The Science Behind the Academy for Eating Disorders' Nine Truths About Eating Disorders

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The Science Behind the Academy for Eating Disorders' Nine Truths About Eating Disorders

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Abstract

Objective: In 2015, the Academy for Eating Disorders (AED) collaborated with international patient, advocacy, and parent organizations to craft the *"Nine Truths About Eating Disorders."* This document has been translated into over 30 languages and has been distributed globally to replace outdated and erroneous stereotypes about eating disorders with factual information. In this paper, we review the state of the science supporting the *Nine Truths*. **Methods:** The literature supporting each of the *Nine Truths* was reviewed, summarized, and richly annotated. **Results:** Most of the *Nine Truths* arise from well-established foundations in the scientific literature. Additional evidence is required to further substantiate some of the assertions in the document. Future investigations are needed in all areas to deepen our understanding of eating disorders, their causes, and their treatments. **Conclusions:** The *"Nine Truths About Eating Disorders"* is a guiding document to accelerate global dissemination of accurate and evidence-informed information about eating disorders.

The Science Behind the Academy for Eating Disorders' Nine Truths About Eating Disorders

Eating disorders are serious mental illnesses that affect millions of individuals worldwide regardless of race, age, nationality, or sex and incur considerable personal, familial, and societal costs. The cumulative lifetime risk by age 80 of anorexia nervosa (AN), bulimia nervosa (BN) and binge-eating disorder (BED) approximates 4.6% (Hudson, Hiripi, Pope, & Kessler, 2007). Inclusion of subthreshold eating disorder behaviors raises this estimate to nearly 10%. Despite the prevalence and toll that eating disorders exact on society, we lack comprehensive understanding of the etiology of eating disorders. We face significant limitations in our ability to prevent, detect, and treat this class of disorders. Stigma surrounding eating disorders has overshadowed the field for decades and has perpetuated misconceptions about their causes, hampered efforts at advancing knowledge, and misdirected lay understanding of these conditions. Perhaps most importantly, stigma surrounding eating disorders has prevented those in need from seeking help (Ali et al., 2017).

In May 2015, the Academy for Eating Disorders (AED) and several international advocacy organizations issued a document entitled "*Nine Truths About Eating Disorders*" (http://www.aedweb.org/index.php/25-press-releases/163-press-release-aed-releases-nine-truths-about-eating-disorders?quot). The AED focused on presenting truths rather than dispelling myths to introduce empirical evidence into the general knowledge base about eating disorders. The document has been translated into over 30 languages and is being disseminated worldwide to transform perceptions and understanding of eating disorders. In this paper, we present an overview of the empirical foundation upon which the *Nine Truths* rest to foster a more accurate understanding of the current state of scientific knowledge about eating disorders for patients, families, professionals, and the public.

The truths span a broad literature. In addition to the review of empirical studies, we also attend to modern theoretical and conceptual models and authoritative reviews to evaluate the current state of the science behind the *Nine Truths*. For each truth, we present supporting statements and a strength of evidence rating (Low, Moderate, or High; see Supplementary Table S1 & S2). A detailed summary of the evidence is presented in Supplementary Table S2. In addition to these tables, online supplementary materials provide a rich source of background information and all references for the main text presented there as an annotated bibliography.

Truth #1: Many people with eating disorders look healthy, yet may be extremely ill.

1.1 Eating disorders are associated with significant somatic, psychosocial, and psychological risk.

Eating disorders are associated with somatic complications in multiple organ systems including the cardiovascular, gastrointestinal, musculoskeletal, dermatologic, endocrine, hematological, and neurological systems (Mehler & Brown, 2015; Mehler & Rylander, 2015; Thornton et al., 2017) as well as psychiatric comorbidities (see Supplementary Table S3). The more chronic and severe the eating disorder, the greater the likelihood of serious somatic complications (Westmoreland, Krantz, & Mehler, 2016). However, severe complications can emerge at any time during the course of illness (Westmoreland et al., 2016). Furthermore, eating disorders are associated with a number of measurable psychological and neurocognitive traits (see Supplementary Table S4 and Statement 4.2).

1.2 Most individuals with eating disorders do not appear emaciated.

Weight loss is a defining characteristic of AN, but not BN or BED. In fact, eating disorders are present in all BMI categories (Duncan, Ziobrowski, & Nicol, 2017; Flament et al., 2015), and AN

is less common than the combined prevalence of other eating disorder diagnoses (Kessler et al., 2013; Lindvall Dahlgren & Wisting, 2016; Qian et al., 2013). On average, the BMI of individuals with AN is lower than the BMI of those with BN, which is lower than the BMI of those with BED. Yet, restrictive eating disorders also occur among normal- and overweight individuals and individuals with BN and BED can be normal weight, overweight, or obese (see 5.4).

1.3 Somatic, psychosocial, and psychological manifestations and comorbidities of eating disorders may be difficult to detect.

Many serious somatic complications of eating disorders are not readily visible to lay observers or recognizable to the affected individual (see Supplementary Table S3). Even experienced healthcare professionals have difficulty accurately identifying complications or may misattribute their causes (Currin et al., 2007b; Currin, Schmidt, & Waller, 2007a; Currin, Waller, & Schmidt, 2009; Gaudiani & Mehler, 2016).

Individuals with eating disorders may fail to report the psychological components of eating disorders or have poor insight into their level of impairment (Dalle Grave, Calugi, & Marchesini, 2008; Griffiths, Mond, Murray, & Touyz, 2015; Nordbø et al., 2012; Santonastaso et al., 2009; Vandereycken, 2006a; Vandereycken, 2006b). However, psychological features are often present, even if at milder levels (Carter & Bewell-Weiss, 2011) with some variation across cultures (Lee, Lee, Ngai, Lee, & Wing, 2001; Pike & Dunne, 2015) and in younger patients (Carter & Bewell-Weiss, 2011; Norris et al., 2014) (see Supplementary Tables S3 & S4). Signs and symptoms of an eating disorder should always be taken seriously and not dismissed or minimized. Immediate attention is warranted, and a comprehensive evaluation should be the first step in treatment planning (American Psychiatric Association, 2006; Hay et al., 2014; National Collaborating Centre for Mental Health, 2004).

1.4 Most individuals with eating disorders do not enter treatment; those who do often do so many years into the course of illness.

Epidemiological studies across the world indicate that only a minority of individuals who meet diagnostic criteria for eating disorders seek treatment (Hoek & van Hoeken, 2003; Hudson et al., 2007; Keski-Rahkonen et al., 2009; Kessler et al., 2013; Preti et al., 2009; Twomey, Baldwin, Hopfe, & Cieza, 2015). Eating disorders thus remain undetected, and, even when detected, may not be viewed as serious issues warranting medical intervention (Keel & Brown, 2010).

Truth #1: Summary and future research directions

Confidence ratings: Moderate (1.3) to High (1.1; 1.2; 1.4) (see Supplementary Table S2)

Looks may deceive, and a healthy appearance and failure to acknowledge the severity of these illnesses can delay help-seeking and detection by friends, family, providers, and even patients themselves. Work is required to push past barriers to detection and care. First, longitudinal research is needed to identify early signs of somatic complications and psychiatric comorbidities in eating disorders. A better understanding of prodromal signs and the illness trajectory will enable early detection. Understanding educational needs for physicians and other front-line providers is necessary for broad dissemination of screening and educational tools. For more information on addressing eating disorders in clinical practice, see the AED Guide to Recognition and Management of Eating Disorders (http://www.aedweb.org/index.php/education/eating-disorder-information/eating-disorder-information-13).

Truth #2: Families are not to blame, and can be the patients' and providers' best allies in treatment.

2.1 Biological risk factors contribute to the development of eating disorders.

Modern etiological models of psychiatric illnesses consider the bidirectional risk between biology and environment (see Truth #4 for summary of biological factors). The assertion that parental characteristics or family dynamics are necessary and sufficient for the development of eating disorders (i.e., "families are to blame") represents an historical and dated model of psychopathology and disregards modern etiological conceptualizations of psychiatric risk. Accordingly, the first part of this truth, "families are not to blame," is empirically and logically justified. This does not imply that evaluation of family functioning in eating disorders is without merit, as such studies may provide actionable information for providers, caregivers, and patients.

2.2 Prototypical family interaction patterns that exist premorbidly among families with eating disorders have not been identified.

A critical methodological issue continues to plague studies of family functioning in eating disorders. Most studies are correlational/differential in nature, precluding causal interpretation. Moreover, the direction of causality has not been examined. Prospective longitudinal designs are necessary to determine whether interactions among family members exist premorbidly or are a consequence of the illness. Some prospective studies have investigated effects of parent and family functioning in predicting later eating disorder onset with mixed results. For example, some evidence suggests that parental factors predict later eating pathology (Johnson, Cohen, Kasen, & Brook, 2002; Nicholls & Viner, 2009; Shoebridge & Gowers, 2000); however, reviews have not identified consistent patterns of risk associated with parenting styles or family interactions (Campbell & Peebles, 2014; Eisler, 2005; Larsen, Strandberg-Larsen, Micali, & Andersen, 2015; le Grange, Lock, Loeb, & Nicholls, 2010; Strober & Humphrey, 1987; Yager, 1982). Indeed, greater family conflict, reduced parental alliance, and increased feelings of depression in families with a child suffering from AN might reflect an accommodation process in response to a severe

and life-threatening condition (Sim et al., 2009). Investigations of parental factors have also been limited by lack of controls with other psychiatric disorders, measurement inconsistencies, and lack of statistical power. For example, certain adverse familial experiences such as sexual abuse may contribute to the risk of pathology in general, and are not eating disorder specific (Kendler et al., 2000).

2.3 Eating disorders place stress on families.

Studies on the experience of caring for a patient with an eating disorder suggest a significant burden and negative impact on the health and well-being of caregivers—especially among mothers and partners (Anastasiadou, Medina-Pradas, Sepulveda, & Treasure, 2014; Kyriacou, Treasure, & Schmidt, 2008). Those caring for patients with AN have reported higher levels of distress than individuals caring for patients with psychoses (Treasure et al., 2001). Parents can initially perceive starvation to be deliberate, which evokes a strong emotional response, significant distress, and can lead to desperate responses in parents in the absence of clear guidance (Whitney et al., 2005). Attributions for these responses should consider the parent's desire to cease the starvation and save their child. Thus, assisting families in developing tools to deal effectively with an eating disorder is imperative. Distress associated with an eating disorder often extends beyond the identified patient. Stresses associated with having a psychiatrically ill child or partner, coupled with the responsibility for collaborating with providers in the treatment of individuals with eating disorders, underscore the importance of self-care for caregivers (Patel, Wheatcroft, Park, & Stein, 2002; Treasure & Nazar, 2016).

2.4 Family-based treatments have demonstrated effectiveness for the treatment of adolescent AN.

Families and support systems are needed as patient allies during treatment (le Grange et al., 2010). The entire family is affected when dealing with chronic and severe illnesses such as AN. Familial organizational changes that emerge may serve to maintain AN and limit access to adaptive resources the family possesses that are necessary to help overcome the eating disorder (Cook-Darzens, 2016; Eisler, 2005). Family-based treatment (FBT), whereby parents reassert control over the child's eating, is a promising approach to the treatment of adolescent AN and has some empirical support for the treatment of adolescent BN (Couturier, Kimber, & Szatmari, 2013; le Grange, Lock, Agras, Bryson, & Jo, 2015). FBT helps families recognize resources and knowledge they possessed prior to the onset of the disorder and re-implement them in the family system (Lock & le Grange, 2015). FBT is recommended by many national guidelines for the treatment of eating disorders in youth (Watson & Bulik, 2013) (see Supplementary Table S5).

The role of the family is also important for adults with eating disorders. Partners can be an asset in treatment of adults since they typically express a strong desire to help, yet fear that anything they do or say will inadvertently exacerbate the situation (Treasure & Nazar, 2016). Couple-based interventions for eating disorders leverage the power of relationships and engage the partner in the recovery process (Bulik, Baucom, Kirby, & Pisetsky, 2011; Kirby, Runfola, Fischer, Baucom, & Bulik, 2015; Schmidt et al., 2013). Initial results of couple-based interventions are promising and suggest that close support from a family member enhances treatment regardless of patient age. However, much of family and couple-based intervention research has focused on patients with AN; additional studies are required to confirm the benefit of engaging family members in the treatment of BN and BED (see Supplementary Table S5).

Truth #2: Summary and future research directions

Confidence ratings: Moderate (2.2; 2.3) to High (2.1; 2.4) (see Supplementary Table S2)

In summary, typical patterns of family functioning or structure that give rise to eating disorders have not been identified. This is consistent with the AED position paper on the role of the family in eating disorders (le Grange et al., 2010). Families are not to blame and in most cases can be the patients' and providers' best allies in treatment.

Research on family functioning has been summarized (Larsen et al., 2015; Saltzman & Liechty, 2016). These reviews point to the need for rigorous prospective designs to help understand how environmental variables, including family systems, may interact with biological risk (as discussed in Truth #7 & #8) to either heighten risk or buffer against the development of eating disorders. Eating disorders place stress on a family system, and future investigations that aim to reduce the burden on caregivers are necessary. Consideration of in-home care may be a useful direction for services. Finally, families represent an important base of support for those in recovery, and the effectiveness of family-based treatments for adolescents highlights how parents and caregivers can be important allies in treatment. Future studies that build on this success by examining how families can be best integrated into care of older adolescents, adults, and those who binge eat are of great interest.

Truth #3: An eating disorder diagnosis is a health crisis that disrupts personal and family functioning.

3.1 Eating disorders interfere with personal and family functioning. 3.2 Eating disorders produce financial burden. 3.3 In adolescence, eating disorders may lead to functional impairment and delays in healthy development. 3.4 In adulthood, eating disorders may interfere with intimate relationships, reproductive health, parenting, and health-related quality of life.

Truth #3 is covered by statements in several other Truths. As discussed in Truth #1, an eating disorder represents a health crisis that affects every aspect of an individual's life. In addition to myriad psychiatric and somatic complications and comorbidities enumerated in Truth #1, eating disorders also lead to considerable psychological distress, as well as isolation, stigmatization, and difficulties with family and other interpersonal relationships (Ali et al., 2017; Caslini et al., 2016; Dimitropoulos, McCallum, Colasanto, Freeman, & Gadalla, 2016; van Langenberg, Sawyer, Le Grange, & Hughes, 2016). Further, eating disorders are associated with financial burden, delays in healthy development, functional impairment, and may interfere with social role functioning including intimate relationships, reproductive health, and parenting (see summaries in Supplementary Tables S2-4).

Truth #3: Summary and future research directions

Confidence ratings: Moderate (3.3; 3.4) to High (3.1; 3.2) (see Supplementary Table S2)

Eating disorders clearly represent a health crisis (see Truth #1); the effects of which disrupt functioning beyond immediate complications of the eating disorder. Financial burden of eating disorders are significant, and they affect all areas of social and economic well-being, along with delaying or preventing healthy childhood and adolescent development. Future investigations that examine the true cost of eating disorders over the long-term are warranted. Longitudinal studies of eating disorders, including intervention studies, are encouraged to include secondary outcomes related to healthy development in youth, education, finances, employment, reproductive health, and overall quality of life. Further, an empirical review of the literature on relationship, role functioning, and quality of life in eating disorders would advance understanding of how eating disorders influence these vital, but understudied, outcomes.

Truth #4: Eating disorders are not choices, but serious biologically influenced illnesses.

4.1 Disordered eating behaviors can be guided by biological processes associated with automatic (unconscious) events.

In vulnerable individuals, biological drives towards automaticity can provoke rigid habits to the point where individuals struggle to regain control over their dysregulated eating and physical activity (Steinglass & Walsh, 2016). For example, altered inhibitory control, the ability to refrain from engaging in prepotent automatic responses, has been shown across eating disorders subtypes (Collantoni et al., 2016; Galimberti, Martoni, Cavallini, Erzegovesi, & Bellodi, 2012) with the greatest support for bulimic subtypes (Lavagnino, Arnone, Cao, Soares, & Selvaraj, 2016; Wu, Hartmann, Skunde, Herzog, & Friederich, 2013) (see Supplementary Table S4 for a review of traits). Such findings are supported by a position paper that reviewed literature identifying alterations in neurobiological pathways related to reward and self-control associated with eating disorders (Wierenga et al., 2014). Further, a recent theoretical model identifies eating behaviors in AN as habitual behaviors, similar to compulsions in obsessive compulsive disorder, supported by case-control studies on neuropsychological and neuroimaging tasks (Godier et al., 2016; Steinglass & Walsh, 2016). Evidence from animal studies and human neuroimaging also supports some shared neurobiology in eating disorders and other habit-related disorders, including addiction (Kaye et al., 2013b; O'Hara, Campbell, & Schmidt, 2015) .

4.2 Biologically-influenced, fundamental personality traits and cognitive styles are associated with eating disorders.

Eating disorders are consistently associated with fundamental personality traits and cognitive styles. These traits are influenced by genetic factors, exist premorbidly, become exacerbated during acute stages of illness, persist after recovery, and/or may affect the prognosis of eating

disorders. Some implicated traits are shared across disorders (e.g., weak central coherence, altered reward sensitivity, anxiety, difficulty with set shifting, altered interoceptive awareness), whereas others are more differentially associated with specific eating disorder phenotypes (e.g., harm avoidance in AN, negative urgency in BN) (see Supplementary Table S4 for overview of associated traits). Overall, identification of genetically influenced personality traits and cognitive styles may reveal core biological risk factors for the development of eating disorders.

4.3 Individuals with eating disorders may experience non-typical responses to eating and activity.

Individuals with eating disorders may have distinct responses to energy restriction and food consumption. For example, individuals with AN may have a paradoxical response to negative energy balance (i.e., taking in less energy than one expends, (Bulik, 2016), such that caloric intake is associated with dysphoric mood (Frank, 2012), whereas caloric restriction evokes a calming, anxiolytic, or euphorigenic effect (Bulik, 2016; Kaye, 2008; Kaye, Wierenga, Bailer, Simmons, & Bischoff-Grethe, 2013a). Non-typical responses to other behaviors such as physical activity and purging (as both positively and negatively reinforcing) are also reported in individuals with eating disorders (Berg et al., 2013; Giel et al., 2013; Kaye, 2008; Klein et al., 2010). Such processes highlight alterations from typical experiences of reinforcement as relevant to development and maintenance of eating disorders, and such patterns may be driven by variations in neurobiology.

4.4 Eating disorders are associated with dysregulation in neurotransmitter availability and function.

Although the precise underlying neurobiology is not fully understood, findings of positron emission tomography (PET) and single-photon emission computed tomography (SPECT) implicate dysregulation in both dopaminergic (DA) and serotonergic (5-HT) systems in eating

disorders (Culbert, Racine, & Klump, 2015; Kaye et al., 2013a; Kaye et al., 2013b; Kaye, 2008; Kessler, Hutson, Herman, & Potenza, 2016; Spies, Knudsen, Lanzenberger, & Kasper, 2015). These systems are central in rewarding aspects of food, motivation, executive functions, and the regulation of mood, satiety, and impulse control.

4.5 Brain structure and function differ between those with active eating disorders and unaffected individuals.

Both human and animal studies have addressed the role of brain anatomy and function in eating disorder psychopathology through use of brain imaging techniques. Studies revealing deviations in structure, function, and activation in the brains of individuals with eating disorders are reviewed comprehensively in several publications (Frank, 2013; Frank, 2015a; Kaye, 2008; O'Hara et al., 2015; Seitz et al., 2014; Seitz, Herpertz-Dahlmann, & Konrad, 2016; Titova, Hjorth, Schiöth, & Brooks, 2013; Van den Eynde et al., 2012).

Structural neuroimaging studies in eating disorders have predominantly shown grey matter reductions in various brain regions that are most pronounced in patients with AN (Seitz et al., 2016). Associations with nutritional abnormalities have been repeatedly demonstrated and in AN volume reductions tend to quickly normalize with weight gain (Bernardoni et al., 2016; Seitz et al., 2016). Functional and structural neuroimaging studies in eating disorders provide evidence that aberrant frontostriatal neural circuitry may represent altered reward pathways, manifesting in impaired regulation of appetite, emotion, and self-control (Frank, 2015b; Friederich, Wu, Simon, & Herzog, 2013; Kaye, Wagner, Fudge, & Paulus, 2011; Kessler et al., 2016; Marsh et al., 2009; Marsh, Maia, & Peterson, 2009). Specifically, altered functioning of limbic regions together with either reduced or exaggerated 'top-down' cognitive control (via the prefrontal cortex) are seen as contributing to impulsive (e.g., BN, BED) or exaggerated self-control (e.g., AN) related

symptoms/behaviors (Ehrlich et al., 2015; Friederich et al., 2013; Hege et al., 2015; Kaye & Strober, 2009; Kessler et al., 2016; King et al., 2016; Marsh et al., 2009; Sanders et al., 2015). Neuroimaging and behavioral findings suggestive of alterations in reward pathways have been shown across eating disorders (see Frank, 2015a for review). Findings are mixed regarding the direction of change and the subregions of the brain reward system, likely due to research design issues such as failure to control for nutritional and medication status, exercise, comorbidity, and inadequate sample sizes (Frank, 2015a).

The persistence of core eating disorder psychopathology may reflect not only preexisting neurobiological vulnerabilities, but also neuroadaptation (Treasure et al., 2015), whereby changes may occur in the brain as a consequence of prolonged eating disorder behaviors (e.g., binge eating or restriction). Adolescence, in particular, is associated with a host of neuronal changes, such as increased synaptogenesis, pruning, and myelination of frontal and limbic areas, which are involved in emotional processing and cognition (Benes, 1998; Blakemore & Choudhury, 2006; Tau & Peterson, 2010). A maturing brain may be particularly vulnerable to the insults caused by extreme food restriction or excessive exercise resulting in negative energy balance or highly variable energy consumption (binge-fast cycles).

Evidence from brain structure and function, though preliminary, advances support for the assertion that eating disorders are biologically influenced. Brain structure and function appears to be altered in the active disease state, though the exact nature and stability of differences requires further investigation. Even if brain structure and function differences only occur after an initial shift in eating behavior, these changes may highlight biologically-driven maintenance patterns that impede recovery.

4.6 Feeding and activity behavior is biologically regulated in animals.

Animal models shed light on highly specific brain pathways implicated in eating disorder features, including restriction and binge eating. Controlled experiments have led to the development of animal models of hunger (Atasoy, Betley, Su, & Sternson, 2012) and binge eating (Murray, Tulloch, Chen, & Avena, 2015), providing evidence of neurobiological origins of eating disorders. In addition, an activity-based anorexia (ABA) rodent model highlights increased physical activity and reduced body weight in response to restricted food access in animals (Chowdhury, Chen, & Aoki, 2015). Using neural circuit-level approaches that enable activation or inhibition of anatomically and genetically defined brain pathways, like optogenetics and chemogenetics, multiple pathways have been identified that regulate different patterns of feeding behavior (Hardaway, Crowley, Bulik, & Kash, 2015; Sternson & Roth, 2014) (see Supplementary Table S7 for specific regions and nuclei). This approach elevates understanding of how discrete neural circuits control feeding and metabolism, and provides additional evidence of how feeding behavior may be biologically influenced. Further study is needed to determine whether these are therapeutic entry points into pathological models of eating disorders.

4.7 Endocrine changes are associated with eating disorder risk.

The risk for eating disorders increases during reproductive milestones (e.g., puberty, pregnancy) and sex hormones play a role in this risk (Baker, Girdler, & Bulik, 2012; Klump, Keel, Sisk, & Burt, 2010). For example, AN in females typically develops around puberty and is rare before the pubertal transition. Earlier pubertal timing is also associated with increased eating disorder symptoms. Increases in estrogen at puberty are hypothesized to activate genes that influence eating disorder development (Culbert et al., 2015; Culbert, Racine, & Klump, 2016; Klump et al., 2010). The increased risk for eating disorder symptoms at puberty is not surprising given that puberty in females involves considerable changes not only in sex hormones, but also in body composition

and in neuropeptides that modulate metabolism (Loomba-Albrecht & Styne, 2009; Siervogel et al., 2003).

