescence.¹ More advanced pubertal development, particularly when it occurs at a relatively young age, confers risk for a broad range of ED-related outcomes, including binge eating, body dissatisfaction, concerns about weight, and clinical diagnoses of bulimia nervosa (reviewed in Refs. 2,3). In this article, we provide a critical analysis of reproductive hormones as one specific mechanism underlying

the relation between puberty and risk for ED symptoms in adolescent females. A number of recently published articles³ have comprehensively summarized the literature on puberty and ED symptoms. The goal of the current article is not to recapitulate these previous reviews, but rather to evaluate critically evidence in support of one hypothesized risk—reproductive hormones. Reflecting the bulk of the empirical and theoretical literature, we limit our article to reproductive hormones in females and to symptoms of disordered eating rather than clinical diagnoses.

First, we describe how hormones change during the course of pubertal development. Second, we describe evidence from studies of animals and adult women that implicate hormones in the etiology and maintenance of ED symptoms. Third, we discuss the methodological challenges associated with isolating the effects of hormonal change in samples of human adolescents, because of the multiplicity of biological and social changes that occur during this critical period of the human lifespan. This section emphasizes perhaps the greatest methodological challenge in puberty research—how puberty is measured—in order to evaluate the extent to which previous studies of puberty and ED

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symptoms have captured hormonal development. Finally, we provide recommendations for future research interested in testing reproductive hormones as a specific risk factor for the development of ED symptoms.

Hormonal Changes of Puberty

Puberty is a complex biopsychosocial process that involves changes in both adrenal and gonadal hormones. During adrenarche, the adrenal cortex increases secretion of androgens: dehydroepiandrosterone (DHEA), DHEA-sulfate (DHEAS), and testosterone. For girls, the increase of adrenal androgens drives the earliest physical changes of puberty, including pubic hair growth, skin changes, and acceleration of growth in height. Gonadal puberty is initiated by activation of the hypothalamic-pituitary-gonadal (HPG) axis, resulting in production of ovarian hormones, the most important of which are progesterone and estradiol. Median serum concentrations of estradiol are approximately nine times higher in adolescence than in childhood. Small amounts of testosterone, although typically conceptualized as a “male” hormone, are also produced in the ovaries; in girls, testosterone levels approximately double across the course of pubertal development. In addition to increases in average circulating concentrations, the cyclical variation in reproductive hormones (LH, FSH, progesterone, and estradiol) necessary for ovulation and menstruation is established over the course of puberty.

The changes of puberty—such as adrenarche and menarche—occur at variable ages. Thus female puberty involves durable increases in the overall circulating levels of multiple reproductive hormones, precipitates new fluctuations in hormones due to menstrual cycle variation, and introduces new individual differences, as females can differ in both the relative timing of these hormonal events and in their ultimate levels. The key question, then, is how these hormonal changes are linked to the rising prevalence of—and emergence of individual differences in—clinical and subclinical eating pathology.

Evidence for Importance of Reproductive Hormones from Non-Pubertal Samples

Discriminating the effects of reproductive hormones (or of any one mechanism) on the development of ED is complicated by the complexity of the pubertal transition. Puberty involves multiple, interconnected changes—not just in hormones but also in body, brain, cognition, affect, self-

perception, and social relationships. Indeed, the developmental and clinical literatures have largely emphasized the unique social challenges accompanying pubertal development.⁴ Girls, particularly those who mature at relatively young ages, may contend with unwanted sexual attention, compare their changing bodies with thin ideals, or face difficulties getting along with peers who have not developed in a comparable way. Given the difficulty of separating these social mechanisms of risk from the effects of hormonal change, it is perhaps not surprising that the strongest evidence for the effects of reproductive hormones on eating- and ED-related outcomes comes from studies that are *not* about pubertal girls. Specifically, three lines of research from experimental studies of animals and correlational studies of adult (postpubertal) women provide convincing evidence for the importance of reproductive hormones in the etiology of ED.

