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Sexuality in eating disorders patients: etiological factors, sexual dysfunction and identity issues. A systematic review

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Abstract: The scientific community appears to be less interested in sexuality of eating disorders (EDs) as compared to other psychiatric or medical comorbidities. However, a clear association between sexual problems and ED psychopathology was reported from different perspectives. The overarching goal of this systematic review was to evaluate the general approach of the scientific literature toward the topic of sexuality and EDs. In particular, four different categories of research have been individuated, encompassing the role of puberty, and sexual abuse in the pathogenesis of the disorders, sexual dysfunctions, and the association between sexual orientation and EDs psychopathology. Timing of puberty with its hormonal consequences and the changes in the way persons perceive their own body represent a crucial period of life for the onset of the disorder. Sexual abuse, and especially childhood sexual abuse are well-recognized risk factors for the development of ED, determining a worse long-term outcome. Recent research overcome the approach that considers sexual activity of EDs patients, in terms of hypersexuality and dangerous sexual behaviors, considering the sexuality of EDs persons in terms of sexual desire, satisfaction, orgasm and pain. Results from this line of research are promising, and describe a clear relationship between sexual dysfunction and the core psychopathological features of EDs, such as body image disturbances. Finally, the analysis of the literature showed an association between sexual orientation and gender dysphoria with EDs psychopathology and pathological eating behaviors, confirming the validity of research developing new models of maintaining factors of EDs related to the topic of self-identity.

Keywords: eating disorders; gender identity; puberty; sexuality; sexual abuse; sexual dysfunction; sexual orientation.

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Introduction

Eating disorders (EDs) are severe psychiatric syndromes affecting especially young women. In most cases, EDs have a long lasting duration (longer than 2 years) and a chronic course [1, 2]. The two well-known diagnoses of anorexia nervosa (AN) and bulimia nervosa (BN) share a similar psychopathological core of extreme concerns about shape and weight that significantly impact the individual's self-esteem, and they are essentially differentiated by the condition of underweight (i.e. their weight must be >85% of ideal), associated with the diagnosis of AN [3]. Typical psychopathological features of EDs are represented by an intense fear of becoming fat or of gaining weight, distortion in the way in which body weight and shape are perceived, undue influence of body weight and/or shape on self-esteem. Abnormal eating behaviors include binge eating, use of inappropriate compensatory behaviors (i.e. self-induced vomiting, or abuse of laxatives, diuretics, or enemas, excessive exercise, fasting), body checking, and body avoidant behaviors.

From an epidemiological point of view, AN and BN full syndromes represent only the tip of an iceberg, with AN occurring in 0.4% of the population and BN in 1%–1.5% [3]. Indeed, the last version of the Diagnostic and Statistical Manual of Mental Disorders (DSM 5) [3] considers also the so-called other specified FED or unspecified FED, and the binge eating disorder (BED), that showed a lifetime prevalence of 0.8% and 1.6% in men and women, respectively [3]. BED is characterized by frequent and persistent overeating episodes that are accompanied by feelings

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of loss of control and marked distress, in the absence of regular compensatory behaviors.

As far as the psychopathological assessment of these syndromes, dimensional rather than categorical models seem to be more appropriate [4, 5], as suggested by two main issues: 1) the higher prevalence of subthreshold conditions, which are the most frequently encountered in clinical [6] and community [7, 8]; 2) the "instability" of the DSM diagnoses, with a substantial crossover between the different EDs categories [9, 10]. Indeed, EDs patients often move in and out of illness states over time, display a high frequency of relapse [10], and the majority of them migrate between different ED diagnoses, without a substantial psychopathological change [9–14].

The pathogenesis of EDs is multi-factorial, and it most likely results from sociocultural, psychological and biological factors [15, 16]. Considering environmental conditions, adverse early life events showed a relevant role as a predisposing factor for EDs [16]. In particular, longitudinal and cross-sectional studies reported that sexual abuse had a significant association with the disorder's onset [16, 17]. These conditions are supposed to affect child development in behavioral, emotional, social, physical, and cognitive areas [18], increasing the risk for EDs adult psychopathology. For example, they can be associated with low self-esteem and depression, severe body image distortion [19], and deficit in emotion regulation [19]. Moreover, chronic stressful conditions occurring during childhood may damage neurobiological and neuroendocrine aspects for long time [18], leading to greater vulnerability in the development of general psychopathology and psychiatric disorders throughout life [17].

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EDs patients show a high rate of comorbidity, especially with mood (including unipolar and bipolar disorder), and anxiety disorders [3]. A large number of studies has been published on this topic, suggesting a common psychopathological trait related to emotion dysregulation between these syndromes. However, considering the psychiatric comorbidities of EDs, very few studies evaluated the co-occurring sexual dysfunction, and sexuality in terms of desire, sexual satisfaction, and relationship with own body.

From a biological point of view, puberty has been one of the most frequently discussed risk periods for the development of EDs. Indeed, puberty is characterized by physical changes such as menarche and increased adiposity, which may lead to body dissatisfaction [20]. At the same time, girls or boys perceive sexual drives for the first time, and their bodies become sexual targets for other persons. Finally, the EDs literature focused a considerable attention on the issue of gender differences in terms of quantitative and qualitative aspects of EDs presentation, as well as on the association between specific expressions of EDs psychopathology and sexual orientation, and gender identity variance.

Indeed, it is noteworthy that, despite the clear association between sexuality and EDs from different perspectives (sexual abuse, puberty as risk factors, sexual orientation), few studies attempted to clarify the nature of this relationship, and sexual dysfunction comorbidity is rarely considered a potential target of treatment or indicator of long-term outcomes of the disorders. The scientific community appeared to be less interested in sexuality of EDs as compared to other psychiatric or medical comorbidities, and the DSM 5 Workgroup considered redundant the criteria for AN of primary amenorrhea or (in postmenarche females) loss of menses for ≥3 consecutive months.

According to this background, the overarching goal of this systematic review was to evaluate the general approach of the scientific literature toward the topic of "sexuality and eating disorders", and synthesize the available data from different points of view.

Methods

Time-frame

A systematic review of the literature was performed using PubMed (U.S. National Library of Medicine, 1995–October 2015) electronic database.

Inclusion criteria

According to the DSM 5 categorization for EDs [3], we used the key words "eating disorder" in combination with "sexuality". Articles were selected according to the following inclusion criteria: original studies, articles in English, human studies effectively related with the mentioned areas of interest. Subsequently, a further research was performed combining "eating disorder" with "puberty", "hormonal levels", "sexual dysfunction", "sexual abuse", and "sexual orientation", "gender identity". We focused on studies reported in the past 20 years but also included commonly referenced and highly regarded older publications. Review articles and book chapters were cited to provide readers with more details and additional references.

Conceptual structure

The structure of the present review was based on the main fields of research associated with sexuality of EDs, taking into consideration etiopathogenic factors such as puberty, role of hormones and sexual abuse, comorbidity with sexual disorders, the issue of self-identity in EDs and its association with gender identity and sexual orientation.

Results

General consideration

The results and the further classification of the first research based on the combination between "eating disorders" AND "sexuality" are reported in Figure 1. Of the 458 articles initially considered, 155 were focused on sexuality and EDs. These studies were categorized into four main categories. The first category included papers considering the relationships between puberty, hormonal factors and their role in the onset and maintenance of EDs (13.5%). The second category included those researches focused on sexual behaviors and sexual quality of life (22.5%); a large proportion of studies was related with the sexual behaviors associated with binge eating and other impulsive behaviors, while few studies were actually focused on sexual satisfaction. The third category is the most represented, including those studies considering the role of sexual abuse (especially childhood sexual abuse) in the pathogenesis of EDs (36.7%). Finally, a large number of studies deal with the complex issue of sexual orientation and EDs, followed by a smaller subcategory including the topic of gender identity and gender role, and their significance in the different expression of EDs

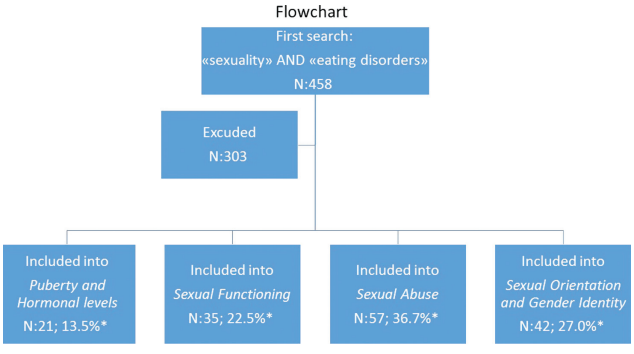

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Figure 1: Flow chart for the details of the resarch "sexuality" and "eating disorder".
*Percentages refer to the total number of included studies (n: 155)

psychopathology (total rate: 27.0%). Secondary researches showed a rate of 24 on 235 initially included studies for "puberty"/"hormonal levels" and "eating disorders" (Table 1), 26 on 116 for "sexual dysfunction" and "eating disorders" (Table 2), 158 on 402 studies for "sexual abuse" and "eating disorders", and finally 32 on 441 studies for "sexual orientation" and "eating disorders", and 11 on 164 studies for "gender identity" and "eating disorders" (Table 3). The results of the present systematic review should be considered in the light of some limitation. First of all, in many studies it is not possible to distinguish between different EDs diagnoses (AN, BN, BED). Indeed, some research considered the association between symptoms or psychopathological dimensions and sexuality. Moreover, further meta-analyses articles should provide a quantitative measure of the many of the mentioned associations. Finally, the choice of the areas of interest was based also on our clinical and scientific experiences and subcategorization or further areas could be individuated in this field.

Puberty and hormonal levels

Most of the reviews focusing on the pathogenesis of EDs [16, 107] agree that puberty is a significant risk period for the development of EDs, especially in women. The relationship between puberty and EDs has been documented by different perspectives. A recent systematic review by Klump [108] examined the role of puberty as a critical period for EDs onset, taking into consideration both animal and human studies. First of all, the effect of early puberty appears to be controversial, as some studies indicated that early puberty increases the risk for EDs onset [21–25, 109, 110], while others have not found such association [34, 43, 44, 111, 112]. Moreover, the prevalence of EDs has been reported to be higher in mid-late adolescence as compared to pre-adolescence, and the pre-pubertal onset of the disorders is rare [113]. Furthermore, several studies highlighted a post-pubertal bias in onset [40, 114], and the earlier onset of the disorders reported by some epidemiological studies has been associated with an earlier puberty onset [114]. Regarding sexual maturation, it has been observed both a delay [33] and an acceleration of psychosexual development [115].

Considering possible mechanisms underlying the association between EDs onset and puberty, as observed by Klump [108], pubertal timing showed a specific association with typical EDs symptoms onset. Most of the studies reported onset of body dissatisfaction, binge eating and purging behaviors at more advanced stages of

Table 1: Puberty and eating disorders.

Study	Diagnostic groups	Type of association
Wichstrom [21]	General population	
Fairburn et al. [22]	BN	
Fairburn et al. [23]	AN	
Kaltiala-Heino et al. [24]	BN	
Ruuska et al. [25]	AN, BN	
Klump et al. [26]	General population	
Cotrufo et al. [27]	General population (adolescent)	Early puberty as risk factor for EDs
Zehr et al. [28]	General population	
Day et al. [29]	General population (adolescent)	
Culbert et al. [30]		
Baker et al. [31]	General population	
McNicholas et al. [32]	General population	
Schmidt et al. [33]	AN, BN	Delays in psychosexual development
Mangweth-Matzek et al. [34]	AN, BN	Bad attitude toward menstruation; no association with early puberty
Vannucci et al. [35]	General population (8–17 years)	
Kaltiala-Heino et al. [36]	General population (14–16 years)	Early puberty as risk factor for bulimic type eating pathology
Tenconi et al. [37]	AN patients	
Tremblay and Lariviere [38]	Gen pop (adolescent)	Early puberty as risk factor for weight control behaviors
Abraham et al. [39]	General population (12–17 years)	Menarche as risk factor for the EDs
Favaro et al. [40]	AN, BN	
Striegel-Moore et al. [41]	General population	Early puberty as risk factors for body image and dieting concerns
Kaczmarek and Trambacz-Ol [42]	General population (12–18 years)	
Stice et al. [43]	General population	No association with early puberty
Ackard and Peterson [44]		

AN, Anorexia nervosa; BN, bulimia nervosa; Eds, eating disorders.

