Eating Disorders Across the Life-Span: From Menstruation to Menopause

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Introduction

In this chapter we review key issues for reproductive mental health in women with anorexia nervosa, bulimia nervosa, binge eating disorder (BED), and purging disorder. We take a developmental lens and cover four main periods across the female life-span that can impact the onset, course, and maintenance of eating disorders. First, we define eating disorders. Second, we address prenatal issues associated with eating disorders in adolescence and young adulthood including the role of estradiol for eating disorder risk, endocrine dysfunction, amenorrhea and oligomenorrhea that accompany the illness, and fertility concerns for women with eating disorders. Third, we cover perinatal issues associated with eating disorders during pregnancy including course of illness during pregnancy, pregnancy outcomes, birth outcomes, and perinatal mental health. Fourth, we discuss the postpartum issues associated with eating disorders including weight retention concerns, postpartum mood disorders, and early child feeding. Finally, we discuss midlife issues for eating disorder onset and relapse. By examining eating disorder behaviors across the life-span, we can explicate the dynamic interplay between women's physical development, eating disorder symptoms, and overall mental health.

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Eating Disorders

Although eating disorders have distinct features, they share common symptoms including the overevaluation of weight for self-worth, dysregulation in eating behavior, and obsessive ruminative thoughts about food, weight, and body shape. In particular, anorexia nervosa (AN) is marked by extremely low body weight of less than 85 % of an ideal body weight for height and age, cognitive distortions related to body shape and weight perception, and severe food restriction. Bulimia nervosa (BN) is marked by periods of binge eating in which a large amount of food is consumed with a perceived sense of loss of control over eating. Binge eating is followed by inappropriate behavior to compensate for the amount of food consumed including purging, excessive exercise, and fasting (American Psychiatric Association, 2013).

The most recent version of the *Diagnostic and Statistical Manual* (DSM-5) also witnessed the addition of a new eating disorder diagnosis: BED. BED is marked by repeated binge eating episodes combined with loss of control but no inappropriate compensatory behavior in response. In addition, although it is not recognized in DSM-5, many researchers also consider purging disorder (PD) to be a possible diagnostic category worthy of further study. PD is marked by purging episodes, but unlike purging in BN, purging does not follow binge eating episodes with a loss of control but instead accompanies average-size meals or snacks (Keel, Haedt, & Edler, 2005). In the best epidemiological studies of eating disorder prevalence, approximately 1 % of women will suffer from AN, 1.5 % will suffer from BN, and 1 % will suffer from PD over their life. BED is by far the most prevalent eating disorder with a lifetime prevalence of 3.5 % for women (Hudson, Hiripi, Pope, & Kessler, 2007).

The medical and psychological consequences of AN, BN, BED, and PD can be both debilitating and life threatening. AN, in particular, has the highest mortality of any psychiatric illness (Berkman, Lohr, & Bulik, 2007; Birmingham, Su, Hlynsky, Goldner, & Gao, 2005; Harris & Barraclough, 1998; Millar et al., 2005; Papadopoulos, Ekbom, Brandt, & Ekselius, 2009; Sullivan, 1995). The psychiatric correlates of eating disorders include depression, anxiety, social withdrawal, and heightened self-consciousness (Birmingham et al., 2005; Fernandez-Aranda et al., 2007; Godart, Flament, Perdereau, & Jeammet, 2002; Halmi et al., 1991; Javaras et al., 2008; Kaplan, 1993; Katzman, 2005; Kaye et al., 2004; Keel, Mitchell, Miller, Davis, & Crow, 1999; Millar et al., 2005; Mitchell, Specker, & de Zwaan, 1991; Papadopoulos et al., 2009; Reichborn-Kjennerud, Bulik, Sullivan, Tambs, & Harris, 2004; Sharp & Freeman, 1993; Sullivan, 1995; Zipfel, Lowe, Reas, Deter, & Herzog, 2000). The medical sequelae include electrolyte imbalances, cardiac arrhythmias, osteoporosis and osteopenia, tooth decay, gastroesophageal reflux disease, and gastric rupture (Brown & Mehler, 2013; Brownell & Fairburn, 1995; Bulik & Reichborn-Kjennerud, 2003; Katzman, 2005). Medical complications such as osteoporosis can persist throughout life even after weight restoration and recovery (Rigotti, Neer, Skates, Herzog, & Nussbaum, 1991). Most notably for reproductive mental health, eating disorders are also associated with significant disruptions to the neuroendocrine system (Abraham, Pettigrew, Boyd, Russell, & Taylor, 2005; Andersen & Ryan, 2009;

Pinheiro et al., 2007; Watson & Andersen, 2003). The impact of malnutrition associated with eating disorders on reproductive hormones is discussed throughout the chapter with a focus on the unique impact at each life stage.

Etiology

For many years eating disorders were conceptualized primarily from a sociocultural model, with much of the blame unfairly directed towards families and parenting as being the primary causal factor for the development of eating disorders. However, over the past 30 years, an enhanced understanding of the biology of eating disorders has resulted in the identification of genetic, neurological, and environmental risk factors (Bulik et al., 2006; Strober, Freeman, Lampert, Diamond, & Kaye, 2000). Thus, the current view maintains a biopsychosocial model of eating disorder etiology in which the development of eating psychopathology is due to biological, psychological, and social factors (Keel, Leon, & Fulkerson, 2001; Rutter, Moffitt, & Caspi, 2006).