First, experimental research with animals confirms the importance of reproductive hormones for the regulation of normal feeding and weight.^{5,6} Estrogen has both tonic and cyclic inhibitory effects on food intake. Removing the ovaries of adult female rats (ovariectomy) increases meal size and thus overall food intake, an effect that can be reversed by estradiol treatment.⁵ Female rats' eating also varies with the estrus cycle; they eat the least amount of food just after estrogen peaks.⁵ Interestingly, the effects of estradiol appear to depend on pubertal maturation, as administration of estradiol does not inhibit eating in prepubertal rats.⁵ In contrast, ovariectomized rats do not show any change in food intake when administered progesterone alone; rather, progesterone antagonizes the effects of estrogen.

Comparatively fewer animal studies have examined disordered eating, but the few extant studies suggest that the inhibitory effects of estrogen may be disrupted by binge eating patterns. Research using an animal model of binge eating, in which rats are given brief intermittent access to high-fat food, found that administration of estrogen initially inhibits binge eating in ovariectomized rats,⁷ but these effects are attenuated as binge-eating patterns are established.⁸ Similarly, an individual differences model of binge-eating in rats found that binge eating proneness (BEP, defined as high consumption of high-fat food) emerged during mid-puberty in a sample of female rats.⁹ However, adult rats identified as high in BEP continue to eat more high-fat food than binge-eating resistant rats even after ovariectomy.¹⁰

Figure 1 Measurement of puberty in studies of ED symptoms. PDS, Pubertal Development Scale; Tanner (self/mother), Tanner staging based on maternal or self-report; Tanner (exam), Tanner staging based on physical exam; Age at Men, Age at menarche; Men Status, Menarcheal status; Hormone, Hormonal concentrations from blood or saliva. For complete listing of identified studies, please see Table 1 in the Online Supporting Information. Studies may measure puberty using more than one method, therefore the sum of the percentages exceeds 100%.



Second, correlational studies in adult women have linked *intraindividual* variation in reproductive hormones to eating behaviors. In clinical samples of women with bulimia nervosa¹¹ and in nonclinical samples,^{12,13} changes in estradiol and progesterone over the menstrual cycle interact to predict within-individual changes in binge eating. Specifically, binge eating is elevated with both estradiol and progesterone are elevated. This interaction effect that may be due to progesterone's antagonistic effects on estrogen, as the lowest levels of binge eating were seen when estradiol with high but progesterone was low. Menstrual cycle changes in estradiol and progesterone have also been linked with body dissatisfaction and drive for thinness.¹⁴ Finally, associations between intraindividual variability in ovarian hormones and dysregulated eating differ for women with a history of clinically-significant binge eating episodes.¹⁵ Among women with no history of binge-eating episodes, the estradiol \times progesterone interaction described above was observed, with higher levels of dysregulated eating when both progesterone and estradiol were high. In contrast, a main effect of low estradiol on dysregulated eating was observed among women with a history of binge-eating episodes (including women with a lifetime history of bulimia nervosa or binge ED), an effect that was amplified by low levels of progesterone.

Third, genetic research has identified associations between EDs and hormone-related genetic polymorphisms. Specifically, polymorphisms in *ESR1* and *ESR2*—which code for the estrogen receptors $ER\alpha$ and $ER\beta$, respectively—have been linked in some studies with bulimia nervosa and anorexia nervosa.¹⁶ Moreover, a single study reported an association between anorexia nervosa and a polymorphism in the *CCK* gene, which codes for cholecystokinin.¹⁷ Cholecystokinin is peptide hormone produced in the small intestine that serves as a peripheral satiety signal, and the CCK satiety signal is a key mechanism mediating the effects of estradiol on food intake.⁵ Lastly, variants of *LEP*, which codes for the production of leptin and plays a role in food intake and satiety, have been correlated with ages of menarche and menopause, and duration of menstrual cycles.¹⁸ It should be noted, however, that genetic research on EDs, like other complex behavioral phenotypes, has been plagued by frequent failures to replicate associations. The failure to identify all the specific polymorphisms underlying genetic influence on EDs (the “missing heritability” problem) may be due to low statistical power, the effects of rare genetic variants, and gene \times gene or gene \times environment interactions.¹⁹