Table 2: Eating disorders and sexual dysfunction.

Study	Diagnostic groups	Correlates of sexual dysfunction
Abraham [45]	AN	Underweight
Morgan et al. [46]	AN	
Meguerditchian et al. [47]	AN	
Wiederman and Pryor [48]	AN, BN	BN better sexuality than AN
Pryor et al. [49]	AN subtypes	No difference
Wiederman et al. [50]	BN	Binge eating and impulsive sexual behaviors
Nagata et al. [51]	BN	
Nagata et al. [52]	BN	
Paul et al. [53]	AN, BN	
Kaltiala-Heino et al. [24]	BN	
Eddy et al. [54]	BN	
Culbert and Klump [55]	BN	
Ackard et al. [56]	BN	
Rodríguez et al. [57]	BN	
Morgan et al. [58]	AN, BN	Bad attitude toward sexuality
Wiederman and Pryor [59]	AN, BN	
Mangweth-Matzek et al. [34]	AN, BN	
Ruuska et al. [25]	AN, BN	
Castellini et al. [60]	BED, obese	Sexual dysfunction and body dissatisfaction
Pinheiro et al. [61]	AN, BN	
Castellini et al. [62]	AN, BN	
Folope et al. [63]	BED, obese	
Castellini et al. [64]	AN, BN	
Sarwer et al. [65]	BED bariatric	Sexual dysfunction and quality of life
Castellini et al. [66]	AN, BN	Sexual abuse, cortisol levels
Brown and Keel [67]	EDs men	Being in a relationship as a protective factor

AN, Anorexia nervosa; BN, bulimia nervosa; EDs, eating disorders.

Table 3: Eating disorders, sexual orientation and gender identity.

Study	Gender	Association between sexual orientation and EDs
Olivardia et al. [68]	Men	Non heterosexuality as risk factor for EDs
Beren et al. [69]	Men	
French et al. [70]	Men	
Carlat et al. [71]	Men	
Russell and Keel [72]	Men	
Yelland and Tiggemann [73]	Men	
Hospers and Jansen [74]	Men	
Feldman and Meyer [75]	Men	
Feldman et al. [76]	Men	
Heinberg et al. [77]	Men	
Brown and Keel [67]	Men	
Matthews et al. [78]	Men/women	
Matthews-ewald et al. [79]	Men/women	
Heffernan et al. [80]	Women	Lack of association
Moore et al. [81]	Women	
Share and Mintz [82]	Women	Non heterosexuality protective for good body image
Owens et al. [83]	Women	
Moore and Keel [81]	Women	Non heterosexuality as risk factor for body uneasiness
Duggan et al. [84]	Men	
Conner et al. [85]	Men/women	
Kaminski et al. [86]	Men	
Morgan and Arcelus [87]	Men	
Carper et al. [88]	Men	
Blashill [89]	Men	
Wiseman and Moradi [90]	Men	
Dakanalis et al. [91]	Men	
Koh and Ross [92]	Women	Higher risk in bisexual than lesbian
Austin et al. [93]	Men/women	Non heterosexuality as risk factor for purging behaviors men not for women
Legenbauer et al. [94]	Men/women	Non heterosexuality as risk factor for men; Lack of association for women
Cella et al. [95]	Men/women	
Austin et al. [96]	Men/women	Non heterosexuality as risk factor for purging behaviors
Brewster et al. [97]	Women	Bisexuality as a risk factor for EDs
Meyer et al. [98]	Men/women	Gender role and EDs
Hepp and Milos [99]	Men/women	
Duggan et al. [78]	Men	
Hepp et al. [100]	Women	
Cella et al. [101]	Men/women	
Diemer et al. [102]	Men/women	
Matthews-ewald et al. [79]	Men/women	
Vocks et al. [103]	Men/women	Higher eating psychopathology in transgender
Ålgars et al. [104]	Men/women	
Bandini et al. [105]	Men/women	
Fisher et al. [106]	Men/women	

EDs, Eating disorders.

pubertal development. In particular, it has been hypothesized that the onset of the disorder would be mediated by the body dissatisfaction due to pubertal changes (e.g. breast development, menarche, increased adiposity) [20, 113, 116]. Indeed, those who early sexually develop are thought to be at particular risk, given that they experience these physical changes earlier than their peers and may therefore experience even more body dissatisfaction than their developmentally on-time counterparts.

Apart from the developmental stage, it has been reported that EDs patients have a more negative subjective perception of menarche, pubertal body changes, and first sexual activities, as compared to healthy women [34, 117]. Indeed, ideal body image was associated with weight status and menstrual cycle phase, and negative body image was significantly associated with different phases of the menstrual cycle and increasing body weight status [42]. Moreover, Kaczmarek and Trambacz-Oleszak [42]

found that body dissatisfaction was more severe during the premenstrual phase of the menstrual cycle. These observation are similar to previous findings by Jappe and Gardner [118] and Racine et al. [119], who found that body image dissatisfaction was likely to be greater during the mid-luteal/premenstrual phases than in the follicular and ovulatory phases in a sample of adult healthy women. Teixtera and colleagues [120] observed that dissatisfaction with body image peaked during the menstrual phase, with a significant increase after the premenstrual phase and a significant decrease after the menstrual phase in a sample of 44 university students.

As expected, few studies took into consideration the role of puberty in males. Klump [108] reported that the majority of them showed that early maturing boys and/or those at advanced stages of puberty had higher rates of AN, BN, and EDs symptoms, especially body dissatisfaction and weight/shape. However, there are also studies reporting that those who sexually develop later had higher rates of body dissatisfaction [121–124], and five studies found that advanced pubertal development was associated with improved body image [121, 125], enhanced self-concept [126, 127], and/or increased perceptions of physical attractiveness and/or decreased social pressures about eating [128]. However, a further support to the strong role of puberty changes in girls as a predisposing factor for ED may be represented by the female to male ratio which ranges from 4:1 [129] up to 10:1 [130]. Sex differences have been also related to a pathogenic effect of gonadal hormones [108].

As far as hormones are concerned, leptin, an adipocyte hormone important in regulating energy homeostasis, interacts with the reproductive axis with stimulatory effects at the hypothalamus and pituitary gland, and inhibitory actions at the gonads [131–139]. Leptin has been shown to play a role in important physiologic processes such as menstruation, pregnancy, and lactation [139, 140]. As a marker of nutritional status, leptin may act in concert with gonadotropins and the growth hormone axis to initiate the complex process of puberty. Conditions in which nutritional status is not adequate, such as eating disorders and exercise-induced amenorrhea, are associated with low serum leptin levels, raising the possibility that relative leptin deficiency may be at least partly responsible for the reproductive abnormalities observed in these conditions [141–144]. Leptin may act as the critical link between adipose tissue and the reproductive system, indicating whether adequate energy reserves are present for normal reproductive function [139, 145].

Although puberty is recognized as an important period of risk for the onset of eating pathology in adolescent females, the extent to which puberty-related hormonal change accounts for elevated risk for disordered eating remains unclear [146]. For example, data from the Swedish Twin Study of Child and Adolescent Development [31] showed that pubertal development in early-to-mid adolescence was significantly associated with EDI scores and dieting in late adolescence, while no significant association was observed between pubertal development and dieting and purging in young adulthood.

In females, at least some of the unique genetic risk may be related to puberty and ovarian hormones. The heritability of disordered eating symptoms in females increases with both pubertal development and increasing levels of estradiol. Although more research is needed in order to elucidate specific mechanisms, gonadal hormones may be promising candidates for understanding sex and developmental effects and the ways in which genes exert their influence on disordered eating [30].

As far as fertility is concerned, it has been suggested that EDs are associated with fertility problems, unplanned pregnancies, negative attitudes to pregnancy and increased risk of induced abortions and miscarriages [147]. Linna et al. [147] reported that unplanned pregnancies were more common in the AN group compared with the general population, and that EDs patients more frequently experienced negative feelings upon discovering their pregnancy. Furthermore, BN patients had an increased risk of induced abortion compared to controls, and BED diagnosis was associated with an high risk of miscarriage. Considering the possible mechanisms underlying fertility problems in AN, they could be due to a combination of anovulation and a rejection of sexual activity. The reduced libido in AN can be associated with both psychological mechanisms (reduced self-esteem, body dissatisfaction; see section 3 of the Results) and low concentrations of circulating sex hormones. The main endocrine dysfunctions leading to amenorrhoea in anorexia nervosa are low concentrations of gonadotrophins and hypoestrogenism [148, 149] caused by inhibition of gonadotrophin-releasing hormone pulsatility [150–152], a blunted response of luteinizing hormone to gonadotrophin-releasing hormone, and diminished pulsatile release of luteinizing hormone [153], loss of feedback effect of estrogen, and multifollicular changes in the ovaries, resulting in the failure of follicle selection and dominance [154, 155]. In premenarchal patients there is a delay in pubertal development due to the continuation of these pre-pubertal secretory patterns [155]. Finally, it is important to note that most of the endocrine disturbances are due to starvation and may be considered a metabolic adaptation of the body to a negative energy balance [156].

Eating disorders and sexual dysfunction

Few studies considered sexual dysfunctions in EDs patients. Sexual functioning is seldom considered an important component of treatment or outcome predictor, and 175 PubMed records on related to the combination of "sexual dysfunction" and "eating disorders" were found. Among them, only 26 were original research actually focused on sexual dysfunctions.

Different studies considered the way sexual function is perceived by persons with EDs, according to different perspectives. In particular, some sexual behaviors have been considered as risky and impulsive behaviors correlated with other pathological EDs features, such as binge eating and purging behaviors [36, 52, 53, 157–162]. Moreover, number of partners and quality of sexual intercourse were taken into account. Sexual functioning in EDs has been considered as either hypersexuality either decrease sexuality [34]. In particular, in the period between 1995 and 2005 a large number of studies supported a model which was based on impulsivity as a mediator of the relationships between trauma, pathological eating behaviors and the tendency to impulsively engage in sexual activities with strangers or in risky situations and without using protections [52, 55, 157, 159, 163, 164]. Different research demonstrated an association between bulimic and purging behaviors and a number of dyscontrol behaviors, namely bullying, truancy, excessive drinking and sexual disinhibition in BN patients [24, 52, 159]. Number of partners and sexual intercourses has also been considered as a manifestation of multi-impulsivity, and the rates of specific disordered eating behaviors were associated with higher numbers of sex partners [165]. Indeed, BN patients and AN binge/purging type patients reported higher rate of multiple partners compared with AN restricting type patients [62]. Therefore, it has been hypothesized that individuals with EDs who are emotionally constricted and over-controlled (as AN restricting patients) report limited sexual functioning, whereas those with emotional dysregulation and low self control report more impulsive and chaotic sexual profiles [166]. However, being in a stable relationship can be considered a protective factor for psychopathology and especially for EDs, for different reasons correlated with the ability to establish emotional bonding, as well as with self-esteem and quality of life. Indeed, several studies showed that married women have lower EDs symptomatology as compared with single women [167–169]. For example, Keel et al. [169] found that being married predicted significant decreases in drive for thinness, bulimic symptoms, and dieting frequency in women, but not in a sample of men that was predominantly heterosexual.

The decrease of sexual interest in AN patients has been associated with hypogonadism and emaciation [170, 171], while weight restoration has been reported to favor an increase in sexual drive [46, 47, 64]. Indeed a specific correlation between the severity of underweight and sexual dysfunction has been reported [61, 117], while Castellini et al. [62] did not confirm such observation.

According to different reports [32, 98, 107–110) sexual dysfunction is not limited to AN but can be found also in BN patients [34, 61, 62, 64, 66] and BED patients [60, 172], who usually do not suffer from hypogonadism. The comparisons of sexuality between AN and BN subjects showed conflicting results, with a similar pattern found in Castellini et al. study [62], while a Mangweth-Matzek et al. [34], demonstrating a worse condition in AN.