Eating disorders run in families due to genetic factors rather than due to parenting behaviors explained by shared environment. Heritability estimates range from 33 to 84 % for AN, 28 to 83 % for BN, and 41 to 57 % for BED, with the remaining variance typically attributable to unique environment factors (Bulik & Tozzi, 2004; Javaras et al., 2008; Thornton, Mazzeo, & Bulik, 2011). No studies have yet been published on the heritability of PD, although self-induced vomiting is highly heritable (72 %; Sullivan, Bulik, & Kendler, 1998). In addition, research on the neurocircuitry of eating disorders has demonstrated that eating disorders are accompanied by altered reward mechanisms including possible anterior ventral striatal pathway dysfunction and altered gustatory processing in the anterior insula (Kaye, Wagner, Fudge, & Paulus, 2011).

Although they can occur throughout the life-span, these disorders primarily begin in mid to late adolescence with an average age of onset between 15 and 22 years of age (Hudson et al., 2007). Puberty and its associated neuroendocrine changes, in particular, may trigger the expression of an eating disorder for those with a genetic predisposition. Cross-sectional and longitudinal studies comparing early-adolescent to middle- and late-adolescent twins have demonstrated a negligible heritability of disordered eating behaviors and weight and shape concerns in younger twins, but higher heritability with the onset of puberty (Baker et al., 2009; Klump et al., 2010; Klump, Keel, Sisk, & Burt, 2010). Although the biological mechanisms that lead to the increase of heritability in puberty are unknown, twin studies have suggested a potential role for estradiol (Klump, Burt et al., 2010; Klump, Keel et al., 2010). However, in some animal models ovarian hormones were not found to contribute to binge eating behavior (Klump, Suisman, Culbert, Kashy, & Sisk, 2011).

Social and psychological risk factors for the development of eating disorders include life trauma (Bulik, Prescott, & Kendler, 2001; Kendler et al., 2000), childhood anxiety (Raney et al., 2008), harm avoidance and perfectionism (Fassino, Amianto, Gramaglia, Facchini, & Abbate Daga, 2004), difficulties with set shifting (Steinglass, Walsh, & Stern, 2006; Tchanturia et al., 2004), impulsivity

(Racine, Culbert, Larson, & Klump, 2009; Wonderlich, Connolly, & Stice, 2004), and problems with emotion regulation (Harrison, Tchanturia, & Treasure, 2010; Heatherton & Baumeister, 1991). However, all biological, psychological, social risk, and protective factors for eating disorders are best conceptualized as probabilistic rather than deterministic (Rutter et al., 2006).

Of special interest for reproductive mental health, environmental risk factors experienced at birth such as preterm birth or neonatal immaturity may also place children and adults at risk for later eating disorders. Research on "fetal programming" posits that environmental influences on the fetal environment could lead to long-term, and perhaps permanent, effects on the structure and functioning of organs leading to risk for the long-term physical and mental health of offspring (Barker, 2004; Gluckman & Hanson, 2004; Schlotz & Phillips, 2009).

Bulik and colleagues have hypothesized that maternal eating disorders during pregnancy lead to a cycle of risk for eating disorder development in subsequent generations (Bulik, Reba, Siega-Riz, & Reichborn-Kjennerud, 2005). In addition to genetic risk factors, maternal eating disorders during pregnancy could lead to fetal undernutrition, overexposure to stress hormones, labor and delivery complications, and low birth weight (Micali & Treasure, 2009). These experiences during the sensitive fetal period could, in turn, place the children of mothers with eating disorders at increased risk for diminished cognitive function, behavioral problems, stress reactivity, and psychopathology throughout their life-span (Schlotz & Phillips, 2009). In fact, with data from large medical birth registries, research has demonstrated that pregnancy and obstetric complications such as gestational diabetes, maternal anemia, and placental infarction are significant risk factors for the development of eating disorders (Favaro, Tenconi, & Santonastaso, 2006). Thus, the combination of genetic risk for eating disorders and increased risk of perinatal malnutrition and birth complications associated with maternal eating disorders may lead to a cycle of long-term and trans-generational risk.

Antenatal Eating Disorders

Prior to conception, there are many ways in which eating disorders can, and do, affect a woman's reproductive viability, her attitude towards pregnancy, and ultimately the outcome of a potential pregnancy. This section addresses some of the antenatal changes and consequences that can result as a consequence of eating disorder behaviors and symptoms.

Amenorrhea/Oligomenorrhea

Although commonly associated with the restrictive subtype of anorexia, amenorrhea or the absence of regular menstrual cycles can also be present in individuals with BN and BED (Abraham et al., 2005; Andersen & Ryan, 2009; Ehrmann, 2005; Naessén &

Hirschberg, 2011; Pinheiro et al., 2007; Watson & Andersen, 2003). Oligomenorrhea, or the presence of menstrual periods that are infrequent or exceptionally light, can also be a consequence of most eating disorders. A survey of 241 inpatients with eating disorders illustrated the prevalence of amenorrhea/oligomenorrhea across diagnosis with 24 % of patients presenting with eating disorder not otherwise specified (EDNOS) and 18 % of patients with BN experiencing menstrual disturbance (Abraham et al., 2005). However, the cause of amenorrhea and oligomenorrhea may emanate from different origins and they have diverging trajectories for each diagnosis.

In anorexia, amenorrhea typically results from low body fat due to restricted caloric and nutrient intake (Pinheiro et al., 2007). With lower body fat content there also comes a decrease in naturally occurring levels of leptin and ghrelin, two important hormones that influence energy balance and menstruation (Andersen & Ryan, 2009; Pinelli & Tagliabue, 2007). As part of the hypothalamic-pituitary-ovarian axis, these changes influence the body's ability to meet the energy requirements needed for menstruation. Even in diagnoses of EDNOS that have AN-like symptoms, there is evidence that women struggle with amenorrhea and oligomenorrhea despite being above the 85th percentile in weight (Watson & Andersen, 2003). Therefore, factors other than weight can play a role in the advent and continuation of amenorrhea or irregular menses, including excessive exercise (Pinheiro et al., 2007).