Method

In light of the evidence of the effects of reproductive hormones on eating, we now turn to studies of how these processes play out in pubertal or adolescent human females. We identified peer-reviewed studies of puberty and ED-related symptoms using electronic search of online databases, including Google Scholar, PubMed, and PsycInfo. Search terms reflecting pubertal development and pubertal hormones (e.g., menarche, pubertal timing, estradiol, and progesterone) were crossed with terms reflecting disordered eating (e.g., anorexia nervosa, binge eating, weight concerns, and body image). In addition, reference sections of previous literature reviews were checked. This review includes studies published through May 2014.

Results

For a complete summary of the extant studies on human pubertal development and ED-related outcomes, please see Table 1 in the Online Supporting Information. As illustrated in **Figure 1**, the most common measures of pubertal development (see “How Puberty is Measured” in Table 1 of the Online Supporting Information) are (1) the Pubertal

Development Scale,²⁰ used in 29 (30%) of studies, (2) menarche, either age at menarche (used in 38 [40%] of studies) or menarcheal status (used in 15 [16%] of studies), and (3) Tanner staging, either based on self- or maternal-report (used in 7 [7%] of studies) or based on physical examination (used in 2 [2%] of studies). Less than 25% ($n = 22$) of studies are longitudinal.

In the following sections, we consider the theoretical implications of these methodological details. Specifically, we describe two issues that preclude any straightforward interpretation of the association between puberty and risk for ED symptoms in terms of hormonal processes. First, many studies have not disentangled the effects of pubertal *status* from pubertal *timing*, a distinction that is important for understanding the extent to which hormones can explain individual differences in ED risk beyond the period of pubertal change. Second, very few studies of pubertal development have incorporated direct measures of reproductive hormones, and, as we describe, commonly used measures of pubertal development (e.g., age at menarche, self-reported bodily changes) have poor correspondence with individual differences in hormones.

Advanced Pubertal Status Versus Early Pubertal Timing

Studies examining the role of puberty in psychopathology commonly compare children or adolescents who vary in their *pubertal status*—or level of development at a given point in time. In the simplest design, pubertal status studies compare girls who are either pre- or postmenarche. But a more common—and nuanced—method of differentiating pubertal status is according to the Tanner system.²¹ Tanner Stages divide observable changes in breast and pubic hair development into a progression ranging from 1 (prepubertal or child-like) to 5 (reproductively mature or adult-like). Thus, in early childhood and in adulthood, everyone has the same pubertal status (excepting individuals with rare medical disorders). There is population variability in pubertal status only during the years when adolescents are undergoing the changes of puberty.

An effect of advanced pubertal status on ED risk implies that the process of becoming reproductively mature increases the likelihood of eating pathology. As later-maturing girls “catch up” to their early-maturing peers, their risk for EDs follows the same parallel curve, just a few months to years later. Because puberty is a uni-

versal transition, pubertal status effects can explain population-level mean differences, such as why adult women have higher ED prevalence than children, but differences in pubertal status cannot explain individual differences in disordered eating beyond adolescence, except as moderators of other individual difference factors, such as genes.

In contrast, *pubertal timing* refers to individual differences in *when* an individual experiences the changes of puberty. Pubertal timing is typically measured in terms of a girl's age when she experiences a discrete transition (most commonly, age at menarche) or by how developed she is at a particular age as *compared to other girls her age*. A pubertal timing effect implies that experiencing the changes of puberty at an earlier age confers greater vulnerability than experiencing the same changes at a later age. Consequently, as later-maturing girls “catch up” in terms of their physical development, their risk for EDs will *not* catch up. Because pubertal timing is an individual difference, rather than a universal transition (everyone goes through puberty, but not everyone goes through puberty early), pubertal timing effects can explain individual differences in ED risk beyond adolescence. Cross-sectional data at a single point in time during adolescence are ambiguous regarding whether observed differences in ED risk represent the effects of status versus timing; in an age-homogenous sample, timing and status are confounded. Ideally, discriminating between timing and status effects requires observing individuals beyond the end of the pubertal transition.