As far as AN is concerned, it is of note that few studies compared AN subtypes, searching for a correlation between pathological behaviors and sexual dysfunction [55, 62, 64, 171]. Castellini et al. [62] demonstrated that purging anorectics had greater sexual drive than non-purging patients, and Rothschild et al. [171] reported that sexual fantasy distinguished AN subgroups, with a paucity of fantasy among restrictive anorectics at normal weight compared with binge/purging anorectics. Castellini et al. [62] found that AN restricting/type patients showed lower female sexual function index arousal, lubrication, orgasm, satisfaction, and pain scores compared with AN binge/purging type and BN patients, confirming that pathological eating behaviors were associated with different levels of sexual dysfunction.

The relationship with own body has been considered as a possible mediator of the sexual dysfunction in EDs. Indeed, a shared psychopathological feature of all EDs is body image distortion and body dissatisfaction [1, 4]. Body image dissatisfaction has been indicated as one of the most relevant predictors of treatment response and long-term outcome of EDs [10, 11, 173, 174], and the most relevant antecedent of both AN and BN [175]. Furthermore, recent research in female sexuality found that several aspects of body satisfaction, sexual attractiveness, body worry during sexual activity, as well as preoccupations about body weight correlates with different extent to sexual satisfaction in healthy women [56, 58, 176]. Persons with a healthy attitude toward their own body have been shown to report more frequent sexual experiences and a wider range of sexual activities, as they feel more sexually desirable and self-confident [177, 178]. This line of research was followed by some groups, which demonstrated that body dissatisfaction significantly correlated with the

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severity of sexual dysfunction [60–62]. In particular, lower female sexual index scores were correlated with higher shape concerns measured by means of the eating disorder examination in AN, BN [62] as well as in BED [60] patients. A further confirmation that EDs specific psychopathology could be considered as a specific maintaining factor for sexual dysfunction in EDs subjects was provided by a 1-year follow-up study after a cognitive behavioral intervention in AN and BN [64]. This kind of intervention is generally targeted to the common core psychopathological features, including dysfunctional attitudes toward eating and overvalued thoughts regarding weight [22, 179]. The above mentioned research confirmed that the psychological intervention allowed an improvement in sexual functioning in both AN and BN groups by the mediation of intrapsychic processes related to the treatment effects on these core features.

Severity of sexual dysfunction has been also associated with frequency of binge eating behaviors in AN binge/purging type, BN [62] and BED [60] patients. In both these studies sexual dysfunction was correlated with emotional eating, a psychological dimension defined as "eating in response to a range of negative emotions" [180]. Therefore, authors hypothesized a shared maintaining factor for both binge eating and sexual dysfunction, associated with dysfunctional mood modulatory mechanism, supported by the relevance of cognitive and emotional dimensions in the modulations of women's sexual desire [181]. This hypothesis is confirmed by the relationship between sexual and the impulsive-compulsive spectrum symptoms, and negative emotions [57, 59, 61]. Indeed, sexual dysfunction has been associated with different adverse emotional states such as sadness, guilt, and anger associated with negative automatic thoughts during sexual activity [182].

As already observed in AN, even in BED patients it can be hypothesized by a relevant role of BMI modification in determining the sexual dysfunction. Obesity has been demonstrated to affect several aspects of sexual functioning [11, 183–186], and the weight loss following bariatric surgery has been reported to be correlated with a significant improvement in hormonal profiles as well as sexual functioning in both men [187, 188] and women [65, 189]. The impaired sexuality in obese subjects can be due to the psychological consequences of obesity as well as to the organic conditions associated with metabolic syndrome [190]. However, also in BED patients it can be hypothesized to be a specific effect of psychopathology, considering that the sexual functioning of obese patients with EDs has been found to be more impaired as compared with obese subjects without EDs [60, 63].

Sexual abuse and eating disorders

The relationship between sexual abuse and EDs has been clearly established from different perspectives. In the last 20 years, 33 reviews and meta-analyses articles on this topic have been published. Although a recent meta-analysis [107] showed that sexual abuse is particularly associated with a diagnosis of BN, most of the available reviews of the literature [16, 18, 107, 191–195] indicated that childhood sexual abuse (CSA) – defined as a sexual encounter in which touching or penetration of the genitals happened before age 16 with someone at least 5 years older [196] – represents a well-demonstrated risk factor for the development of all EDs in a trans-diagnostic fashion.

It is important to note that most of the meta-analyses demonstrated that the strength of the association varied across the large amount of studies in this field, as well as the moderators of the relationship between sexual abuse and EDs. Several methodological issues account for such differences in the literature. Most of the studies adopted a cross-sectional design based on case-control approach [107] and the strongest association was reported in the non-clinical control groups [107, 197, 198]. However, only longitudinal studies can demonstrate the role of a life event as a risk factor for the onset of a disorder, clarifying causal priority, and avoiding recall and sampling bias. Indeed, the retrospective design may be biased by the poor reliability of the memories relevant to childhood, memory distortions, and influence of adulthood psychopathology [199–201]. The population taken into account is also a relevant issue, as evidence of greater reliability and generalizability can be derived from studies utilizing large random community samples, birth cohorts, and twin cohorts.

Therefore, evidence for the association between CSA and EDs increase from reliable and representative community studies such as those performed by Sanci et al. [198], Cutajar et al. [202], and Jonas et al. [203]. Cutajar et al. [202] evaluated almost 3000 children whose sexual abuse was assessed at the time by forensic medical examinations, using the records of the Victorian Institute of Forensic Medicine (VIFM), which, since 1957, has provided medical examinations in cases of suspected CSA. This strategy allowed establishing a very reliable evaluation of the life event. Jonas et al. [203] performed a representative study for the general population, avoiding the bias of a selected clinical sample. Authors used the detailed information available from the 2007 Adult Psychiatric Morbidity Survey of England (APMS 2007), including a large random sample of the English household population. Another

reliable survey was the one by Sanci et al. [198], based on the Victorian Adolescent Health Cohort Study, and providing evidence of a significant association of CSA with bulimic symptoms in young females.

Most of these studies demonstrated that CSA and sexual abuse in adulthood [201] are risk factors for many forms of comorbid psychopathological conditions. Furthermore, EDs have a multifactorial pathogenesis, and therefore are caused by a sequence or combination of risk factors rather than a single influence. Accordingly, sexual abuse may combine with certain other risk factors such as other types of abuse (emotional, physical, neglect) resulting in different psychopathological phenotypes [204]. The reason why CSA may result in EDs could be the combination of the trauma with different environmental or biological conditions, which represent the so-called "moderators". Moderator analyses allow identifying specific sub-populations of subjects in which a certain relationship is stronger than in the general population or allowed to predict in which persons the EDs outcome after a CSA is more likely. For example, Vanderlynden and Vandereyken [205] hypothesized that CSA occurring in early developmental stages leads to a higher probability to develop an ED rather than other psychiatric conditions. From this perspective, EDs represent a severe consequence of CSA occurring when the foundation of brain architecture is being wired and it can disrupt the development of neural circuits that interfere with typical patterns of brain development, heightening vulnerability to psychopathology. Therefore, it compromises a child's ability to successfully master stage-salient developmental tasks, including secure attachments, emotion regulation, stress response, and his/her relationship with ones own body [206–208].

Furthermore, several studies considered the synergic effects of sexual abuse and repeated traumatic experience [209–211]. Sanci et al. [198] found that two or more episodes of CSA before the age of 16 years predicted a >5-fold elevated cumulative risk of new bulimic syndrome during adolescence. Moreover, Favaro et al. [212] took into consideration traumas preceding the CSA, demonstrating a synergic effect of neonatal dysmaturity and childhood abuse in increasing the risk for AN, which might be explained by the hypothesis that a prenatal programming of stress response systems can result in an impairment of the individual's resilience to severe stressful events.

Even though the scientific literature converges on a clear association between sexual abuse and EDs psychopathology, no conclusive explanation was provided regarding the possible mechanisms mediating such relationship. It is well known that sexual abuse and especially CSA determine a pattern of several concomitant psychopathological conditions, characterized by pathological eating behaviors, sexual dysfunction [62, 213], dissociative experiences [214, 215], mood disorders [202], and suicidal thoughts [107].

Given that a person's sexuality, mood regulation and eating behaviors are multifaceted and interconnected phenomena, several models of interaction related with sexual abuse have been proposed involving biologic, cognitive, and affective processes [210, 213, 216–218]. Therefore, common underlying maintaining factors of these symptoms which are typical consequences of traumatic sexual experience, have been proposed, such as severe body image disturbances [19, 44, 219] and emotion dysregulation [197, 220].

Several psychopathological trajectories have been proposed for the development of EDs among women with a history of sexual abuse or CSA. Sexual abuse may interfere with sexual maturation [221], leading a woman to feel revulsion about her body in a way that may manifest with concerns about body weight, shape, and size [19]. Furthermore, sexual trauma might act specifically by inducing feelings of poor self-esteem, triggering self-starvation, as a reflection of the individual's effort at regaining control on her life [107].

Dissociation, which consists in "a disruption in the usually integrated functions of consciousness, memory, identity, and perception of the environment" [214], has been considered as a shared maintaining factor of both sexual dysfunction and pathological eating behaviors in sexual abuse survivors [62, 204]. For example, a disconnection between body and mind and the dissociation during sex reported by sexual abuse survivors have been associated with individual's subjective experience of sexual arousal inhibition [222]. Indeed, sexually abused persons experiencing sexual difficulties may have learned to automatically pair sexual stimuli with fear (implicit sexual memories) [213]. As a partial confirmation of this hypothesis, Castellini et al. [66] found a significant association between sexual desire and cortisol levels in a group of EDs reporting a history of CSA, and Rellini et al. [213] found a greater cortisol response associated with greater perceived states of physiological sexual arousal in subjects reporting abuse. The authors hypothesized that in people who have been abused during childhood, the emotional involvement in the sexual relationship would result in activation of the mechanisms of stress. In the same way, dissociation has been implicated in the pathogenesis of bulimic symptoms [214, 215], according with the "mood modulation" theory [223] and the "escape" theory [224]. Several authors suggested that binge eating occurs in dissociative states [214, 215, 225] as a way to manage the

effect of a sexual abuse. In particular, the awareness of the traumatic experience creates negative affects, and favors the onset of the escape mechanism, which occurs by the patient narrowing his or her awareness from abstract levels (self-evaluation) to the level of the physical surroundings or stimulus (i.e. food) [215].

Indeed, CSA has been supposed to determine emotion dysregulation – encompassing the ability to adaptively identify and cope with negative mood states – which resulted in several psychopathological outcomes associated with eating disorder behaviors (including binge eating, purging, or starving), such as self-harm and suicide attempts [197]. In this model, disordered eating behaviors are conceptualized as a maladaptive means of dealing with negative affect. Binge eating behavior likely acts as a negative reinforcement via this pathway because it acts to decrease or block negative affect, at least in the short term, by relieving the experience of painful emotions [180].

Moreover, body image disturbance and body dissatisfaction represent common psychopathological consequence of sexual abuse both associated with sexual dysfunction and EDs. The relationship with one's own body has been often reported among victims of sexual abuse [19, 219].

Finally, recent studies suggest a mediating effect of sexual dysfunction in the relationship between CSA and dangerous sexual behaviors [218]. Indeed, the body is the real battleground for EDs who experienced sexual abuse, who are more likely to engage in self-destructive behaviours [212]. The recourse to self-harming behavior in these patients has been interpreted as a way to regain control on a compromised interoceptive awareness by cutting and burning the body [219]. Compulsive cleaning behaviors have also been associated with a history of CSA by the mediation of a disease related to ones own body, and specific compulsive behaviors could be interpreted as an attempt to symbolically clean out the body of the impurity associated with the past abuse [19]. Finally, according to a psychodynamic perspective, it is possible that the relationship between sexual dysfunction, eating disorders and history of abuse can be interpreted in the light of a denial of adult sexuality in relation to either childhood conflicts or on the refusal of the body as a symbolic refusal of the abuse instead.