Polycystic ovarian syndrome (PCOS) has also routinely been found to be associated with eating disorders, especially BN and BED. PCOS modifies both sex hormones and menstrual cycles significantly and has also been correlated with craving sweets and binge eating due to the disrupted hormones (Hirschberg, Naessen, Stridsberg, Bystrom, & Holtet, 2004). High androgen levels as a result of PCOS may also lead to bulimic behavior through increased cravings and diminished impulse control (Resch, Szendei, & Haasz, 2004a, 2004b). Binge eating is actually cited as a more common occurrence among individuals with oligomenorrhea, secondary to their eating disorder (Pinheiro et al., 2007).

Prenatal and Perinatal Eating Disorders

Sexual Functioning

Pregnancy in women with AN was long thought to be rare due to the psychological and psychosocial features of the disorder coupled with the endocrinological disturbances and amenorrhea associated with malnutrition. In general, women with eating disorders report higher levels of sexual dysfunction. A significant percentage of women with AN, BN, and PD report a loss of libido (75 %, 39 %, and 45.4 %, respectively), and overall women with active eating disorders report decreased sexual desire (66.9 %) and increased sexual anxiety (59.2 %; Pinheiro et al., 2010). In particular, a lower body mass index (BMI) predicts a greater loss of libido, more sexual anxiety, and fewer sexual relationships.

From a biological perspective, low body weight affects the physiological functioning of sexual organs, and changes in BMI have been directly associated with fluctuations in sexual interest (Beumont, Abraham, & Simson, 1981; Hsu, 1980; Morgan, Lacey, & Reid, 1999). From a psychological perspective, women who attain a lower BMI body may also feel more dissatisfaction with body weight and body shape and experience more distortion of body size. Thus, they may also experience more discomfort with physical contact and physical exposure, which contributes to a loss of libido and an increase in sexual anxiety. With weight restoration and recovery, women with eating disorders commonly report an increase in sexual drive, likely due to both biological and psychosocial factors (Morgan, Lacey, & Reid, 1999). Changes in sexual functioning are likely to be multifactorial in patients with eating disorders and represent a convergence of physiological and psychological factors.

Fertility

Clinically, worries about current or future fertility represent one of the most reported motivators for treatment and weight restoration in women. However, studies on fertility outcomes in women with eating disorders have been equivocal. Some have not found differences in fertility between women with histories of AN or BN when compared with controls, demonstrating that despite high levels of amenorrhea and oligomenorrhea, women with AN can become pregnant (Brinch, Isager, & Tolstrup, 1988; Bulik et al., 1999). However, others have found that women with AN have children at one-third the rate of women without an eating disorder history (Brinch et al., 1988). Moreover, there is an especially high prevalence (16–20 %) of women meeting eating disorder criteria within a fertility specialist clinic setting in comparison to the lifetime prevalence of eating disorders in women within the general population (~5–6 %; Bulik et al., 1999; Freizinger, Franko, Dacey, Okun, & Domar, 2010; Hudson et al., 2007). In a large population-based longitudinal study, women with AN were more likely to have seen a doctor for fertility concerns, taken longer than 6 months to conceive, and needed fertility treatment in order to conceive (Easter, Treasure, & Micali, 2011). Thus, at an individual clinical level, assessing whether lifetime eating disorder status will have a long-term effect on fertility is difficult, and there are no clear predictive algorithms to determine fertility during the eating disorder or after recovery.

Attitudes Towards Pregnancy

Upon becoming pregnant, many women with eating disorders and eating disorder histories struggle in adapting to this significant life transition. Women with eating disorders are more likely to report that they experienced negative feelings upon discovering that they were pregnant (Easter et al., 2011). However, by 18 weeks of gestation, there is no significant difference in their attitudes when compared to women without eating disorders. Pregnant women with eating disorders have also

been found to be two times more likely than referent women to endorse that motherhood meant giving up something important and were more likely to view motherhood as a personal sacrifice (Easter et al., 2011).

Again, the difficulty that women with eating disorders face during the transition to motherhood may be due to a combination of factors, both physical and psychological. First, women with AN have been found significantly more likely to be younger than the referent group at their first pregnancy and are perhaps not as prepared emotionally and developmentally for the challenges associated with pregnancy and parenting (Bulik et al., 2009; Micali, Treasure, & Simonoff, 2007). Second, the likelihood of an unplanned pregnancy is markedly higher for women with AN and BN than for women without eating disorders. There is a twofold increase in the risk of unplanned pregnancy for women with AN and a 30-fold increase of unplanned pregnancy for women with BN (Bulik et al., 2010; Morgan, Lacey, & Chung, 2006). Women with AN are also significantly more likely to have terminated a pregnancy at some point in their lives than women without an eating disorder (24.2 % vs. 14.6 %; Bulik et al., 2010). Although the reasons for a higher likelihood of an unplanned and terminated pregnancy are unclear, researchers speculate that it may be due to patients' belief that amenorrhea or oligomenorrhea conveys a lack of fertility and thus they do not arrange for adequate contraception under the mistaken belief that they cannot become pregnant (Bulik et al., 2010). An unplanned pregnancy may reduce the likelihood that women with eating disorders can arrange for the critical nutritional and emotional support needed to manage the demands of pregnancy and motherhood, especially early in their pregnancy.