An additional complication is that many studies of persistent pubertal timing effects are retrospective, asking late adolescents or adult women to recall their age at menarche. As we describe below, however, retrospective designs may have limited utility for researchers specifically interested in the effects of pubertal hormones, because of the uncertain correspondence between self-report measures of pubertal development (such as age at menarche) and hormonal levels. A more rigorous alternative—which to our knowledge has not been done in any previous study—would be to characterize hormonal differences during pubertal development and then prospectively follow participants past the end of the pubertal transition. Yet even among studies not directly measuring hormones, such longitudinal prospective designs are in the minority; less than a quarter ($n = 22$) of studies on puberty and ED symptoms (summarized in Table 1 of the Online Supporting Information) are longitudinal.

The status/timing distinction has been most extensively discussed in terms of visible changes of puberty (e.g., growth of breasts or changes in body shape), but a similar conceptual distinction can be applied to the effects of pubertal hormones. In a sample of adolescent girls still undergoing pubertal change, hormonal variation will reflect both developmental processes (older girls will have higher levels of estradiol than younger girls) and individual differences (earlier developing girls will have higher hormone levels than later maturing girls). A key theoretical question is whether vulnerability conferred by pubertal hormones represents an effect of hormone levels (e.g., adult-like levels are “riskier” than child-like levels, regardless of when hormonal changes occur), hormonal timing (e.g., hormonal transitions that occur earlier are riskier than hormonal transitions occurring later), or some combination of both processes.

Status Versus Timing Effects in Hormone Studies

Of nearly 100 studies of pubertal development and ED-related outcomes, only *two* include a direct measure of hormones. The dearth of direct evidence linking hormones and eating phenotypes in pubertal or adolescent samples is surprising, particularly considering the volume of theoretical work that has implicated hormonal change as a key mechanism of developmental risk. Davison et al.²² found that estradiol was significantly but modestly correlated with lower body esteem at ages 11 ($r = -.19$) and age 13 ($r = -.18$). Moreover, they included two other measures of pubertal development, a nurse exam and maternal report, and their primary analyses reported relations with a latent factor representing variance in pubertal development shared across the three measures. Such multivariate measurement stands out as a strength in the puberty-eating literature.

In contrast, Klump et al.,²³ found that, among 10- to 15-year-old adolescent girls, low-estradiol and high-estradiol groups (based on a median split) did not differ in *mean* levels of disordered eating symptoms. Rather, using a biometric twin design, they found evidence for a genotype \times estradiol interaction. Among low estradiol girls, heritable influence on disordered eating was minimal, whereas there was substantial heritability among girls with high estradiol. The genotype \times estradiol interaction effect was interpreted in terms of hormonal “status.” Higher estradiol concentrations activate genetic risk, with the expectation that later maturing girls will eventually catch up to their early

maturing peers. Given the relatively small number of twin pairs and the cross-sectional design, however, it is not possible to determine whether heritable influences on disordered eating are particularly accentuated among girls who experience estradiol increases at a relatively young age (i.e., an effect of hormonal “timing”).