Sexual orientation and gender identity

When searching for "sexuality" and "eating disorders", a considerable proportion of original studies (27.0%) deal with the relationship between EDs, sexual orientation and gender identity. Several studies indicated that non heterosexual men (homosexual and bisexual persons) are at higher risk to develop an ED [67, 69–72, 85, 226, 227]. Considering specific pathological behaviors, Austin et al. [96] found that non-heterosexual persons reported an elevated odds of purging and diet pill use in both girls and boys (odds ratios [OR] range=1.9–6.8). From a different perspective it has been reported that up to 42% of men seeking treatment for an eating disorder have self-identified as homosexual or bisexual [68, 228].

As a general explanation for all non-exclusively heterosexual persons, the Minority Stress Model suggest that sexual minorities might experience more stress and therefore are more susceptible to developing pathological eating behaviors [75, 76, 229, 230]. However, more specific explanation has been provided. For example, it has been observed that gays or bisexual men develop a higher body shape concern due to the pressure to obtain a lean physique to attract a male partner, as men emphasize physical appearance when selecting mates [226]. This excessive preoccupation with one's physical appearance might result in an ED as well as for heterosexual women. Furthermore, it has been noted that homosexual men have a heightened emphasis on a thin, muscular, and youthful figure [73, 231, 232]. This could lead to a greater drive for thinness as compared to heterosexual men [67]. Indeed, the presence of EDs behaviors in gays appeared to be mediated by a higher body dissatisfaction in homosexual than heterosexual men [86, 233]. Gay men scored significantly higher on drive for thinness, body dissatisfaction, and body image-related anxiety than their straight counterparts [88, 95]. The objectification theory might represent a possible explanation: gay men tend to report higher levels of exposure to sexually objectifying media, body surveillance, body shame, which in turn leads to disordered eating behaviors than heterosexual men [88, 90, 91]. It has been suggested that the gay men's increased vulnerability to media influence partially accounts for the relatively high rate of eating pathology observed in this population, as perceptions of media influence were higher for gay men, and it has been found to significantly mediate the relation between sexual orientation and eating and body image concerns [88].

Less evidence has been found in women [79, 81, 82, 93]. Some studies reported that lesbian sexual orientation is predictive of positive body image and fewer negative attitudes toward eating and weight [80, 83, 110]. Matthews-Ewald et al. [79] found that lesbians reported more dieting to lose weight compared to those identifying as heterosexual. Authors speculated that the risk of dieting

may be due to the increased incidence in overweight and obesity among lesbian women [234]. Regarding a possible mechanism involved in women, Brewster et al. [97] examined the roles of antibisexual discrimination and internalized biphobia in a group of bisexual women. They found that antibisexual discrimination and internalization of sociocultural standards of attractiveness were correlated with body surveillance, body shame, and eating disorder symptoms. Finally, a moderating role of childhood sexual abuse has been hypothesized, as, for example, men with a history of childhood sexual abuse showed a higher rate of bulimic symptoms compared with men who do not have a history of childhood sexual abuse [75].

The issue of the relationship between gender identity and EDs has been more recently addressed by the scientific literature, as compared to the sexual orientation one. A population study performed on 289,024 students from 223 US universities [97] showed that transgender students had greater odds of past-year self-reported EDs diagnosis, use of diet pills, and vomiting or laxatives, confirming previous observations [99, 102]. As for sexual orientation, it has been postulated that the rate of psychopathology can be attributed to the high rates of discrimination suffered by transgender persons [235, 236]. Indeed, several authors support the hypothesis that transgender persons lack a primary psychopathology [237–240], and the psychiatric disorders often reported may be the consequence of difficulties in coping with social stigma [241, 242] or rejection by family and friends [243].

Q8: In the original manuscript ref. 206 (now ref. 235) is not cited in the text. Please check and confirm the insertion of ref. 235 in text citation is correct here

However, it has been noted that transgender persons may be at increased risk of body dissatisfaction, which may predispose them to disordered eating [99]. This is especially true for gender dysphoria (GD) persons who show a strong and persistent identification with the opposite sex, discomfort with one's own sex, and a sense of inappropriateness in the gender role of that sex [3]. GD subjects live in a cognitive state where their physical body is in contrast with their self-perceived identity [244], and this experience is a source of deep and chronic suffering [245]. While several case reports have been published on GD patients with EDs [e.g. [246–248]], few larger studies took into consideration this issue. Vocks et al. [103] and Bandini et al. [105] reported that both male-to-female and male-to-female persons show higher levels of EDs specific psychopathology, as compared to a control group. In a recent study, Witcomb et al. [249] demonstrated that trans individuals showed greater body dissatisfaction than controls, and trans males had comparable body dissatisfaction scores to eating disordered males. According to Pauly and Lindgren's [250] position, the authors supported the position that the body is the primary source of suffering in these persons, even with regard to nonsexual body parts and aspects. A further confirmation of the centrality of the body in GD is also demonstrated by the fact that its successful treatment is also capable of relieving these symptoms [106].

Q9: Please check "both male-to-female and male-to-female persons" do you mean male-to-female and female-to-male persons?

Several studies [98, 100, 101], took into consideration the relationship between gender role EDs pathological behaviors and body dissatisfaction. In general, masculinity seems to be a protective factor, independently by the biological gender, while femininity appears to be more associated with body dissatisfaction and in turn with pathological eating behaviors [100, 101].

Expert opinion

Sexuality in EDs does not have a unique significance and it has been considered according to different lines of research in the scientific literature. From an etiological point of view, the timing of puberty with its hormonal consequences and the changes in the way persons perceive their own body represent a crucial period of life for EDs onset. Even though several review articles have been provided on this topic, still there are few research studies which clarify the mechanisms underlying this association. Furthermore, there are few studies which took into consideration how the attitude toward one's own body changes across this period, together with the eating and physical exercise habits. Indeed, in most Western countries (especially in the south of Europe) sexual education is almost neglected at school.

According to our research of the literature, the multifactorial etiopathogenesis of EDs include sexual abuse, and CSA is a well-recognized risk factor for the onset of all forms of EDs. The underlying mechanisms of the several psychopathological consequences of sexual abuse have been studied in different ways, providing conflicting and non-conclusive results. However, it has been demonstrated that CSA and sexual abuse identified a sub-population of EDs with a worse long-term outcome. According to our research, an adequate clinical approach should always consider the possibility of sexual abuse when treatment of ED does not follow the expected course or improvement. Furthermore, it would be crucial to specifically target the comorbidities of CSA before useful treatment of an ED can begin. Finally, it is reasonable to hypothesize that effective treatments specifically targeted on body image perception and cognitive/emotional consequences of sexual abuse would improve not just sexual activity and pathological eating behaviors but the long-term recovery process too.

It is noteworthy that the evaluation of sexual functioning in EDs patients represents a limited proportion of the studies included in the category of "sexuality and eating disorders". Sexuality of EDs has been studied in terms of hypersexuality as a form of dangerous consequence of impulsivity. Therefore, the scientific literature followed an approach that sounds like: "EDs patients do not have sex and, if they have it, they use it in a compulsive self-damaging way". Recent researches overcome this perspective considering the sexuality of EDs persons in terms of sexual desire, satisfaction, orgasm and pain. In other words, authors evaluated sexual functioning of patients who really have a form of sexual activity, even though damaged. Results from this line of research are promising, and describe a clear relationship between sexual dysfunction and the core psychopathological features of EDs, such as body image disturbances. Therefore, a relationship with one's own body, as well as sexual function, should become the target of psychological interventions, as well as the pathological eating behaviors.

As far as the relationships between sexual orientation, gender identity and EDs are concerned, the analyses of the literature showed that the majority of the studies were carried out by experts in the field of sexual minorities or gender dysphoria. In most cases they took into consideration EDs psychopathology in groups of homosexual and gender dysphoric persons. However, from the opposite point of view, the issue of sexual orientation and gender identity in EDs is barely considered by clinicians and research studying EDs. However, several lines of research are developing new models of maintaining factors of EDs psychopathology related the topic of self-identity. Accordingly, impairments in overall identity development and the failure to establish multiple and diverse domains of self-definition have been suggested as the core pathoplastic mechanism of EDs.

Outlook

Taking into consideration the complex relationship between sexuality and EDs would significantly improve the way of recognizing and treating these patients in the future.

For example, longitudinal studies should take into account the temporal relationship between hormonal effects, body modification induced by puberty, subjective perception of increasing sexual drives and the changes in eating habits as predisposing factors for EDs onset. As extensively reported in the puberty and sexual abuse section, from a methodological point of view, these kind of studies should be based on a general population survey, in order to provide a more comprehensive and representative explanation of the phenomena. The main reason for this choice would be that the main EDs diagnoses provided by the DSM 5 represents only the tip of an iceberg, while the reality of EDs psychopathology in the general population is much more complex, and it should be considered in a dimensional rather than categorical perspective.

From a clinical point of view, future research focused on treatment and long-term outcome of EDs patients should take into account the complex relationship between psychopathological core features such as body image distortion and sexual dysfunction. In particular, there is a need for clinical trials which take into consideration the role of sexual dysfunction (and amenorrhea for AN patients) as potential moderators and mediators of the efficacy of the psychological and pharmacological interventions. Future researches should evaluate whether the recovery of a good sexuality represent a prognostic factor for the treatment, or in which extent relationship with one's own body and sexual functioning could be considered as the final hurdle in the recovery process of persons with EDs.

Highlights

- Sexuality in EDs has been considered in a heterogeneous way, including different lines of research.
- Puberty is a risk factor for the onset of EDs, and timing of puberty with its hormonal consequences represent a crucial period of life for EDs onset.
- Few studies which took into consideration how the attitude toward one's own body change during puberty, together with the eating and physical exercise habits.
- Sexual abuse and especially childhood sexual abuse are well-demonstrated risk factors for the onset of EDs, and they determine long-lasting psychopathological consequences, which could interfere with the treatment of the disorders.
- From a clinical point of view, an adequate clinical approach should always consider the possibility of sexual abuse when treatment does not follow the expected course or improvement.
- A large proportion of the studies focused on sexuality in EDs patients considered the relationship between

impulsivity, pathological eating behaviors and compulsive sexuality.
– Recent research focused on the sexual satisfaction of eating disorders patients, demonstrating a clear relationship between sexual dysfunction and core psychopathological features, such as body image disturbances.

Several studies documented that non heterosexual orientation is associated with body uneasiness and pathological eating behaviors in men; transgender persons may be at increased risk of body dissatisfaction, which may predispose them to disordered eating.