Pregnancy has also been suggested as both a risk and protective period for eating disorder symptoms. Women with acute AN and BN often report symptom improvement or remission during pregnancy, whereas pregnancy increases risk for relapse for those in remission from AN (Kimmel, Ferguson, Zerwas, Bulik, & Meltzer-Brody, 2016). Pregnancy may also mark a vulnerable time for BED onset (Bulik et al., 2007). Eating disorder symptoms fluctuate across the menstrual cycle in a manner that mirrors changes in sex hormones (Baker et al., 2012; Edler, Lipson, & Keel, 2007; Klump, Keel, Culbert, & Edler, 2008; Racine et al., 2012). Paralleling these findings, a direct association between diminishing estrogen and increasing progesterone levels and eating disorder symptoms has been observed (Edler et al., 2007; Klump et al., 2008). The menopause transition, which involves prolonged and erratic changes in sex hormones, may represent an additional vulnerability period for the development or re-emergence of an eating disorder (Baker & Runfola, 2016; Mangweth-Matzek et al., 2013).

Much less is known about the role of reproductive milestones and sex hormones in the risk for eating disorders in males. Some studies suggest that boys who experience either early or late puberty are at increased risk for eating disorder symptoms (Ricciardelli & McCabe, 2004). Testosterone may be a protective factor against eating disorder development, but findings are inconclusive (Baker et al., 2012).

In addition, aberrant blood and cerebrospinal fluid levels of various appetite-regulating peptides have been observed in individuals suffering from AN or BN (Monteleone & Maj, 2013). Most of these studies, however, are limited both by small sample sizes and their sampling process because plasma levels of appetite-regulating peptides may not reflect the concentrations in the

central nervous system. Serum leptin levels have also been tied with eating disturbances. Serum leptin levels correspond with fat mass in healthy, energy-balanced humans (Hebebrand, Muller, Holtkamp, & Herpertz-Dahlmann, 2007). As would be expected due to their low BMI and fat mass, in acute stages of the illness, individuals with AN generally have low serum leptin levels (Föcker et al., 2011). The observed levels in AN are typically lower than those in BMI-matched healthy lean individuals, most likely due to differences in fat mass (Hebebrand et al., 2007). Intriguingly, hypoleptinemia in AN has also been associated with characteristic hyperactivity (Ehrlich et al., 2009; Holtkamp et al., 2006). Hypoleptinemia is considered to be a state biomarker for AN and together with BMI may represent a useful diagnostic test to distinguish constitutional thinness from AN (Föcker et al., 2011). Additional endocrine changes observed in eating disorders are presented in Supplementary Table S3.

Truth #4: Summary and future research directions

Confidence ratings: Moderate (4.3; 4.4; 4.7); Moderate to High (4.1); High (4.2, 4.5; 4.6) (see Supplementary Table S2)

The precise nature of underlying biological signatures is an active area of investigation and evidence in support of Truth #4 is accumulating rapidly. In-depth work concentrating on personality traits, cognition, neurobiology, brain anatomy and function, endocrinology, genomics and other -omics (see Truths #7 and 8) contributes to improved understanding of the biological underpinnings of eating disorders. Future research directions for this truth include: 1) examining neuropsychologically-based treatment approaches and outcomes; 2) treatment matching based on phenotypic psychobiological profiles; 3) evaluation of childhood behavioral and neurobiological traits; 4) systematic reviews on altered response to food and exercise in eating disorders and brain

function; 5) additional investigation of neurotransmitter availability and function in eating disorders using methods including postmortem brain analyses, measures of cerebrospinal fluid, PET imaging, and magnetic imaging spectroscopy; 6) basic science and animal research to further probe neural circuitry associated with eating disorder risk; and 7) further examination of the role of longitudinal endocrine changes in eating disorders, including the menopause transition along with the role of hormonal changes in men's eating disorder risk.

Truth #5: Eating disorders affect people of all genders, ages, races, ethnicities, body shapes and weights, sexual orientations, and socioeconomic statuses.

5.1 Eating disorders affect both males and females.

Since research on eating disorders has historically focused on women, the nosology of eating disorders has evolved based on female symptom profiles (Anderson & Bulik, 2004) and normative data on males are lacking (see Supplementary Figure S1 for lifetime prevalence of eating disorders by sex). Available evidence suggests that males may also be less likely to seek treatment (Striegel, Bedrosian, Wang, & Schwartz, 2012), less likely to be diagnosed with an eating disorder even when presenting with identical symptoms as females (Currin et al., 2007a), and less likely to access treatment even with similar clinical severity (Austin et al., 2008).

5.2 Eating disorders occur across the lifespan.

The typical age of onset of both AN and BN is in adolescence or early adulthood (Currin, Schmidt, Treasure, & Jick, 2005; Keski-Rahkonen et al., 2007; Keski-Rahkonen et al., 2009; Smink, van Hoeken, & Hoek, 2012; Zerwas et al., 2015). Childhood-onset AN is seen clinically from about age 7 years upwards, whereas BN before puberty is quite rare (Nicholls & Bryant-Waugh, 2009). Likewise, BED often begins in late adolescence or early adulthood (Hudson et al., 2007; Kessler

et al., 2013; Mustelin, Raevuori, Hoek, Kaprio, & Keski-Rahkonen, 2015; Preti et al., 2009), though some people report that they began binge eating early childhood—even before going on their first diet (Grilo & Masheb, 2000). Overall, however, BED commonly begins later than AN and BN, with new cases steadily arising up to age 40-60 years in the population (Hudson et al., 2007; Preti et al., 2009).

Eating disorders in midlife are either recurring or persisting early-onset disorders or new late-onset disorders (Baker & Runfola, 2016; Gagne et al., 2012; Peat, Peyerl, & Muehlenkamp, 2008). Bulimic symptoms in particular are relatively common in midlife women (Baker et al., 2017; Gagne et al., 2012), with one study finding that, among 2,000 women above age 50, 13% endorsed an eating disorder symptom (Gagne et al., 2012). Although the etiology of midlife eating disorders remains poorly understood, life events such as divorce, loss of family members, or somatic illness could serve as triggers (Kally & Cumella, 2008; Peat et al., 2008), and pregnancy or menopause with accompanying biological changes may increase vulnerability for onset or recurrence of eating disorders (Baker & Runfola, 2016; Baker et al., 2017; Bulik et al., 2007; Peat et al., 2008). Very little is known about eating disorders in men in midlife and beyond.

5.3 Eating disorders occur in all races and ethnicities.

A review of community studies from 30 countries found no systematic association between ethnicity/race and eating disorder occurrence (see Supplementary Figure S2) (Mitchison & Hay, 2014). Although eating disorders were initially considered to be limited to Western culture, accumulating evidence ties eating disorders more generally to economic development, urbanization, and industrialization across the globe (Pike, Dunne, & Addai, 2013; Pike, Hoek, & Dunne, 2014). Rising incidences of eating disorders have been reported in numerous countries, particularly in Asia and the Middle East (Pike & Dunne, 2015; Pike et al., 2014). In the United

States, the prevalence of eating disorders in ethnic and racial minority groups is similar to non-Latino whites, while ethnic minority groups more frequently report binge-eating behavior compared with non-Latino whites (Marques et al., 2011). AN has been found to be somewhat less common among Black than White Americans (Pike et al., 2013; Striegel-Moore & Franko, 2003). Importantly, racial and ethnic minorities are underrepresented in specialist eating disorder services, possibly due to underdetection in primary care (Striegel-Moore et al., 2003).

5.4 Eating disorders occur in individuals of all shapes and sizes.

Weight and BMI can vary substantially across the different types of eating disorders. In a sample of over 3,000 adolescents, eating disorders were present in all BMI categories (Flament et al., 2015). Restrictive eating disorders in normal- and overweight individuals are increasingly being acknowledged. The DSM-5 facilitates the diagnosis of atypical AN in individuals who meet all criteria for AN with the exception of low weight (American Psychiatric Association, 2013). This diagnosis is appropriate, for example, in individuals who begin at high weights and lose weight precipitously. A substantial portion of treatment-seeking adolescents with restrictive eating disorders have a history of overweight or obesity (Lebow, Sim, & Kransdorf, 2015), and there is a well-established relationship among dietary restriction, obesity, and eating disorders (Field et al., 2003; Neumark-Sztainer et al., 2006). In a review of clinical trials of BN, baseline BMI was most commonly in the normal range (Berkman et al., 2006), whereas community studies indicate that BN is prevalent in overweight and obese adolescents (Flament et al., 2015) and predicts weight gain over time (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000; Micali et al., 2015). Individuals with BED are commonly overweight or obese (Hudson et al., 2007; Kessler et al., 2013), yet a substantial minority of individuals with BED are normal-weight, particularly early in

the course of illness (Fairburn et al., 2000; Mustelin et al., 2015) (see Statement 1.2 for additional information on BMI and eating disorders).

5.5 Eating disorders are present across different sexual orientations and gender identities. Homosexual orientation is regarded as a risk factor for eating disorders in men: gay and bisexual men report more body dissatisfaction and disordered eating, and are more likely to be diagnosed with an eating disorder than heterosexual men (Brown & Keel, 2012; French, Story, Remafedi, Resnick, & Blum, 1996; Russell & Keel, 2002). In women, the evidence on sexual orientation and disordered eating is mixed. Lower body dissatisfaction among homosexual women have been observed in some, but not all studies (Alvy, 2013; French et al., 1996; Moore & Keel, 2003; Morrison, Morrison, & Sager, 2004). In a population-based cohort of adolescents, unhealthy weight control behaviors (e.g., laxative use, fasting, and vomiting) were significantly more prevalent among sexual minority males and females than in their heterosexual peers (Hadland, Austin, Goodenow, & Calzo, 2014).

Most research on eating-related pathology has focused on cisgender individuals (i.e., those whose gender identity matches the sex they were assigned at birth). A study of over 280,000 American college students indicated that transgender individuals may have particularly high eating disorder risk: 16% of transgender youth reported being diagnosed with an eating disorder in the past year, compared with 2% and 4% of cisgender sexual minority men and women, respectively (Diemer, Grant, Munn-Chernoff, Patterson, & Duncan, 2015). Similarly, studies of Canadian transgender youth and of UK transgender adults have found high rates of endorsement of disordered eating behaviors, particularly among trans males (Watson, Veale, & Saewyc, 2016; Witcomb et al., 2015).

5.6 There is no consistent association between socioeconomic status and risk for eating disorders.

Although higher parental education has been associated with increased risk of being diagnosed with an eating disorder in registry studies (Ahrén et al., 2013; Goodman, Heshmati, & Koupil, 2014), evidence suggests that this association may be genetically rather than socially mediated (Duncan et al., 2017). No consistent association has been observed between socioeconomic status and risk of eating disorders (Mitchison & Hay, 2014). In Australian population surveys, both binge eating and purging increased more in low-income than high-income individuals during a 10-year time period, suggesting an ongoing shift in the demographics of disordered eating (Mitchison, Hay, Slewa-Younan, & Mond, 2014).

Truth #5: Summary and future research directions

Confidence ratings: Moderate (5.5; 5.6); Moderate to High (5.2); High (5.1; 5.3; 5.4) (see Supplementary Table S2)

In summary, no dominant pattern of age, body size, sexual orientation or gender identity, race, ethnicity, or socioeconomic status is associated with eating disorder risk. Providers should remain vigilant to eating disorders in all individuals regardless of demographic characteristics. Further research on socioeconomic status and eating disorders are needed to clarify inconsistent patterns observed and proposed genetic associations.

Longitudinal studies that consider weight trajectories as they relate to eating disorder symptom development are needed, as it is clear that individuals may develop eating disorders from any premorbid weight. More research on eating disorders among sexual minorities is also necessary for the development of targeted prevention and intervention efforts, specifically

longitudinal studies that examine how sexual and gender identity development in youth may impact eating disorder risk.

Truth #6: Eating disorders carry an increased risk for both suicide and medical complications.

6.1 Eating disorders are associated with premature death.

The most significant medical complication of an eating disorder is premature death. The standardized mortality ratio (SMR) associated with AN ranges between 5.9 and 6.2, meaning the risk of death for individuals with AN is up to 6.2 times greater than the risk in the general population, and the weighted annual mortality rate of AN is reported as 5.1 per 1000 person years (Chesney, Goodwin, & Fazel, 2014; Papadopoulos, Ekbom, Brandt, & Ekselius, 2009). Additionally, for females with AN between the ages of 15-24 years old, the mortality rate is 12 times higher than the death rate of all other causes of death (Klump, Bulik, Kaye, Treasure, & Tyson, 2009). Notably, AN also has one of the highest mortality rates of any psychiatric illness (Chesney et al., 2014), and one in five deaths in AN is attributable to suicide (Arcelus, Mitchell, Wales, & Nielsen, 2011).

The mortality rate for BN is also significantly elevated relative to the general population, with meta-analyses estimating the SMR for BN to be 1.9 (Chesney et al., 2014). For those with BN, mortality risk may increase with severity (Huas et al., 2013). One clinical follow-up study in Finland found the all-cause mortality hazard ratio for BED to be 1.77 (0.60, 5.27) (Suokas et al., 2013). Though similar in effect size to reported SMRs for BN, this hazard ratio for BED was not significant. With the inclusion of BED in the DSM-5, more studies on epidemiology, course, and outcome of BED are likely.

6.2 Risk of suicide is elevated in eating disorders.

The risk of suicide attempts is also elevated in eating disorders. In the Swedish population born between 1979 and 2001, the odds ratio (OR) of suicide attempts was estimated to be 5.3 (95% CI: 5.0, 5.5) for any eating disorder, meaning that the risk of suicide attempts in people with eating disorders is 5.3 times the risk in individuals without an eating disorder. The ORs for suicide were 4.4 (95% CI: 4.1, 4.7) for AN and 6.3 (95% CI: 5.7, 6.9) for BN (Yao et al., 2016). Similar relative risks have been reported in the Danish population for the period between 1989 and 2006 (Zerwas et al., 2015). A large clinical study found that 35.6% of eating disorder patients had attempted suicide at least once, and patients with binge eating and/or purging behaviors were associated with an elevated risk for suicide attempts compared with patients without such behaviors (Fedorowicz et al., 2007; Foulon et al., 2007). In Sweden, 13.6% of women with a lifetime history of BED had at least one lifetime suicide attempt (Pisetsky, Thornton, Lichtenstein, Pedersen, & Bulik, 2013; Runfola, Thornton, Pisetsky, Bulik, & Birgegård, 2014).

Based on a meta-analysis, the *suicide-specific* SMR is 18.1 (95% CI: 11.5, 28.7) for AN (Keshaviah et al., 2014). Among female AN patients in specialized care, this ratio could be as high as 31.0 (95% CI: 21.0, 44.0) (Preti, Rocchi, Sisti, Camboni, & Miotto, 2011). The suicide-specific SMR is reported as 7.5 (95% CI: 1.6, 11.6) for BN (Preti et al., 2011) and no deaths by suicide in individuals with BED were reported; however, more data for BED are expected to emerge as recognition and reporting of BED increases. Familial co-aggregation of eating disorders and suicide attempt has been observed in nationwide population data (Yao et al., 2016). Two studies from Australia (Wade, Fairweather-Schmidt, Zhu, & Martin, 2015) and Sweden (Thornton, Welch, Munn-Chernoff, Lichtenstein, & Bulik, 2016) have reported that the co-occurrence of eating disorders and suicide may be in part due to shared genetic factors.

Whereas women with disordered eating in the community may be more likely to attempt suicide than males (Davison, Marshall-Fabien, & Gondara, 2014), no sex differences have been found for the risk of suicide attempts or death by suicide in eating disorders (Yao et al., 2016).

Truth #6: Summary and future research directions

Confidence ratings: High (6.1;6.2) (see Supplementary Table S2)

Increased risk of premature death, including suicide, among eating disorders is well established; however, little is known about the mechanism underlying this association. Future investigations should consider why eating disorders specifically display increased risk for suicide and examine how psychobiological models of suicide (Anestis et al., 2016) may pertain to those with eating disorders, including how unique complications associated with eating disorders, such as nutritional status, may influence risk as proposed by these models.

Truth #7: Genes and environment play important roles in the development of eating disorders.

7.1 Eating disorders run in families.

Family, twin, and genetic research has established that eating disorders run in families and genes play a role in this familial pattern (Yilmaz, Hardaway, & Bulik, 2015). Familial history of AN increases the risk of AN development fourfold compared with the general population (Steinhausen, Jakobsen, Helenius, Munk-Jørgensen, & Strober, 2015). Furthermore, AN, BN, and eating disorder not otherwise specified (EDNOS) track together in families, suggesting a lack of specificity (Lilenfeld et al., 1998; Strober, Freeman, Lampert, Diamond, & Kaye, 2000). BED also aggregates in families independent of obesity (Fowler & Bulik, 1997; Hudson et al., 2006). Twin studies cannot identify which genes influence risk, but they have identified a strong genetic

contribution in AN, BN, and BED. Specifically, 48-74% of the total variance in liability to AN, 55-62% to BN, and 39-45% to BED is attributable to genetic factors (Yilmaz et al., 2015).

7.2 Genes play a role in eating disorder risk.

Genome-wide association studies (GWAS), which scan the entire genome in a hypothesis-free manner, and related approaches such as exome sequencing and whole genome sequencing have rapidly accelerated the field. The Eating Disorders Working Group of the Psychiatric Genomics Consortium (PGC-ED) recently identified the first genome-wide significant locus for AN (Duncan et al., 2017) in an area that harbors genes previously implicated in type 1 diabetes and other autoimmune disorders. We expect this will mark an inflection point in genomic discovery if AN follows the same progression of findings as other psychiatric disorders such as schizophrenia, where increased sample size has led to fruitful genomic discovery (Schizophrenia Working Group of the Psychiatric Genomics Consortium, 2014). GWAS represent a starting point for genomic discovery, as post-GWAS science reveals causative biological pathways and the functional significance of implicated genes and epigenetic enhancer regions. No GWAS of BN or BED have been conducted to date. In addition to GWAS approaches, familial linkage analysis with whole-genome and exome sequencing has identified two potential missense mutations (Cui et al., 2013), which evidence a connection with eating-disordered behaviors in a recent mouse model (Lutter et al., 2017).

7.3 Environmental factors play a role in eating disorder risk.

Genes do not act alone: environment plays an important role. Cross-sectional and longitudinal twin studies also indicate that nonshared environmental factors account for variance in eating disorder symptoms. Cultural pressure for thinness has been identified as a specific risk factor for eating disorders, and clinical trials of interventions that reduce thin-ideal internalization have led to

reductions in eating disorder symptoms (Culbert et al., 2015). While thin-ideal internalization may have some genetic influence, one longitudinal twin study indicates that nonshared environmental influences were most important in the etiology of thin-ideal internalization (Suisman et al., 2014).

7.4 Only a small portion of individuals exposed to environmental risk develop eating disorders.

Dieting, drive for thinness, and portion size escalation are widespread in industrialized countries and may represent risk scenarios for the development of eating disorders (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; Steenhuis & Vermeer, 2009; Striegel-Moore & Bulik, 2007); however, despite nearly ubiquitous exposure, threshold illnesses are disproportionately rare. A current hypothesis is that individuals *genetically predisposed* to eating disorders are most vulnerable to societal pressures and environmental insults. Eating disorders are "complex traits," meaning that multiple genetic and environmental factors—each of small to moderate effect— act together to increase risk. Genetic and environmental factors may not only act in an additive manner, but may co-act in other ways (see Truth #8).

Truth #7: Summary and future research directions

Confidence rating: Moderate (7.4); Moderate to High (7.1); High (7.2; 7.3) (see Supplementary Table S2)

Genomic discovery in AN is accelerating rapidly, but work on BN and BED is woefully behind. Very large sample sizes (in the tens of thousands) are key to discovering genetic variants associated with risk, and global cooperation is underway to achieve such sample sizes. Advances in genetic methodology, coupled with increasing knowledge about environmental risk factors, will provide a more complete and accurate picture of eating disorder etiology.

Truth #8: Genes alone do not predict who will develop eating disorders.

8.1 Eating disorders do not follow Mendelian transmission patterns.

Inheritance patterns for eating disorders do not follow the traditional Mendelian patterns where variation in one gene results in one disorder (e.g., Huntington's chorea). Rather, hundreds (or perhaps thousands) of genes act in concert and are influenced by environmental factors. An individual's risk is a composite of the cumulative number of genetic and environmental risk and protective factors to which they are exposed. This pattern is supported by several case-control studies examining candidate genes that show inconsistent effects (see Yilmaz et al., 2015 for a review).

8.2 Many cases of eating disorders are sporadic, meaning there is no known family member who suffers from an eating disorder.

Family studies indicate that the relative risk for eating disorders is higher in family members of affected individuals; however, the majority of affected individuals have no known affected family members (Bould et al., 2015; Steinhausen et al., 2015; Strober et al., 2000). This literature is limited in that eating disorder history among relatives may not be fully known or accurately captured.

8.3 Genes and environment may co-act to influence risk for eating disorders.

Genes represent probabilities in all complex traits, such as eating disorders. Individuals with a high genetic susceptibility for disordered eating may be protected by other factors, whereas individuals at relatively low genetic risk may be burdened with cumulative or extreme environmental insults leading to possible eating disorder development despite their favorable genetic profile. Understanding the role that genes and environment play in eating disorders requires a deep acceptance of probability and of uncertainty.

Genes and environment may co-act to influence risk for eating disorders (Trace, Baker, Peñas-Lledó, & Bulik, 2013). First, in most families, parents and extended family provide both genes and shared environment, meaning that these two factors are confounded. Second, individuals with a stronger genetic susceptibility for eating disorders might be more sensitive to environmental factors (dieting, bullying, teasing, or overeating). Whereas many adolescents may try dieting, only for a few does it serve as an environmental trigger for an underlying genetic predisposition. Third, an individual who is genetically predisposed to traits associated with eating disorders (e.g., perfectionism, persistence, high physical activity) can seek out environments that may serve as triggers (e.g., sports that have a lean body type ideal, certain social media content) (Carrotte, Vella, & Lim, 2015; Giel et al., 2016; Rousselet et al., 2017). This phenomenon is known as an active gene-environment correlation (Plomin, DeFries, & Loehlin, 1977). Genetic research combined with ambulatory assessment may help understand how environmental influences affect risk for eating disorders by pinpointing specificity of risk factors.

Rigorous studies of gene-environment interaction in eating disorders are sparse. Some developmental twin studies have examined gene-environment interaction (Culbert et al., 2015). For example, contribution of genetic risk to the emergence of dysfunctional eating attitudes and disordered eating varies with developmental stage, with higher genetic effects observed in mid-to-late adolescence and mid-to-late puberty (Culbert et al., 2015; Culbert, Burt, McGue, Iacono, & Klump, 2009; Klump, Burt, McGue, & Iacono, 2007). More sophisticated analytic techniques that examine *interplay* between genetic risk and family environment provide indication that fit between an individual's genotype and their family environment may be relevant for eating disorder risk (Culbert et al., 2015). In human studies, large samples using genome-wide and phenome-wide data are required for credible conclusions. Following a report of a rare missense mutation being

associated with the development of eating disorders, Lutter et al. (2017) found that group (vs. individually) housed transgenic female mice displayed irregular feeding and anxiety behaviors, preliminarily revealing both sex-specific and gene by environment effects.

Additional ways in which genes and environment interact are via mechanisms collectively called epigenetics—the modification of DNA, RNA, or proteins by biological or environmental factors. These mechanisms alter gene expression without changing the DNA sequence. Importantly, epigenetic changes such as DNA methylation are tissue specific and can rarely be directly studied in the brain. Therefore, it is important to determine whether epigenetic changes seen in blood are good proxies for epigenetic changes in brain (Walton et al., 2016).

Preliminary epigenetic studies have reported changes in dopaminergic genes and genes for proopiomelancortin (*POMC*), cannabinoid receptor 1 (*CNR1*, also referred to as *CB1*), atrial natriuretic peptide (*NPPA*, also referred to as *ANP*), alpha synuclein (*SNCA*), and oxytocin receptor (*OXTR*) (Ehrlich et al., 2010; Ehrlich et al., 2012; Frieling et al., 2007; Frieling et al., 2008; Frieling et al., 2010; Kim, Kim, Kim, & Treasure, 2014; Schroeder et al., 2012). If replicated, epigenetic findings could make important contributions to understanding the role of of non-DNA elements in eating disorder susceptibility.

Truth #8: Conclusions and future research directions

Confidence ratings: Low (8.2;8.3); Moderate (8.1) (see Supplementary Table S2)

A complex interplay between genetic and environmental factors underlies the development of eating disorders. Future research on genetic pathways and their interplay with environmental factors is an exciting and emerging area of research—and one that has the potential to provide key understanding of the multiple and nuanced facets by which individuals may develop eating pathology. In the short-term, large population-based studies with both genotypic and phenotypic

information to probe gene-environment interactions, along with case-control studies to examine potential epigenetic effects represent key areas for advancing knowledge regarding complex risk patterns.