Correspondence Between Common Puberty Measures and Hormones

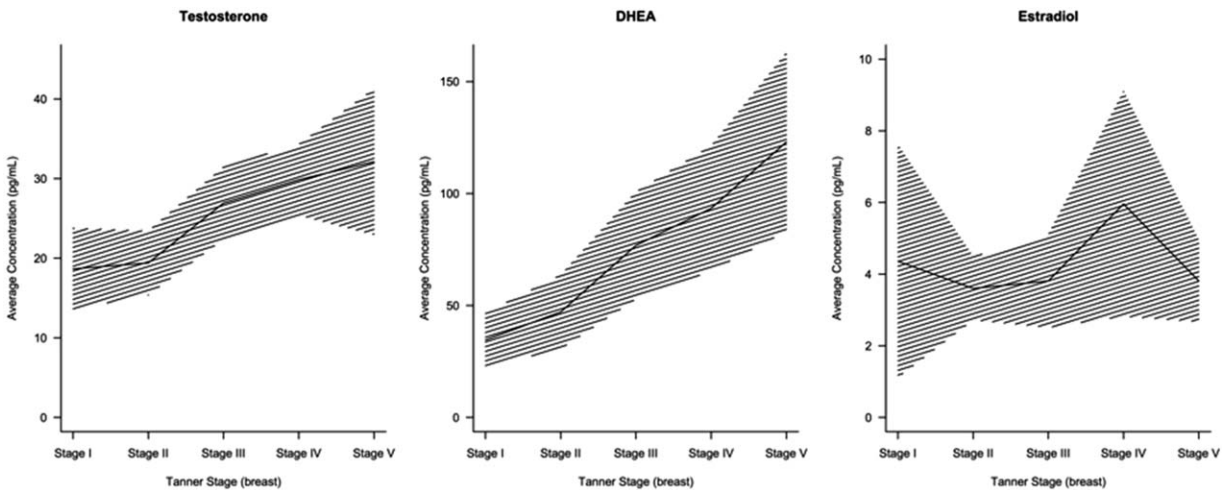
Because so few studies include hormonal measures, understanding the role of hormonal change during puberty in the development of eating pathology requires particular attention to how puberty is measured and how well these measures capture hormonal variation.

Tanner Staging

Tanner staging based on a physical examination by a trained clinician is often claimed as the “gold standard” of pubertal development²⁴; however, few studies have considered the extent to which Tanner Staging by physical exam captures the underlying endocrine changes of puberty. Among early adolescents (ages 9–14), Tanner stages based on physician exam accounted for only 5% ($r = .23$) of the overall variation in estradiol, which is the hormone most commonly implicated by experimental animal research as important for eating²⁵ (data plotted in **Fig. 2**). The strongest hormonal correlate of physician-rated breast Tanner Stage was DHEA—an adrenal androgen—and to a lesser extent, testosterone. A similarly modest relation between exam-based Tanner stages and estradiol was found by Davison et al.²² In contrast, Huang et al.²⁶ found a more substantial relation between estradiol and Tanner stage based on physical exam (Spearman’s $\rho = 0.56$) in 9- to 14-year-olds, although the majority of variance in estradiol was still unique of Tanner stage. The poor correspondence between exam-based Tanner stages and individual differences in hormones may be partially driven by clinicians’ diagnostic errors.^{27,28}

An alternative to physical exam is to provide drawings or pictures representing each Tanner stage and ask adolescents and/or their parents to report, which picture best represents their current stage of development. Shirtcliff et al.²⁵ found that picture-based self-reports of Tanner stage captured variation in estradiol ($R^2 = 15\%$) somewhat better than physical exam, and Huang et al.²⁶ found that picture-based self- and parent- reports of Tanner stage were as strongly correlated with serum

Figure 2 Average salivary hormonal concentrations by tanner stage. Data from Table 4 of Shirtcliff et al.²⁵ Lines represent means; shaded areas represent ± 2 SE. Tanner Stages were determined from physical exam. Testosterone and DHEA concentrations are averages across 32 salivary samples. Estradiol concentrations are averages across 5 morning salivary samples.



estradiol concentrations as exam-based Tanner stage ($\rho = 0.51$ for self-report and 0.55 for parent-report).

Pubertal Development Scale

The Pubertal Development Scale (PDS²⁰) asks adolescents and/or their parents to rate observed change in various secondary sex characteristics. Inter-rater reliability between parents and children is generally around $\rho = 0.5$ range but can be as high as $\rho = 0.8$.²⁹ The PDS has many advantages: it is short and cheap, requires minimal training for accurate administration, avoids stimuli (such as pictures of genitals or breasts) that some parents or children might find objectionable, and can be easily scaled-up for large epidemiological studies of both genders, including studies with distance populations. Davison et al.²² found that estradiol among European American 11-year-olds was more strongly correlated with maternal report on the PDS ($r = .40$) than with Tanner stages based on nurse exams. Similarly, Shirtcliff et al.²⁵ reported that the PDS items that described characteristics of gonadal development predicted variation in estradiol better than the physical exam ($R^2 = 15\%$ as compared to 5% for physical exam).