References

1. Fairburn CG, Harrison PJ. Eating disorders. Lancet 2003;i;361:407–16.
2. Fichter MM, Quadflieg N, Hedlund S. Twelve-year course and outcome predictors of anorexia nervosa. Int J Eat Disord 2006;39:87–100.
3. American Psychiatric Association. Diagnostic and statistical manual of mental disorders.5th ed. Arlington, VA: American Psychiatric Association, 2013.
4. Fairburn CG. Eating disorders: the transdiagnostic view and the cognitive behavioral theory. In: Fairburn CG, editor. Cognitive behavior therapy and eating disorders. New York, NY: Guilford Press, 2008:7–22.
5. Williamson DA, Womble LG, Smeets MA, Netemeyer RG, Thaw JM, Kutlesic V, Gleaves DH. Latent structure of eating disorder symptoms: a factor analytic and taxometric investigation. Am J Psychiatry 2002;159:412–8.
6. Thomas JJ, Vartanian LR, Brownell KD. The relationship between eating disorder not otherwise specified (EDNOS) and officially recognized eating disorders: meta-analysis and implications for DSM. Psychol Bull 2009;135:407–33.
7. Fairburn CG, Bohn K. Eating disorder NOS (EDNOS): an example of the troublesome "not otherwise specified" (NOS) category in DSM-IV. Behav Res Ther 2005;43:691–701.
8. Machado PP, Machado BC, Gonçalves S, Hoek HW. The prevalence of eating disorders not otherwise specified. Int J Eat Disord 2007;40:212–7.
9. Tozzi F, Thornton LM, Klump KL, Fichter MM, Halmi KA, Kaplan AS, Strober M, Woodside DB, Crow S, Mitchell J, Rotondo A, Mauri M,Cassano G, Keel P, Plotnicov KH, Pollice C, Lilenfeld LR, Berrettini WH, Bulik CM, Kaye WH. Symptom fluctuation in eating disorders: correlates of diagnostic crossover. Am J Psychiatry 2005;162:732–40.
10. Castellini G, Lo Sauro C, Mannucci E, Ravaldi C, Rotella CM, Faravelli C, Ricca V. Diagnostic crossover and outcome predictors in eating disorders according to DSM-IV and DSM-V proposed criteria: a 6-year follow-up study. Psychosom Med 2011;73:270–9.
11. Ricca V, Castellini G, Lo Sauro C, Mannucci E, Ravaldi C, Rotella F, Faravelli C. Cognitive-behavioral therapy for threshold and subthreshold anorexia nervosa: a three-year follow-up study. Psychother Psychosom 2010;79:238–48.
12. Milos GF, Baur V, Muehlebach S, Spindler A. Axis-I comorbidity is linked to prospective instability of diagnoses within eating disorders. BMC Psychiatry 2013;13:295.
13. Keel PK, Mitchell JE, Miller KB, Davis TL, Crow SJ. Long-term outcome of bulimia nervosa. Arch Gen Psychiatry 1999;56:63–9.
14. Fairburn CG, Cooper Z, Doll HA, Norman P, O'Connor M. The natural course of bulimia nervosa and binge eating disorder in young women. Arch Gen Psychiatry 2000;57:659–65.
15. Klump KL, Bulik CM, Kaye WH, Treasure J, Tyson E. Academy for eating disorders position paper: eating disorders are serious mental illnesses. Int J Eat Disord 2009;42:97–103.
16. Jacobi C, Hayward C, De Zwaan M, Kraemer HC, Agras WS. Coming to terms with risk factors for eating disorders: application of risk terminology and suggestions for a general taxonomy. Psychol Bull 2004;130:19–65.
17. Johnson JG, Cohen P, Brown J, Smailes EM, Bernstein DP. Childhood maltreatment increases risk for personality disorders during early adulthood. Arch Gen Psychiatry 1999;56:600–6.
18. Carr CP, Martins CM, Stingel AM, Lemgruber VB, Juruena MF. The role of early life stress in adult psychiatric disorders: a systematic review according to childhood trauma subtypes. J Nerv Ment Dis 2013;201:1007–20.
19. Kearney-Cooke A, Ackard DM. The effects of sexual abuse on body image, self-image, and sexual activity of women. J Gend Specif Med 2000;3:54–60.
20. Fornari V, Dancyger IF. Psychosexual development and eating disorders. Adolesc Med 2003;14:61–75.
21. Wichstrom L. Social, psychological and physical correlates of eating problems. A study of the general adolescent population in Norway. Psychol Med 1995;25:567–79.
22. Fairburn CG, Welch SL, Doll HA, Davis BA, O'Connor ME. Risk factors for bulimia nervosa. Arch Gen Psychiatry 1997;54: 509–17.
23. Fairburn CG, Cooper Z, Doll HA, Welch SL. Risk factors for anorexia nervosa: three integrated case-control comparisons. Arch Gen Psychiatry 1999;56:468–76.
24. Kaltiala-Heino R, Rissanen A, Rimpelä M, Rantanen P. Bulimia and impulsive behaviour in middle adolescence. Psychother Psychosom 2003;72:26–33.
25. Ruuska J, Kaltiala-Heino R, Koivisto AM, Rantanen P. Puberty, sexual development and eating disorders in adolescent outpatients. Eur Child Adolesc Psychiatry 2003;12:214–20.
26. Klump KL, Perkins PS, Alexandra Burt S, McGue M, Iacono WG. Puberty moderates genetic influences on disordered eating. Psychol Med 2007;37:627–34.
27. Cotrufo P, Cella S, Cremato F, Labella AG. Eating disorder attitude and abnormal eating behaviours in a sample of 11-13-year-old school children: the role of pubertal body transformation. Eat Weight Disord 2007;12:154–60.
28. Zehr JL, Culbert KM, Sisk CL, Klump KL. An association of early puberty with disordered eating and anxiety in a population of undergraduate women and men. Horm Behav 2007;52: 427–35.
29. Day J, Schmidt U, Collier D, Perkins S, Van den Eynde F, Treasure J, Yi I, Winn S, Robinson P, Murphy R, Keville S, Johnson-Sabine E, Jenkins M, Frost S, Dodge L, Berelowitz M, Eisler I. Risk factors, correlates, and markers in early-onset bulimia nervosa and EDNOS. Int J Eat Disord 2011;44:287–94.

Q10: Refs. [20] and [41] and [24] and [200] and [90] and [234] were the same in the original manuscript – the duplications were deleted and following references were re-ordered according to numerical order in text and reference list. Please check and confirm

30. Culbert KM, Racine SE, Klump KL. The influence of gender and puberty on the heritability of disordered eating symptoms. Curr Top Behav Neurosci 2011;6:177–85.
31. Baker JH, Thornton LM, Lichtenstein P, Bulik CM. Pubertal development predicts eating behaviors in adolescence. Int J Eat Disord 2012;45:819–26.
32. McNicholas F, Dooley B, McNamara N, Lennon R. The impact of self-reported pubertal status and pubertal timing on disordered eating in Irish adolescents. Eur Eat Disord Rev 2012;20:355–62.
33. Schmidt U, Evans K, Tiller J, Treasure J. Puberty, sexual milestones and abuse: how are they related in eating disorder patients? Psychol Med 1995;25:413–7.
34. Mangweth-Matzek B, Rupp CI, Hausmann A, Kemmler G, Biebl W. Menarche, puberty, and first sexual activities in eating-disordered patients as compared with a psychiatric and a non psychiatric control group. Int J Eat Disord 2007;40:705–10.
35. Vannucci A, Tanofsky-Kraff M, Ranzenhofer LM, Kelly NR, Hannallah LM, Pickworth CK, Grygorenko MV, Brady SM,Condarco TA, Kozlosky M, Demidowich AP, Yanovski SZ, Shomaker LB, Yanovski JA. Puberty and the manifestations of loss of control eating in children and adolescents. Int J Eat Disord 2014;47:738–47.
36. Kaltiala-Heino R, Rimpelä M, Rissanen A, Ratanen P. Early puberty and early sexual activities are associated with bulimic type eating pathology in middle adolescence. J Adolesc Health 2001;28:346–52.
37. Tenconi E, Lunardi N, Zanetti T, Santonastaso P, Favaro A. Predictors of binge eating in restrictive anorexia nervosa patients in Italy. J Nerv Ment Dis 2006;194:712–5.
38. Tremblay L, Lariviere M. The influence of puberty onset, body mass index, and pressure to be thin on disordered eating behaviors in children and adolescents. Eat Behav 2009;10: 75–83.
39. Abraham S, Boyd C, Lal M, Luscombe G, Taylor A. Time since menarche, weight gain and body image awareness among adolescent girls: onset of eating disorders? J Psychosom Obstet Gynaecol 2009;30:89–94.
40. Favaro A, Caregaro L, Tenconi E, Bosello R, Santonastaso P. Time trends in age at onset of anorexia nervosa and bulimia nervosa. J Clin Psychiatry 2009;70:1715–21.
41. Striegel-Moore RH, McMahon RP, Biro FM, Schreiber G, Crawford PB, Voorhees C. Exploring the relationship between timing of menarche and eating disorder symptoms in Black and White adolescent girls. Int J Eat Disord 2001;30:421–33.
42. Kaczmarek M, Trambacz-Oleszak S. The association between menstrual cycle characteristics and perceived body image: a cross-sectional survey of polish female adolescents. J Biosoc Sci 2015;29:1–17.
43. Stice E, Presnell K, Bearman SK. Relation of early menarche to depression, eating disorders, substance abuse, and comorbid psychopathology among adolescent girls. Dev Psychol 2001; 37:608–19.
44. Ackard DM, Peterson CB. Association between puberty and disordered eating, body image, and other psychological variables. Int J Eat Disord 2001;29:187–94.
45. Abraham S. Sexuality and reproduction in bulimia nervosa patients over 10 years. J Psychosom Res 1998;44:491–502.
46. Morgan JF, Lacey JH, Reid F. Anorexia nervosa: Changes in sexuality during weight restoration. Psychosom Med 1999;61: 541–5.
47. Meguerditchian C, Samuelian-Massat C, Valéro R, Begu-Le Corroller A, Fromont I, Mancini J, Sparrow JD, Poinso F, Vialettes B. The impact of weight normalization on quality of recovery in anorexia nervosa. J Am Coll Nutr 2009;28:397–404.
48. Wiederman MW, Pryor T. Substance use and impulsive behaviors among adolescents with eating disorders. Addict Behav 1996;21:269–72.
49. Pryor T, Wiederman MW, McGilley B. Clinical correlates of anorexia nervosa subtypes. Int J Eat Disord 1996;19:371–9.
50. Wiederman MW, Pryor T, Morgan CD. The sexual experience of women diagnosed with anorexia nervosa or bulimia nervosa. Int J Eat Disord 1996;19:109–18.
51. Nagata T, Kiriike N, Iketani T, Kawarada Y, Tanaka H. History of childhood sexual or physical abuse in Japanese patients with eating disorders: relationship with dissociation and impulsive behaviours. Psychol Med 1999;29:935–42.
52. Nagata T, Kawarada Y, Kiriike N, Iketani T. Multi-impulsivity of Japanese patients with eating disorders: primary and secondary impulsivity. Psychiatry Res 2000;94:239–50.
53. Paul T, Schroeter K, Dahme B, Nutzinger DO. Self-injurious behavior in women with eating disorders. Am J Psychiatry 2002;159:408–11.
54. Eddy KT, Novotny CM, Westen D. Sexuality, personality, and eating disorders. Eat Disord 2004;12:191–208.
55. Culbert KM, Klump KL. Impulsivity as an underlying factor in the relationship between disordered eating and sexual behaviour. Int J Eat Disord 2005;38:361–6.
56. Ackard DM, Kearney-Cooke A, Peterson CB. Effect of body image and self-image on women's sexual behaviors. Int J Eat Disord 2000;28:422–9.
57. Rodríguez S, Mata JL, Moreno S, Fernández MC, Vila J. Psychophysiological mechanisms involved in the affective regulation and food restriction of women at risk of suffering from bulimia nervosa. Psicothema 2007;19:30–6.
58. Morgan CD, Wiederman MW, Pryor TL. Sexual functioning and attitudes of eating-disordered women: a follow-up study. J Sex Marital Ther 1995;21:67–77.
59. Wiederman MW, Pryor T. A comparison of ever-married and never-married women with anorexia or bulimia nervosa. Int J Eat Disord 1997;22:395–401.
60. Castellini G, Mannucci E, Mazzei C, Lo Sauro C, Faravelli C, Rotella CM, Maggi M, Ricca V. Sexual function in obese women with and without binge eating disorder. J Sex Med 2010;7: 3969–78.
61. Pinheiro AP, Raney TJ, Thornton LM, Fichter MM, Berrettini WH, Goldman D, Halmi KA, Kaplan AS, Strober M, Treasure J, Woodside DB, Kaye WH, Bulik CM. Sexual functioning in women with eating disorders. Int J Eat Disord 2010;43:123–9.
62. Castellini G, Lelli L, Lo Sauro C, Fioravanti G, Vignozzi L, Maggi M, Faravelli C, Ricca V. Anorectic and bulimic patients suffer from relevant sexual dysfunctions. J Sex Med 2012;9: 2590–9.
63. Folope V, Chapelle C, Grigioni S, Coëffier M, Déchelotte P. Impact of eating disorders and psychological distress on the quality of life of obese people. Nutrition 2012;28:7–13.
64. Castellini G, Lo Sauro C, Lelli L, Godini L, Vignozzi L, Rellini AH, Faravelli C, Maggi M, Ricca V. Childhood sexual abuse moderates the relationship between sexual functioning and eating disorder psychopathology in anorexia nervosa and bulimia nervosa: a 1-year follow-up study. J Sex Med 2013;10:2190–200.