9. Truth #9: Full recovery from an eating disorder is possible. Early detection and intervention are important.

9.1 A substantial portion of individuals with eating disorders achieve recovery.

Full recovery from an eating disorder is not only possible, but indeed probable. A substantial portion of individuals with eating disorders achieve recovery, some without seeking treatment (Eddy et al., 2016; Keel & Brown, 2010; Steinhausen & Weber, 2009; Steinhausen, 2009). Five-year clinical recovery rates have been estimated at 67% for AN (Keski-Rahkonen et al., 2007) and 55% for BN (Keski-Rahkonen et al., 2009) in community samples, and by 10 years after eating disorder onset 70% of individuals are recovered (Berkman, Lohr, & Bulik, 2007). Although recovery is attainable, there is a lack of consensus on the exact definition of recovery, making it difficult to compare recovery rates across studies (Bardone-Cone et al., 2010; Emanuelli, Waller, Jones-Chester, & Ostuzzi, 2012). Traditionally, these definitions focus on physical and behavioral recovery. Physical recovery refers to the resumption and maintenance of a healthy body weight and a normalization of all physical parameters affected by the eating disorder, whereas behavioral recovery means the absence of eating-disorder related behaviors such as food restriction, binge eating, and purging. Psychological recovery, including the attainment of normal attitudes toward food and the body, is important yet often overlooked. It has been proposed that full recovery is achieved only when patients are indistinguishable from healthy controls on all eating disorder related measures, including psychological aspects (Bardone-Cone et al., 2010). Although this definition may seem stringent, it is attainable. Full recovery from an eating disorder is possible,

and given that lingering eating disorder attitudes predict relapse (Helverskov et al., 2010), the psychological component of recovery is clinically relevant.

9.2 Early detection and intervention may improve prognosis.

For some, recovery from an eating disorder is possible without treatment; however, early detection and intervention are preferred for all eating disorders (Treasure et al., 2015). For AN, a longer duration of illness before presentation for treatment is associated with poor outcome (Keel & Brown, 2010; Pike, 1998; Richard, Bauer, & Kordy, 2005), and the probability of recovering decreases as a function of duration of illness, irrespective of treatment (Pike, 1998). For BN, some studies find that a longer duration of illness is associated with poor outcome, whereas others observe that severity of illness and additional psychiatric comorbidities are more significant predictors of outcome (Steinhausen & Weber, 2009). However, in general, the sooner an eating disorder is identified and treatment can begin, the better prognosis there is for full recovery.

9.3 Effective psychological interventions for eating disorders exist. Many, but not all, patients benefit. & 9.4 Medication can be an effective treatment component for eating disorders.

Treatment for an eating disorder typically includes psychological treatment and may include medication (Zipfel, Giel, Bulik, Hay, & Schmidt, 2015). For AN, weight restoration is an essential first step in treatment. Inpatient renourishment for AN is typically directed by clinical guidelines that advocate for a "low and slow" approach, due to concerns about refeeding syndrome (Solomon & Kirby, 1990). However, this approach is being challenged in favor of more aggressive renourishment techniques, leading to shorter hospital stays and a favorable safety profile (Garber et al., 2013; Madden et al., 2015; Redgrave et al., 2015). Once medical stabilization of an eating disorder is established, patients may step down to other levels of care. The evidence base has been thoroughly reviewed for psychotherapeutic and medication interventions for eating disorders.

Supplementary Tables S5 & S6 provide an overview of psychotherapeutic and medication treatments.

Truth #9: Summary and future research directions

Confidence Ratings: Low (9.4 for AN); Moderate (9.2); High (9.1; 9.3; 9.4 for BN/BED) (see Supplementary Table S2)

Increasing understanding of the mechanisms underlying eating disorders will facilitate the development of more effective and personalized prevention and treatment options, eventually leading to increased recovery rates and shorter recovery times. Some evidence-based treatments have proven efficacy. Importantly, recovery from eating disorders can and does occur at any age and for those who do not achieve complete remission, quality of life and somatic status may be improved, monitored, and stabilized (Treasure, Stein, & Maguire, 2015). Future research goals include development of strategies for early detection and intervention, development of a provider's toolbox that includes psychological and pharmacological interventions that are effective for a range of eating disorders in diverse populations, drug development or repurposing investigations to target core biological pathology of AN, studies of long-term efficacy of medication interventions for all eating disorders, and studies of the effectiveness of medications for eating disorders in community settings.

General conclusion

We summarize the available literature that led to the development of the "*Nine Truths About Eating Disorders."* Eating disorders are not choices and do affect individuals from all walks of life. They result from a combination of biological (including genetic) and environmental factors. Eating disorders increase the risk for suicide and medical complications, and interrupt personal and family functioning. Families are not to blame and can be critical sources of support in recovery.

Clearly, additional work is needed to better understand risk factors, course of illness, and treatment of eating disorders. Important for advancing science in this area is the ability to remain flexible in thinking about causal factors and acknowledge accumulating evidence underlying these truths to eliminate misconceptions that have plagued the field for decades. In addition, providers should be mindful of the multitude of ways eating disorders can arise and be especially vigilant to signs of somatic and psychiatric complications resulting from AN, BN, and BED. As scientists, providers, patients, family, and friends, we need to continue educating others in the community about these truths in order to detect and treat eating disorders as soon as possible.

Yet, the science of this field cannot be advanced in the absence of appropriate investment and financial support from organizations worldwide that fund research. A 2015 blog post by the former director of the US National Institute of Mental Health, Thomas Insel, MD, revealed how woefully underfunded research on eating disorders was relative to the disability-adjusted life years associated with the illnesses (http://www.nimh.nih.gov/funding/funding-strategy-for-research-grants/white-paper_149362.pdf). Despite the dire morbidity and mortality statistics, eating disorders continue to be low-priority illnesses, we contend, in part due to long-standing misconceptions about their causes and consequences. Funding is required for larger more definitive collaborative studies to avoid the confusion that arises from conflicting results from small, underfunded, underpowered, and unreplicated investigations. Far too often, such small-budget studies are all that investigators can afford to conduct.

Science is constantly evolving, and novel methods will enhance our ability to clarify the etiology of eating disorders and to develop scientifically informed and effective treatments for these debilitating illnesses. With adequate support for science, emerging information will facilitate the refinement of the *Nine Truths* and may in fact uncover new truths. Ultimately, it is our hope

that dissemination of the *Nine Truths* will serve to reduce stigma and misunderstanding, and, via their impact on science and practice, reduce illness burden, improve quality of life, and eliminate mortality from eating disorders.

Annotated Bibliography

Ahrén, J. C., Chiesa, F., Koupil, I., Magnusson, C., Dalman, C., & Goodman, A. (2013). We are family--parents, siblings, and eating disorders in a prospective total-population study of 250,000 Swedish males and females. *International Journal of Eating Disorders*, *46*, 693-700. https://doi.org/10.1002/eat.22146

Ali, K., Farrer, L., Fassnacht, D. B., Gulliver, A., Bauer, S., & Griffiths, K. M. (2017). Perceived barriers and facilitators towards help-seeking for eating disorders: A systematic review. *International Journal of Eating Disorders*, *50*, 9-21. https://doi.org/10.1002/eat.22598

Alvy, L. M. (2013). Do lesbian women have a better body image? Comparisons with heterosexual women and model of lesbian-specific factors. *Body Image*, *10*, 524-534. https://doi.org/10.1016/j.bodyim.2013.06.002

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed). Arlington, VA: American Psychiatric Publishing.

American Psychiatric Association. (2006). Practice guideline for the treatment of patients with eating disorders. Retrieved from http://psychiatryonline.org/pb/assets/raw/sitewide/practice_guidelines/guidelines/eatingdisorders.pdf

Anastasiadou, D., Medina-Pradas, C., Sepulveda, A. R., & Treasure, J. (2014). A systematic review of family caregiving in eating disorders. *Eating Behaviors*, *15*, 464-477. https://doi.org/10.1016/j.eatbeh.2014.06.001

Anderson, C. B., & Bulik, C. M. (2004). Gender differences in compensatory behaviors, weight and shape salience, and drive for thinness. *Eating Behaviors*, *5*, 1-11. https://doi.org/10.1016/j.eatbeh.2003.07.001

Anestis, J. C., Anestis, M. D., Rufino, K. A., Cramer, R. J., Miller, H., Khazem, L. R., & Joiner, T. E. (2016). Understanding the relationship between suicidality and psychopathy: An examination of the Interpersonal-Psychological Theory of Suicidal Behavior. *Archives of Suicide Research*, *20*, 349-368. https://doi.org/10.1080/13811118.2015.1048399

Arcelus, J., Mitchell, A. J., Wales, J., & Nielsen, S. (2011). Mortality rates in patients with anorexia nervosa and other eating disorders. A meta-analysis of 36 studies. *Archives of General Psychiatry*, *68*, 724-731. https://doi.org/10.1001/archgenpsychiatry.2011.74

Authors meta-analyzed 36 quantitative studies covering the mortality of eating disorders. The weighted mortality rates (i.e., deaths per 1000 person-years) were 5.1 for AN, 1.7 for BN, and 3.3 for EDNOS. The standardized mortality ratios were 5.86 for AN, 1.93 for BN, and 1.92 for EDNOS. One in 5 individuals with AN who died had committed suicide.

Atasoy, D., Betley, J. N., Su, H. H., & Sternson, S. M. (2012). Deconstruction of a neural circuit for hunger. *Nature*, 488,172-177. https://doi.org/10.1038/nature11270

Demonstration that cell and pathway specific optogenetic activation of a pathway from Agouti related peptide-expressing neurons in the arcuate nucleus to the paraventricular nucleus of the hypothalamus induces hunger.

Austin, S. B., Ziyadeh, N. J., Forman, S., Prokop, L. A., Keliher, A., & Jacobs, D. (2008). Screening high school students for eating disorders: Results of a national initiative. *Prevention of Chronic Disease*, *5*, A114.

Baker, J. H., Girdler, S. S., & Bulik, C. M. (2012). The role of reproductive hormones in the development and maintenance of eating disorders. *Expert Review of Obstetrics and Gynecology*, *7*, 573-583. https://doi.org/10.1586/eog.12.54

Baker, J. H., Peterson, C. M., Thornton, L., Brownley, K. A., Bulik, C. M., Girdler, S. S., . . . Bromberger, J. T. (2017). Reproductive and appetite hormones and bulimic symptoms during midlife. *European Eating Disorders Review*, *25*, 188-194. https://doi.org/10.1002/erv.2510 *Authors compared bulimic symptoms in premenopausal and perimenopausal midlife women and examined the association between these symptoms and reproductive and appetite hormones. No mean differences in bulimic symptoms were observed between premenopause and perimenopause. A significant positive association between leptin and binge eating was observed.*

Baker, J. H., & Runfola, C. D. (2016). Eating disorders in midlife women: A perimenopausal eating disorder. *Maturitas*, *85*, 112-116. https://doi.org/10.1016/j.maturitas.2015.12.017

Bardone-Cone, A. M., Harney, M. B., Maldonado, C. R., Lawson, M. A., Robinson, D. P., Smith, R., & Tosh, A. (2010). Defining recovery from an eating disorder: Conceptualization, validation, and examination of psychosocial functioning and psychiatric comorbidity. *Behavior Research and Therapy*, *48*, 194-202. https://doi.org/10.1016/j.brat.2009.11.001

Benes, F. M. (1998). Brain development, VII. Human brain growth spans decades. *American Journal of Psychiatry*, *155*, 1489. https://doi.org/10.1176/ajp.155.11.1489

Berg, K. C., Crosby, R. D., Cao, L., Peterson, C. B., Engel, S. G., Mitchell, J. E., & Wonderlich, S. A. (2013). Facets of negative affect prior to and following binge-only, purge-only, and binge/purge events in women with bulimia nervosa. *Journal of Abnormal Psychology*, *122*, 111-118. https://doi.org/10.1037/a0029703

Berkman, N. D., Bulik, C. M., Brownley, K. A., Lohr, K. N., Sedway, J. A., Rooks, A., & Gartlehner, G. (2006). Management of eating disorders. *Evidence Report/Technology Assessment*, *135*, 1-166.

Berkman, N. D., Lohr, K. N., & Bulik, C. M. (2007). Outcomes of eating disorders: A systematic review of the literature. *International Journal of Eating Disorders*, *40*, 293-309. https://doi.org/10.1002/eat.20369

Bernardoni, F., King, J. A., Geisler, D., Stein, E., Jaite, C., Nätsch, D., . . . Ehrlich, S. (2016). Weight restoration therapy rapidly reverses cortical thinning in anorexia nervosa: A longitudinal study. *Neuroimage*, *130*, 214-222. https://doi.org/10.1016/j.neuroimage.2016.02.003

Grey matter reductions which are typically found in acutely underweight anorexia nervosa patients were found to be reversed at a rate much faster than previously thought upon successful weight gain.

Blakemore, S. J., & Choudhury, S. (2006). Development of the adolescent brain: Implications for executive function and social cognition. *Journal of Child Psychology and Psychiatry*, *47*, 296-312. https://doi.org/10.1111/j.1469-7610.2006.01611.x

Bould, H., Sovio, U., Koupil, I., Dalman, C., Micali, N., Lewis, G., & Magnusson, C. (2015). Do eating disorders in parents predict eating disorders in children? Evidence from a Swedish cohort. *Acta Psychiatrica Scandinavica*, *132*, 51-59. https://doi.org/10.1111/acps.12389

In a sample of 158,697 children born in Stockholm county 1984-1995, Sweden, the authors tested whether the diagnosis of an eating disorder in a parent was predictive of a diagnosis of an eating disorder in the offspring. Due to low rates of eating disorders in males, analyses were restricted to females who were found to be at increased risk of being diagnosed with an eating disorder.

Brown, T. A., & Keel, P. K. (2012). The impact of relationships on the association between sexual orientation and disordered eating in men. *International Journal of Eating Disorders*, *45*, 792-799. https://doi.org/10.1002/eat.22013

Bulik, C. M. (2016). Towards a science of eating disorders: Replacing myths with realities: The fourth Birgit Olsson lecture. *Nordic Journal of Psychiatry*, *70*, 224-230. https://doi.org/10.3109/08039488.2015.1074284

Bulik, C. M., Baucom, D. H., Kirby, J. S., & Pisetsky, E. (2011). Uniting Couples (in the treatment of) Anorexia Nervosa (UCAN). *International Journal of Eating Disorders*, *44*, 19-28. https://doi.org/10.1002/eat.20790

Uniting Couples (in the treatment of) Anorexia Nervosa (UCAN) is a couple based intervention founded on cognitive-behavioral couple therapy principles. The authors discuss the delivery of the treatment and highlight its potential to enhance both retention and treatment outcome.

Bulik, C. M., Von Holle, A., Hamer, R., Knoph Berg, C., Torgersen, L., Magnus, P., . . . Reichborn-Kjennerud, T. (2007). Patterns of remission, continuation and incidence of broadly defined eating disorders during early pregnancy in the Norwegian Mother and Child Cohort Study (MoBa). *Psychological Medicine*, *37*, 1109-1118. https://doi.org/10.1017/S0033291707000724

Campbell, K., & Peebles, R. (2014). Eating disorders in children and adolescents: State of the art review. *Pediatrics*, *134*, 582-592. https://doi.org/10.1542/peds.2014-0194

Carrotte, E. R., Vella, A. M., & Lim, M. S. (2015). Predictors of "liking" three types of health and fitness-related content on social media: A cross-sectional study. *Journal of Medical Internet Research*, *17*, e205. https://doi.org/10.2196/jmir.4803

Carter, J. C., & Bewell-Weiss, C. V. (2011). Nonfat phobic anorexia nervosa: Clinical characteristics and response to inpatient treatment. *International Journal of Eating Disorders*, *44*, 220-224. https://doi.org/10.1002/eat.20820

Caslini, M., Crocamo, C., Dakanalis, A., Tremolada, M., Clerici, M., & Carrà, G. (2016). Stigmatizing attitudes and beliefs about anorexia and bulimia nervosa among Italian undergraduates. *Journal of Nervous and Mental Disease*, *204*, 916-924. https://doi.org/10.1097/NMD.0000000000000606

Chesney, E., Goodwin, G. M., & Fazel, S. (2014). Risks of all-cause and suicide mortality in mental disorders: A meta-review. *World Psychiatry*, *13*, 153-160. https://doi.org/10.1002/wps.20128

Chowdhury, T. G., Chen, Y. W., & Aoki, C. (2015). Using the activity-based anorexia rodent model to study the neurobiological basis of anorexia nervosa. *Journal of Visualized Experiments*, *105*, e52927. https://doi.org/10.3791/52927

Collantoni, E., Michelon, S., Tenconi, E., Degortes, D., Titton, F., Manara, R., . . . Favaro, A. (2016). Functional connectivity correlates of response inhibition impairment in anorexia nervosa. *Psychiatry Research*, *247*, 9-16. https://doi.org/10.1016/j.pscychresns.2015.11.008

Cook-Darzens, S. (2016). The role of family meals in the treatment of eating disorders: A scoping review of the literature and implications. *Eating and Weight Disorders*, *21*, 383-393. https://doi.org/10.1007/s40519-016-0263-y

Couturier, J., Kimber, M., & Szatmari, P. (2013). Efficacy of family-based treatment for adolescents with eating disorders: A systematic review and meta-analysis. *International Journal of Eating Disorders*, *46*, 3-11. https://doi.org/10.1002/eat.22042

Cui, H., Moore, J., Ashimi, S. S., Mason, B. L., Drawbridge, J. N., Han, S., . . . Lutter, M. (2013). Eating disorder predisposition is associated with ESRRA and HDAC4 mutations. *Journal of Clinical Investigation*, *123*, 4706-4713. https://doi.org/10.1172/JCI71400

Culbert, K. M., Burt, S. A., McGue, M., Iacono, W. G., & Klump, K. L. (2009). Puberty and the genetic diathesis of disordered eating attitudes and behaviors. *Journal of Abnormal Psychology*, *118*, 788-796. https://doi.org/10.1037/a0017207

Culbert, K. M., Racine, S. E., & Klump, K. L. (2015). Research Review: What we have learned about the causes of eating disorders - a synthesis of sociocultural, psychological, and biological research. *Journal of Child Psychology and Psychiatry*, *56*, 1141-1164. https://doi.org/10.1111/jcpp.12441

Culbert, K. M., Racine, S. E., & Klump, K. L. (2016). Hormonal factors and disturbances in eating disorders. *Current Psychiatry Reports*, *18*, 65. https://doi.org/10.1007/s11920-016-0701-6

The role of hormonal factors influencing eating disorders is still unknown. However, the strongest evidence for etiologic effects has emerged for ovarian hormones, suggesting that estradiol reduces food intake whereas progesterone and testosterone increase food intake.

Currin, L., Schmidt, U., Treasure, J., & Jick, H. (2005). Time trends in eating disorder incidence. *British Journal of Psychiatry*, *186*, 132-135. https://doi.org/10.1192/bjp.186.2.132

Currin, L., Schmidt, U., & Waller, G. (2007a). Variables that influence diagnosis and treatment of the eating disorders within primary care settings: A vignette study. *International Journal of Eating Disorders*, *40*, 257-262. https://doi.org/10.1002/eat.20355

Currin, L., Waller, G., & Schmidt, U. (2009). Primary care physicians' knowledge of and attitudes toward the eating disorders: Do they affect clinical actions. *International Journal of Eating Disorders*, *42*, 453-458. https://doi.org/10.1002/eat.20636

Currin, L., Waller, G., Treasure, J., Nodder, J., Stone, C., Yeomans, M., & Schmidt, U. (2007b). The use of guidelines for dissemination of "best practice" in primary care of patients with eating

disorders. *International Journal of Eating Disorders*, *40*, 476-479. https://doi.org/10.1002/eat.20385

Dalle Grave, R., Calugi, S., & Marchesini, G. (2008). Underweight eating disorder without over-evaluation of shape and weight: Atypical anorexia nervosa. *International Journal of Eating Disorders*, *41*, 705-712. https://doi.org/10.1002/eat.20555

Davison, K. M., Marshall-Fabien, G. L., & Gondara, L. (2014). Sex differences and eating disorder risk among psychiatric conditions, compulsive behaviors and substance use in a screened Canadian national sample. *General Hospital Psychiatry*, *36*, 411-414. https://doi.org/10.1016/j.genhosppsych.2014.04.001

Diemer, E. W., Grant, J. D., Munn-Chernoff, M. A., Patterson, D. A., & Duncan, A. E. (2015). Gender identity, sexual orientation, and eating-related pathology in a national sample of college students. *Journal of Adolescent Health*, *57*, 144-149. https://doi.org/10.1016/j.jadohealth.2015.03.003

In a sample of nearly 300,000 U.S. college students, transgender and cisgender sexual minority young adults reported a higher prevalence of past-year self-reported eating disorder diagnosis and past-month compensatory behaviors (i.e., self-induced vomiting, diet pills, and laxatives) than their cisgender heterosexual peers.

Dimitropoulos, G., McCallum, L., Colasanto, M., Freeman, V. E., & Gadalla, T. (2016). The effects of stigma on recovery attitudes in people with anorexia nervosa in intensive treatment. *Journal of Nervous and Mental Disease*, *204*, 370-380. https://doi.org/10.1097/NMD.0000000000000480

Duncan, A. E., Ziobrowski, H. N., & Nicol, G. (2017). The prevalence of past 12-month and lifetime DSM-IV eating disorders by BMI category in US men and women. *European Eating Disorders Review*, https://doi.org/10.1002/erv.2503

In a U.S. general population sample, lifetime eating disorder prevalence was 2.22% in men and 4.93% in women. The prevalence of any lifetime and past 12-month ED, binge eating disorder and recurrent binge eating was highest among obese individuals.

Duncan, L., Yilmaz, Z., Gaspar, H., Walters, R., Goldstein, J., Antilla, V., Bulik-Sullivan, B., . . . Bulik, C. M. (in press). Genome-wide association study reveals first locus for anorexia nervosa and metabolic correlations. *American Journal of Psychiatry.*

First identified genome-wide significant locus for anorexia nervosa on chromosome 12 (rs4622308) in a region harboring a previously reported type 1 diabetes and autoimmune disorder locus. Significant positive genetic correlations observed between anorexia nervosa and schizophrenia, neuroticism, educational attainment, and high-density lipoprotein cholesterol, and significant negative genetic correlations were observed between anorexia nervosa and body mass index, insulin, glucose, and lipid phenotypes.

Eddy, K. T., Tabri, N., Thomas, J. J., Murray, H. B., Keshaviah, A., Hastings, E. R., . . . Franko, D. L. (2017). Recovery from anorexia nervosa and bulimia nervosa at 22-year follow-up. *Journal of Clinical Psychiatry*, *78*, 184-189. https://doi.org/10.40888/JCP.15m10393

Edler, C., Lipson, S. F., & Keel, P. K. (2007). Ovarian hormones and binge eating in bulimia nervosa. *Psychological Medicine*, *37*, 131-141. https://doi.org/10.1017/S0033291706008956

Ehrlich, S., Burghardt, R., Schneider, N., Broecker-Preuss, M., Weiss, D., Merle, J. V., . . . Hebebrand, J. (2009). The role of leptin and cortisol in hyperactivity in patients with acute and weight-recovered anorexia nervosa. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *33*, 658-662. https://doi.org/10.1016/j.pnpbp.2009.03.007

Ehrlich, S., Geisler, D., Ritschel, F., King, J. A., Seidel, M., Boehm, I., . . . Kroemer, N. B. (2015). Elevated cognitive control over reward processing in recovered female patients with anorexia nervosa. *Journal of Psychiatry and Neuroscience*, *40*, 307-315.

Individuals recovered from anorexia nervosa showed elevated brain activity in the dorsolateral prefrontal cortex (DLPFC) as well as greater functional coupling between the DLPFC and the orbitofrontal cortex during the anticipation phase of a monetary reward paradigm. The findings are suggestive of elevated self-regulatory processes in response to rewarding stimuli in patients recovered from anorexia nervosa.

Ehrlich, S., Walton, E., Roffman, J. L., Weiss, D., Puls, I., Doehler, N., . . . Frieling, H. (2012). Smoking, but not malnutrition, influences promoter-specific DNA methylation of the proopiomelanocortin gene in patients with and without anorexia nervosa. *Canadian Journal of Psychiatry*, *57*, 168-176.