Finally, the demands of pregnancy and motherhood represent an immense challenge for women already struggling to recover both medically and psychologically from an eating disorder (Easter et al., 2011). Pregnancy requires a radical transformation in weight, size, and shape. In general, women with eating disorders report that they worry about gestational weight gain more than women without eating disorders. Specifically, 62.9 % of those with AN, 61.5 % of those with BN, 50 % of those with PD, and 24 % of those with BED endorse being "very worried" about gestational weight gain in comparison to 6.9 % of those without eating disorders (Swann et al., 2009). They may perceive the loss of the pre-pregnancy body that accompanies motherhood as a "sacrifice" (Easter et al., 2011). Pregnant women with eating disorders may feel additional stress as they struggle to adapt to "eating for the baby" while also managing the negative emotions and cognitions that accompany an increase in gestational weight and radical changes to their shape.

Eating Disorder Course

Fortunately, despite the anticipatory worry that many women report at the beginning of their pregnancies, large population-based prospective studies have found that the most common course of eating disorders during pregnancy is remission. For full remission, rates are 78 % for PD and 34 % for BN with an additional 29 % of women with BN attaining partial remission during pregnancy (Bulik et al., 2007).

However, estimates of remission from AN are extremely difficult to obtain because there are no clear criteria to establish maternal underweight during pregnancy (Bulik et al., 2007). Cohort studies have also found that weight and shape concerns decrease during pregnancy in women with active eating disorders (Fairburn, Stein, & Jones, 1992; Micali et al., 2007). Some women with eating disorders also report believing that pregnancy could be a means to recover from their disorder because they view gestational weight gain and a larger body size as more acceptable during pregnancy than under other circumstances (Lemberg & Phillips, 1989).

Although eating disorder diagnoses most often remit during pregnancy, women with eating disorders report greater continued use of laxatives, self-induced vomiting, and higher levels of vigorous exercise (defined as greater than 1 h of moderate–vigorous activity daily) during pregnancy than women without eating disorders (Micali et al., 2007). Women with AN are also more likely to smoke during pregnancy (37.1 % compared to 9.2 % in women without eating disorders), possibly due to concerns about weight and appetite control (Bulik et al., 2009).

Notably, women with BED are most likely to continue their eating disorder behaviors during pregnancy. In a large population-based Norwegian sample (the Mother and Baby Cohort Study—MoBa), a full 61 % of women with BED continued to have binge eating episodes with a loss of control while pregnant. Moreover, incident cases of BED are more common than any other eating disorder. In MoBa, 2 % of the ~42,000 mothers in the sample developed a new diagnosis of BED with repeated binge eating episodes and loss of control over eating. Thus, pregnancy may be a window of vulnerability for binge eating (Bulik et al., 2007).

From a biological perspective, a cascade of adaptive neuroendocrine changes occur during pregnancy that can affect brain functioning, metabolism, appetite, and mood (Russell, Douglas, & Ingram, 2001). Also, from a nutritional perspective, binge eating episodes often immediately follow a period of food restriction (Hagan et al., 2002). Women who attempt to eat with the same frequency and amount as prior to their pregnancy may unwittingly set themselves up for periods of intense hunger and as a result trigger a dysregulated eating pattern (Bulik et al., 2007).

From a psychological perspective, the emotional, financial, and social stress of pregnancy and anticipated motherhood may also trigger binge eating episodes. In the MoBa cohort, the mothers at greatest risk for incident BED were also more likely to have fewer years of education, more previous pregnancies, and lower income (Bulik et al., 2007).

New onset of AN, BN, and PD during pregnancy is extremely rare. However, in AN, there is also evidence that of those who had experienced a recovery from AN before becoming pregnant, approximately a third (33 %) relapsed in their eating disorder behavior during pregnancy (Koubaa, Hallstrom, Lindholm, & Hirschberg, 2005). In addition, some women with a history of eating disorders reported that they experienced an increase in overall weight and shape concerns during pregnancy (Micali et al., 2007). Thus, for women with histories of AN and BN, pregnancy can also be a sensitive period for the reemergence of symptoms.

Hyperemesis Gravidarum

Some have questioned whether self-induced vomiting prior to pregnancy could be a risk factor for hyperemesis gravidarum during pregnancy. Indeed, women with purging-type eating disorders are more likely to report both nausea and vomiting during pregnancy than those without an eating disorder (Torgersen et al., 2008), and some have also found an increased risk of hyperemesis gravidarum (Koubaa et al., 2005). Underweight pregnant women are more likely to use antiemetic drugs during their pregnancy and are also more likely to be hospitalized for the weight loss and electrolyte dysfunction associated with hyperemesis (Cedergren, Brynhildsen, Josefsson, Sydsjo, & Sydsjo, 2008). However, the mechanism predisposing women with pre-pregnancy vomiting and underweight to hyperemesis is unclear. Some women with pre-pregnancy eating disorders may attempt to hide their ongoing struggle with purging behaviors under the guise of hyperemesis (Lingam & McCluskey, 1996). However, neurobiological mechanisms and/or automatic learned behaviors could also contribute to the risk of both hyperemesis and continued self-induced vomiting. Self-induced vomiting is highly heritable which may suggest a biological pathway, although the neurobiology of hyperemesis is unclear (Goodwin, 2002; Sullivan et al., 1998).

Women with repeated experiences of self-induced purging may also have lower thresholds for vomiting in response to pregnancy-associated nausea than women without a history of purging. Nevertheless, women with a history of intentional purging behaviors should be counseled that intentional purging during pregnancy can have a significant impact on fetal nutrition and development, despite the fact that pregnancy-related vomiting is commonplace. Qualitatively, women with eating disorders report that they are also able to distinguish between pregnancy-related vomiting and intentional compensatory purging, a distinction, which could be leveraged in clinical care (Lacey & Smith, 1987).