65. Sarwer DB, Spitzer JC, Wadden TA, Rosen RC, Mitchell JE, Lancaster K, Courcoulas A, Gourash W, Christian NJ. Sexual functioning and sex hormones in persons with extreme obesity and seeking surgical and nonsurgical weight loss. Surg Obes Relat Dis 2013;9:997–1007.
66. Castellini G, Lelli L, Lo Sauro C, Vignozzi L, Maggi M, Faravelli C, Ricca V. Childhood abuse, sexual function and cortisol levels in eating disorders. Psychother Psychosom 2012;81:380–2.
67. Brown TA, Keel PK. The impact of relationships, friendships, and work on the association between sexual orientation and disordered eating in men. Eat Disord 2013;21:342–59.
68. Olivardia R, Pope HG Jr, Mangweth B, Hudson JI. Eating disorders in college men. Am J Psychiatry 1995;152:1279–85.
69. Beren SE, Hayden HA, Wilfley DE, Grilo CM. The influence of sexual orientation on body dissatisfaction in adult men and women. Int J Eat Disord 1996;20:135–41.
70. French SA, Story M, Remafedi G, Resnick MD, Blum RW. Sexual orientation and prevalence of body dissatisfaction and eating disordered behaviors: a population-based study of adolescents. Int J Eat Disord 1996;19:119–26.
71. Carlat DJ, Camargo CA Jr, Herzog DB. Eating disorders in males: A report on 135 patients. Am J Psychiatry 1997;154: 1127–32.
72. Russell CJ, Keel PK. Homosexuality as a specific risk factor for eating disorders in men. Int J Eat Disord 2002;31:300–6.
73. Yelland C, Tiggemann M. Muscularity and the gay ideal: body dissatisfaction and disordered eating in homosexual men. Eat Behav 2003;4:107–16.
74. Hospers HJ, Jansen A. Why homosexuality is a risk factor for eating disorders in males. J Soc Clin Psych 2005;24:1188–201.
75. Feldman MB, Meyer IH. Eating disorders in diverse lesbian, gay, and bisexual populations. Int J Eat Disord 2007;40:218–26.
76. Feldman MB, Meyer IH. Childhood abuse and eating disorders in gay and bisexual men. Int J Eat Disord 2007;40:418–23.
77. Heinberg LJ, Pike E, Loue S. Body image and eating disturbance in African-American men who have sex with men: preliminary observations. J Homosex 2009;56:839–48.
78. Matthews DD, Blosnich JR, Farmer GW, Adams BJ. Operational definitions of sexual orientation and estimates of adolescent health risk behaviors. LGBT Health 2014;1:42–49.
79. Matthews-Ewald MR, Zullig KJ, Ward RM. Sexual orientation and disordered eating behaviors among self-identified male and female college students. Eat Behav 2014;15:441–4.
80. Heffernan K. Eating disorders and weight concern among lesbians. Int J Eat Disord 1996;19:127–38.
81. Moore F, Keel PK. Influence of sexual orientation and age on disordered eating attitudes and behaviors in women. Int J Eat Disord 2003;34:370–4.
82. Share TL, Mintz LB. Differences between lesbians and heterosexual women in disordered eating and related attitudes. J Homosex 2002;42:89–106.
83. Owens LK, Hughes TL, Owens-Nicholson D. The effects of sexual orientation on body image and attitudes about eating and weight. J Lesbian Stud 2003;7:15–33.
84. Duggan SJ, McCreary DR. Body image, eating disorders, and the drive for muscularity in gay and heterosexual men: the influence of media images. J Homosex 2004;47:45–58.
85. Conner M, Johnson C, Grogan S. Gender, sexuality, body image and eating behaviours. J Health Psychol 2004;9:505–15.
86. Kaminski PL, Chapman BP, Haynes SD, Own L. Body image, eating behaviors, and attitudes toward exercise among gay and straight men. Eat Behav 2005;6:179–87.
87. Morgan JF, Arcelus J. Body image in gay and straight men: a qualitative study. Eur Eat Disord Rev 2009;17:435–43.
88. Carper TL, Negy C, Tantleff-Dunn S. Relations among media influence, body image, eating concerns, and sexual orientation in men: a preliminary investigation. Body Image 2010;7:301–9.
89. Blashill AJ. Elements of male body image: prediction of depression, eating pathology and social sensitivity among gay men. Body Image 2010;7(4):310–6.
90. Wiseman MC, Moradi B. Body image and eating disorder symptoms in sexual minority men: a test and extension of objectification theory. J Couns Psychol 2010;57:154–66.
91. Dakanalis A, Di Mattei VE, Bagliacca EP, Prunas A, Sarno L, Riva G, Zanetti MA. Disordered eating behaviors among Italian men: objectifying media and sexual orientation differences. Eat Disord 2012;20:356–67.
92. Koh AS, Ross LK. Mental health issues: a comparison of lesbian, bisexual and heterosexual women. J Homosex 2006;51:33–57.
93. Austin SB, Ziyadeh NJ, Corliss HL, Rosario M, Wypij D, Haines J, Camargo CA Jr, Field AE. Sexual orientation disparities in purging and binge eating from early to late adolescence. J Adolesc Health 2009;45:238–45.
94. Legenbauer T, Vocks S, Schafer C, Schutt-Stromel S, Hiller W, Wagner C, Vögele C. Preference for attractiveness and thinness in a partner: influence of internalization of the thin ideal and shape/weight dissatisfaction in heterosexual women, heterosexual men, lesbians, and gay men. Body Image 2009;6: 228–34.
95. Cella S, Iannaccone M, Ascione R, Cotrufo P. Body dissatisfaction, abnormal eating behaviours and eating disorder attitude in homo- and heterosexuals. Eat Weight Disord 2010;15:180–5.
96. Austin SB, Nelson LA, Birkett MA, Calzo JP, Everett B. Eating disorder symptoms and obesity at the intersections of gender, ethnicity, and sexual orientation in US high school students. Am J Public Health 2013;103:16–22.
97. Brewster ME, Velez BL, Esposito J, Wong S, Geiger E, Keum BT. Moving beyond the binary with disordered eating research: a test and extension of objectification theory with bisexual women. J Couns Psychol 2014;61:50–62.
98. Meyer C, Blissett J, Oldfield C. Sexual orientation and eating psychopathology: the role of masculinity and femininity. Int J Eat Disord 2001;29:314–8.
99. Hepp U, Milos G. Gender identity disorder and eating disorders. Int J Eat Disord 2002;32:473–8.
100. Hepp U, Spindler A, Milos G. Eating disorder symptomatology and gender role orientation. Int J Eat Disord 2005;37:227–33.
101. Cella S, Iannaccone M, Cotrufo P. Influence of gender role orientation (masculinity versus femininity) on body satisfaction and eating attitudes in homosexuals, heterosexuals and transsexuals. Eat Weight Disord 2013;18:115–24.
102. Diemer EW, Grant JD, Munn-Chernoff MA, Patterson DA, Duncan AE. Gender identity, sexual orientation, and eating-related pathology in a national sample of college students. J Adolesc Health 2015;57:144–9.
103. Vocks S, Stahn C, Loenser K, Legenbauer T. Eating and body image disturbances in male-to-female and female-to-male transsexuals. Arch Sex Behav 2009;38:364–77.

104. Ålgars M, Alanko K, Santtila P, Sandnabba NK. Disordered eating and gender identity disorder: a qualitative study. Eat Disord 2012;20:300–11.
105. Bandini E, Fisher AD, Castellini G, Lo Sauro C, Lelli L, Meriggiola MC, Casale H, Benni L, Ferruccio N, Faravelli C, Dettore D, Maggi M, Ricca V. Gender identity disorder and eating disorders: similarities and differences in terms of body uneasiness. J Sex Med 2013;10:1012–23.
106. Fisher AD, Castellini G, Bandini E, Casale H, Fanni E, Benni L, Ferruccio N, Meriggiola MC, Manieri C, Gualerzi A, Jannini E, Oppo A, Ricca V, Maggi M, Rellini AH. Cross-sex hormonal treatment and body uneasiness in individuals with gender dysphoria. J Sex Med 2014;11:709–19.
107. Smolak L, Murnen SK. Meta-analytic examination of the relationship between child sexual abuse and eating disorders. Int J Eat Disord 2002;31:136–50.
108. Klump KL. Puberty as a critical risk period for eating disorders: a review of human and animal studies. Horm Behav 2013;64:399–410.
109. Killen JD, Hayward C, Litt I, Hammer LD, Wilson DM, Miner B, Taylor CB, Varady A, Shisslak C. Is puberty a risk factor for eating disorders? Am J Dis Child 1992;146:323–5.
110. Koff E, Rierdan J. Advanced pubertal development and eating disturbance in early adolescent girls. J Adolesc Health 1993;14:433–99.
111. Johnson-Sabine E, Wood K, Patton G, Mann A, Wakeling A. Abnormal eating in London schoolgirls: A prospective epidemiological study: factors associated with abnormal response on screening questionnaires. Psychol Med 1988;18:615–22.
112. Leon GR, Fulkerson JA, Perry CL, Cudeck R. Personality and behavioral vulnerabilities associated with risk status for eating disorders in adolescent girls. J Abnorm Psychol 1993;102: 438–44.
113. Culbert KM, Burt SA, McGue M, Iacono WG, Klump KL. Puberty and the genetic diathesis of disordered eating attitudes and behaviors. J Abnorm Psychol 2009;118:788–96.
114. Favaro A, Ferrara S, Santonastaso P. The spectrum of eating disorders in young women: a prevalence study in a general population sample. Psychosom Med 2003;65:701–8.
115. Raboch J. Sexual development and life of psychiatric female patients. Arch Sex Behav 1986;15:341–53.
116. Brooks-Gunn J, Warren MP. Mother-daughter differences in menarcheal age in adolescent girls attending national dance company schools and non-dancers. Ann Hum Biol 1988;15: 35–43.
117. Beumont P, Abraham S, Simson K. The psychosexual histories of adolescent girls and young women with anorexia nervosa. Psychol Med 1981;11:131–40.
118. Jappe LM, Gardner RM. Body-image perception and dissatisfaction throughout phases of the female menstrual cycle. Percept Mot Skills 2009;108:74–80.
119. Racine SE, Culbert KM, Keel PK, Sisk CL, Burt SA, Klump KL. Differential associations between ovarian hormones and disordered eating symptoms across the menstrual cycle in women. Int J Eat Disord 2012;45:333–44.
120. Teixeira AL, Fernandes JV, Maques FA, Lacio ML, Dias MR. Influence of different phases of menstrual cycle on flexibility of young women. Revista Brasileira de Medicina do Esporte 2012;18:361–4.
121. McCabe MP, Ricciardelli LA. A longitudinal study of pubertal timing and extreme body change behaviors among adolescent boys and girls. Adolescence 2004 Spring;39:145–66.
122. Michaud PA, Suris JC, Deppen A. Gender-related psychological and behavioural correlates of pubertal timing in a national sample of Swiss adolescents. Mol Cell Endocrinol 2006; 254–255:172–8.
123. Petersen AC. Psychological and social issues during adolescence. Depression and body image problems in adolescence. Womens Health Issues 1994;4:63–5.
124. Siegel JM, Yancey AK, Aneshensel CS, Schuler R. Body image, perceived pubertal timing, and adolescent mental health. J Adolesc Health 1999;25:155–65.
125. Suka M, Sugimori H, Yoshida K, Kanayama H, Sekine M, Yamagami T, Kagamimori S. Body image and body satisfaction play important roles in the path to dieting behavior in Japanese preadolescents: The Toyama birth cohort study. Environ Health Prev Med 2005;10:324–30.
126. O'Dea JA, Abraham S. Onset of disordered eating attitudes and behaviors in early adolescence: interplay of pubertal status, gender, weight, and age. Adolescence 1999;34:671–9.
127. O'Dea JA, Abraham S. Association between self-concept and body weight, gender, and pubertal development among male and female adolescents. Adolescence 1999;34:69–79.
128. Muris P, Meesters C, van de Blom W, Mayer B. Biological, psychological, and sociocultural correlates of body change strategies and eating problems in adolescent boys and girls. Eat Behav 2005;6:11–22.
129. Hudson JI, Hiripi E, Pope HG Jr, Kessler RC. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. Biol Psychiatry 2007;61:348–58.
130. Diagnostic and statistical manual of mental disorders. IV ed. Arlington, VA: American Psychiatric Association, 2000.
131. Yu WH, Walczewska A, Karanth S, McCann SM. Nitric oxide mediates leptin-induced luteinizing hormone-releasing hormone (LHRH) and LHRH and leptin-induced LH release from the pituitary gland. Endocrinology 1997;138: 5055–8.
132. Karlsson C, Lindell K, Svensson E, Bergh C, Lind P, Billig H, Carlsson LM, Carlsson B. Expression of functional leptin receptors in the human ovary. J Clin Endocrinol Metab 1997;82:4144–8.
133. Cunningham MJ, Clifton DK, Steiner RA. Leptin's actions on the reproductive axis: perspectives and mechanisms. Biol Reprod 1999;60:216–22.
134. Jin L, Burguera BG, Couce ME, Scheithauer BW, Lamsan J, Eberhardt NL, Kulig E, Lloyd RV. Leptin and leptin receptor expression in normal and neoplastic human pituitary: evidence of a regulatory role for leptin on pituitary cell proliferation. J Clin Endocrinol Metab 1999;84:2903–11.
135. Brannian JD, Zhao Y, McElroy M. Leptin inhibits gonadotrophinstimulated granulosa cell progesterone production by antagonizing insulin action. Hum Reprod 1999;14:1445–8.
136. Agarwal SK, Vogel K, Weitsman SR, Magoffin DA. Leptin antagonizes the insulin-like growth factor-I augmentation of steroidogenesis in granulosa and theca cells of the human ovary. J Clin Endocrinol Metab 1999;84:1072–6.
137. Barkan D, Jia H, Dantes A, Vardimon L, Amsterdam A, Rubinstein M. Leptin modulates the glucocorticoid-induced ovarian steroidogenesis. Endocrinology 1999;140:1731–8.