Ehrlich, S., Weiss, D., Burghardt, R., Infante-Duarte, C., Brockhaus, S., Muschler, M. A., . . . Frieling, H. (2010). Promoter specific DNA methylation and gene expression of POMC in acutely underweight and recovered patients with anorexia nervosa. *Journal of Psychiatric Research*, *44*, 827-833. https://doi.org/10.1016/j.jpsychires.2010.01.011

Eisler, I. (2005). The empirical and theoretical base of family therapy and multiple family day therapy for adolescent anorexia nervosa. *Journal of Family Therapy*, *27*, 104-131. https://doi.org/10.1111/j.1467-6427.2005.00303.x

Emanuelli, F., Waller, G., Jones-Chester, M., & Ostuzzi, R. (2012). Recovery from disordered eating: Sufferers' and clinicians' perspectives. *European Eating Disorders Review*, *20*, 363-372. https://doi.org/10.1002/erv.2159

In a checklist study, individuals with eating difficulties and clinicians ranked factors associated with recovery. Domains included psychological-emotional-social, weight-controlling behaviors, non-life-threatening and life-threatening features, and evaluation of one's own appearance. Ill individuals and clinicians agreed on the ranking of importance of these factors, but those with eating disturbances, considered 'psychological-emotional-social' and 'evaluation of one's own appearance' criteria as more important to recovery than clinicians.

Fairburn, C. G., Cooper, Z., Doll, H. A., Norman, P., & O'Connor, M. (2000). The natural course of bulimia nervosa and binge eating disorder in young women. *Archives of General Psychiatry*, *57*, 659-665. https://doi.org/10-1001/pubs.Arch Gen Psychiatry-ISSN-0003-990x-57-7-yoa9404

Fedorowicz, V. J., Falissard, B., Foulon, C., Dardennes, R., Divac, S. M., Guelfi, J. D., & Rouillon, F. (2007). Factors associated with suicidal behaviors in a large French sample of inpatients with eating disorders. *International Journal of Eating Disorders*, *40*, 589-595. https://doi.org/10.1002/eat.20415

Field, A. E., Austin, S. B., Taylor, C. B., Malspeis, S., Rosner, B., Rockett, H. R., . . . Colditz, G. A. (2003). Relation between dieting and weight change among preadolescents and adolescents. *Pediatrics*, *112*, 900-906. https://doi.org/10.1542/peds.112.4.900

Flament, M. F., Henderson, K., Buchholz, A., Obeid, N., Nguyen, H. N., Birmingham, M., & Goldfield, G. (2015). Weight status and DSM-5 diagnoses of eating disorders in adolescents from the community. *Journal of the American Academy of Child and Adolescent Psychiatry*, *54*, 403-411.e2. https://doi.org/10.1016/j.jaac.2015.01.020

Föcker, M., Timmesfeld, N., Scherag, S., Bühren, K., Langkamp, M., Dempfle, A., . . . Hebebrand, J. (2011). Screening for anorexia nervosa via measurement of serum leptin levels. *Journal of Neural Transmission*, *118*, 571-578. https://doi.org/10.1007/s00702-010-0551-z

Foulon, C., Guelfi, J. D., Kipman, A., Adès, J., Romo, L., Houdeyer, K., . . . Gorwood, P. (2007). Switching to the bingeing/purging subtype of anorexia nervosa is frequently associated with suicidal attempts. *European Psychiatry*, *22*, 513-519. https://doi.org/10.1016/j.eurpsy.2007.03.004

Fowler, S. J., & Bulik, C. M. (1997). Family environment and psychiatric history in women with binge-eating disorder and obese controls. *Behaviour Change*, 14, 106-112.

Frank, G.K. (2013). Altered brain reward circuits in eating disorders: Chicken or egg. *Current Psychiatry Reports*, *15*, 396. https://doi.org/10.1007/s11920-013-0396-x

Frank, G. K. (2012). Advances in the diagnosis of anorexia nervosa and bulimia nervosa using brain imaging. *Expert Opinion in Medical Diagnosis*, *6*, 235-244. https://doi.org/10.1517/17530059.2012.673583

Frank, G. K. (2015a). Recent advances in neuroimaging to model eating disorder neurobiology. *Current Psychiatry Reports*, *17*, 559. https://doi.org/10.1007/s11920-015-0559-z

Frank, G. K. (2015b). Advances from neuroimaging studies in eating disorders. *CNS Spectrums*, *20*, 391-400. https://doi.org/10.1017/S1092852915000012

French, S. A., Story, M., Remafedi, G., Resnick, M. D., & Blum, R. W. (1996). Sexual orientation and prevalence of body dissatisfaction and eating disordered behaviors: A population-based study of adolescents. *International Journal of Eating Disorders*, *19*, 119-126. https://doi.org/10.1002/(SICI)1098-108X(199603)19:2<119::AID-EAT2>3.0.CO;2-Q

Friederich, H. C., Wu, M., Simon, J. J., & Herzog, W. (2013). Neurocircuit function in eating disorders. *International Journal of Eating Disorders*, *46*, 425-432. https://doi.org/10.1002/eat.22099

Frieling, H., Bleich, S., Otten, J., Römer, K. D., Kornhuber, J., de Zwaan, M., . . . Hillemacher, T. (2008). Epigenetic downregulation of atrial natriuretic peptide but not vasopressin mRNA expression in females with eating disorders is related to impulsivity. *Neuropsychopharmacology*, *33*, 2605-2609. https://doi.org/10.1038/sj.npp.1301662

Frieling, H., Gozner, A., Römer, K. D., Lenz, B., Bönsch, D., Wilhelm, J., . . . Bleich, S. (2007). Global DNA hypomethylation and DNA hypermethylation of the alpha synuclein promoter in females with anorexia nervosa. *Molecular Psychiatry*, *12*, 229-230. https://doi.org/10.1038/sj.mp.4001931

Frieling, H., Römer, K. D., Scholz, S., Mittelbach, F., Wilhelm, J., De Zwaan, M., . . . Bleich, S. (2010). Epigenetic dysregulation of dopaminergic genes in eating disorders. *International Journal of Eating Disorders*, *43*, 577-583. https://doi.org/10.1002/eat.20745

Gagne, D. A., Von Holle, A., Brownley, K. A., Runfola, C. D., Hofmeier, S., Branch, K. E., & Bulik, C. M. (2012). Eating disorder symptoms and weight and shape concerns in a large web-based convenience sample of women ages 50 and above: Results of the Gender and Body Image (GABI) study. *International Journal of Eating Disorders*, *45*, 832-844. https://doi.org/10.1002/eat.22030

Galimberti, E., Martoni, R. M., Cavallini, M. C., Erzegovesi, S., & Bellodi, L. (2012). Motor inhibition and cognitive flexibility in eating disorder subtypes. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *36*, 307-312. https://doi.org/10.1016/j.pnpbp.2011.10.017

Garber, A. K., Mauldin, K., Michihata, N., Buckelew, S. M., Shafer, M. A., & Moscicki, A. B. (2013). Higher calorie diets increase rate of weight gain and shorten hospital stay in hospitalized

adolescents with anorexia nervosa. *Journal of Adolescent Health*, *53*, 579-584. https://doi.org/10.1016/j.jadohealth.2013.07.014

Gaudiani, J. L., & Mehler, P. S. (2016). Rare medical manifestations of severe restricting and purging: "Zebras," missed diagnoses, and best practices. *International Journal of Eating Disorders*, *49*, 331-344. https://doi.org/10.1002/eat.22475

Giel, K. E., Hermann-Werner, A., Mayer, J., Diehl, K., Schneider, S., Thiel, A., . . . GOAL study group. (2016). Eating disorder pathology in elite adolescent athletes. *International Journal of Eating Disorders*, *49*, 553-562. https://doi.org/10.1002/eat.22511

Giel, K. E., Kullmann, S., Preißl, H., Bischoff, S. C., Thiel, A., Schmidt, U., . . . Teufel, M. (2013). Understanding the reward system functioning in anorexia nervosa: Crucial role of physical activity. *Biological Psychology*, *94*, 575-581. https://doi.org/10.1016/j.biopsycho.2013.10.004

Godier, L. R., de Wit, S., Pinto, A., Steinglass, J. E., Greene, A. L., Scaife, J., . . . Park, R. J. (2016). An investigation of habit learning in anorexia nervosa. *Psychiatry Research*, *244*, 214-222. https://doi.org/10.1016/j.psychres.2016.07.051

Exploration of the role of habits, similar to those reported in compulsive disorders, that are hypothesized to play a role in the development and maintenance of anorexia nervosa. In two parallel studies, individuals with the binge/purge subtype of anorexia nervosa, restricting subtype of anorexia nervosa, and individuals recovered from anorexia nervosa did not show reliance on habits compared to healthy controls. Intact goal-directed learning was evident across all subtypes of anorexia nervosa.

Goodman, A., Heshmati, A., & Koupil, I. (2014). Family history of education predicts eating disorders across multiple generations among 2 million Swedish males and females. *PLoS One*, *9*, e106475. https://doi.org/10.1371/journal.pone.0106475

Griffiths, S., Mond, J. M., Murray, S. B., & Touyz, S. (2015). Positive beliefs about anorexia nervosa and muscle dysmorphia are associated with eating disorder symptomatology. *Australian and New Zealand Journal of Psychiatry*, *49*, 812-820. https://doi.org/10.1177/0004867415572412

Grilo, C. M., & Masheb, R. M. (2000). Onset of dieting vs binge eating in outpatients with binge eating disorder. *International Journal of Obesity and Related Metabolic Disorders*, *24*, 404-409.

Hadland, S. E., Austin, S. B., Goodenow, C. S., & Calzo, J. P. (2014). Weight misperception and unhealthy weight control behaviors among sexual minorities in the general adolescent population. *Journal of Adolescent Health*, *54*, 296-303. https://doi.org/10.1016/j.jadohealth.2013.08.021

Hardaway, J. A., Crowley, N. A., Bulik, C. M., & Kash, T. L. (2015). Integrated circuits and molecular components for stress and feeding: Implications for eating disorders. *Genes Brain and Behavior*, *14*, 85-97. https://doi.org/10.1111/gbb.12185

Exploration of how stress modulates different forms of feeding in animal models and identification of molecularly defined brain circuits that regulate feeding. Authors discuss the potential impact of these interactions and circuits for eating disorder biology.

Hay, P., Chinn, D., Forbes, D., Madden, S., Newton, R., & Surgenor, L. (2014). Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for the treatment of eating disorders. *Australian & New Zealand Journal of Psychiatry*, *48*, 977-1008. https://doi.org/10.1177/0004867414555814

Hebebrand, J., Muller, T. D., Holtkamp, K., & Herpertz-Dahlmann, B. (2007). The role of leptin in anorexia nervosa: Clinical implications. *Molecular Psychiatry*, *12*, 23-35. https://doi.org/10.1038/sj.mp.4001909

Hege, M. A., Stingl, K. T., Kullmann, S., Schag, K., Giel, K. E., Zipfel, S., & Preissl, H. (2015). Attentional impulsivity in binge eating disorder modulates response inhibition performance and frontal brain networks. *International Journal of Obesity*, *39*, 353-360. https://doi.org/10.1038/ijo.2014.99

Helverskov, J. L., Clausen, L., Mors, O., Frydenberg, M., Thomsen, P. H., & Rokkedal, K. (2010). Trans-diagnostic outcome of eating disorders: A 30-month follow-up study of 629 patients. *European Eating Disorders Review*, *18*, 453-463. https://doi.org/10.1002/erv.1025

Using a naturalistic design, the authors investigated the 30-months outcome (remission and relapse) of various treatments and predictors of outcome in 629 patients (adolescents and adults) with different eating disorders. Almost half the patients provided data at follow-up of which 42% attained full remission, and 30% partial remission (no longer fulfilling the criteria for an eating disorder diagnosis). A total of 22% or 35% of those obtaining full or partial remission relapsed. Adult patients with AN-like conditions had the poorest outcome, and low BMI emerged as predictor of poor outcome in AN. The frequency of bingeing and purging was a predictor of poor outcome in BN.

Hoek, H. W., & van Hoeken, D. (2003). Review of the prevalence and incidence of eating disorders. *International Journal of Eating Disorders*, *34*, 383-396. https://doi.org/10.1002/eat.10222

Holtkamp, K., Herpertz-Dahlmann, B., Hebebrand, K., Mika, C., Kratzsch, J., & Hebebrand, J. (2006). Physical activity and restlessness correlate with leptin levels in patients with adolescent anorexia nervosa. *Biological Psychiatry*, *60*, 311-313. https://doi.org/10.1016/j.biopsych.2005.11.001

Huas, C., Godart, N., Caille, A., Pham-Scottez, A., Foulon, C., Divac, S. M., . . . Rouillon, F. (2013). Mortality and its predictors in severe bulimia nervosa patients. *European Eating Disorders Review*, *21*, 15-19. https://doi.org/10.1002/erv.2178

The mortality risk of bulimia nervosa was estimated by following 258 individuals admitted to a hospital in France between 1988 and 2004. The mean follow-up duration for subjects was 10.5 years. A total of 10 deaths were recorded during the follow-up time period, with the majority of deaths from suicide. The results show that individuals with bulimia nervosa are at an increased risk for death, specifically suicide.

Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry*, *61*, 348-358. https://doi.org/10.1016/j.biopsych.2006.03.040

Hudson, J. I., Lalonde, J. K., Berry, J. M., Pindyck, L. J., Bulik, C. M., Crow, S. J., . . . Pope, H. G. (2006). Binge-eating disorder as a distinct familial phenotype in obese individuals. *Archives of General Psychiatry*, *63*, 313-319. https://doi.org/10.1001/archpsyc.63.3.313

Jacobi, C., Hayward, C., de Zwaan, M., Kraemer, H. C., & Agras, W. S. (2004). Coming to terms with risk factors for eating disorders: Application of risk terminology and suggestions for a general taxonomy. *Psychological Bulletin*, *130*, 19-65. https://doi.org/10.1037/0033-2909.130.1.19

Johnson, J. G., Cohen, P., Kasen, S., & Brook, J. S. (2002). Childhood adversities associated with risk for eating disorders or weight problems during adolescence or early adulthood. *American Journal of Psychiatry*, *159*, 394-400. https://doi.org/10.1176/appi.ajp.159.3.394

Kally, Z., & Cumella, E. J. (2008). 100 midlife women with eating disorders: A phenomenological analysis of etiology. *Journal of General Psychology*, *135*, 359-377. https://doi.org/10.3200/GENP.135.4.359-378

Kaye, W.H. (2008). Neurobiology of anorexia and bulimia nervosa. *Physiology & Behavior*, *94*, 121-135. https://doi.org/10.1016/j.physbeh.2007

Kaye, W.H., & Strober, M. (2009). Neurobiology of eating disorders. In *The American Psychiatric Publishing Textbook of Psychopharmacology*. Arlington, VA: American Psychiatric Association.

Kaye, W. H., Wagner, A., Fudge, J. L., & Paulus, M. (2011). Neurocircuity of eating disorders. *Current Topics in Behavioral Neuroscience*, *6*, 37-57. https://doi.org/10.1007/7854_2010_85

Kaye, W. H., Wierenga, C. E., Bailer, U. F., Simmons, A. N., & Bischoff-Grethe, A. (2013a). Nothing tastes as good as skinny feels: The neurobiology of anorexia nervosa. *Trends in Neuroscience*, *36*, 110-120. https://doi.org/10.1016/j.tins.2013.01.003

Kaye, W. H., Wierenga, C. E., Bailer, U. F., Simmons, A. N., Wagner, A., & Bischoff-Grethe, A. (2013b). Does a shared neurobiology for foods and drugs of abuse contribute to extremes of food ingestion in anorexia and bulimia nervosa. *Biological Psychiatry*, *73*, 836-842. https://doi.org/10.1016/j.biopscyh.2013.01.002

Keel, P. K., & Brown, T. A. (2010). Update on course and outcome in eating disorders. *International Journal of Eating Disorders*, *43*, 195-204. https://doi.org/10.1002/eat.20810/abstract

Kendler, K. S., Bulik, C. M., Silberg, J., Hettema, J. M., Myers, J., & Prescott, C. A. (2000). Childhood sexual abuse and adult psychiatric and substance use disorders in women: An epidemiological and cotwin control analysis. *Archives of General Psychiatry*, *57*, 953-959. https://doi.org/10.1001/archpsyc.57.10.953

Keshaviah, A., Edkins, K., Hastings, E. R., Krishna, M., Franko, D. L., Herzog, D. B., . . . Eddy, K. T. (2014). Re-examining premature mortality in anorexia nervosa: A meta-analysis redux. *Comprehensive Psychiatry*, *55*, 1773-1784. https://doi.org/10.1016/j.comppsych.2014.07.017

Keski-Rahkonen, A., Hoek, H. W., Linna, M. S., Raevuori, A., Sihvola, E., Bulik, C. M., . . . Kaprio, J. (2009). Incidence and outcomes of bulimia nervosa: A nationwide population-based study. *Psychological Medicine*, *39*, 823-831. https://doi.org/10.1017/S0033291708003942

Keski-Rahkonen, A., Hoek, H. W., Susser, E. S., Linna, M. S., Sihvola, E., Raevuori, A., . . . Rissanen, A. (2007). Epidemiology and course of anorexia nervosa in the community. *American Journal of Psychiatry*, *164*, 1259-1265. https://doi.org/10.1176/appi.ajp.2007.06081388

Kessler, R. C., Berglund, P. A., Chiu, W. T., Deitz, A. C., Hudson, J. I., Shahly, V., . . . Xavier, M. (2013). The prevalence and correlates of binge eating disorder in the World Health Organization World Mental Health Surveys. *Biological Psychiatry*, *73*, 904-914. https://doi.org/10.1016/j.biopsych.2012.11.020

Kessler, R. M., Hutson, P. H., Herman, B. K., & Potenza, M. N. (2016). The neurobiological basis of binge-eating disorder. *Neuroscience and Biobehavioral Reviews*, *63*, 223-238. https://doi.org/10.1016/j.neubiorev.2016.01.013

Kim, Y. R., Kim, J. H., Kim, M. J., & Treasure, J. (2014). Differential methylation of the oxytocin receptor gene in patients with anorexia nervosa: A pilot study. *PLoS One*, *9*, e88673. https://doi.org/10.1371/journal.pone.0088673

Kimmel, M. C., Ferguson, E. H., Zerwas, S., Bulik, C. M., & Meltzer-Brody, S. (2016). Obstetric and gynecologic problems associated with eating disorders. *International Journal of Eating Disorders*, *49*, 260-275. https://doi.org/10.1002/eat.22483

King, J. A., Geisler, D., Bernardoni, F., Ritschel, F., Böhm, I., Seidel, M., . . . Ehrlich, S. (2016). Altered neural efficiency of decision making during temporal reward discounting in anorexia nervosa. *Journal of the American Academy of Child and Adolescent Psychiatry*, *55*, 972-979. https://doi.org/10.1016/j.jaac.2016.08.005

Decreased activation in frontoparietal regions involved in decision making, but faster and more consistent choice behavior of acute patients with acute anorexia nervosa in a temporal delay

discounting task, suggests that the altered efficiency of neural resource allocation might underlie an increased level of self-control.

Kirby, J. S., Runfola, C. D., Fischer, M. S., Baucom, D. H., & Bulik, C. M. (2015). Couple-based interventions for adults with eating disorders. *Eating Disorders*, *23*, 356-365. https://doi.org/10.1080/10640266.2015.1044349

Klein, D. A., Schebendach, J. E., Gershkovich, M., Bodell, L. P., Foltin, R. W., & Walsh, B. T. (2010). Behavioral assessment of the reinforcing effect of exercise in women with anorexia nervosa: Further paradigm development and data. *International Journal of Eating Disorders*, *43*, 611-618. https://doi.org/10.1002/eat.20758

Klump, K. L., Bulik, C. M., Kaye, W. H., Treasure, J., & Tyson, E. (2009). Academy for Eating Disorders position paper: Eating disorders are serious mental illnesses. *International Journal of Eating Disorders*, *42*, 97-103. https://doi.org/10.1002/eat.20589

Klump, K. L., Burt, S. A., McGue, M., & Iacono, W. G. (2007). Changes in genetic and environmental influences on disordered eating across adolescence: A longitudinal twin study. *Archives of General Psychiatry*, *64*, 1409-1415. https://doi.org/10.1001/archpsyc.64.12.1409

Changes in genetic and environmental influences on disordered eating across early-, mid-, and late- adolescence were examined. Significant changes in genetic and shared environmental effects across early- to mid-adolescence were observed. Specifically, genetic factors accounted for a small proportion of variance during early adolescence, but increased in importance and accounted for approximately 50% of the variance in disordered eating at mid- and late-adolescence. Shared environmental influences decreased substantially from early- to mid-adolescence.

Klump, K. L., Keel, P. K., Culbert, K. M., & Edler, C. (2008). Ovarian hormones and binge eating: Exploring associations in community samples. *Psychological Medicine*, *38*, 1749-1757. https://doi.org/10.1017/S0033291708002997

Klump, K. L., Keel, P. K., Sisk, C., & Burt, S. A. (2010). Preliminary evidence that estradiol moderates genetic influences on disordered eating attitudes and behaviors during puberty. *Psychological Medicine*, *40*, 1745-1753. https://doi.org/10.1017/S0033291709992236

In a sample of 198 female adolescent twins, this study explored if estradiol levels moderated disordered eating by comparing twin correlations in low vs. high estradiol groups. They found similar MZ and DZ correlations in the low estradiol group, indicating no genetic effect. In the high estradiol group the MZ twin correlation was more than double the DZ twin correlation indicating genetic effects.

Kyriacou, O., Treasure, J., & Schmidt, U. (2008). Understanding how parents cope with living with someone with anorexia nervosa: Modelling the factors that are associated with carer distress. *International Journal of Eating Disorders*, *41*, 233-242. https://doi.org/10.1002/eat.20488

Larsen, P. S., Strandberg-Larsen, K., Micali, N., & Andersen, A. M. (2015). Parental and child characteristics related to early-onset disordered eating: A systematic review. *Harvard Review of Psychiatry*, *23*, 395-412. https://doi.org/10.1097/HRP.0000000000000073

Lavagnino, L., Arnone, D., Cao, B., Soares, J. C., & Selvaraj, S. (2016). Inhibitory control in obesity and binge eating disorder: A systematic review and meta-analysis of neurocognitive and neuroimaging studies. *Neuroscience and Biobehavioral Reviews*, *68*, 714-726. https://doi.org/10.1016/j.neubiorev.2016.06.041

le Grange, D., Lock, J., Agras, W. S., Bryson, S. W., & Jo, B. (2015). Randomized clinical trial of family-based treatment and cognitive-behavioral therapy for adolescent bulimia nervosa. *Journal*

of the American Academy of Child and Adolescent Psychiatry, *54*, 886-94.e2. https://doi.org/10.1016/j.jaac.2015.08.008

le Grange, D., Lock, J., Loeb, K., & Nicholls, D. (2010). Academy for Eating Disorders position paper: the role of the family in eating disorders. *International Journal of Eating Disorders*, *43*, 1-5. https://doi.org/10.1002/eat.20751

Lebow, J., Sim, L. A., & Kransdorf, L.N. (2015). Prevalence of a history of overweight and obesity in adolescents with restrictive eating disorders. *Journal of Adolescent Health*, *56*, 19-24. https://doi.org/10.1016/j.jadohealth.2014.06.005

Lee, S., Lee, A. M., Ngai, E., Lee, D. T., & Wing, Y. K. (2001). Rationales for food refusal in Chinese patients with anorexia nervosa. *International Journal of Eating Disorders*, *29*, 224-229. https://doi.org/10.1002/1098-108X(200103)29:2<224::AID-EAT1012>3.0.CO;2-R

Lilenfeld, L. R., Kaye, W. H., Greeno, C. G., Merikangas, K. R., Plotnicov, K., Pollice, C., . . . Nagy, L. (1998). A controlled family study of anorexia nervosa and bulimia nervosa: Psychiatric disorders in first-degree relatives and effects of proband comorbidity. *Archives of General Psychiatry*, *55*, 603-610. https://doi.org/10.1001/archpsyc.55.7.603

Lindvall Dahlgren, C., & Wisting, L. (2016). Transitioning from DSM-IV to DSM-5: A systematic review of eating disorder prevalence assessment. *International Journal of Eating Disorders*, *49*, 975-997. https://doi.org/10.1002/eat.22596

Lock, J., & le Grange, D. (2015). *Treatment Manual for Anorexia Nervosa: A Family-Based Approach, Second Edition*. New York: Guildford Press.

Loomba-Albrecht, L. A., & Styne, D. M. (2009). Effect of puberty on body composition. *Current Opinion in Endocrinology Diabetes and Obesity*, *16*, 10-15.