Age at Menarche

Age at menarche was the most commonly used measure in the studies we reviewed (used in 40%). Like the PDS, age at menarche is typically assessed using self-report, providing investigators with an

inexpensive and brief measure of pubertal timing. Unlike the PDS, age at menarche can be reliably and validly reported on by adult samples. As menarche occurs relatively later in the process of pubertal development, age at menarche or menarcheal status may not be a useful marker for pubertal changes that happen relatively early in development. Moreover, there is not an obvious, reliably-reported analog to age at menarche for boys. Despite these limitations, some evidence suggests that age at menarche indexes individual differences in estrogen exposure. Vihko and Apter³⁰ found that postmenarcheal girls with early menarche (<12 years) had significantly higher estradiol than girls with average (12 – 13 years) or late menarche; these differences were apparent both premenarcheally and throughout adolescence. Among young adults (ages 20 – 30), earlier age at menarche (prior to age 12) predicted more rapid rises in estradiol and higher peak levels of estradiol during the follicular phase of the menstrual cycle.³¹

Discussion

Recommendations for Future Research

EDs are relatively uncommon in childhood and rise precipitously as children—and particularly girls—transition through puberty and adolescence. The striking coincidence of ED symptomatology and female puberty has been attributed to numerous factors, most commonly the discrepancy

between cultural beauty ideals and physiological changes in weight and body shape.³² Despite clichés of “raging hormones” and widespread acknowledgement of substantial biological change during this period of the lifespan, hardly any studies of ED-related outcomes in pubertal or adolescent samples have directly measured reproductive hormones. Because of the low base rates of clinically-defined EDs and the frequent use of convenience populations in research, the current literature has overwhelmingly focused on continuous dimensions of ED symptoms, broadly defined. Therefore, generalizability of past findings (and the inferences we have drawn from these findings) to clinical-level pathology remains both uncertain and an important vein for future research. Moreover, commonly used measures of pubertal development—including Tanner staging by medical exam—are relatively weak indicators of the underlying hormonal “signal,” capturing modest amounts of hormonal variation at best. Therefore, the voluminous literature linking advanced pubertal status and/or pubertal timing with ED-related outcomes cannot be unambiguously interpreted as evidence for the importance of reproductive hormones. Instead current understanding of hormonal effects on ED development during puberty is largely inferred from correlational studies of adult women and experimental studies of animals. As interest in developmental risk for EDs moves forward, there is a strong need for prospective longitudinal studies that incorporate repeated measures of reproductive hormones and multimethod measures of other aspects of pubertal development.

Currently, studies of the relation between reproductive hormones and ED-related outcomes in adult women have focused on *intraindividual variability*—relatively short-term within-person fluctuations in hormones over the course of the menstrual cycle.^{12,13} In contrast, research on pubertal development (both the few empirical studies that have directly measured hormones and the theoretical work interpreting puberty-ED associations in terms of hormonal mechanisms) has largely emphasized *intraindividual change* in “basal” or average levels of circulating hormones, which rise over the course of puberty. Intraindividual change and intraindividual variability were memorably referred to by Nesselrode³³ as “the warp and the woof of the developmental fabric,” woven together as the “key constituents of our everyday lives” (pp. 213–214). This analogy seems particularly apt when applied to hormonal change, as advancing pubertal development involves both enduring changes in average levels of reproductive

hormones and the establishment of cyclic variability—and new *interindividual* differences in both. For instance, in their longitudinal study, Vihko and Apter³⁰ noted that early age at menarche was associated with both elevated overall levels of estradiol even prior to menstruation and with more frequent cycle-related hormonal variability, due to the earlier establishment of regular ovulation. They further hypothesized that it was the regular exposure to estradiol spikes during the follicular phase of the menstrual cycle (rather than higher overall basal levels) that contributed to the elevated breast cancer risk seen in women who had early menarche. Thus the dynamic interaction between intraindividual change and intraindividual variability is likely to be critical for understanding how reproductive hormones relate to ED risk.