Q11: Please supply authors name for ref. 130

138. Kitawaki J, Kusuki I, Koshiba H, Tsukamoto K, Honjo H. Leptin directly stimulates aromatase activity in human luteinized granulosa cells. Mol Hum Reprod 1999;5:708–13.
139. Moschos S, Chan JL, Mantzoros CS. Leptin and reproduction: a review. Fertil Steril 2002;77:433–44.
140. Schubring C, Englaro P, Siebler T, Blum WF, Demirakca T, Kratzsch J, Kiess W. Longitudinal analysis of maternal serum leptin levels during pregnancy, at birth and up to six weeks after birth: relation to body mass index, skinfolds, sex steroids and umbilical cord blood leptin levels. Horm Res 1998;50:276–83.
141. Grinspoon S, Gulick T, Askari H, Landt M, Lee K, Anderson E, Ma Z, Vignati L, Bowsher R, Herzog D, Klibanski A. Serum leptin levels in women with anorexia nervosa. J Clin Endocrinol Metab 1996;81:3861–3.
142. Mantzoros C, Flier JS, Lesem MD, Brewerton TD, Jimerson DC. Cerebrospinal fluid leptin in anorexia nervosa: correlation with nutritional status and potential role in resistance to weight gain. J Clin Endocrinol Metab 1997;82:1845–51.
143. Ballauff A, Ziegler A, Emons G, Sturm G, Blum WF, Remschmidt H, Hebebrand J. Serum leptin and gonadotropin levels in patients with anorexia nervosa during weight gain. Mol Psychiatry 1999;4:71–5.
144. Weimann E, Blum WF, Witzel C, Schwidergall S, Bohles HJ. Hypoleptinemia in female and male elite gymnasts. Eur J Clin Invest 1999;29:853–60.
145. Pinelli G, Tagliabue A. Nutrition and fertility. Minerva Gastroenterol Dietol 2007;53:375–82.
146. Harden KP, Kretsch N, Moore SR, Mendle J. Descriptive review: hormonal influences on risk for eating disorder symptoms during puberty and adolescence. Int J Eat Disord 2014;47:718–26.
147. Linna MS, Raevuori A, Haukka J, Suvisaari JM, Suokas JT, Gissler M. Reproductive health outcomes in eating disorders. Int J Eat Disord 2013;46:826–33.
148. Carpenter SE. Psychosocial menstrual disorders: stress, exercise and diet's effect on the menstrual cycle. Curr Opin Obstet Gynecol 1994;6:536–9.
149. Reid RL, Van Vugt DA. Weight-related changes in reproductive function. Fertil Steril 1987;48:905–13.
150. Schweiger U. Menstrual function and luted-phase deficiency in relation to weight changes and dieting. Clh Obstet Gynecol 1991;34:191–7.
151. Allouche J, Bennet A, Barbe P, Plantavid M, Caron P, Louvet JP. LH pulsatility and in vitro bioactivity in women with anorexia nervosa-related hypothalamic amenorrhea. Actu Endocrinol 1991;125:614–20.
152. Treasure J. The biochemical and hormonal sequelae of the eating disorders. Br J Hosp Med 1987;37:301–3.
153. Golden NH, Shenker IR. Amenorrhea in anorexia nervosa. Neuroendocrine control of hypothalamic dysfunction. Int J Eating Disord 1994;16:53.
154. Danziger Y, Mukamel M, Zeharia A, Dinari G, Mimouni M. Stunting of growth in anorexia nervosa during the prepubertal and pubertal Deriod. Isr J Med Sci 1994;30:581–4.
155. Katz MG, Vollenhoven B. The reproductive endocrine consequences of anorexia nervosa. BJOG 2000;107:707–13.
156. Wassif WS, Ross AR. Steroid metabolism and excretion in anorexia nervosa. Vitam Horm 2013;92:125–40.
157. Liang KY, Meg Tseng MC. Impulsive behaviors in female patients with eating disorders in a university hospital in northern Taiwan. J Formos Med Assoc 2011;110:607–10.
158. Favaro A, Ferrara S, Santonastaso P. Self-injurious behavior in a community sample of young women: relationship with childhood abuse and other types of self-damaging behaviors. J Clin Psychiatry 2007;68:122–31.
159. Favaro A, Santonastaso P, Monteleone P, Bellodi L, Mauri M, Rotondo A, Erzegovesi S, Maj M. Self-injurious behavior and attempted suicide in purging bulimia nervosa: associations with psychiatric comorbidity. J Affect Disord 2008;105: 285–9.
160. Corstorphine E, Waller G, Lawson R, Ganis C. Trauma and multi-impulsivity in the eating disorders. Eat Behav 2007;8:23–30.
161. Lockwood R, Lawson R, Waller G. Compulsive features in the eating disorders: a role for trauma? J Nerv Ment Dis 2004;192:247–9.
162. Favaro A, Santonastaso P. Self-injurious behavior in anorexia nervosa. J Nerv Ment Dis 2000;188:537–42.
163. Irving L, McClusky-Fawcett K, Thissen D. Sexual attitudes and behavior of bulimic women: a preliminary investigation. J Youth Adolesc 1990;19:395–411.
164. Hayes RD, Dennerstein L, Bennett CM, Koochaki PE, Leiblum SR, Graziottin A. Relationship between hypoactive sexual desire disorder and aging. Fertil Steril 2007;87: 107–12.
165. Ackard DM, Fedio G, Neumark-Sztainer D, Britt HR. Factors associated with disordered eating among sexually active adolescent males: gender and number of sexual partners. Psychosom Med 2008;70:232–8.
166. Westen D, Harnden-Fischer J. Personality profiles in eating disorders: Rethinking the distinction between axis I and axis II. Am J Psychiatry 2001;158:547–62.
167. Vogeltanz-Holm ND, Wonderlich SA, Lewis BA, Wilsnack SC, Harris TR, Wilsnack RW, Kristjanson AF. Longitudinal predictors of binge eating, intense dieting, and weight concerns in a national sample of women. Behav Ther 2000;31:221–35.
168. Hay P. The epidemiology of eating disorder behaviors: An Australian community-based survey. Int J Eat Disord 1998;23:371–82.
169. Keel PK, Baxter MG, Heatherton TF, Joiner TE Jr. A 20-year longitudinal study of body weight, dieting, and eating disorder symptoms. J Abnorm Psychol 2007;116:422–32.
170. Tuiten A, Panhuysen G, Everaerd W, Koppeschaar H, Krabbe P, Zelissen P. The paradoxical nature of sexuality in anorexia nervosa. J Sex Marital Ther 1993;19:259–75.
171. Rothschild BS, Fagan P, Woodall C, Andersen AE. Sexual functioning of female eating disordered patients. Int J Eat Disord 1991;10:389–94.
172. Grilo CM, Masheb RM. Childhood psychological, physical, and sexual maltreatment in outpatients with binge eating disorder: frequency and associations with gender, obesity, and eating-related psychopathology. Obes Res 2001;9:320–5.
173. Vall E, Wade TD. Predictors of treatment outcome in individuals with eating disorders: A systematic review and meta-analysis. Int J Eat Disord 2015;48:946–71.
174. Löwe B, Zipfel S, Buchholz C, Dupont Y, Reas DL, Herzog W. Long-term outcome of anorexia nervosa in a prospective 21-year follow-up study. Psychol Med 2001;31:881–90.
175. Rosen JC. Assessment and treatment of body image disturbance. In: Brownell KD, Fairburn CG, editors. Eating disorders and obesity. A comprehensive handbook. New York, NY: The Guilford Press, 1995:369–73.