Lutter, M., Khan, M. Z., Satio, K., Davis, K. C., Kidder, I. J., McDaniel, L., . . . Cui, H. (2017). The eating-disorder associated HDAC4(A778T) mutation alters feeding behaviors in female mice. *Biological Psychiatry*, 81,770-777. https://doi.org/10.1016/j.biopsych.2016.09.024

Madden, S., Miskovic-Wheatley, J., Clarke, S., Touyz, S., Hay, P., & Kohn, M. R. (2015). Outcomes of a rapid refeeding protocol in adolescent anorexia nervosa. *Journal of Eating Disorders*, *3*, 8. https://doi.org/10.1186/s40337-015-0047-1

Mangweth-Matzek, B., Hoek, H. W., Rupp, C. I., Kemmler, G., Pope, H. G., & Kinzl, J. (2013). The menopausal transition--a possible window of vulnerability for eating pathology. *International Journal of Eating Disorders*, *46*, 609-616. https://doi.org/10.1002/eat.22157

Marques, L., Alegria, M., Becker, A. E., Chen, C. N., Fang, A., Chosak, A., & Diniz, J. B. (2011). Comparative prevalence, correlates of impairment, and service utilization for eating disorders across US ethnic groups: Implications for reducing ethnic disparities in health care access for eating disorders. *International Journal of Eating Disorders*, *44*, 412-420. https://doi.org/10.1002/eat.20787

Marsh, R., Maia, T. V., & Peterson, B. S. (2009). Functional disturbances within frontostriatal circuits across multiple childhood psychopathologies. *American Journal of Psychiatry*, *166*, 664-674. https://doi.org/10.1176/appi.ajp.2009.08091354

Marsh, R., Steinglass, J. E., Gerber, A. J., Graziano O'Leary, K., Wang, Z., Murphy, D., . . . Peterson, B. S. (2009). Deficient activity in the neural systems that mediate self-regulatory control in bulimia nervosa. *Archives of General Psychiatry*, *66*, 51-63. https://doi.org/10.1001/archgenpsychiatry.2008.504

Mehler, P. S., & Brown, C. (2015). Anorexia nervosa - medical complications. *Journal of Eating Disorders*, *3*, 11. https://doi.org/10.1186/s40337-015-0040-8

Mehler, P. S., & Rylander, M. (2015). Bulimia nervosa - medical complications. *Journal of Eating Disorders*, *3*, 12. https://doi.org/10.1186/s40337-015-0044-4

Comprehensive review of the literature on medical complications associated with purging behaviors in bulimia nervosa, focusing on self-induced vomiting and laxative abuse. While complications of laxative abuse involve mainly the gastrointestinal system and electrolyte disturbances, complications of self-induced vomiting also include cutaneous, dental, throat, cardiac, reproductive, and pulmonary domains. Effects are dependent on the mode and frequency of purging.

Micali, N., Solmi, F., Horton, N. J., Crosby, R. D., Eddy, K. T., Calzo, J. P., . . . Field, A. E. (2015). Adolescent eating disorders predict psychiatric, high-risk behaviors and weight outcomes in young adulthood. *Journal of the American Academy of Child and Adolescent Psychiatry*, *54*, 652-659.e1. https://doi.org/10.1016/j.jaac.2015.05.009

Mitchison, D., Hay, P., Slewa-Younan, S., & Mond, J. (2014). The changing demographic profile of eating disorder behaviors in the community. *BMC Public Health*, *14*, 943. https://doi.org/10.1186/1471-2458-14-943

Using two Australian household surveys, one in 1998 (n=3010) and the other in 2008 (n=3034), the authors interrogated the stereotype that eating disorders are diseases of young, white, socioeconomically privileged women. Between the two samples, the most drastic increases in prevalence of various eating-disorder symptoms were in participants who were male, >45 years old, or from lower socioeconomic brackets. All disordered eating traits had similar negative impact on health-related quality of life regardless of demographic factors.

Mitchison, D., & Hay, P. J. (2014). The epidemiology of eating disorders: Genetic, environmental, and societal factors. *Clinical Epidemiology*, *6*, 89-97. https://doi.org/10.2147/CLEP.S40841

Monteleone, P., & Maj, M. (2013). Dysfunctions of leptin, ghrelin, BDNF and endocannabinoids in eating disorders: Beyond the homeostatic control of food intake. *Psychoneuroendocrinology*, *38*, 312-330. https://doi.org/10.1016/j.psyneuen.2012.10.021

Moore, F., & Keel, P. K. (2003). Influence of sexual orientation and age on disordered eating attitudes and behaviors in women. *International Journal of Eating Disorders*, *34*, 370-374. https://doi.org/10.1002/eat.10198

Morrison, M. A., Morrison, T. G., & Sager, C. L. (2004). Does body satisfaction differ between gay men and lesbian women and heterosexual men and women? A meta-analytic review. *Body Image*, *1*, 127-138. https://doi.org/10.1016/j.bodyim.2004.01.002

Murray, S. M., Tulloch, A. J., Chen, E. Y., & Avena, N. M. (2015). Insights revealed by rodent models of sugar binge eating. *CNS Spectrums*, *20*, 530-536. https://doi.org/10.1017/S1092852915000656

Mustelin, L., Raevuori, A., Hoek, H. W., Kaprio, J., & Keski-Rahkonen, A. (2015). Incidence and weight trajectories of binge eating disorder among young women in the community. *International Journal of Eating Disorders*, *48*, 1106-1112. https://doi.org/10.1002/eat.22409 *In a community sample of young women, lifetime prevalence of DSM-5 BED was 0.7%, mean age of onset was 19 years, comorbid major depressive disorder was common, and BED was frequently preceded by relative overweight in adolescence.*

National Collaborating Centre for Mental Health. (2004). Eating Disorders: Core interventions in the treatment of anorexia nervosa, bulimia nervosa and related eating disorders. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/23346610

Neumark-Sztainer, D., Wall, M., Guo, J., Story, M., Haines, J., & Eisenberg, M. (2006). Obesity, disordered eating, and eating disorders in a longitudinal study of adolescents: How do dieters

fare 5 years later? *Journal of the American Dietetics Association*, *106*, 559-568. https://doi.org/10.1016/j.jada.2006.01.003

Nicholls, D., & Bryant-Waugh, R. (2009). Eating disorders of infancy and childhood: Definition, symptomatology, epidemiology, and comorbidity. *Child and Adolescent Psychiatric Clinics of North America*, *18*, 17-30. https://doi.org/10.1016/j.chc.2008.07.008

Nicholls, D.E., & Viner, R. M. (2009). Childhood risk factors for lifetime anorexia nervosa by age 30 years in a national birth cohort. *Journal of the American Academy of Child and Adolescent Psychiatry*, *48*, 791-799. https://doi.org/10.1097/CHI.0b013e3181ab8b75

Nordbø, R. H., Espeset, E. M., Gulliksen, K. S., Skårderud, F., Geller, J., & Holte, A. (2012). Reluctance to recover in anorexia nervosa. *European Eating Disorders Review*, *20*, 60-67. https://doi.org/10.1002/erv.1097

In depth-interviews from 36 women with anorexia nervosa were collected to explore what makes individuals with anorexia nervosa ambivalent about recovery. Core obstacles reported by patients with anorexia included: perceiving judgements, feeling stuck, feeling distressed, denying anorexia nervosa, eating, gaining weight, and perceived benefits of the illness.

Norris, M. L., Robinson, A., Obeid, N., Harrison, M., Spettigue, W., & Henderson, K. (2014). Exploring avoidant/restrictive food intake disorder in eating disordered patients: A descriptive study. *International Journal of Eating Disorders*, *47*, 495-499. https://doi.org/10.1002/eat.22217

O'Hara, C. B., Campbell, I. C., & Schmidt, U. (2015). A reward-centered model of anorexia nervosa: A focused narrative review of the neurological and psychophysiological literature. *Neuroscience & Biobehavioral Reviews*, *52*, 131-152. https://doi.org/10.1016/j.neubiorev.2015.02.012

Papadopoulos, F. C., Ekbom, A., Brandt, L., & Ekselius, L. (2009). Excess mortality, causes of death and prognostic factors in anorexia nervosa. *British Journal of Psychiatry*, *194*, 10-17. https://doi.org/10.1192/bjp.bp.108.054742

Patel, P., Wheatcroft, R., Park, R. J., & Stein, A. (2002). The children of mothers with eating disorders. *Clinical Child and Family Psychological Review*, *5*, 1-19.

Peat, C. M., Peyerl, N. L., & Muehlenkamp, J. J. (2008). Body image and eating disorders in older adults: A review. *Journal of General Psychology*, *135*, 343-358. https://doi.org/10.3200/GENP.135.4.343-358

Pike, K. M. (1998). Long-term course of anorexia nervosa: response, relapse, remission, and recovery. *Clinical Psychology Review*, *18*, 447-475. https://doi.org/10.1016/S0272-7358(98)00014-2

Pike, K. M., & Dunne, P. E. (2015). The rise of eating disorders in Asia: A review. *Journal of Eating Disorders*, *3*, 33. https://doi.org/10.1186/s40337-015-0070-2

Pike, K. M., Dunne, P. E., & Addai, E. (2013). Expanding the boundaries: reconfiguring the demographics of the "typical" eating disordered patient. *Current Psychiatry Reports*, *15*, 411. https://doi.org/10.1007/s11920-013-0411-2

Pike, K. M., Hoek, H. W., & Dunne, P. E. (2014). Cultural trends and eating disorders. *Current Opinions in Psychiatry*, *27*, 436-442. https://doi.org/10.1097/YCO.0000000000000100

Pisetsky, E. M., Thornton, L. M., Lichtenstein, P., Pedersen, N. L., & Bulik, C. M. (2013). Suicide attempts in women with eating disorders. *Journal of Abnormal Psychology*, *122*, 1042-1056. https://doi.org/10.1037/a0034902

Plomin, R., DeFries, J. C., & Loehlin, J. C. (1977). Genotype-environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin*, *84*, 309-322. https://doi.org/10.1037/0033-2909.84.2.309

Examination of the effect of genotype-environment interaction and correlation in behavioral genetic studies (twin and adoption studies).

Preti, A., Girolamo, G., Vilagut, G., Alonso, J., Graaf, R., Bruffaerts, R., . . . the ESEMeD-WMH investigators. (2009). The epidemiology of eating disorders in six European countries: Results of the ESEMeD-WMH project. *Journal of Psychiatric Research*, *43*, 1125-1132. https://doi.org/10.1016/j.jpsychires.2009.04.003

In a cross-sectional survey of six European countries, lifetime estimated prevalence of AN, BN, BED, sub-threshold BED, and any binge eating were 0.48%, 0.51%, 1.12%, 0.72%, and 2.15%, respectively, and they were 3–8 times higher among women for all eating disorders. Age of onset for the majority of eating disorders was between 10 and 20 years of age. Comorbidity with other mental disorders was common. Only a minority of individuals with a lifetime eating disorder requested medical treatment.

Preti, A., Rocchi, M. B., Sisti, D., Camboni, M. V., & Miotto, P. (2011). A comprehensive meta-analysis of the risk of suicide in eating disorders. *Acta Psychiatrica Scandinavica*, *124*, 6-17. https://doi.org/10.1111/j.1600-0447.2010.01641.x

Qian, J., Hu, Q., Wan, Y., Li, T., Wu, M., Ren, Z., & Yu, D. (2013). Prevalence of eating disorders in the general population: A systematic review. *Shanghai Archives of Psychiatry*, *25*, 212-223. https://doi.org/10.3969/j.issn.1002-0829.2013.04.003

Racine, S. E., Culbert, K. M., Keel, P. K., Sisk, C. L., Burt, S. A., & Klump, K. L. (2012). Differential associations between ovarian hormones and disordered eating symptoms across the menstrual cycle in women. *International Journal of Eating Disorders*, *45*, 333-344. https://doi.org/10.1002/eat.20941

Redgrave, G. W., Coughlin, J. W., Schreyer, C. C., Martin, L. M., Leonpacher, A. K., Seide, M., . . . Guarda, A. S. (2015). Refeeding and weight restoration outcomes in anorexia nervosa: Challenging current guidelines. *International Journal of Eating Disorders*, *48*, 866-873. https://doi.org/10.1002/eat.22390

Ricciardelli, L. A., & McCabe, M. P. (2004). A biopsychosocial model of disordered eating and the pursuit of muscularity in adolescent boys. *Psychological Bulletin*, *130*, 179-205. https://doi.org/10.1037/0033-2909.130.2.179

Richard, M., Bauer, S., & Kordy, H. (2005). Relapse in anorexia and bulimia nervosa—a 2.5-year follow-up study. *European Eating Disorders Review, 13*, 180-190. https://doi.org/10.1002/erv.638

In Project TR-EAT, the symptomatic status of eating-disordered patients (AN, N = 233, BN, N = 422) was tracked after inpatient treatment over a 2.5-year follow-up period. The distribution of time to relapse for both disorders and possible predictors for relapse are investigated by means of discrete time survival analysis. Fifty-eight per cent of the patients with AN and 74% of those with BN achieved partial remission before end of treatment, and thus were at risk for relapse. The relapse rates within 2.5 years were 32.6% for AN and 37.4% for BN. For both eating disorders, the highest risk of relapse was within the first 6 or 7 months after achieving partial remission.

Rousselet, M., Guérineau, B., Paruit, M. C., Guinot, M., Lise, S., Destrube, B., . . . Prétagut, S. (2017). Disordered eating in French high-level athletes: Association with type of sport, doping behavior, and psychological features. *Eating and Weight Disorders*, *22*, 61-68. https://doi.org/10.1007/s40519-016-0342-0

Runfola, C. D., Thornton, L. M., Pisetsky, E. M., Bulik, C. M., & Birgegård, A. (2014). Self-image and suicide in a Swedish national eating disorders clinical register. *Comprehensive Psychiatry*, *55*, 439-449. https://doi.org/10.1016/j.comppsych.2013.11.007

Russell, C. J., & Keel, P. K. (2002). Homosexuality as a specific risk factor for eating disorders in men. *International Journal of Eating Disorders*, *31*, 300-306.

Saltzman, J. A., & Liechty, J. M. (2016). Family correlates of childhood binge eating: A systematic review. *Eating Behaviors*, *22*, 62-71. https://doi.org/10.1016/j.eatbeh.2016.03.027

Sanders, N., Smeets, P. A., van Elburg, A. A., Danner, U. N., van Meer, F., Hoek, H. W., & Adan, R. A. (2015). Altered food-cue processing in chronically ill and recovered women with anorexia nervosa. *Frontiers in Behavioral Neuroscience*, *9*, 46. https://doi.org/10.3389/fnbeh.2015.00046

Santonastaso, P., Bosello, R., Schiavone, P., Tenconi, E., Degortes, D., & Favaro, A. (2009). Typical and atypical restrictive anorexia nervosa: Weight history, body image, psychiatric symptoms, and response to outpatient treatment. *International Journal of Eating Disorders*, *42*, 464-470. https://doi.org/10.1002/eat.20706

Schizophrenia Working Group of the Psychiatric Genomics Consortium. (2014). Biological insights from 108 schizophrenia-associated genetic loci. *Nature*, *511*, 421-427. https://doi.org/10.1038/nature13595

Schmidt, U., Renwick, B., Lose, A., Kenyon, M., Dejong, H., Broadbent, H., . . . Landau, S. (2013). The MOSAIC study - comparison of the Maudsley Model of Treatment for Adults with Anorexia Nervosa (MANTRA) with Specialist Supportive Clinical Management (SSCM) in outpatients with anorexia nervosa or eating disorder not otherwise specified, anorexia nervosa type: Study protocol for a randomized controlled trial. *Trials*, *14*, 160. https://doi.org/10.1186/1745-6215-14-160

Schroeder, M., Eberlein, C., de Zwaan, M., Kornhuber, J., Bleich, S., & Frieling, H. (2012). Lower levels of cannabinoid 1 receptor mRNA in female eating disorder patients: Association with wrist cutting as impulsive self-injurious behavior. *Psychoneuroendocrinology*, *37*, 2032-2036. https://doi.org/10.1016/j.psyneuen.2012.03.025

Seitz, J., Bühren, K., von Polier, G. G., Heussen, N., Herpertz-Dahlmann, B., & Konrad, K. (2014). Morphological changes in the brain of acutely ill and weight-recovered patients with anorexia nervosa. A meta-analysis and qualitative review. *Zeitschrift für Kinder- und Jugendpsychiatrie Psychotherapie*, *42*, 7-17. https://doi.org/10.1024/1422-4917/a000265

Seitz, J., Herpertz-Dahlmann, B., & Konrad, K. (2016). Brain morphological changes in adolescent and adult patients with anorexia nervosa. *Journal of Neural Transmission*, *123*, 949-959. https://doi.org/10.1007/s00702-016-1567-9

Shoebridge, P., & Gowers, S. G. (2000). Parental high concern and adolescent-onset anorexia nervosa. A case-control study to investigate direction of causality. *British Journal of Psychiatry*, *176*, 132-137. https://doi.org/10.1192/bjp.176.2.132

Siervogel, R. M., Demerath, E. W., Schubert, C., Remsberg, K. E., Chumlea, W. C., Sun, S., . . . Towne, B. (2003). Puberty and body composition. *Hormone Research*, *60*, 36-45. https://doi.org/71224

Sim, L. A., Homme, J. H., Lteif, A. N., Vande Voort, J. L., Schak, K. M., & Ellingson, J. (2009). Family functioning and maternal distress in adolescent girls with anorexia nervosa. *International Journal of Eating Disorders*, *42*, 531-539. https://doi.org/10.1002/eat.20654

Smink, F. R., van Hoeken, D., & Hoek, H. W. (2012). Epidemiology of eating disorders: Incidence, prevalence and mortality rates. *Current Psychiatry Reports*, *14*, 406-414. https://doi.org/10.1007/s11920-012-0282-y

Solomon, S. M., & Kirby, D. F. (1990). The refeeding syndrome: A review. *Journal of Parenteral and Enteral Nutrition*, *14*, 90-97.

Spies, M., Knudsen, G. M., Lanzenberger, R., & Kasper, S. (2015). The serotonin transporter in psychiatric disorders: Insights from PET imaging. *Lancet Psychiatry*, *2*, 743-755. https://doi.org/10.1016/S2215-0366(15)00232-1

Steenhuis, I. H., & Vermeer, W. M. (2009). Portion size: Review and framework for interventions. *International Journal of Behavioral Nutrition and Physical Activity*, *6*, 58. https://doi.org/10.1186/1479-5868-6-58

Steinglass, J. E., & Walsh, B. T. (2016). Neurobiological model of the persistence of anorexia nervosa. *Journal of Eating Disorders*, *4*, 19. https://doi.org/10.1186/s40337-016-0106-2

Steinhausen, H. C. (2009). Outcome of eating disorders. *Child and Adolescent Psychiatric Clinics of North America*, *18*, 225-242. https://doi.org/10.1016/j.chc.2008.07.013

Steinhausen, H. C., Jakobsen, H., Helenius, D., Munk-Jørgensen, P., & Strober, M. (2015). A nation-wide study of the family aggregation and risk factors in anorexia nervosa over three generations. *International Journal of Eating Disorders*, *48*, 1-8. https://doi.org/10.1002/eat.22293

Steinhausen, H. C., & Weber, S. (2009). The outcome of bulimia nervosa: Findings from one-quarter century of research. *American Journal of Psychiatry*, *166*, 1331-1341. https://doi.org/10.1176/appi.ajp.2009.09040582

Sternson, S. M., & Roth, B. L. (2014). Chemogenetic tools to interrogate brain functions. *Annual Review of Neuroscience*, *37*, 387-407. https://doi.org/10.1146/annurev-neuro-071013-014048

Striegel-Moore, R. H., & Bulik, C. M. (2007). Risk factors for eating disorders. *American Psychologist*, *62*, 181-198. https://doi.org/10.1037/0003-066X.62.3.181

Striegel-Moore, R. H., Dohm, F. A., Kraemer, H. C., Taylor, C. B., Daniels, S., Crawford, P. B., & Schreiber, G. B. (2003). Eating disorders in White and Black women. *American Journal of Psychiatry*, *160*, 1326-1331. https://doi.org/10.1176/appi.ajp.160.7.1326

Striegel-Moore, R. H., & Franko, D. L. (2003). Epidemiology of binge eating disorder. *International Journal of Eating Disorders*, *34*, S19-S29.

Striegel, R. H., Bedrosian, R., Wang, C., & Schwartz, S. (2012). Why men should be included in research on binge eating: Results from a comparison of psychosocial impairment in men and women. *International Journal of Eating Disorders*, *45*, 233-240. https://doi.org/10.1002/eat.20962

Strober, M., Freeman, R., Lampert, C., Diamond, J., & Kaye, W. (2000). Controlled family study of anorexia nervosa and bulimia nervosa: Evidence of shared liability and transmission of partial syndromes. *American Journal of Psychiatry*, *157*, 393-401.

Strober, M., & Humphrey, L. L. (1987). Familial contributions to the etiology and course of anorexia nervosa and bulimia. *Journal of Consulting and Clinical Psychology*, *55*, 654-659.

Suisman, J. L., Thompson, J. K., Keel, P. K., Burt, S. A., Neale, M., Boker, S., . . . Klump, K. L. (2014). Genetic and environmental influences on thin-ideal internalization across puberty and preadolescent, adolescent, and young adult development. *International Journal of Eating Disorders*, *47*, 773-783. https://doi.org/10.1002/eat.22321

Suokas, J. T., Suvisaari, J. M., Gissler, M., Löfman, R., Linna, M. S., Raevuori, A., & Haukka, J. (2013). Mortality in eating disorders: A follow-up study of adult eating disorder patients treated in tertiary care, 1995-2010. *Psychiatry Research*, *210*, 1101-1106. https://doi.org/10.1016/j.psychres.2013.07.042

Tau, G. Z., & Peterson, B. S. (2010). Normal development of brain circuits. *Neuropsychopharmacology*, *35*, 147-168. https://doi.org/10.1038/npp.2009.115

Thornton, L. M., Watson, H. J., Jangmo, A., Welch, E., Wiklund, C., von Hausswolff-Juhlin, Y., . . . Bulik, C. M. (2017). Binge-eating disorder in the Swedish national registers: Somatic comorbidity. *International Journal of Eating Disorders*, 50, 58-65 https://doi.org/10.1002/eat.22624

Swedish register data were used to identify a sample of 850 individuals diagnosed with binge-eating disorder (BED). Associations were examined between BED and neurologic, immune, respiratory, gastrointestinal, skin, musculoskeletal, genitourinary, circulatory, and endocrine system diseases. Compared with controls matched on sex, year, month, and county of birth, BED was associated with an increased risk for most classes of disease. The strongest associations were found between BED and diabetes and cardiovascular system diseases.

Thornton, L. M., Welch, E., Munn-Chernoff, M. A., Lichtenstein, P., & Bulik, C. M. (2016). Anorexia nervosa, major depression, and suicide attempts: Shared genetic factors. *Suicide and Life Threatening Behaviors*, *46*, 525-534. https://doi.org/10.1111/sltb.12235

This is one of the first studies to investigate overlapping genetic and environmental risk factors for anorexia nervosa, major depression, and suicide attempts. Findings suggest that a portion of the genetic factors underlying anorexia nervosa also contribute to liability to major depression and suicide attempts in adult women. Individual-specific environmental factors, however, may not overlap but rather are trait specific.

Titova, O. E., Hjorth, O. C., Schiöth, H. B., & Brooks, S. J. (2013). Anorexia nervosa is linked to reduced brain structure in reward and somatosensory regions: A meta-analysis of VBM studies. *BMC Psychiatry*, *13*, 110. https://doi.org/10.1186/1471-244X-13-110

Trace, S. E., Baker, J. H., Peñas-Lledó, E., & Bulik, C. (2013). The genetics of eating disorders. *Annual Review of Clinical Psychology*, *9*, 589-620. https://doi.org/10.1146/annurev-clinpsy-050212-185546

Treasure, J., Murphy, T., Szmukler, G., Todd, G., Gavan, K., & Joyce, J. (2001). The experience of caregiving for severe mental illness: A comparison between anorexia nervosa and psychosis. *Social Psychiatry and Psychiatric Epidemiology*, *36*, 343-347.

