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Review

# Puberty as a critical risk period for eating disorders: A review of human and animal studies

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ABSTRACT

This article is part of a Special Issue "Puberty and Adolescence".

Puberty is one of the most frequently discussed risk periods for the development of eating disorders. Prevailing theories propose environmentally mediated sources of risk arising from the psychosocial effects (e.g., increased body dissatisfaction, decreased self-esteem) of pubertal development in girls. However, recent research highlights the potential role of ovarian hormones in phenotypic and genetic risk for eating disorders during puberty. The goal of this paper is to review data from human and animal studies in support of puberty as a critical risk period for eating disorders and evaluate the evidence for hormonal contributions. Data are consistent in suggesting that both pubertal status and pubertal timing significantly impact risk for most eating disorders in girls, such that advanced pubertal development and early pubertal timing are associated with increased rates of eating disorders and their symptoms in both cross-sectional and longitudinal research. Findings in boys have been much less consistent and suggest a smaller role for puberty in risk for eating disorders in boys. Twin and animal studies indicate that at least part of the female-specific risk is due to genetic factors associated with estrogen activation at puberty. In conclusion, data thus far support a role for puberty in risk for eating disorders and highlight the need for additional human and animal studies of hormonal and genetic risk for eating disorders during puberty.



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## Introduction

Historically, puberty has been one of the most frequently discussed risk periods for the development of eating disorders (i.e., anorexia nervosa (AN), bulimia nervosa (BN)) and their symptoms (e.g., body dissatisfaction, weight concerns, dieting, binge eating). Traditional theories of risk focused on the psychosocial effects (e.g., increased body dissatisfaction) of the physical changes associated with puberty (e.g., increased adiposity) and their potential consequences for eating disorder development (Fornari & Dancyger, 2003). However, human and animal data are converging to suggest that biological and genetic factors also significantly contribute to eating disorder risk during the pubertal transition. The overarching goal of this review is to evaluate the evidence in support of biological and genetic risk and synthesize these data with existing neuroendocrinological models of puberty's effects on behavior. Perhaps not surprisingly, most theories focus on gonadal hormones and their influence on sex-differentiated behaviors during and after puberty. This review will be no exception.

The review begins with an overview of the types of eating disorders and symptoms that have been examined in puberty studies. Data in support of puberty as a critical risk period for these phenotypes will then be reviewed, with a particular emphasis on differentiating pubertal status (i.e., pubertal stage at a given point in time) from pubertal timing (i.e., onset of puberty relative to peers, including early, on-time, and late onset) effects. Evidence from human and animal studies suggesting that biological and/or genetic factors contribute to puberty's effects will then be evaluated and integrated into existing behavioral neuroendocrinological models of sex-differentiated behaviors during puberty.

## Eating disorder definitions

To date, studies of puberty have primarily focused on AN and BN and their component symptoms. DSM-IV criteria for each disorder (American Psychiatric Association, 2000) are presented in Table 1. Notably, two subtypes of AN exist, the restricting subtype which is characterized by strict dieting and/or excessive exercise only, and the binge/purge subtype which is characterized by strict dieting and/or excessive exercise as well as binge eating, purging, or binge eating and purging. Thus, despite common misconceptions, AN frequently includes binge eating and purging behaviors commonly thought to be exclusive to BN. Likewise, BN includes behaviors and cognitions that are commonly attributed to AN, including strict dieting and extreme concerns about shape/weight that significantly impact the individual's self-esteem (see Table 1). To avoid overlap between the two disorders, DSMIV stipulates that individuals with BN cannot be underweight (i.e., their weight must be >85% of ideal), otherwise they would receive a diagnosis of AN, binge/purge subtype.

Both AN and BN are relatively rare disorders, with AN occurring in only .5% of the population and BN occurring in 1–3% (American Psychiatric Association, 2000). However, subthreshold cases of the disorders (see Table 1) are much more common with estimates as high as 10% (Fairburn and Bohn, 2005). These and other symptom presentations (i.e., those with binge eating disorder who binge eat but do not purge) are categorized as eating disorders not otherwise specified (EDNOS) within DSMIV (American Psychiatric Association, 2000). The fact that EDNOS diagnoses are the most frequently encountered eating disorders in clinical (Thomas et al., 2009) and community (Machado et al., 2007) samples has led the DSM 5 Workgroup to consider broadening the criteria for AN and

**Table 1**
Definitions of DSM IV diagnoses and eating disorder symptoms that have been investigated in studies of puberty.

| Diagnoses/symptoms | Definition |
|---|---|
| *Diagnoses* | |
| Anorexia nervosa (AN) | All of the following are present:<br>1) Low body weight (i.e., <85% of ideal for age and height).<br>2) Fear of becoming fat or of gaining weight.<br>3) Distortion in the way in which body weight and shape are perceived (i.e., thinks part(s) of body too fat, undue influence of body weight/shape on self-esteem, denial of seriousness of low body weight).<br>4) Primary amenorrhea or (in post-menarcheal females) loss of menses for ≥3 consecutive months. |
| Bulimia nervosa (BN) | All of the following are present:<br>1) Binge eating (i.e., the consumption of a large amount of food in short period of time (i.e., 2 h) with a loss of control over the binge episode).<br>2) Use of inappropriate compensatory behaviors including purging (i.e., self-induced vomiting, or abuse of laxatives, diuretics, or enemas) and non-purging (e.g., excessive exercise, fasting) behaviors.<br>3) Binge eating and inappropriate compensatory behaviors occur, on average, ≥2 times per week for 3 months.<br>4) Undue influence of body weight and/or shape on self-esteem.<br>5) The BN symptoms do not occur exclusively during periods of AN. |
| Eating Disorder Not Otherwise Specified (EDNOS) | Clinically significant syndromes that do not meet criteria for either AN or BN—examples include:<br>1) A woman who would meet criteria for BN but who engages in binge eating/purging episodes only 1 time per week for 3 months.<br>2) A woman who would meet criteria for AN except that her body weight is 90% of ideal.<br>3) Binge eating disorder, i.e., a woman who binge eats but does not regularly engage in compensatory behaviors. |
| *Symptoms* | |
| Body dissatisfaction, weight/shape concerns | Dissatisfaction with the size/shape of one's body or body parts, and/or preoccupation with weight and a desire to lose weight |
| Dieting/weight management | Behavioral attempts to restrict food intake and/or engage in other behaviors to lose weight (e.g., exercise). |
| Dietary restraint | A cognitive intent to diet (i.e., desire to lose weight, plans to restrict food intake) as well as actual attempts to lose weight through dieting, avoidance of high fat foods, and/or fasting. |
| Binge eating | See definition above under BN |
| Purging behaviors | See definition above under BN |
| Drive for muscularity | Both an intent/desire to increase muscle mass as well as actual attempts to increase muscle mass (e.g., weight lifting, protein shakes). |