Untreated Eating Disorders During Pregnancy

One of the primary challenges in knowing how to help a patient suffering with an eating disorder during pregnancy is that the physicians must know that their patient either has an eating disorder or has a history of one. Many obstetricians do not inquire into eating disorder histories and statuses, and expectant mothers do not always volunteer this information. Due to the heightened risk for caesarian sections and a trifold likelihood of postpartum depression among women with active eating disorders when compared to the general population, women with eating disorders should be considered to be in high-risk pregnancies and monitored closely by physicians both during and after pregnancy (Franko et al., 2001).

Postpartum Eating Disorders

The complications that ensue due to the presence of a maternal eating disorder do not end at delivery. Women with a lifetime or a recent history of an eating disorder are at greater risk for postpartum depression than the general population, and the risk of relapse and problems with breastfeeding are considerable concerns.

Postpartum Depression and Relapse

Multiple researchers have found that among pregnant women with active eating disorders, the likelihood of postpartum depression or postpartum distress is considerably higher than the general population (Abraham, Taylor, & Conti, 2001; Franko et al., 2001). BED and BN have the strongest correlation with postpartum depression with a two- to threefold increased risk, but there is still a trend seen in AN, which is in need of greater study (Mazzeo et al., 2006). Regardless, one of the hallmark traits of AN, perfectionism, seems to play a role in determining the severity of postpartum depression (Mazzeo, Slof-Op't Landt et al., 2006). Mothers with eating disorders also appear to struggle more with adjusting to lives with their newborns. Among women who were suffering from eating disorders (specifically AN or BN) prior to pregnancy, 92 % reported problems adjusting compared to only 13 % in the control group ($p < 0.001$; Koubaa, Hallstrom, & Hirschberg, 2008).

Women with eating disorders may have a similar or a greater risk of developing perinatal and postpartum depression compared to women with a history of major depressive disorder and no eating disorder (Gavin et al., 2005; Mazzeo, Mitchell, Gerke, & Bulik, 2006). Active BN during pregnancy is a strong predictor of postpartum depression, inferring almost a threefold risk compared with women with quiescent BN during pregnancy (Morgan et al., 2006; Morgan, Lacey, & Sedgwick, 1999). Moreover, women with eating disorders are over-represented in women seeking postpartum depression treatment. In one study, 37 % of women seeking treatment for perinatal depression reported a lifetime diagnosis of an eating disorder, a three- to fourfold higher lifetime prevalence than in national general population samples (Meltzer-Brody et al., 2011). In addition, women with lifetime eating disorders, particularly those with a history of BN and PD, have also reported more severe perinatal depression (as measured by the Edinburgh Depression Inventory) than women with no eating disorder history (Meltzer-Brody et al., 2011).

Women with a higher residual postpartum weight—due to the likelihood of greater weight gain among women with eating disorders—may have a stronger desire to lose weight, thus falling back on some maladaptive weight-loss strategies they used in the past and some perhaps relapsing during this fragile time (Bulik et al., 2009). The presence of postpartum depression magnifies the risk of recurrence of a previous eating disorder or even crossover to a new diagnosis. Remission is the most typical course in pregnant women, but it is essential to understand that pregnancy does not offer a lasting protective effect for everyone (Bulik et al., 2009).

In a large-scale population-based study of postpartum eating disorders, only 59 % of women with AN, 30 % with BN, 57 % with PD, and 42 % with BED prior to pregnancy were in remission 3 years postpartum (Knoph et al., 2013). Both social support and psychological factors were associated with continuation of eating disorder symptoms. Women who reported less relationship satisfaction and greater psychological distress during the postpartum period were more likely to experience a continuation of eating disorder symptoms (in particular, BED symptoms; Knoph et al., 2013). These results highlight the need to provide women with eating disorders adequate social support and treatment for postpartum mood in order to preserve the symptom remission often seen during pregnancy.

Postpartum Dieting, Nutrition, and Body Image

Weight gain during pregnancy can be a source of stress for many women, and most women attempt to lose residual gestational weight in the weeks and months following delivery. In general, concerns about weight and weight loss attempts are normative with 75 % of women concerned about their weight in the first few weeks postpartum (Hiser, 1987; Stein & Fairburn, 1996) and an additional 70 % of women worried about their "flabby figure" (Hiser, 1987; Stein & Fairburn, 1996).

Perhaps because of this heightened awareness of weight and shape concerns among the majority of postpartum women, it is not surprising that almost three-quarters are still trying to lose weight at 4 months postpartum (Baker, Carter, Cohen, & Brownell, 1999; Stein & Fairburn, 1996). Therefore, this pattern of weight concerns and desire for weight loss postpartum are not solely restricted to women with histories of eating disorders. However, among individuals with eating disorders, especially anorexia, these feelings are exponentially heightened due to the preexisting hyper-focus on body weight and shape that defines the disease. There is also evidence that women with AN, BN, PD, and BED gain more gestational weight than women without eating disorders (Bulik et al., 2009). Therefore, it is not surprising that among women with eating disorders, Eating Disorder Examination Questionnaire (EDE-Q) scores are higher at postpartum than among women without an eating disorder diagnosis (Stein & Fairburn, 1996). Consequently, although weight concerns and weight loss are a prevalent concern among new mothers, the presence of an eating disorder exacerbates and exponentially heightens the distress around weight and the compulsion to lose gestational weight.