Treasure, J., & Nazar, B. P. (2016). Interventions for the carers of patients with eating disorders. *Current Psychiatry Reports*, *18*, 16. https://doi.org/10.1007/s11920-015-0652-3

Treasure, J., Stein, D., & Maguire, S. (2015). Has the time come for a staging model to map the course of eating disorders from high risk to severe enduring illness? An examination of the evidence. *Early Intervention in Psychiatry*, *9*, 173-184. https://doi.org/10.1111/eip.12170

Treasure, J., Zipfel, S., Micali, N., Wade, T., Stice, E., Claudino, A., . . . Wentz, E. (2015). Anorexia nervosa. *Nature Reviews Disease Primers*, *1*, 15074. https://doi.org/10.1038/nrdp.2015.74

Twomey, C. D., Baldwin, D. S., Hopfe, M., & Cieza, A. (2015). A systematic review of the predictors of health service utilisation by adults with mental disorders in the UK. *BMJ Open*, *5*, e007575. https://doi.org/10.1136/bmjopen-2015-007575

van den Eynde, F., Suda, M., Broadbent, H., Guillaume, S., van den Eynde, M., Steiger, H., . . . Schmidt, U. (2012). Structural magnetic resonance imaging in eating disorders: A systematic review of voxel-based morphometry studies. *European Eating Disorders Review*, *20*, 94-105. https://doi.org/10.1002/erv.1163

van Langenberg, T., Sawyer, S. M., le Grange, D., & Hughes, E. K. (2016). Psychosocial well-being of siblings of adolescents with anorexia nervosa. *European Eating Disorders Review*, *24*, 438-445. https://doi.org/10.1002/erv.2469

Eighty-five parents and 55 siblings of adolescents with anorexia nervosa completed the Strengths and Difficulties Questionnaire at diagnosis. In addition, 88 parents and 46 siblings completed the Strengths and Difficulties Questionnaire after finishing treatment. Mothers and fathers reported siblings to have lower levels of conduct problems in comparison with population norms. Mothers also reported lower levels of prosocial behaviors. Siblings reported higher levels of emotional difficulties and hyperactivity in comparison with their peers.

Vandereycken, W. (2006a). Denial of illness in anorexia nervosa—a conceptual review: Part 1 diagnostic significance and assessment. *European Eating Disorders Review*, *14,* 341-351. https://doi.org/10.1002/erv.721

Vandereycken, W. (2006b). Denial of illness in anorexia nervosa—a conceptual review: Part 2 different forms and meanings. *European Eating Disorders Review*, *14*, 352-368. https://doi.org/10.1002/erv.722

Wade, T. D., Fairweather-Schmidt, A. K., Zhu, G., & Martin, N. G. (2015). Does shared genetic risk contribute to the co-occurrence of eating disorders and suicidality? *International Journal of Eating Disorders*, *48*, 684-691. https://doi.org/10.1002/eat.22421

Overlapping genetic and environmental risk factors for a composite measure of eating disorders (including AN, BN, BED, and purging disorder) and suicidality were examined in female twins. Results suggest that some of the genetic factors influencing vulnerability to eating disorders also influence suicidality.

Walton, E., Hass, J., Liu, J., Roffman, J. L., Bernardoni, F., Roessner, V., . . . Ehrlich, S. (2016). Correspondence of DNA methylation between blood and brain tissue and its application to schizophrenia research. *Schizophrenia Bulletin*, *42*, 406-414. https://doi.org/10.1093/schbul/sbv074

Blood and temporal lobe biopsy samples were obtained from twelve epilepsy patients during neurosurgical treatment. Findings indicate that most DNA methylation markers in peripheral blood do not reliably predict brain DNA methylation status, but a subset of peripheral data may proxy methylation status of brain tissue. Restricting the analysis to these markers may identify meaningful epigenetic differences in brain disorders.

Watson, H. J., & Bulik, C. M. (2013). Update on the treatment of anorexia nervosa: Review of clinical trials, practice guidelines and emerging interventions. *Psychological Medicine*, *43*, 2477-2500. https://doi.org/10.1017/S0033291712002620

Watson, R. J., Veale, J. F., & Saewyc, E. M. (2016). Disordered eating behaviors among transgender youth: Probability profiles from risk and protective factors. *International Journal of Eating Disorders*. https://doi.org/10.1002/eat.22627

Westmoreland, P., Krantz, M. J., & Mehler, P. S. (2016). Medical complications of anorexia nervosa and bulimia. *American Journal of Medicine*, *129*, 30-37. https://doi.org/10.1016/j.amjmed.2015.06.031

Review of medical complications associated with anorexia nervosa and bulimia nervosa and how the complications can be treated. Epidemiology and psychiatric comorbidities of eating disorders are discussed.

Whitney, J., Murray, J., Gavan, K., Todd, G., Whitaker, W., & Treasure, J. (2005). Experience of caring for someone with anorexia nervosa: Qualitative study. *British Journal of Psychiatry*, *187*, 444-449. https://doi.org/10.1192/bjp.187.5.444

Wierenga, C. E., Ely, A., Bischoff-Grethe, A., Bailer, U. F., Simmons, A. N., & Kaye, W. H. (2014). Are extremes of consumption in eating disorders related to an altered balance between reward

and inhibition. *Frontiers in Behavioral Neuroscience*, *8*, 410. https://doi.org/10.3389/fnbeh.2014.00410

Witcomb, G. L., Bouman, W. P., Brewin, N., Richards, C., Fernandez-Aranda, F., & Arcelus, J. (2015). Body image dissatisfaction and eating-related psychopathology in trans individuals: A matched control study. *European Eating Disorders Review*, *23*, 287-293. https://doi.org/10.1002/erv.2362

Eating disorder traits and body dissatisfaction are compared across three different groups of people: 200 trans people, 200 people with eating disorders, and 200 healthy controls. Results from the study are in line with previous literature; trans individuals do not score as highly on measures of body dissatisfaction as those with clinical eating disorders. However, compared with healthy controls, trans individuals report higher eating disorder symptoms. Particular attention is paid to gender differences, as trans males had similarly high scores on measures of body dissatisfaction as males with eating disorders.

Wu, M., Hartmann, M., Skunde, M., Herzog, W., & Friederich, H. C. (2013). Inhibitory control in bulimic-type eating disorders: A systematic review and meta-analysis. *PLoS One*, *8*, e83412. https://doi.org/10.1371/journal.pone.0083412

Yager, J. (1982). Family issues in the pathogenesis of anorexia nervosa. *Psychosomatic Medicine*, 44, 43-60.

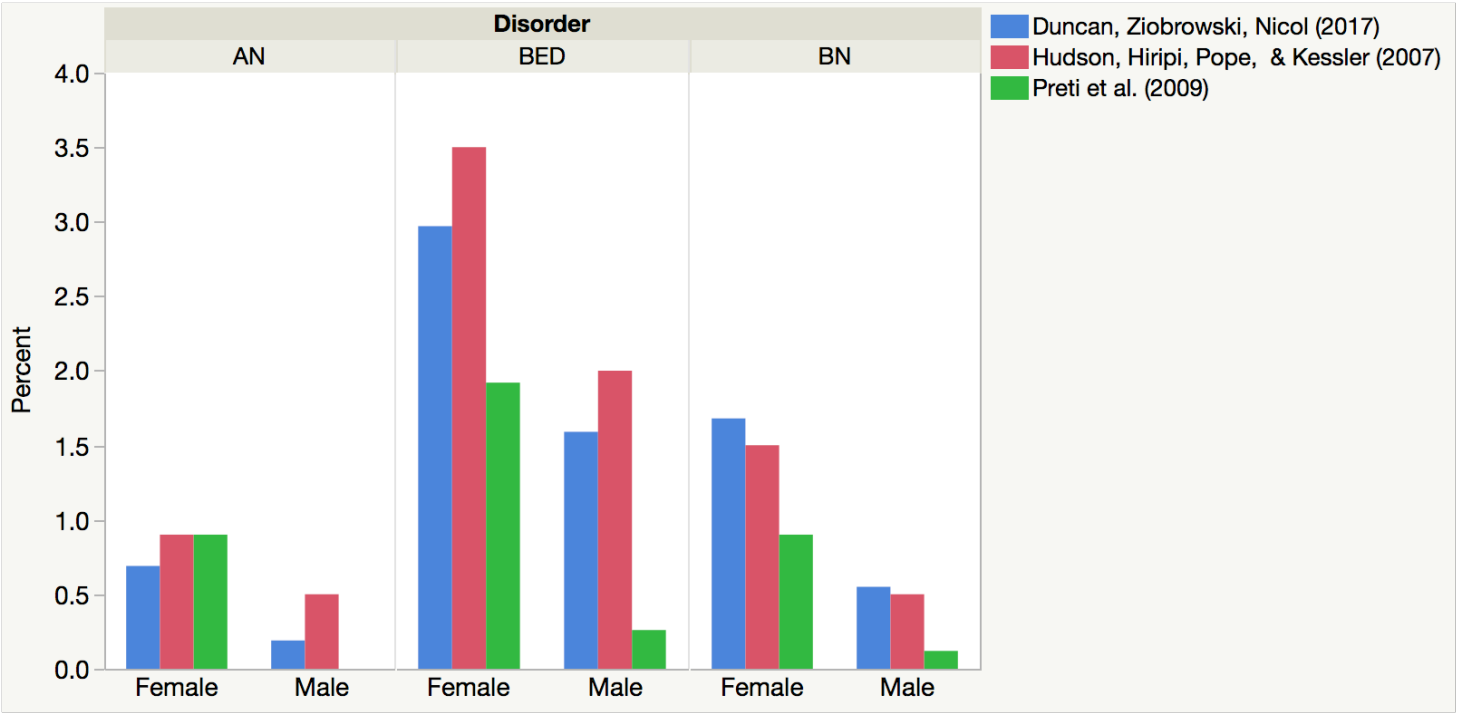
Yao, S., Kuja-Halkola, R., Thornton, L. M., Runfola, C. D., D'Onofrio, B. M., Almqvist, C., . . . Bulik, C. M. (2016). Familial liability for eating disorders and suicide attempts: Evidence from a population registry in Sweden. *JAMA Psychiatry*, *73*, 284-291. https://doi.org/10.1001/jamapsychiatry.2015.2737

The association between eating disorders and suicide attempts was examined using national register data from Sweden. Significantly elevated risks of suicide attempts were observed in individuals with any eating disorders, anorexia nervosa, and bulimia nervosa. Elevated risks of suicide attempts were also found in relatives (full-siblings, half-siblings, and cousins) of the individuals with the aforementioned eating disorders, suggesting familial liability for eating disorders and suicide attempts.

Yilmaz, Z., Hardaway, J. A., & Bulik, C. M. (2015). Genetics and epigenetics of eating disorders. *Advances in Genomics and Genetics*, *5*, 131-150. https://doi.org/10.2147/AGG.S55776

Zerwas, S., Larsen, J. T., Petersen, L., Thornton, L. M., Mortensen, P. B., & Bulik, C. M. (2015). The incidence of eating disorders in a Danish register study: Associations with suicide risk and mortality. *Journal of Psychiatric Research*, *65*, 16-22. https://doi.org/10.1016/j.jpsychires.2015.03.003

Zipfel, S., Giel, K. E., Bulik, C. M., Hay, P., & Schmidt, U. (2015). Anorexia nervosa: Aetiology, assessment, and treatment. *Lancet Psychiatry*, *2*, 1099-1111. https://doi.org/10.1016/S2215-0366(15)00356-9



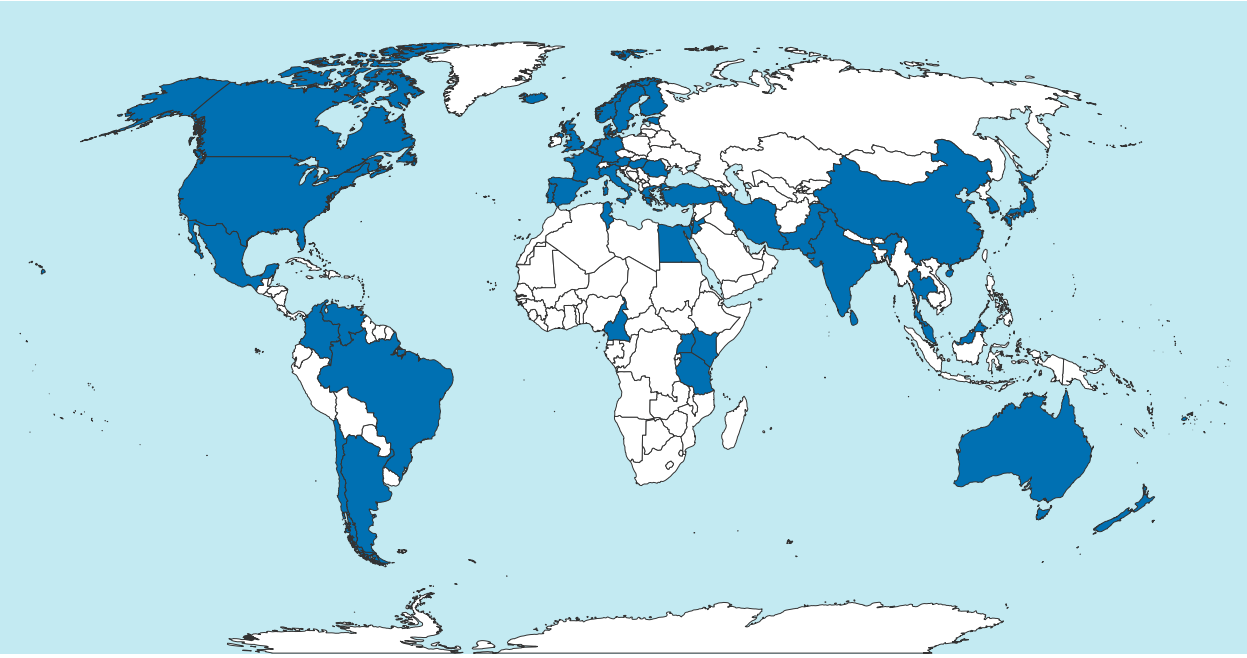
Supplementary Figure S1. Lifetime Prevalence of Eating Disorders by Sex

References for Supplementary Figure S1

Duncan, A. E., Ziobrowski, H. N., & Nicol, G. (2017). The prevalence of past 12-month and lifetime DSM-IV eating disorders by BMI category in US men and women. *European Eating Disorders Review*, *25*, 165-171. https://doi.org/10.1002/erv.2503

Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry*, *61*, 348-358. https://doi.org/10.1016/j.biopsych.2006.03.040

Preti, A., Girolamo, G., Vilagut, G., Alonso, J., Graaf, R., Bruffaerts, R., . . . ESEMeD-WMH Investigators. (2009). The epidemiology of eating disorders in six European countries: Results of the ESEMeD-WMH project. *Journal of Psychiatric Research*, *43*, 1125-1132. https://doi.org/10.1016/j.jpsychires.2009.04.003



Supplementary Figure S2. World map of countries with reported prevalence or cases of eating disorders (dark blue = positive report).

References for Supplementary Figure S2

Ben Salem, H., Gaigi, I., El Fray, H., Gaigi, S., & El Ati, J. (2011). [Bulimia and anorexia among the teenagers]. *La Tunisie Medicale*, *89*, 820–824. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/22179916

Hoek, H. W., van Harten, P. N., Hermans, K. M. E., Katzman, M. A., Matroos, G. E., & Susser, E. S. (2005). The incidence of anorexia nervosa on Curaçao. *American Journal of Psychiatry*, *162*, 748–752. https://doi.org/10.1176/appi.ajp.162.4.748

Kolar, D. R., Rodriguez, D. L. M., Chams, M. M., & Hoek, H. W. (2016). Epidemiology of eating disorders in Latin America: a systematic review and meta-analysis. *Current Opinion in Psychiatry*, *29*, 363–371. https://doi.org/10.1097/YCO.0000000000000279

Lindvall Dahlgren, C., & Wisting, L. (2016). Transitioning from DSM-IV to DSM-5: A systematic review of eating disorder prevalence assessment. *The International Journal of*

Eating Disorders, *49*, 975–997. https://doi.org/10.1002/eat.22596

Mbassa Menick, D., & Moukouta, C. S. (2016/1). Quand manger n'est plus manger… Une clinique émergente en Afrique ? *Neuropsychiatrie de L'enfance et de L'adolescence*, *64*, 22–30. Retrieved from http://www.sciencedirect.com/science/article/pii/S0222961715001853

Mitchison, D., & Hay, P. J. (2014). The epidemiology of eating disorders: genetic, environmental, and societal factors. *Clinical Epidemiology*, *6*, 89–97. https://doi.org/10.2147/CLEP.S40841

Pike, K. M., & Dunne, P. E. (2015). The rise of eating disorders in Asia: a review. *Journal of Eating Disorders*, *3*, 33. https://doi.org/10.1186/s40337-015-0070-2

Thomas, J. J., Lee, S., & Becker, A. E. (2016). Updates in the epidemiology of eating disorders in Asia and the Pacific. *Current Opinion in Psychiatry*, *29*, 354–362. https://doi.org/10.1097/YCO.0000000000000288

van Hoeken, D., Burns, J. K., & Hoek, H. W. (2016). Epidemiology of eating disorders in Africa. *Current Opinion in Psychiatry*, *29*, 372–377. https://doi.org/10.1097/YCO.0000000000000274

Supplementary Table S1. Confidence Matrix for Review

Level of Confidence	Description
High	>1 systematic review and/or meta-analysis of available evidence providing support >1 randomized controlled trial with effects in a consistent direction When appropriate to the research question, >2 large epidemiological studies with consistent evidence from samples in >1 area of the world
Moderate	Converging body of evidence, studied across multiple sites and/or outcomes
Low	Singular studies as the only supporting evidence, a body of evidence with equivocal or conflicting findings, exclusive reliance on indirect outcome measures
Very Low	Expert opinion or theory only

Supplementary Table S2. Detailed Evidence for Confidence Ratings of Supportive Statements

Supportive Statements	Confidence Rating	Basis for Confidence Rating	Relevant Future Investigations
Truth 1: Many people with eating disorders look healthy, yet may be extremely ill.			
1.1 Eating disorders are associated with significant somatic, psychosocial and psychological risk.	High	Multiple systematic reviews across several outcomes, including: GI complications (Norris et al., 2016); refeeding syndrome (O'Connor & Nicholls, 2013); cardiovascular complications (Sachs, Harnke, Mehler, & Krantz, 2016); mortality (Arcelus, Mitchell, Wales, & Nielsen, 2011); and psychological outcomes (Berkman, Lohr, & Bulik, 2007; Sheehan & Herman, 2015). Additional narrative reviews summarize medical complications in eating disorders, e.g. (Mehler & Brown, 2015; Mehler & Rylander, 2015; Westmoreland, Krantz, & Mehler, 2016). (see Supplementary Tables S3 & S4)	Case-control and longitudinal studies distinguishing specific relationships between medical and psychological comorbidities and eating disorders to identify direction of causality.
1.2 Most individuals with eating disorders do not appear emaciated.	High	Eating disorders are present across the whole BMI range and weight is only a criterion in AN. On average BMI: AN<BN<BED. The prevalence of AN is lower than other eating disorders. One systematic review (Lindvall Dahlgren & Wisting, 2016) and one meta-analysis (Qian et al., 2013) indicate that AN is less prevalent than BN and BED. Epidemiological data from several countries indicate that lifetime prevalence of BED is higher than that of BN, which is consistent across several countries (Kessler et al., 2013), and estimates from a large community sample of adolescents indicate that risk for BN is greater in obese as compared to normal weight adolescents. (Flament et al., 2015). Also see Statement 5.4: Eating disorders occur in individuals of all shapes and sizes.	Meta-analyses to provide accurate estimates of incidence and lifetime prevalence of DSM-5 eating disorders. RCTs of interventions that address both disordered eating and risk for excess weight gain in vulnerable populations. Animal research, case-control, and longitudinal studies that consider the role of metabolic dysfunction in understanding eating pathology. Longitudinal research to consider potential differences in course and outcome of eating disorders based on weight status (e.g. normal weight vs. obese individuals who binge eat).
1.3 Somatic, psychosocial, and psychological manifestations and comorbidities of	Moderate	Narrative literature reviews indicate that patients with eating disorders may present to emergency departments with symptoms such as dizziness, fatigue, syncope, and seizures due to eating disorder complications (Mascolo, Trent, Colwell, & Mehler, 2012), and highlights potential for misdiagnosis of problems	Cross-sectional investigations of providers' abilities to detect eating disorders in pediatric, primary care, and obstetrics and gynecology settings.

	eating disorders may be difficult to detect.		associated with restriction and purging (Gaudiani & Mehler, 2016). National survey of Accreditation Council for Graduate Medical Education programs indicates that training in eating disorders for United States resident physicians is limited (Mahr et al., 2015). Cross-sectional studies of UK family physicians indicate that primary care physicians do not utilize national clinical practice guidelines (Currin et al., 2007), have gaps in knowledge of eating disorders (Currin, Waller, & Schmidt, 2009), and that nonclinical features of case presentations (e.g., gender) may influence diagnosis and treatment recommendations (Currin, Schmidt, & Waller, 2007). Cross-sectional investigation of fertility specialists in Australia indicates some misconceptions about eating disorders and low rates of screening for eating disorders in practice (Rodino, Byrne, & Sanders, 2016). See Supplementary Tables S3 & S4 for an overview of somatic, psychosocial, psychological and neurocognitive manifestations and comorbidities associated with eating disorders.	Development and evaluation of specific screening recommendations for primary care providers. Improvement of education about eating disorders for health care professionals. Programs to enhance the adherence of caregivers to medical guidelines.
1.4	Most individuals with eating disorders do not enter treatment; those that do often do so many years into the course of illness.	High	Several epidemiological studies across many countries indicate that only a minority of individuals who meet criteria for eating disorders seek treatment (Hoek & van Hoeken, 2003; Hudson, Hiripi, Pope, & Kessler, 2007; Keski-Rahkonen et al., 2009; Kessler et al., 2013; Preti et al., 2009).	Develop and disseminate methods for early detection and referral. Cross-sectional identification of factors that relate treatment initiation across eating disorder diagnoses. Increasing reach of available interventions.
Truth 2: Families are not to blame, and can be the patients' and providers' best allies in treatment.				
2.1	Biological risk factors contribute to the development of eating disorders.	High	See Truth 4: Eating disorders are not choices, but serious biologically influenced illnesses.	
2.2	Prototypical family interaction patterns that exist premorbidly among families with	Moderate	While a few studies have found parental factors associated with eating disorder onset (Johnson, Cohen, Kasen, & Brook, 2002; Nicholls & Viner, 2009; Shoebridge & Gowers, 2000), reviews and position papers that summarize longitudinal, case-control,	Longitudinal epidemiological studies to more rigorously assess parental and family factors associated with eating risk.

eating disorders have not been identified.		and cross-sectional research on the role of family functioning in eating disorders have not identified consistent patterns of risk associated with parenting or family interaction styles (Campbell & Peebles, 2014; Eisler, 2005; le Grange, Lock, Loeb, & Nicholls, 2010; Strober & Humphrey, 1987; Yager, 1982).	Recent systematic reviews call for additional longitudinal investigations (Larsen, Strandberg-Larsen, Micali, & Andersen, 2015; Saltzman & Liechty, 2016).
2.3 Eating disorders place stress on families.	Moderate	Case-control studies indicate burden of caring for AN and highlight that parental distress may be a consequence of the disorder (Anastasiadou, Medina-Pradas, Sepulveda, & Treasure, 2014; Sim et al., 2009; Treasure et al., 2001). Cross-sectional studies indicate impairments in quality of life and high burden among caregivers (Anastasiadou et al., 2014; Martín et al., 2011; Whitney et al., 2005).	Strategies for aiding caregivers and reducing burden. Patient-initiated admissions. In-home care.
2.4 Family-based treatments have demonstrated effectiveness for the treatment of adolescent AN.	High	Several randomized controlled trials [e.g.; (Eisler et al., 2016; Lock et al., 2010)] and one meta-analysis (Couturier, Kimber, & Szatmari, 2013) support the use of FBT for adolescent AN. Recent research also indicates that FBT may be efficacious for adolescent BN (Le Grange, Lock, Agras, Bryson, & Jo, 2015; Murray et al., 2015). See Supplementary Table S5 for an overview of psychological interventions in eating disorder treatment.	Additional studies evaluating family-based treatment for BN and BED. Studies extending family and couple-based treatment to older adolescents and adults.
Truth 3: An eating disorder diagnosis is a health crisis that disrupts personal and family functioning.			
3.1 Eating disorders have significant medical and psychological risk.	High	See Statement 1.1: Eating disorders are associated with significant somatic, psychosocial and psychological risk.	
3.2 Eating disorders produce financial burden.	High	Two small, cross-sectional studies indicate high rates of economic hardship (Gatt et al., 2014) and significant financial costs (Crow et al., 2009) associated with eating disorders. A survey of the cost of mental disorders in the UK estimated costs of eating disorders at £50.6 million in 2007, with an estimated increase to £76.4 million by 2026. The majority of cost was accounted for by loss of employment (McCrone, Dhanasiri, Patel, Knapp, & Lawton-Smith, 2007). A systematic review of cost-of-illness studies and cost-effectiveness analyses in eating disorders estimated substantial	Case-control studies examining discrepancy in food and medical expenses. Studies examining the long-term financial burden of eating disorders over time.

		annual costs per patient ranging from $1,288-$8,042 US (2008) (Stuhldreher et al., 2012). Recent systematic reviews indicate economic burden of all eating disorders is substantial (Ágh et al., 2015; Ágh et al., 2016).	
3.3 In adolescence, eating disorders may lead to functional impairment and delays in healthy development.	Moderate	Eight-year prospective investigation found that youth with eating disorders report greater functional impairment, suicidality, mental health treatment, and unhealthy BMIs compared with unaffected youth (Stice, Marti, & Rohde, 2013). Narrative review summarizes case-control and cross-sectional research on potential delays in healthy development for adolescents and young adults with eating disorders (Stice & Bohon, 2013).	Inclusion of secondary outcomes related to healthy development in intervention trials with child and adolescent samples.
3.4 In adulthood, eating disorders may interfere with intimate relationships, reproductive health, parenting, and health-related quality of life.	Moderate	A review summarizing case-control and cross sectional studies found gynecologic problems including menstrual disturbances across all eating disorders, unplanned pregnancy, greater gestational weight gain, obstetric complications including risk for preterm birth and low birth weight infants, higher rates of miscarriage in BN and BED; poor nutrition during pregnancy, associated polycystic ovarian syndrome in those with BN and BED; and sexual dysfunction across all eating disorders (see review (Kimmel, Ferguson, Zerwas, Bulik, & Meltzer-Brody, 2016). Studies specific to fertility have produced mixed findings (Kimmel et al., 2016), with some case-control and cross-sectional studies finding fertility issues, and others finding comparable rates of fertility and reproduction in those with and without an eating disorder history. One case-control study indicates higher incidence of marital problems in women with BED (Whisman, Dementyeva, Baucom, & Bulik, 2012). Systematic reviews indicate impaired health-related quality of life among individuals with eating disorders (Ágh et al., 2015; Ágh et al., 2016).	Prospective cohort studies that examine and follow outcomes secondary to eating disorder onset. Systematic review or meta-analysis of relationship and role functioning in eating disorders.
Truth #4: Eating disorders are not choices, but serious biologically influenced illnesses.			