176. Pujols Y, Cindy MM, Seal Brooke N. The association between sexual satisfaction and body image in women. J Sex Med 2010;7:905–16.
177. Holmes T, Chamberlin P, Young M. Relations of exercise to body image and sexual desirability among a sample of university students. Psychol Rep 1994;74:920–2.
178. Kinzl JF, Trefalt E, Fiala M, Hotter A, Biebl W, Aigner F. Partnership, sexuality, and sexual disorders in morbidly obese women: consequences of weight loss after gastric banding. Obes Surg 2001;11:455–8.
179. Garner DM, Vitousek K, Pike KM. Cognitive behavioral therapy for anorexia nervosa. In: Garner DM, Garfinkel PE, editors. Handbook of treatment for eating disorders. 2nd edition. Chichester: Wiley, 1997.
180. Arnow B, Kenardy J, Agras WS. The emotional eating scale: The development of a measure to assess coping with negative affect by eating. Int J Eat Disord 1995;18:79–90.
181. Carvalho J, Nobre P. Predictors of women's sexual desire: The role of psychopathology, cognitive-emotional determinants, relationship dimensions, and medical factors. J Sex Med 2010;7:928–37.
182. Nobre PJ, Pinto-Gouveia J. Emotions during sexual activity: differences between sexually functional and dysfunctional men and women. Arch Sex Behav 2006;35:8–15.
183. Derogatis LR, Lipman RS, Covi L. SCL-90. An outpatient psychiatric rating scale-preliminary report. Psychopharmacol Bull 1973;9:13–28.
184. Kolotkin RL, Binks M, Crosby RD, Østbye T, Gress RE, Adams TD. Obesity and sexual quality of life. Obesity 2006;14:472–9.
185. Esposito K, Ciotola M, Giugliano F, Bisogni C, Schisano B, Autorino R, Cobellis L, De Sio M, Colacurci N, Giugliano D. Association of body weight with sexual function in women. Int J Impot Res 2007;19:353–7.
186. Kolotkin RL, Zunker C, Østbye T. Sexual functioning and obesity: a review. Obesity 2012;20:2325–33.
187. Reis LO. Sexual functioning in men who underwent bariatric surgery: far beyond sex hormones. Surg Obes Relat Dis 2015;11:651–2.
188. Sarwer DB, Spitzer JC, Wadden TA, Rosen RC, Mitchell JE, Lancaster K, Courcoulas A, Gourash W, Christian NJ. Sexual functioning and sex hormones in men who underwent bariatric surgery. Surg Obes Relat Dis 2015;11:643–51.
189. Sarwer DB, Spitzer JC, Wadden TA, Mitchell JE, Lancaster K, Courcoulas A, Gourash W, Rosen RC, Christian NJ. Changes in sexual functioning and sex hormone levels in women following bariatric surgery. JAMA Surg 2014;149:26–33.
190. Borges R, Temido P, Sousa L, Azinhais P, Conceição P, Pereira B, Leão R, Retroz E, Brandão A, Cristo L, Sobral F. Metabolic syndrome and sexual (dys)function. J Sex Med 2009;6:2958–75.
191. Chen LP, Murad MH, Paras ML, Colbenson KM, Sattler AL, Goranson EN, Elamin MB, Seime RJ, Shinozaki G, Prokop LJ, Zirakzadeh A. Sexual abuse and lifetime diagnosis of psychiatric disorders: systematic review and meta-analysis. Mayo Clin Proc 2010;85:618–29.
192. Wilson DR. Health consequences of childhood sexual abuse. Perspect Psychiatr Care 2010;46:56–64.
193. Danielson CK, Holmes MM. Adolescent sexual assault: an update of the literature. Curr Opin Obstet Gynecol 2004;16:383–8.
194. Stice E. Risk and maintenance factors for eating pathology: a meta-analytic review. Psychol Bull 2002;128:825–48.
195. Kendall-Tackett K. The health effects of childhood abuse: four pathways by which abuse can influence health. Child Abuse Negl 2002;26:715–29.
196. Finkelhor D, Hotaling GT, Lewis IA, Smith C. Sexual abuse and its relationship to later sexual satisfaction, marital status, religion, and attitudes. J Interpers Violence 1989;4:379–99.
197. Wonderlich SA, Brewerton TD, Jocic Z, Danskey BS, Abbott DW. Relationship of childhood sexual abuse and eating disorders. J AmAcad Child Adolesc Psychiatry 1997;36:1107–15.
198. Sanci L, Coffey C, Olsson C, Reid S, Carlin JB, Patton G. Childhood sexual abuse and eating disorders in females: findings from the Victorian Adolescent Health Cohort Study. Arch Pediatr Adolesc Med 2008;162:261–7.
199. Zlotnick C, Johnson J, Kohn R, Vincente B, Rioseco P, Saldivia S. Childhood trauma, trauma in adulthood, and psychiatric diagnoses: results from a community sample. Comprehensive Psychiatry 2008;49:163–9.
200. Copeland WE, Keeler G, Angold A, Costello EJ. Traumatic events and posttraumatic stress in childhood. Arch Gen Psychiatry 2007;64:577–84.
201. Faravelli C, Gorini Amedei S, Rotella F, Faravelli L, Palla A, Consoli G, Ricca V, Batini S, Lo Sauro C, Spiti A, Catena Dell'osso M. Childhood traumata, Dexamethasone Suppression Test and psychiatric symptoms: a trans-diagnostic approach. Psychol Med 2010;40:2037–48.
202. Cutajar MC, Mullen PE, Ogloff JR, Thomas SD, Wells DL, Spataro J. Psychopathology in a large cohort of sexually abused children followed up to 43 years. Child Abuse Negl 2010;34:813–22.
203. Jonas S, Bebbington P, McManus S, Meltzer H, Jenkins R, Kuipers E, Cooper C, King M, Brugha T. Sexual abuse and psychiatric disorder in England: results from the 2007 Adult Psychiatric Morbidity Survey. Psychol Med 2011;41:709–19.
204. Castellini G, Maggi M, Ricca V. Childhood sexual abuse and psychopathology. In: Corona G, Jannini EA, Maggi M, editors. Emotional, physical and sexual abuse. Impact in children and social minorities. Eds Springer International Publishing, 2014.
205. Vanderlinden J, Vandereycken W. Trauma, dissociation, and impulse dyscontrol in eating disorders. Eds Brunner Mazel, 1997.
206. McCrory E, DeBrito SA, Viding E. Research review: the neurobiology and genetics of maltreatment and adversity. J Child Psychol Psychiatry 2010;51:1079–95.
207. Dunn EC, McLaughlin KA, Slopen N, Rosand J, Smoller JW. Developmental timing of child maltreatment and symptoms of depression and suicidal ideation in young adulthood: results from the National Longitudinal Study of Adolescent Health. Depress Anxiety 2013;30:955–64.
208. Cicchetti D. How research on child maltreatment has informed the study of child maltreatment: Perspectives from developmental psychopathology. In Cicchetti D, Carlson V, editors. Child maltreatment: theory and research on the causes and consequences of child abuse and neglect. New York, NY: Cambridge University Press, 1989: 377–431.

Q12: Please supply publisher location for refs. 204, 205

209. Seng JS, Low LK, Sparbel KJ, Killion C. Abuse-related post-traumatic stress during the childbearing year. J Adv Nurs 2004;46:604–13.
210. Ackard DM, Neumark-Sztainer D. Multiple sexual victimizations among adolescent boys and girls: prevalence and associations with eating behaviors and psychological health. J Child Sex Abus 2003;12:17–37.
211. Chou KL. Childhood sexual abuse and psychiatric disorders in middle-aged and older adults: evidence from the 2007 Adult Psychiatric Morbidity Survey. J Clin Psychiatry 2012;73:1365–71.
212. Favaro A, Tenconi E, Santonastaso P. The interaction between perinatal factors and childhood abuse in the risk of developing anorexia nervosa. Psychol Med 2010;40:657–65.
213. Rellini AH, Hamilton LD, Delville Y, Meston CM. The cortisol response during physiological sexual arousal in adult women with a history of childhood sexual abuse. J Trauma Stress 2009;22:557–65.
214. Vanderlinden J, Van Dyck R, Vandereycken W, Vertommen H. Dissociation and traumatic experiences in the general population of the Netherlands. Hosp Community Psychiatry 1993;44:786–8.
215. La Mela C, Maglietta M, Castellini G, Amoroso L, Lucarelli S. Dissociation in eating disorders: relationship between dissociative experiences and binge-eating episodes. Compr Psychiatry 2010;51:393–400.
216. Schloredt KA, Heiman JR. Perceptions of sexuality as related to sexual functioning and sexual risk in women with different types of childhood abuse histories. J Trauma Stress 2003;16:275–84.
217. Rellini AH, Meston CM. Psychophysiological sexual arousal in women with a history of childhood sexual abuse. J Sex Marital Ther 2006;32:1–18.
218. Rellini AH, Meston CM. Sexual function and satisfaction in adults based on the definition of child sexual abuse. J Sex Med 2007;4:1312–21.
219. Preti A, Incani E, Camboni MV, Petretto DR, Masala C Lockwood R, Lawson R, Waller G. Compulsive features in the eating disorders: a role for trauma? J Nerv Ment Dis 2004;192:247–9.
220. Burns EE, Fischer S, Jackson JL, Harding HG. Deficits in emotion regulation mediate the relationship between childhood abuse and later eating disorder symptoms. Child Abuse Negl 2012;36:32–9.
221. Walsh J, Burns F. Sexual maturation and control issues among sexually abused and non-abused anorexia patients. Br J Clin Psychol 2000;39:307–10.
222. Chivers M, Seto M, Lalumiere M, Laan E, Grimbos T. Agreement of genital and subjective measures of sexual arousal: Preliminary results of a meta-analysis. Paper presented at the annual meeting of the International Academy of Sex Research, Ottawa, Canada, 2005.
223. McManus F, Waller G. A functional analysis of binge-eating. Clin Psychol Rev 1995;15:845–63.
224. Heatherton TF, Baumeister RF. Binge eating as escape from selfawareness. Psychol Bull 1991;110:86–108.
225. Meyer C, Waller G, Waters A. Emotional states and bulimic psychopathology. In: Hoek HW, Treasure JL, Katzman MA, editors. Neurobiology in the treatment of eating disorders. Chichester (NY): Wiley, 1998:265–80.
226. Siever MD. Sexual orientation and gender as factors in socioculturally acquired vulnerability to body dissatisfaction and eating disorders. J Consult Clin Psychol 1994;62:252–60.
227. Boisvert JA, Harrell WA. Homosexuality as a risk factor for eating disorder symptomatology in men. J Men's Stud 2009;17:210–25.
228. Herzog DB, Norman DK, Gordon C, Pepose M. Sexual conflict and eating disorders in 27 males. Am J Psychiatry 1984;141:989–90.
229. Meyer IH. Prejudice, social stress, and mental health in lesbian, gay, and bisexual populations: conceptual issues and research evidence. Psychological Bulletin 2003;129:674–97.
230. Feldman MB, Meyer IH. Comorbidity and age of onset of eating disorders in gay men, lesbians, and bisexuals. Psychiatry Res 2010;180:126–31.
231. Tiggemann M, Martins Y, Kirkbride A. Oh to be lean and muscular: body image ideals in gay and heterosexual men. Psychol Men Masculin 2007;8:15–24.
232. Blashill AJ. Gender roles, eating pathology, and body dissatisfaction in men: a meta-analysis. Body Image 2011;8:1–11.
233. Silberstein LR, Mishkind ME, Striegel-Moore RH, Timko C, Rodin J. Men and their bodies: a comparison of homosexual and heterosexual men. Psychosom Med 1989;51:337–46.
234. Boehmer U, Bowen DJ, Bauer GR. Overweight and obesity in sexualminority women: Evidence from population-based data. Am J Public Health 2007;97:1134–40.
235. Surgenor LJ, Fear JL. Eating disorder in a transgendered patient: a case report. Int J Eat Disord 1998;24:449–52.
236. Bradford J, Reisner SL, Honnold JA, Xavier J. Experiences of transgender-related discrimination and implications for health: results from the Virginia Transgender Health Initiative Study. Am J Public Health 2013;103:1820–9.
237. Cole CM, O'Boyle M, Emory LE, Meyer WJ 3rd. Comorbidity of GID and other major psychiatric diagnoses. Arch Sex Behav 1997;26:13–26.
238. Hoshiai M, Matsumoto Y, Sato T, Ohnishi M, Okabe N, Kishimoto Y, Terada S, Kuroda S. Psychiatric comorbidity among patients with gender identity disorder. Psychiatry Clin Neurosci 2010;64:514–9.
239. Gómez-Gil E, Trilla A, Salamero M, Godás T, Valdés M. Sociodemographic, clinical, and psychiatric characteristics of transsexuals from Spain. Arch Sex Behav 2009;38:378–92.
240. Haraldsen IR, Dahl AA. Symptom profiles of GID patients of transsexual type compared to patients with personality disorders and healthy adults. Acta Psychiatr Scand 2000;102:276–81.
241. Matsumoto Y, Sato T, Ohnishi M, Kishimoto Y, Terada S, Kuroda S. Stress coping strategies of patients with gender identity disorder. Psychiatry Clin Neurosci 2009;63:715–20.
242. Nuttbrock L, Hwahng S, Bockting W, Rosenblum A, Mason M, Macri M, Becker J. Psychiatric impact of gender-related abuse across the life course of male-to-female transgender persons. J Sex Res 2010;47:12–23.
243. Factor RJ, Rothblum ED. A study of transgender adults and their non-transgender siblings on demographic characteristics, social support, and experiences of violence. J LGBT Health Res 2007;3:11–30.

244. Gooren L. The biology of human psychosexual differentiation. Horm Behav 2006;50:589–601.
245. Gooren L. Clinical practice. Care of transsexual persons. N Engl J Med 2011;364:1251–7.
246. Couturier J, Pindiprolu B, Findlay S, Johnson N. Anorexia nervosa and gender dysphoria in two adolescents. Int J Eat Disord 2015;48:151–5.
247. Ewan LA, Middleman AB, Feldmann J. Treatment of anorexia nervosa in the context of transsexuality: a case report. Int J Eat Disord 2014;47:112–5.
248. Winston AP, Acharya S, Chaudhuri S, Fellowes L. Anorexia nervosa and gender identity disorder in biologic males: a report of two cases. Int J Eat Disord 2004;36:109–13.
249. Witcomb GL, Bouman WP, Brewin N, Richards C, Fernandez-Aranda F, Arcelus J. Body image dissatisfaction and eating-related psychopathology in trans individuals: a matched control study. Eur Eat Disord Rev 2015;23: 287–93.
250. Pauly LB, Lindgren TW. Body image and gender identity. J Homosex 1976;2:133–42.

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