Breastfeeding and Infant Feeding

There is significant evidence that breastfeeding is the optimal practice for both maternal and child health due to immunological benefits for the child and a reduced risk of maternal breast and ovarian cancer, hypertension, and type 2 diabetes

(Galson, 2008; Schwarz et al., 2010; Stuebe et al., 2010, 2011). The American Academy of Pediatrics recommends exclusive breastfeeding for the first 6 months with continuation through the first year or longer to ensure optimal benefit (AAP, 2012). However, breastfeeding is still not utilized as the primary source of feeding for many mothers; among mothers with a history of an eating disorder, breastfeeding is often more truncated due to multiple concerns.

While one study found that women with lifetime histories of AN and BN are more likely to initiate breastfeeding and breastfeed just as long as or longer than women in the general population (Micali, Simonoff, & Treasure, 2009), others have found that those women with prenatal AN or EDNOS (including PD) are approximately two times more likely to stop breastfeeding early when compared to women without eating disorders (Blais et al., 2000; Larsson & Andersson-Ellstrom, 2003; Torgersen et al., 2010). This premature cessation of breastfeeding in mothers with a history of eating disorders begins soon after delivery. In a Scandinavian sample of women who self-reported histories of eating disorders, by the time their child was 3 months old, 19 % had ceased breastfeeding compared to only 7 % of mothers without a history of an eating disorder (Larsson & Andersson-Ellstrom, 2003).

As partial explanation for their shortened duration of breastfeeding, women with eating disorders often find the act of breastfeeding embarrassing and worry that this could alter their appearance (Stein & Fairburn, 1989; Waugh & Bulik, 1999). Some mothers with eating disorders do not even initiate breastfeeding due to these concerns (Waugh & Bulik, 1999). Mothers with eating disorders also endorse worrying that their breast milk will be insufficient for their infant's needs and that their infant may be allergic to their breast milk and are more likely to rigidly adhere to a prescribed feeding schedule, experiencing anxiety when their infant signals hunger cues outside of the prescribed feeding window (Evans & le Grange, 1995).

These differences also continue to rise in infant feeding. Women with a history of an eating disorder or a current episode during pregnancy are more likely to have infants with feeding difficulties, primarily due to the presence of maternal distress (Micali, Simonoff, Stahl, & Treasure, 2011). Maternal distress from perinatal depression and anxiety also partially mediates the relationship between eating disorder status and early infant feeding difficulties (Micali et al., 2011). Maternal emotional dysregulation, specifically in response to food or weight concerns, impairs the mother's ability to feed her newborn without a certain level of anxiety, which potentially could be conveyed to the infant. Women with eating disorders are also more likely to rate their infants as having a more difficult and fussy temperament, which may also contribute to feeding difficulties (Zerwas et al., 2012).

Midlife Eating Disorders

Typically eating disorders have been conceptualized as diseases that are bracketed within the teenage and young adult years. However, there is increasing evidence to suggest that the landscape for eating disorders has considerably changed to include an older age range than previously thought. Midlife eating disorders also comprise

a more heterogeneous mix than eating disorders earlier in life (Bulik, 2013). For many, eating disorders in midlife could be the progression of a chronic eating disorder that began many years earlier in adolescence or early adulthood. For a smaller minority, it could represent a new diagnosis in a formerly healthy individual or a relapse from recovery (Bulik, 2013). Consequently, recognizing and treating these varied presentations and establishing effective treatments for chronic, recurrent, and new-onset eating disorders in midlife women are crucial.

Although there is little epidemiological data documenting increased eating disorders in midlife clinically, treatment centers report that they are witnessing greater numbers of women in midlife than seen previously. Among women over age 50, the greatest predictors for eating disorder symptoms, concerns, and behaviors are a younger age and a higher BMI (Gagne et al., 2012). Therefore it seems that the fear of aging and weight may be more pronounced in middle age and then decrease in the elderly population (Lewis & Cachelin, 2001).

This may be due to a cohort effect. Because an older generation of women did not have as much exposure to fat talk, "old talk," and unrealistic representations of beauty that dominate the current media landscape, they may have experienced less environmental pressure towards developing eating disorder symptoms (Becker, Diedrichs, Jankowski, & Werchan, 2013). This fear of aging has been directly tied to disordered eating and can emanate from both a personal source, but also in response to media focus on defying the effects of age, as well as "old talk" by friends and family members (Becker et al., 2013; Lewis & Cachelin, 2001). "Old talk" is defined as a parallel but distinct form of "fat talk" that specifically addresses nonconformance with the thin young ideal of female beauty (Becker et al., 2013; Lewis & Cachelin, 2001). Having this type of talk perpetuates body- and age-related anxiety and increases body image disturbances as well as eating disorder pathology among women most exposed to it (Becker et al., 2013). It also suggests that when women are young, the most salient feature of beauty is weight, whereas youth becomes a much more significant and salient concern as women age.

The hyper-focus on eating disorders in youth can be problematic for older individuals dealing with eating disorders for multiple reasons. Stigma and embarrassment about experiencing these problems in midlife could keep women suffering with eating disorders from seeking help. Because of the focus on youth eating disorders and eating disorder risk, they may feel ignored both by public attitudes on eating disorders and by treatment providers. Additionally, the majority of evidence-based treatments for eating disorders are targeted towards and based on findings from a younger population.

Menopause

In relation to understanding eating disorders in an older age range of women than previously studied, it is important to acknowledge one of the major life changes women face that may be associated with eating pathology. Menopause marks the cessation of menstruation and also changing hormone levels that affect metabolism

and weight in women, thus contributing to factors that could increase the risk for disordered eating.