4.1 Disordered eating behaviors can be guided by biological processes associated with automatic (unconscious) events.	Moderate-High	One systematic review and meta-analysis identified difficulties with inhibitory control associated with bulimic-type eating disorders (Wu, Hartmann, Skunde, Herzog, & Friederich, 2013). A recent theoretical model identifies eating behaviors in anorexia nervosa as habitual behaviors, similar to compulsions in OCD, supported by case-control studies on neuropsychological and neuroimaging tasks (Godier et al., 2016; Steinglass & Walsh, 2016). Evidence from animal studies and human neuroimaging support some shared neurobiology in eating disorders and addiction (Kaye et al., 2013b; O'Hara, Campbell, & Schmidt, 2015). A position paper reviews literature (primarily case-control studies) that identifies alterations in neurobiological pathways related to reward and self-control associated with eating disorders (Wierenga et al., 2014).	Development of neuropsychologically based treatment approaches. Longitudinal examination of neuropsychological outcomes during the course of illness and intervention.
4.2 Biologically-influenced, fundamental personality traits and cognitive styles are associated with eating disorders.	High	Several systematic reviews and meta-analyses converge on the idea that fundamental personality traits (e.g. impulsivity, perfectionism) and cognitive styles (e.g. difficulties with set shifting) are associated with eating disorders (Cassin & von Ranson, 2005; Lang, Lopez, Stahl, Tchanturia, & Treasure, 2014; Lopez, Tchanturia, Stahl, & Treasure, 2008; Roberts, Tchanturia, Stahl, Southgate, & Treasure, 2007). Recent case-control studies and narrative reviews of the literature support and extend these findings (Balodis et al., 2013; Ehrlich et al., 2015; Kaye, Wierenga, Bailer, Simmons, & Bischoff-Grethe, 2013a; Klabunde, Acheson, Boutelle, Matthews, & Kaye, 2013; Lavender et al., 2015; Vall & Wade, 2015). Also see Supplementary Table S4 for an overview of psychological and neurocognitive traits associated with eating disorders.	Updated conceptualization of eating disorder etiology for patients and caregivers. Longitudinal studies examining RCTs involving treatment matching based on phenotypic psychobiological profiles. Longitudinal investigations of specific behavioral traits that occur in childhood, prior to ED onset.
4.3 Individuals with eating disorders may experience non-typical responses to eating and activity.	Moderate	Case-control studies find increased attention to and value of physical activity in patients with AN (Giel et al., 2013; Klein et al., 2010).	Systematic reviews and meta-analyses that empirically summarize altered response to food and exercise related experiences in those with eating disorders.

		Ecological Momentary Assessment (EMA) data indicate that negative affect increases prior to episodes of binge eating and purging (Berg et al., 2013), though a meta-analysis found that negative affect increases, rather than decreases, after binge eating episodes (Haedt-Matt & Keel, 2011). Conclusions remain conflicted on the exact nature of affect changes after binge eating and purging episodes. Case-control experimental paradigms that provide food, amino acid drinks, or food images to participants indicate that individuals with AN show altered neural response to food anticipation and food-related reward (Kaye et al., 2013a; Kaye, 2008; O'Hara et al., 2015). Case-control and cross-sectional fMRI studies examining food anticipation and response in individuals with BN and BED appear to have mixed findings (Bohon & Stice, 2011; Bohon & Stice, 2012; Skunde et al., 2016; Van den Eynde et al., 2013; Wagner et al., 2015; Weygandt, Schaefer, Schienle, & Haynes, 2012). This literature has not yet been summarized.	Basic science research identifying neural circuitry associated with eating disorder risk. Longitudinal ambulatory assessment in the general population or birth cohorts to quantify the degree of physical activity in patients and healthy controls prior to the onset of the disorder and during the course of illness.
4.4 Eating disorders are associated with dysregulation in neurotransmitter availability and function.	Moderate	Case-control and cross-sectional studies of ill and recovered patients indicate that individuals with eating disorders have disturbances of dopamine and serotonin systems [see reviews (Kaye et al., 2005; Kaye et al., 2013a; Kaye, 2008)]. Other case-control and cross-sectional research supports the role of leptin, ghrelin, BDNF, and endocannabinoids in eating disorders [see reviews, (Monteleone & Maj, 2013; Scherma, Fattore, Castelli, Fratta, & Fadda, 2014)].	Additional investigation of neurotransmitter availability and function in eating disorders using methods including post mortem brain analyses, measures of cerebrospinal fluid, PET imaging, and magnet imaging spectroscopy. Longitudinal investigations that examine the role of neurotransmitter availability in the onset and maintenance of eating disorders.
4.5 Brain structure and function differ between those with active eating disorders and unaffected individuals.	High	Systematic reviews and meta-analyses of individuals with AN indicate alterations in brain structure during illness (Seitz et al., 2014; Titova, Hjorth, Schiöth, & Brooks, 2013; Van den Eynde et al., 2012). Neuroimaging research indicates altered brain function in individuals with eating disorders**,** which may predispose individuals to or arise as a result of illness [see reviews (Frank, 2013; Frank, 2015; Kaye, 2008; O'Hara et al., 2015)].	Systematic reviews and meta-analyses of brain function in those with eating disorders. Longitudinal investigations to distinguish temporal sequence of changes in brain function in relation to disorder onset and maintenance.

4.6 Feeding and activity behavior is biologically regulated in animals.	High	Controlled experiments have resulted in the development of animal models of hunger (Atasoy, Betley, Su, & Sternson, 2012) and binge eating (Murray, Tulloch, Chen, & Avena, 2015), providing evidence that eating disorders have neurobiological origins. Controlled experiments of an activity-based anorexia rodent model (Chowdhury, Chen, & Aoki, 2015) highlight increased physical activity and reduced body weight in response to restricted food access. See Supplementary Table S7 for an overview of brain circuitry regions involved in the regulation of feeding and eating in animal models.	Additional research is needed to determine if regions identified in animal models of feeding and eating are therapeutic entry points.
4.7 Endocrine changes are associated with eating disorder risk.	Moderate	A growing body of longitudinal research, twin studies, case-control studies, cross-sectional studies, and animal research supports the role of endocrine changes in the onset of disordered eating in females (Baker & Runfola, 2016; Baker, Girdler, & Bulik, 2012; Klump, 2013). See Supplementary Table S3 for an overview of endocrine changes associated with eating disorders.	Longitudinal examination of eating disorder risk during the menopause transition in women. Investigations on reproductive milestones and sex hormones and eating disorders risk in males. Longitudinal investigations on appetite-regulating hormones.
Truth #5: Eating disorders affect people of all genders, ages, races, ethnicities, body shapes and weights, sexual orientations, and socioeconomic statuses.			
5.1 Eating disorders affect both males and females.	High	Large epidemiological studies indicate that males are affected by eating disorders, though at lower rates than females (Hudson et al., 2007; Javaras et al., 2015; Kessler et al., 2013; Preti et al., 2009; Zerwas et al., 2015). Males and females with eating disorders may have different clinical characteristics (Welch, Ghaderi, & Swenne, 2015).	Epidemiological studies in large population-based registers applying DSM5 criteria globally to assess global distribution and region-specific risk factors.
5.2 Eating disorders occur across the lifespan.	Moderate-High	Large epidemiological studies indicate that eating disorder risk fluctuates with age, though eating disorders occur at all ages (Keski-Rahkonen et al., 2009; Munkholm et al., 2016; Preti et al., 2009) with binge-eating disorder being more common in older individuals (Pike, Dunne, & Addai, 2013; Smink, van Hoeken, & Hoek, 2012).	Longitudinal examination of eating disorder development during midlife and later life.

5.3 Eating disorders occur in all races and ethnicities.	High	A systematic review of sociodemographic correlates of eating disorders found that ethnicity was not associated with eating disorder epidemiology (Mitchison & Hay, 2014). A large epidemiological study across several countries found eating disorders in all parts of the world (Kessler et al., 2013). Narrative review of epidemiological studies in specific countries likewise suggests stable or decreasing rates of eating disorders among Caucasian groups in Western Europe and North America, with increasing rates of eating pathology in other countries and among some minority groups in North America (Pike et al., 2013; Pike, Hoek, & Dunne, 2014). One meta-analysis of differences in Black and White females in North America supports that differences in body dissatisfaction among these ethnic groups are decreasing (Roberts, Cash, Feingold, & Johnson, 2006).	RCTs establishing the efficacy of intervention for minority populations. Global epidemiological studies especially in Africa and Asia to evaluate prevalence, incidence, and mortality globally.
5.4 Eating disorders occur in individuals of all shapes and sizes.	High	Longitudinal studies indicate that unhealthy weight control methods and binge eating prospectively predict increases in BMI and risk for obesity over time (Field et al., 2003; Neumark-Sztainer et al., 2006; Stice, Cameron, Killen, Hayward, & Taylor, 1999; Stice, Presnell, & Spangler, 2002). A longitudinal investigation found that the course of individual eating disorder symptoms, including body weight, is quite variable and does not conform to initial diagnosis (Lavender et al., 2011). A cross-sectional study of adolescents found increased risk for BN in obese, compared with normal-weight, boys and girls (Flament et al., 2015). Also see Statement 1.2: Most individuals with eating disorders do not appear emaciated.	Longitudinal studies that consider weight trajectory as it may relate to eating disorder symptom development using latent class analysis.
5.5 Eating disorders are present across different sexual orientations and gender identities.	Moderate	Cross-sectional studies indicate that gay and bisexual males may be at increased risk for eating disorders (Brown & Keel, 2012b; French, Story, Remafedi, Resnick, & Blum, 1996; Hadland, Austin, Goodenow, & Calzo, 2014; Russell & Keel, 2002).	Longitudinal studies of adolescent sexual and gender identity development and eating disorder risk.

		Some cross-sectional evidence indicates that lesbian and bisexual women have elevated eating disorder risk as compared with heterosexual women (Hadland et al., 2014; Moore & Keel, 2003). A cross-sectional study of college students indicates that transgender individuals are at heightened risk for eating disorders compared with cisgender sexual minority and cisgender heterosexual youth (Diemer, Grant, Munn-Chernoff, Patterson, & Duncan, 2015).	Further examination of the relationship between eating disorder risk and gender identity.
5.6 There is no consistent association between socioeconomic status and risk for eating disorders.	Moderate	A systematic review of sociodemographic correlates of eating disorders found that socioeconomic status was not associated with eating disorder epidemiology (Mitchison & Hay, 2014).	Further longitudinal examination of the relationship between socioeconomic status and eating disorders to clarify inconsistent patterns and proposed genetic associations.
Truth #6: Eating disorders carry an increased risk for both suicide and medical complications.			
6.1 Eating disorders are associated with premature death.	High	A meta-review examined all-cause mortality in mental disorders, finding very high all-cause mortality in AN, BN, and EDNOS also evidenced elevated all-cause mortality (Chesney, Goodwin, & Fazel, 2014). A meta-analysis of mortality rates among eating disorders found significantly elevated mortality for AN, BN, and EDNOS (Arcelus et al., 2011). A meta-analysis hypothesizing inflated mortality estimates in AN re-estimated after methodological corrections and continued to find elevated all-cause mortality in AN (Keshaviah et al., 2014).	Further international studies of mortality associated with eating disorder to identify global patterns and regional differences.
6.2 Risk of suicide is elevated in eating disorders.	High	Several meta-analyses find elevated suicide risk in individuals with eating disorders (Chesney et al., 2014; Keshaviah et al., 2014; Preti, Rocchi, Sisti, Camboni, & Miotto, 2011). Recent epidemiological data suggest that comorbid psychiatric conditions increase suicide risk (Pisetsky, Thornton, Lichtenstein, Pedersen, & Bulik, 2013) and that family history of an eating disorder may relate to risk of suicide (Yao et al., 2016).	Investigations exploring the mechanism underlying the association between eating disorders and suicide.
Truth #7: Genes and environment play important roles in the development of eating disorders.			
7.1 Eating disorders run in families.	Moderate-High	Family and twin studies consistently indicate that eating disorders aggregate within families. Heritability estimates range from 0.48-0.74 in AN, 0.55-0.62 in BN, and 0.39-0.45 in BED	

		(Bulik, Kleiman, & Yilmaz, 2016; Trace, Baker, Peñas-Lledó, & Bulik, 2013; Yilmaz, Hardaway, & Bulik, 2015).	
7.2 Genes play a role in eating disorder risk.	High	Evidence consistently indicates that genetics play a role in eating disorders. (Bulik et al., 2016; Culbert, Racine, & Klump, 2011; Trace et al., 2013; Yilmaz et al., 2015)]. The first genome-wide significant locus for AN has been discovered which is likely to represent the turning point for genomic discovery (Duncan et al., 2017).	Global efforts to increase sample size and statistical power are underway. Increase sample size in AN GWAS. Conduct BN and BED GWAS to understand role of genetics in all eating disorders. Examination of potential rare genetic variants that occur in densely affected pedigrees.
7.3 Environmental factors play a role in eating disorder risk.	High	Cross-sectional and longitudinal twin studies indicate that nonshared environmental factors account for variance in eating disorder symptoms that are not accounted for by genetic effects. Cultural pressure for thinness has been identified as one specific risk factor for eating disorders, and randomized controlled trials of interventions that reduce thin-ideal internalization have led to reductions in eating disorder symptoms (Culbert, Racine, & Klump, 2015). While thin-ideal internalization may have some genetic influence, one longitudinal twin study indicates that nonshared environmental influences were most important in the etiology of thin-ideal internalization (Suisman et al., 2014).	Longitudinal studies in birth cohorts to identify risk factors of eating disorder pathology.
7.4 Only a small portion of individuals exposed to environmental risk develop eating disorders.	Moderate	While exposure to some environmental risk factors, such as the sociocultural thin ideal, are pervasive, relatively few exposed individuals develop eating disorders, providing indirect evidence that environmental risk does not act alone (Culbert et al., 2015).	Longitudinal studies that examine how environmental exposure may influence eating disorder risk differentially across individuals, including gene by environment interaction.
Truth #8: Genes alone do not predict who will develop eating disorders.			
8.1 Eating disorders do not follow Mendelian transmission patterns.	Moderate	Case-control studies examining candidate genes in eating disorders have not shown consistent effects (Yilmaz et al., 2015).	Investigation of possible rare variants of strong effect.
8.2 Many cases of eating disorders are sporadic, meaning there is no known family member who	Low	Family studies indicate that the relative risk for eating disorders is higher in family members of affected individuals; however, the majority of affected individuals have no reported diagnosis in affected family members (Bould et al., 2015; Steinhausen, Jakobsen, Helenius, Munk-Jørgensen, & Strober, 2015; Strober, Freeman, Lampert, Diamond, & Kaye, 2000). This literature is	Further examination of eating disorder history among relatives.

suffers from an eating disorder.		limited in that eating disorder history among relatives may not be fully known or accurately captured.	
8.3 Genes and environment may co-act to influence risk for eating disorders.	Low	Twin studies indicate that genetic risk for eating disorders may be activated by hormonal changes, such as puberty [see review (Baker et al., 2012)]. Longitudinal research indicates that the learned expectations about eating and thinness mediate the relationship between personality risk and eating disorder symptoms (Combs, Smith, Flory, Simmons, & Hill, 2010; Pearson & Smith, 2015). Longitudinal twin studies have found some statistical evidence of gene by environment interaction mainly stressing developmental stages as environmental moderators. Findings are currently inconsistent across studies (Culbert, Racine, & Klump, 2015). Preliminary case-control studies of methylation and expression of candidate genes indicate the possibility of epigenetic effects that relate to eating disorder affection status (Yilmaz et al., 2015).	Large population-based studies with both genotypic and phenotypic information to probe gene x environment interplay. Case-control studies to examine potential for epigenetic effects.
Truth #9: Full recovery from an eating disorder is possible. Early detection and intervention are important.			
9.1 A substantial portion of individuals with eating disorders achieve recovery.	High	Systematic reviews and meta-analyses evaluating the longitudinal course and outcome of eating disorders in clinical samples indicate that many individuals achieve remission/recovery (Keel & Brown, 2010; Steinhausen & Weber, 2009). An 8-year longitudinal study of a community sample of adolescents found that one-year recovery rates ranged from 91%-96% (Stice, Marti, Shaw, & Jaconis, 2009).	Establishment of uniform definitions of remission, recovery, and relapse. Using these universal definitions to re-evaluate recovery rates and update prognosis estimates.
9.2 Early detection and intervention may improve prognosis.	Moderate	Cross-sectional and longitudinal studies indicate that recovery is less likely as illness progresses (Keel & Brown, 2010; Pike, 1998) and that length of illness is associated with medical, neurobiological and social deteriorations that can negatively impact the course of the disorder (Treasure, Stein, & Maguire, 2015).	Develop and evaluate strategies for early detection, intervention, and relapse prevention.
9.3 Effective psychological interventions for eating disorders exist. Many, but not all, patients benefit.	High	Several systematic reviews and meta-analyses support the efficacy of psychological interventions, including family-based treatment for adolescent AN (Couturier et al., 2013), cognitive behavioral treatment (Groff, 2015; Hay, 2013; Peat, Brownley, Berkman, & Bulik, 2012), including Internet-based guided self-help approaches for BN and BED (Dölemeyer, Tietjen, Kersting,	Aim for a clinician's toolbox that includes psychological and pharmacological interventions that are effective for a range of eating disorders in diverse populations.

		& Wagner, 2013; Peat et al., 2012) and several forms of prevention including media literacy, cognitive-behavioral therapy, healthy weight, and dissonance-based approaches (Watson et al., 2016). See Supplementary Table S5 for an overview of psychological interventions in eating disorder treatment.	
9.4 Medication can be an effective treatment component for eating disorders.	High for BN/BED Low for AN	Systematic reviews indicate that medication can be effective for the treatment of BED and BN (Brown & Keel, 2012a; Brownley, Berkman, Sedway, Lohr, & Bulik, 2007; Hay & Claudino, 2012; Reas & Grilo, 2008; Shapiro et al., 2007). Systematic reviews and meta-analyses have found little evidence that medications improve AN outcomes (Dold, Aigner, Klabunde, Treasure, & Kasper, 2015; Lebow, Sim, Erwin, & Murad, 2013). See Supplementary Table S6 for an overview of medications in eating disorder treatment.	Drug development and repurposing investigations to target core biological pathology of AN; studies of long-term efficacy of medication interventions for all eating disorders; study of the effectiveness of medications for eating disorders in community settings.

AN: Anorexia nervosa; BED: Binge-eating disorder; BMI: Body mass index; BN: Bulimia nervosa; DSM-5: Diagnostic and Statistical Manual of Mental Disorders; EDNOS: Eating disorder not otherwise specified; FBT: Family based treatment; GI: Gastrointestinal; OCD: Obsessive-compulsive disorder; PET: Positron emission tomography; RCT: Randomized-controlled trial

References for Supplementary Table S2

Ágh, T., Kovács, G., Pawaskar, M., Supina, D., Inotai, A., & Vokó, Z. (2015). Epidemiology, health-related quality of life and economic burden of binge eating disorder: A systematic literature review. *Eating and Weight Disorders*, *20*, 1-12. https://doi.org/10.1007/s40519-014-0173-9

Ágh, T., Kovács, G., Supina, D., Pawaskar, M., Herman, B. K., Vokó, Z., & Sheehan, D. V. (2016). A systematic review of the health-related quality of life and economic burdens of anorexia nervosa, bulimia nervosa, and binge eating disorder. *Eating and Weight Disorders*, *21*, 353-364. https://doi.org/10.1007/s40519-016-0264-x

Anastasiadou, D., Medina-Pradas, C., Sepulveda, A. R., & Treasure, J. (2014). A systematic review of family caregiving in eating disorders. *Eating Behaviors*, *15*, 464-477. https://doi.org/10.1016/j.eatbeh.2014.06.001

Arcelus, J., Mitchell, A. J., Wales, J., & Nielsen, S. (2011). Mortality rates in patients with anorexia nervosa and other eating disorders. A meta-analysis of 36 studies. *Archives of General Psychiatry*, *68*, 724-731. https://doi.org/10.1001/archgenpsychiatry.2011.74

Atasoy, D., Betley, J. N., Su, H. H., & Sternson, S. M. (2012). Deconstruction of a neural circuit for hunger, *488*, 172-177. *Nature*, https://doi.org/10.1038/nature11270

Baker, J. H., Girdler, S. S., & Bulik, C. M. (2012). The role of reproductive hormones in the development and maintenance of eating disorders. *Expert Reviews in Obstetrics and Gynecology*, *7*, 573-583. https://doi.org/10.1586/eog.12.54

Baker, J. H., & Runfola, C. D. (2016). Eating disorders in midlife women: A perimenopausal eating disorder. *Maturitas*, *85*, 112-116. https://doi.org/10.1016/j.maturitas.2015.12.017

Balodis, I. M., Molina, N. D., Kober, H., Worhunsky, P. D., White, M. A., Rajita,

S., . . . Potenza, M. N. (2013). Divergent neural substrates of inhibitory control in binge eating disorder relative to other manifestations of obesity. *Obesity*, *21*, 367-377. https://doi.org/10.1002/oby.20068

Berg, K. C., Crosby, R. D., Cao, L., Peterson, C. B., Engel, S. G., Mitchell, J. E., & Wonderlich, S. A. (2013). Facets of negative affect prior to and following binge-only, purge-only, and binge/purge events in women with bulimia nervosa. *Journal of Abnormal Psychology*, *122*, 111-118. https://doi.org/10.1037/a0029703

Berkman, N. D., Lohr, K. N., & Bulik, C. M. (2007). Outcomes of eating disorders: A systematic review of the literature. *International Journal of Eating Disorders*, *40*, 293-309. https://doi.org/10.1002/eat.20369

Bohon, C., & Stice, E. (2011). Reward abnormalities among women with full and subthreshold bulimia nervosa: A functional magnetic resonance imaging study. *International Journal of Eating Disorders*, *44*, 585-595. https://doi.org/10.1002/eat.20869

Bohon, C., & Stice, E. (2012). Negative affect and neural response to palatable food intake in bulimia nervosa. *Appetite*, *58*, 964-970. https://doi.org/10.1016/j.appet.2012.02.051

Bould, H., Sovio, U., Koupil, I., Dalman, C., Micali, N., Lewis, G., & Magnusson, C. (2015). Do eating disorders in parents predict eating disorders in children? Evidence from a Swedish cohort. *Acta Psychiatrica Scandinavica*, *132*, 51-59. https://doi.org/10.1111/acps.12389

Brown, T. A., & Keel, P. K. (2012a). Current and emerging directions in the treatment of eating disorders. *Subst Abuse*, *6*, 33-61. https://doi.org/10.4137/SART.S7864

Brown, T. A., & Keel, P. K. (2012b). The impact of relationships on the association between sexual orientation and disordered eating in men. *International Journal of Eating Disorders*, *45*, 792-799. https://doi.org/10.1002/eat.22013

Brownley, K. A., Berkman, N. D., Sedway, J. A., Lohr, K. N., & Bulik, C. M. (2007). Binge eating disorder treatment: A systematic review of randomized controlled trials. *International Journal of Eating Disorders*, *40*, 337-348. https://doi.org/10.1002/eat.20370

Bulik, C. M., Kleiman, S. C., & Yilmaz, Z. (2016). Genetic epidemiology of eating disorders. *Current Opinion in Psychiatry*, *29*, 383-388. https://doi.org/10.1097/YCO.0000000000000275

Campbell, K., & Peebles, R. (2014). Eating disorders in children and adolescents: State of the art review. *Pediatrics*, *134*, 582-592. https://doi.org/10.1542/peds.2014-0194

Cassin, S. E., & von Ranson, K. M. (2005). Personality and eating disorders: a decade in review. *Clinical Psychology Review*, *25*, 895-916. https://doi.org/10.1016/j.cpr.2005.04.012

Chesney, E., Goodwin, G. M., & Fazel, S. (2014). Risks of all-cause and suicide mortality in mental disorders: A meta-review. *World Psychiatry*, *13*, 153-160. https://doi.org/10.1002/wps.20128

Chowdhury, T. G., Chen, Y. W., & Aoki, C. (2015). Using the activity-based anorexia rodent model to study the neurobiological basis of anorexia nervosa. *Journal of Visualized Experiments*, *105*, e52927. https://doi.org/10.3791/52927

Combs, J. L., Smith, G. T., Flory, K., Simmons, J. R., & Hill, K. K. (2010). The acquired preparedness model of risk for bulimic symptom development. *Psychology of Addictive Behaviors*, *24*, 475-486. https://doi.org/10.1037/a0018257

Couturier, J., Kimber, M., & Szatmari, P. (2013). Efficacy of family-based treatment for adolescents with eating disorders: A systematic review and meta-analysis. *International Journal of Eating Disorders*, *46*, 3-11. https://doi.org/10.1002/eat.22042

Crow, S. J., Frisch, M. J., Peterson, C. B., Croll, J., Raatz, S. K., & Nyman, J. A. (2009).