Longitudinal studies are further necessary to resolve the extent to which hormonal influences on ED-related outcomes depend on the relative age at which pubertal development occurs. On the basis of current research findings, it seems that progressing through puberty creates vulnerability for all children (e.g., pubertal status) and progressing through puberty earlier than peers amplifies this general risk (e.g., pubertal timing). However, the status-timing distinction has not generally been explored with regards to hormonal mechanisms specifically: Do developmental increases in estradiol confer risk for ED-related outcomes generally, or are girls who experience these hormonal changes at an earlier age particularly vulnerable? Understanding this question requires studies that are capable of disentangling universal developmental experiences from individual differences. Cross-sectional studies of pubertal adolescents, which represent the majority of research on pubertal development and ED-related outcomes, cannot fully discriminate status versus timing effects, whereas retrospective studies (which usually use age at menarche as an index of pubertal timing) cannot fully capture individual differences in hormones during the pubertal period.

Cross-sectional studies are additionally ambiguous about other individual differences in pubertal development aside from pubertal timing. Perhaps the most intriguing of these is pubertal tempo, or the rate of physical development.³⁴ Although the process of physical maturation typically takes four years, there is considerable variability, with extremes ranging from one to seven or more years. To date, pubertal tempo has not been investigated in relation to EDs. Yet rapid tempo might reflect hormonal differences important for eating behavior, as a quicker pubertal progression has been

associated with higher levels of circulating estrogen and follicle-stimulating hormone in girls.³⁵

Prospective longitudinal studies linking pubertal development and EDs face considerable methodological challenges: Pubertal development spans several years; the risk for EDs continues to escalate for several years after that; clinically-defined EDs are low base rate phenomena; and direct measures of hormones and hormonal cycling are expensive and require multiple repeated measurements. In the face of these challenges, we have five recommendations for increasing the feasibility of research with large longitudinal samples. First, it may help to maintain focus on dimensional variation in ED symptoms rather than clinical diagnoses *per se*, as this approach is both more statistically powerful³⁶ and—pragmatically—consistent with NIH funding priorities.³⁷ Second, integrative data analysis approaches can be used to pool data from multiple studies that have measured similar constructs, thus expanding construct coverage and the developmental span captured by the data.³⁸ Third, researchers can use a planned missingness “two-method measurement” design,³⁹ in which a randomly selected subset of individuals is characterized using an “expensive” measure (e.g., multiple daily hormone samples) whereas the entire sample is characterized on a “cheaper” measure (e.g., a single hormone sample or self-report survey). Both measures are then modeled as indicators of a latent factor, yielding lower standard errors and higher statistical power than either measure alone. Fourth, although Tanner stage exams have been claimed as the “gold standard” in puberty research, evidence suggests that Tanner stage exams do not capture hormonal variation better than self-reports, which are considerably cheaper and easier to administer. For researchers specifically interested in hormonal mechanisms, we do not recommend devoting scarce research resources to clinical Tanner stage exams. Combining multiple reports of puberty (e.g., both the PDS and picture-based Tanner stages), modeled in a latent variable framework, will allow researchers to devote research funds toward increasing sample size or including additional hormonal measures while still obtaining a valid and reliable measure of development. Fifth, researchers should explore new technologies for the measurement of hormones in hair.⁴⁰ Hair samples index monthly accumulations of hormones and can potentially be used to capture relatively stable individual differences while reducing participant burden and remaining sensitive to pubertal change.

Conclusions

Despite a consistent correlation of puberty with disordered eating, the diversity of research methods and conflation of different pubertal indicators has made mechanistic inferences challenging. It is unclear how well hormones map onto current methods of assessing puberty. Hormonal assay is perhaps the only method of assessing pubertal change that is fully independent of perception. Yet until we understand more about human puberty, particularly the cyclical variation of female hormones across the menstrual cycle and interactions between different hormones, it is difficult to rely exclusively on conclusions from hormone-only studies. Studies that link ED outcomes with multi-method measures of puberty, innovative technologies for measuring hormones, and sophisticated longitudinal designs represent the best chance for discriminating social and biological pathways of risk.

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