This is a relatively new area of study, and therefore the research concerning menopause and eating disorders is still rather limited; however, *anorexia tardive* was a term established in the 1980s to describe late onset of anorexia, occurring anytime after a woman's marriage and containing diagnoses that happened at or after menopause (Dally, 1984). This loss of weight was, at the time, seen as a desire to die, rather than expressing marital crises (Dally, 1984). A more recent conception of eating disorders around menopause suggests that the biological changes to the body and its deviation from both the thin and young ideal could cause women experiencing these changes to adopt maladaptive strategies to control their weight and slow the aging process (Becker et al., 2013).

Recently, a large-scale survey about shape and weight concerns was conducted in an older population of women, aged 50 and older. More than 70 % said that they were currently trying to lose weight, and 41 % said that they scrutinized their body at least once a day (Gagne et al., 2012). Additionally, several responses to an open-ended question reflected frustration with menopause-related weight gain (Gagne et al., 2012). Therefore, there is evidence to suggest that menopausal women are at risk for disordered eating and body image disturbances as well as possible eating disorders. Thus far, only one study has examined eating disorders, body image, and menopausal status in women at midlife (Mangweth-Matzek et al., 2006).

Perimenopausal women were significantly more likely to report eating disorder symptoms when compared to a referent group of premenopausal women (Mangweth-Matzek et al., 2006). Moreover, they were significantly more likely to report body image concerns including "feeling fat" and dissatisfaction with their weight (Mangweth-Matzek et al., 2006). Women with menopause due to hysterectomies also reported significantly higher eating and body image concerns (Mangweth-Matzek et al., 2006). Clearly, however, more research is needed to clarify the specific triggers and prognosis for eating disorders during the menopausal transition, and examining eating disorders in midlife could be a rich area of future study.

Clinical Implications

Fostering a greater understanding and screening of eating disorders across the lifespan are essential for women's physical and mental health and for the health of future generations. Clinicians in primary care and gynecology are an untapped resource to detect and treat women who are struggling with eating disorders (Mitchell-Gieleghem, Mittelstaedt, & Bulik, 2002; Sim et al., 2010). However, all too often, large-scale surveys have demonstrated that clinicians do not ask about eating disorder symptoms, and patients frequently do not discuss their struggle with eating disorders. For example, among patients who presented for treatment to an infertility clinic with oligomenorrhea or amenorrhea, 58–76.4 % of women met clinical indicators for eating disorders, but none had disclosed symptoms to their

providers (Freizinger et al., 2010; Stewart, Robinson, Goldbloom, & Wright, 1990). Furthermore, 64 % of pregnant women did not report their eating disorder status to their obstetrician-gynecologists (OB-GYN), and of those who discussed their eating disorders with their OB-GYNs, only half found this to be helpful (Lemberg & Phillips, 1989).

Obstetrician-gynecologists also report that they are not confident in making an eating disorder diagnosis. Only 20 % report confidence in their ability to diagnose eating disorders, and only 54 % of gynecologists report that eating disorder assessment and screening fell within their scope of practice. However, 90.8 % reported that eating disorders could have a negative impact on pregnancy and birth outcomes (Leddy, Jones, Morgan, & Schulkin, 2009; Morgan, 1999). Much of the reluctance to screen for eating disorders may be due to lack of education, with an overwhelming majority reporting (88.5–96.2 %) that their training in diagnosing and treating eating disorders was barely adequate (Leddy et al., 2009).

Paradoxically, although primary care providers surveyed express reluctance to screen for eating disorders, the perinatal period could be viewed as a unique window of opportunity for recovery. Routine perinatal care gives OB-GYN providers the chance to screen for mental health issues and engage their patients in repeated monthly or even weekly visits (Borri et al., 2008; Hawkins & Gottlieb, 2013; Reck et al., 2008). In addition, women with eating disorders express motivation to eat and avoid inappropriate compensatory measures "for the baby" during their pregnancies and view weight gain during this period as more "socially acceptable." Moreover, postpartum women will report disordered eating symptoms when directly asked by providers (Broussard, 2012). Treatment and support may also be especially acute in the postpartum period, as the pressure to lose gestational weight and stress accompanying the demands of caring for a newborn often leads to the reemergence of symptoms (Knoph et al., 2013; Lacey & Smith, 1987; Morgan, Lacey, & Sedgwick, 1999).

Given the increased risk of perinatal depression and anxiety in women with eating disorders, additional screening for symptoms of depression and anxiety is critical. Although the awareness and assessment of perinatal and postpartum depression within the OB-GYN community have dramatically improved (Yonkers et al., 2009), raising awareness and screening of eating disorder symptoms during pregnancy are still desperately needed in order to capitalize on this unique window for eating disorder remission and recovery (Franko et al., 2001; Franko & Spurrell, 2000). Currently, screening for domestic violence and depression in pregnant and postpartum women is routine, but screening for eating disorders has only been recommended in women with a documented history (Agency for Healthcare Research and Policy, 2013). Given the fact that many providers do not ask about eating disorder histories and many patients do not tell, limiting screening to a documented history could fail to capture many women who are struggling during this important juncture.

Clinicians in primary care should also be educated about the risk of eating disorder emergence or reemergence during the perimenopausal period. Creating a safe environment for disclosure of symptoms also means identifying and addressing clinician biases about who can and cannot be affected by eating disorders. Although the highest risk of eating disorder onset is in mid-adolescence to early adulthood,

increasingly women have been presenting with eating disorder symptoms in midlife (Hudson et al., 2007). Educating providers about the role of the menopausal transition for eating psychopathology may be necessary in order to ensure that providers do not assume that eating disorders are restricted only to young women.

Screening

Routine uniform screening for eating disorders across all levels of primary care and in OB-GYN offices would be the best way to improve their detection and treatment (Harris, 2010). The SCOFF questions (Luck et al., 2002; Morgan, Reid, & Lacey, 1999) have universally been identified as an excellent measure to assist providers when screening for eating disorders (Harris, 2010; Hawkins & Gottlieb, 2013; Hoffman, Zerwas, & Bulik, 2011; Sim et al., 2010). The SCOFF includes five short yes/no questions about possible eating disorder symptoms, and the acronym originates from the words used in the questions:

- S—Do you make yourself *sick/vomit* because you are uncomfortably full?
- C—Do you worry about loss of *control* over your eating?
- O—Have you recently lost *one* stone (14 lb) in 3 months?
- F—Do you believe you are *fat* although others say that you are thin?
- F—Would you say *food* predominates your life? (Morgan, Reid, & Lacey, 1999)

A positive screen (answering yes to two or more questions) suggests a referral to an eating disorder specialist and nutrition providers is necessary in order to ensure continued assessment and care.

Future Directions

The director of the National Institute of Mental Health, Dr. Thomas Insel, cites the mathematician and physicist Freeman Dyson to describe how innovations in technology have disrupted the past 10 years of mental health research, "New directions in science are launched by new tools much more often than by new concepts (Dyson, 1997)." Because of new tools, we have witnessed the launch of a new era of inquiry into the biological bases of mental health and a dramatic change in our understanding of genetic and epigenetic mechanisms for the transmission of psychiatric illness. The combination of the publication of the human genome sequence by the international Human Genome Project (HGP) in the spring of 2001 and the rapidly accelerating pace of computing power paved the way for genome-wide association studies (GWAS), a new form of genetic case–control association studies. In turn, results from GWAS have pointed towards new biological pathways that confer the risk for mental illness and possible new treatments (Kim, Zerwas, Trace, & Sullivan, 2011; Lander et al., 2001).

In eating disorder research, two GWAS are under way. In one GWAS, funded by the International Wellcome Trust Case Control Consortium (WTCCC3), a consortium of investigators from 17 countries is combining 3,000 blood samples from women with AN (Bulik, Collier, & Sullivan, 2011). In the second, the Anorexia Nervosa Genetics Initiative (ANGI), a second consortium of investigators from four countries is currently collecting 8,000 blood samples with AN (Bulik & Baker, 2013). Understanding the genes that predispose individuals to eating disorders will be a powerful tool for understanding the biology of eating psychopathology and ultimately elucidate how genetic risk markers are expressed over the life-span from menses to menopause.

Particularly relevant for reproductive mental health and eating disorders are the new tools to understand epigenetic modulation of the genome. Epigenetic modulation or epigenetics refers to any factor that affects gene expression such as DNA methylation, histone modification, and small interfering RNA (Campbell, Mill, Uher, & Schmidt, 2011). In particular, reduced representation bisulfite sequencing allows researchers to analyze DNA methylation on a genome-wide basis (Fouse, Nagarajan, & Costello, 2010). The prenatal period is considered a critical window for modulation of the epigenome because prenatal nutrition, particularly deficiencies in micronutrients, can alter gene expression of offspring through methylation (Davis et al., 2007; Entringer, Kumsta, Hellhammer, Wadhwa, & Wust, 2009; Lemberg & Phillips, 1989; Micali & Treasure, 2009; O'Connor et al., 2005; Seckl, 2008). When micronutrients are deficient during pregnancy, children are more likely to have impairments in their development (Chmurzynska, 2010). For example, when mothers experienced the Dutch Famine early in their pregnancies, their children had less DNA methylation of the imprinted insulin-like growth factor 2 (IGF2) gene as adults, even though the children's weight was within the normal range at birth (Heijmans et al., 2008). Moreover, since epigenetic marks may be heritable, these effects may not be limited just to immediate offspring but could be passed on to grandchildren (Champagne, 2008; Lim & Ferguson-Smith, 2010; Richards, 2006).

Although this research has not been extended to eating disorders during pregnancy, women struggling with restrictive eating disorders during pregnancy may be more likely to have micronutrient deficiencies. Unplanned pregnancies may be more common in women with AN and prenatal vitamin consumption prior to conception or in early pregnancy unlikely (Bulik et al., 2010; Dellava et al., 2011). Moreover, there is some evidence to suggest that women with eating disorders have systematic differences in their DNA methylation. Global DNA hypermethylation of the *DRD2* promoter, associated with dopamine regulation, has been found in AN and BN, and hypermethylation of the atrial natriuretic peptide (ANP) gene promoter region which regulates corticotropin and cortisol has been found in BN (Frieling et al., 2008, 2010; Groleau et al., 2013). Understanding how methylation is programmed in utero and varies across development constitutes a new biological pathway for understanding the interaction between genetic risk for eating disorders and environmental influences on gene expression. New technologies in genetics and epigenetics combined with prospective longitudinal studies measuring these

biomarkers across multiple time points will pave the way towards a new understanding of the interaction between reproductive hormones and genetics as risk and resilience factors for the development of eating disorders.

In sum, eating disorder prevention and treatment can have a tremendous impact on public health. Preconception, antenatal, postpartum, and perimenopausal care should include screening in order to reduce the medical and financial burden these devastating disorders place on individuals, families, and communities. Leveraging disruptive new technologies to understand how genetics and epigenetics interact in development will create new ways of understanding the biological bases of eating disorders across the life-span.

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