Monetary costs associated with bulimia. *International Journal of Eating Disorders*, *42*, 81-83. https://doi.org/10.1002/eat.20581

Culbert, K. M., Racine, S. E., & Klump, K. L. (2015). Research Review: What we have learned about the causes of eating disorders - a synthesis of sociocultural, psychological, and biological research. *Journal of Child Psychology and Psychiatry*, *56*, 1141-1164. https://doi.org/10.1111/jcpp.12441

Culbert, K. M., Racine, S. E., & Klump, K. L. (2011). The influence of gender and puberty on the heritability of disordered eating symptoms. *Current Topics in Behavioral Neuroscience*, *6*, 177-185. https://doi.org/10.1007/7854_2010_80

Currin, L., Schmidt, U., & Waller, G. (2007). Variables that influence diagnosis and treatment of the eating disorders within primary care settings: a vignette study. *International Journal of Eating Disorders*, *40*, 257-262. https://doi.org/10.1002/eat.20355

Currin, L., Waller, G., & Schmidt, U. (2009). Primary care physicians' knowledge of and attitudes toward the eating disorders: Do they affect clinical actions. *International Journal of Eating Disorders*, *42*, 453-458. https://doi.org/10.1002/eat.20636

Currin, L., Waller, G., Treasure, J., Nodder, J., Stone, C., Yeomans, M., & Schmidt, U. (2007). The use of guidelines for dissemination of "best practice" in primary care of patients with eating disorders. *International Journal of Eating Disorders*, *40*, 476-479. https://doi.org/10.1002/eat.20385

Diemer, E. W., Grant, J. D., Munn-Chernoff, M. A., Patterson, D. A., & Duncan, A. E. (2015). Gender identity, sexual orientation, and eating-related pathology in a national sample of college students. *Journal of Adolescent Health*, *57*, 144-149. https://doi.org/10.1016/j.jadohealth.2015.03.003

Dold, M., Aigner, M., Klabunde, M., Treasure, J., & Kasper, S. (2015). Second-generation antipsychotic drugs in anorexia nervosa: A Meta-analysis of randomized controlled trials. *Psychotherapy and Psychosomatics*, *84*, 110-116. https://doi.org/10.1159/000369978

Dölemeyer, R., Tietjen, A., Kersting, A., & Wagner, B. (2013). Internet-based interventions for eating disorders in adults: A systematic review. *BMC Psychiatry*, *13*, 207. https://doi.org/10.1186/1471-244X-13-207

Duncan, L., Yilmaz, Z., Gaspar, H., Walters, R., Goldstein, J., Antilla, V., Bulik-Sullivan, B., . . . Bulik, C. M. (in press). Genome-wide association study reveals first locus for anorexia nervosa and metabolic correlations. *American Journal of Psychiatry.* https://doi.org/10.1101/088815

Ehrlich, S., Lord, A. R., Geisler, D., Borchardt, V., Boehm, I., Seidel, M., . . . Walter, M. (2015). Reduced functional connectivity in the thalamo-insular subnetwork in patients with acute anorexia nervosa. *Human Brain Mapping*, *36*, 1772-1781. https://doi.org/10.1002/hbm.22736

Eisler, I. (2005). The empirical and theoretical base of family therapy and multiple family day therapy for adolescent anorexia nervosa. *Journal of Family Therapy*, https://doi.org/10.1111/j.1467-6427.2005.00303.x

Eisler, I., Simic, M., Hodsoll, J., Asen, E., Berelowitz, M., Connan, F., . . . Landau, S. (2016). A pragmatic randomised multi-centre trial of multifamily and single family therapy for adolescent anorexia nervosa. *BMC Psychiatry*, *16*, 422. https://doi.org/10.1186/s12888-016-1129-6

Field, A. E., Austin, S. B., Taylor, C. B., Malspeis, S., Rosner, B., Rockett, H. R., . . . Colditz, G. A. (2003). Relation between dieting and weight change among preadolescents and

adolescents. *Pediatrics*, *112*, 900-906.

Flament, M. F., Henderson, K., Buchholz, A., Obeid, N., Nguyen, H. N., Birmingham, M., & Goldfield, G. (2015). Weight status and DSM-5 diagnoses of eating disorders in adolescents from the community. *Journal of the American Academy of Child and Adolescent Psychiatry*, *54*, 403-411.e2. https://doi.org/10.1016/j.jaac.2015.01.020

Frank, G. (2013). Altered brain reward circuits in eating disorders: Chicken or egg. *Current Psychiatry Reports*, *15*, 396. https://doi.org/10.1007/s11920-013-0396-x

Frank, G. K. (2015). Recent advances in neuroimaging to model eating disorder neurobiology. *Current Psychiatry Reports*, *17*, 559. https://doi.org/10.1007/s11920-015-0559-z

French, S. A., Story, M., Remafedi, G., Resnick, M. D., & Blum, R. W. (1996). Sexual orientation and prevalence of body dissatisfaction and eating disordered behaviors: A population-based study of adolescents. *International Journal of Eating Disorders*, *19*, 119-126. https://doi.org/10.1002/(SICI)1098-108X(199603)19:2<119::AID-EAT2>3.0.CO;2-Q

Gatt, L., Jan, S., Mondraty, N., Horsfield, S., Hart, S., Russell, J., . . . Essue, B. (2014). The household economic burden of eating disorders and adherence to treatment in Australia. *BMC Psychiatry*, *14*, 338. https://doi.org/10.1186/s12888-014-0338-0

Gaudiani, J. L., & Mehler, P. S. (2016). Rare medical manifestations of severe restricting and purging: "Zebras," missed diagnoses, and best practices. *International Journal of Eating Disorders*, *49*, 331-344. https://doi.org/10.1002/eat.22475

Giel, K. E., Kullmann, S., Preißl, H., Bischoff, S. C., Thiel, A., Schmidt, U., . . . Teufel, M. (2013). Understanding the reward system functioning in anorexia nervosa: Crucial role of physical activity. *Biological Psychology*, *94*, 575-581. https://doi.org/10.1016/j.biopsycho.2013.10.004

Godier, L. R., de Wit, S., Pinto, A., Steinglass, J. E., Greene, A. L., Scaife, J., . . . Park, R. J. (2016). An investigation of habit learning in anorexia nervosa. *Psychiatry Research*, *244*, 214-222. https://doi.org/10.1016/j.psychres.2016.07.051

Groff, S. E. (2015). Is enhanced cognitive behavioral therapy an effective intervention in eating disorders? A review. *Journal of Evidence Informed Social Work*, *12*, 272-288.

Hadland, S. E., Austin, S. B., Goodenow, C. S., & Calzo, J. P. (2014). Weight misperception and unhealthy weight control behaviors among sexual minorities in the general adolescent population. *Journal of Adolescent Health*, *54*, 296-303. https://doi.org/10.1016/j.jadohealth.2013.08.021

Haedt-Matt, A. A., & Keel, P. K. (2011). Revisiting the affect regulation model of binge eating: a meta-analysis of studies using ecological momentary assessment. *Psychological Bulletin*, *137*, 660-681. https://doi.org/10.1037/a0023660

Hay, P. (2013). A systematic review of evidence for psychological treatments in eating disorders: 2005-2012. *International Journal of Eating Disorders*, *46*, 462-469. https://doi.org/10.1002/eat.22103

Hay, P. J., & Claudino, A. M. (2012). Clinical psychopharmacology of eating disorders: a research update. *International Journal of Neuropsychopharmacology*, *15*, 209-222. https://doi.org/10.1017/S1461145711000460

Hoek, H. W., & van Hoeken, D. (2003). Review of the prevalence and incidence of eating disorders. *International Journal of Eating Disorders*, *34*, 383-396. https://doi.org/10.1002/eat.10222

Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry*,

61, 348-358. https://doi.org/10.1016/j.biopsych.2006.03.040

Javaras, K. N., Runfola, C. D., Thornton, L. M., Agerbo, E., Birgegård, A., Norring, C., . . . Bulik, C. M. (2015). Sex- and age-specific incidence of healthcare-register-recorded eating disorders in the complete swedish 1979-2001 birth cohort. *International Journal of Eating Disorders*, *48*, 1070-1081. https://doi.org/10.1002/eat.22467

Johnson, J. G., Cohen, P., Kasen, S., & Brook, J. S. (2002). Childhood adversities associated with risk for eating disorders or weight problems during adolescence or early adulthood. *American Journal of Psychiatry*, *159*, 394-400. https://doi.org/10.1176/appi.ajp.159.3.394

Kaye, W. (2008). Neurobiology of anorexia and bulimia nervosa. *Physiology & Behavior*, *94*, 121-135. https://doi.org/10.1016/j.physbeh.2007

Kaye, W. H., Frank, G. K., Bailer, U. F., Henry, S. E., Meltzer, C. C., Price, J. C., . . . Wagner, A. (2005). Serotonin alterations in anorexia and bulimia nervosa: New insights from imaging studies. *Physiology & Behavior*, *85*, 73-81.

Kaye, W. H., Wierenga, C. E., Bailer, U. F., Simmons, A. N., & Bischoff-Grethe, A. (2013a). Nothing tastes as good as skinny feels: the neurobiology of anorexia nervosa. *Trends in Neuroscience*, *36*, 110-120. https://doi.org/10.1016/j.tins.2013.01.003

Kaye, W. H., Wierenga, C. E., Bailer, U. F., Simmons, A. N., Wagner, A., & Bischoff-Grethe, A. (2013b). Does a shared neurobiology for foods and drugs of abuse contribute to extremes of food ingestion in anorexia and bulimia nervosa. *Biological Psychiatry*, *73*, 836-842. https://doi.org/10.1016/j.biopscyh.2013.01.002.

Keel, P. K., & Brown, T. A. (2010). Update on course and outcome in eating disorders. *International Journal of Eating Disorders*, *43*, 195-204. https://doi.org/10.1002/eat.20810/abstract

Keshaviah, A., Edkins, K., Hastings, E. R., Krishna, M., Franko, D. L., Herzog, D. B., . . . Eddy, K. T. (2014). Re-examining premature mortality in anorexia nervosa: A meta-analysis redux. *Comprehensive Psychiatry*, *55*, 1773-1784. https://doi.org/10.1016/j.comppsych.2014.07.017

Keski-Rahkonen, A., Hoek, H. W., Linna, M. S., Raevuori, A., Sihvola, E., Bulik, C. M., . . . Kaprio, J. (2009). Incidence and outcomes of bulimia nervosa: A nationwide population-based study. *Psychological Medicine*, *39*, 823-831. https://doi.org/10.1017/S0033291708003942

Kessler, R. C., Berglund, P. A., Chiu, W. T., Deitz, A. C., Hudson, J. I., Shahly, V., . . . Xavier, M. (2013). The prevalence and correlates of binge eating disorder in the World Health Organization World Mental Health Surveys. *Biological Psychiatry*, *73*, 904-914. https://doi.org/10.1016/j.biopsych.2012.11.020

Kimmel, M. C., Ferguson, E. H., Zerwas, S., Bulik, C. M., & Meltzer-Brody, S. (2016). Obstetric and gynecologic problems associated with eating disorders. *International Journal of Eating Disorders*, *49*, 260-275. https://doi.org/10.1002/eat.22483

Klabunde, M., Acheson, D. T., Boutelle, K. N., Matthews, S. C., & Kaye, W. H. (2013). Interoceptive sensitivity deficits in women recovered from bulimia nervosa. *Eating Behaviors*, *14*, 488-492. https://doi.org/10.1016/j.eatbeh.2013.08.002

Klein, D. A., Schebendach, J. E., Gershkovich, M., Bodell, L. P., Foltin, R. W., & Walsh, B. T. (2010). Behavioral assessment of the reinforcing effect of exercise in women with anorexia nervosa: further paradigm development and data. *International Journal of Eating Disorders*, *43*, 611-618. https://doi.org/10.1002/eat.20758

Klump, K. L. (2013). Puberty as a critical risk period for eating disorders: A review of human

and animal studies. *Hormones and Behavior*, 64, 399-410. https://doi.org/10.1016%2Fj.yhbeh.2013.02.019

Lang, K., Lopez, C., Stahl, D., Tchanturia, K., & Treasure, J. (2014). Central coherence in eating disorders: an updated systematic review and meta-analysis. *World Journal of Biological Psychiatry*, *15*, 586-598. https://doi.org/10.3109/15622975.2014.909606

Larsen, P. S., Strandberg-Larsen, K., Micali, N., & Andersen, A. M. (2015). Parental and child characteristics related to early-onset disordered eating: A systematic review. *Harvard Review of Psychiatry*, *23*, 395-412. doi:10.1097/HRP. 0000000000000073

Lavender, J. M., De Young, K. P., Franko, D. L., Eddy, K. T., Kass, A. E., Sears, M. S., & Herzog, D. B. (2011). An investigation of the joint longitudinal trajectories of low body weight, binge eating, and purging in women with anorexia nervosa and bulimia nervosa. *International Journal of Eating Disorders*, *44*, 679-686. https://doi.org/10.1002/eat.20880

Lavender, J. M., Shaw, J. A., Crosby, R. D., Feig, E. H., Mitchell, J. E., Crow, S. J., . . . Lowe, M. R. (2015). Associations between weight suppression and dimensions of eating disorder psychopathology in a multisite sample. *Journal of Psychiatric Research*, *69*, 87-93. https://doi.org/10.1016/j.jpsychires.2015.07.021

le Grange, D., Lock, J., Agras, W. S., Bryson, S. W., & Jo, B. (2015). Randomized clinical trial of family-based treatment and cognitive-behavioral therapy for adolescent bulimia nervosa. *Journal of the American Academy of Child and Adolescent Psychiatry*, *54*, 886-94.e2. doi:10.1016/j.jaac.2015.08.008

le Grange, D., Lock, J., Loeb, K., & Nicholls, D. (2010). Academy for Eating Disorders position paper: the role of the family in eating disorders. *International Journal of Eating Disorders*, *43*, 1-5. https://doi.org/10.1002/eat.20751

Lebow, J., Sim, L. A., Erwin, P. J., & Murad, M. H. (2013). The effect of atypical antipsychotic medications in individuals with anorexia nervosa: a systematic review and meta-analysis. *International Journal of Eating Disorders*, *46*, 332-339. https://doi.org/10.1002/eat.22059

Lindvall Dahlgren, C., & Wisting, L. (2016). Transitioning from DSM-IV to DSM-5: A systematic review of eating disorder prevalence assessment. *International Journal of Eating Disorders*, *49*, 975-997. https://doi.org/10.1002/eat.22596

Lock, J., Grange, D. L., Agras, W. S., Moye, A., Bryson, S., & Booil, J. (2010). Randomized clinical trial comparing family-based treatment with adolescent-focused individual therapy for adolescents with anorexia nervosa. *Archives of General Psychiatry*, *67*, 1025-1032. https://doi.org/10.1001/archgenpsychiatry.2010.128.

Lopez, C., Tchanturia, K., Stahl, D., & Treasure, J. (2008). Central coherence in eating disorders: A systematic review. *Psychological Medicine*, *38*, 1393-1404. https://doi.org/10.1017/S0033291708003486

Mahr, F., Farahmand, P., Bixler, E. O., Domen, R. E., Moser, E. M., Nadeem, T., . . . Halmi, K. A. (2015). A national survey of eating disorder training. *International Journal of Eating Disorders*, *48*, 443-445. https://doi.org/10.1002/eat.22335

Martín, J., Padierna, A., Aguirre, U., Quintana, J. M., Hayas, C. L., & Muñoz, P. (2011). Quality of life among caregivers of patients with eating disorders. *Quality of Life Research*, *20*, 1359-1369. https://doi.org/10.1007/s11136-011-9873-z

Mascolo, M., Trent, S., Colwell, C., & Mehler, P. S. (2012). What the emergency department needs to know when caring for your patients with eating disorders. *International Journal of Eating Disorders*, *45*, 977-981. https://doi.org/10.1002/eat.22035

McCrone, P., Dhanasiri, S., Patel, A., Knapp, M., & Lawton-Smith, S. (2007). Paying the price:

The cost of mental health care in England to 2026. Retrieved from https://www.kingsfund.org.uk/sites/files/kf/Paying-the-Price-the-cost-of-mental-health-care-England-2026-McCrone-Dhanasiri-Patel-Knapp-Lawton-Smith-Kings-Fund-May-2008_0.pdf

Mehler, P. S., & Brown, C. (2015). Anorexia nervosa - medical complications. *Journal of Eating Disorders*, *3*, 11. https://doi.org/10.1186/s40337-015-0040-8

Mehler, P. S., & Rylander, M. (2015). Bulimia Nervosa - medical complications. *Journal of Eating Disorders*, *3*, 12. https://doi.org/10.1186/s40337-015-0044-4

Mitchison, D., & Hay, P. J. (2014). The epidemiology of eating disorders: Genetic, environmental, and societal factors. *Clinical Epidemiology*, *6*, 89-97. https://doi.org/10.2147/CLEP.S40841

Monteleone, P., & Maj, M. (2013). Dysfunctions of leptin, ghrelin, BDNF and endocannabinoids in eating disorders: beyond the homeostatic control of food intake. *Psychoneuroendocrinology*, *38*, 312-330. https://doi.org/10.1016/j.psyneuen.2012.10.021

Moore, F., & Keel, P. K. (2003). Influence of sexual orientation and age on disordered eating attitudes and behaviors in women. *International Journal of Eating Disorders*, *34*, 370-374. https://doi.org/10.1002/eat.10198

Munkholm, A., Olsen, E. M., Rask, C. U., Clemmensen, L., Rimvall, M. K., Jeppesen, P., . . . Skovgaard, A. M. (2016). Early predictors of eating problems in preadolescence-a prospective birth cohort study. *Journal of Adolescent Health*, *58*, 533-542. https://doi.org/10.1016/j.jadohealth.2016.01.006

Murray, S. B., Anderson, L. K., Cusack, A., Nakamura, T., Rockwell, R., Griffiths, S., & Kaye, W. H. (2015). Integrating family-based treatment and dialectical behavior therapy for

adolescent bulimia nervosa: Preliminary outcomes of an open pilot trial. *Eating Disorders*, *23*, 336-344. https://doi.org/10.1080/10640266.2015.1044345

Murray, S. M., Tulloch, A. J., Chen, E. Y., & Avena, N. M. (2015). Insights revealed by rodent models of sugar binge eating. *CNS Spectrums*, *20*, 530-536. https://doi.org/10.1017/S1092852915000656

Neumark-Sztainer, D., Wall, M., Guo, J., Story, M., Haines, J., & Eisenberg, M. (2006). Obesity, disordered eating, and eating disorders in a longitudinal study of adolescents: How do dieters fare 5 years later? *Journal of the American Dietitics Association*, *106*, 559-568. https://doi.org/10.1016/j.jada.2006.01.003

Nicholls, D. E., & Viner, R. M. (2009). Childhood risk factors for lifetime anorexia nervosa by age 30 years in a national birth cohort. *Journal of the American Academy of Child and Adolescent Psychiatry*, *48*, 791-799. https://doi.org/10.1097/CHI.0b013e3181ab8b75

Norris, M. L., Harrison, M. E., Isserlin, L., Robinson, A., Feder, S., & Sampson, M. (2016). Gastrointestinal complications associated with anorexia nervosa: A systematic review. *International Journal of Eating Disorders*, *49*, 216-237. https://doi.org/10.1002/eat.22462

O'Connor, G., & Nicholls, D. (2013). Refeeding hypophosphatemia in adolescents with anorexia nervosa: A systematic review. *Nutrition in Clinical Practice*, *28*, 358-364. https://doi.org/10.1177/0884533613476892

O'Hara, C. B., Campbell, I. C., & Schmidt, U. (2015). A reward-centered model of anorexia nervosa: A focused narrative review of the neurological and psychophysiological literature. *Neuroscience & Biobehavioral Reviews*, *52*, 131-152. https://doi.org/10.1016/j.neubiorev.2015.02.012

Pearson, C. M., & Smith, G. T. (2015). Bulimic symptom onset in young girls: A longitudinal

trajectory analysis. *Journal of Abnormal Psychology*, *124*, 1003-1013. https://doi.org/10.1037/abn0000111

Peat, C. M., Brownley, K. A., Berkman, N. D., & Bulik, C. M. (2012). Binge eating disorder: Evidence-based treatments. *Current Psychiatry*, *11*, 32-39.

Pike, K. M. (1998). Long-term course of anorexia nervosa: Response, relapse, remission, and recovery. *Clinical Psychology Review*, *18*, 447-475.

Pike, K. M., Dunne, P. E., & Addai, E. (2013). Expanding the boundaries: Reconfiguring the demographics of the "typical" eating disordered patient. *Current Psychiatry Reports*, *15*, 411. https://doi.org/10.1007/s11920-013-0411-2

Pike, K. M., Hoek, H. W., & Dunne, P. E. (2014). Cultural trends and eating disorders. *Current Opinion in Psychiatry*, *27*, 436-442. https://doi.org/10.1097/YCO.0000000000000100

Pisetsky, E. M., Thornton, L. M., Lichtenstein, P., Pedersen, N. L., & Bulik, C. M. (2013). Suicide attempts in women with eating disorders. *Journal of Abnormal Psychology*, *122*, 1042-1056. https://doi.org/10.1037/a0034902

Preti, A., Girolamo, G., Vilagut, G., Alonso, J., Graaf, R., Bruffaerts, R., . . . ESEMeD-WMH, I. (2009). The epidemiology of eating disorders in six European countries: Results of the ESEMeD-WMH project. *Journal of Psychiatric Research*, *43*, 1125-1132. https://doi.org/10.1016/j.jpsychires.2009.04.003

Preti, A., Rocchi, M. B., Sisti, D., Camboni, M. V., & Miotto, P. (2011). A comprehensive meta-analysis of the risk of suicide in eating disorders. *Acta Psychiatrica Scandinavica*, *124*, 6-17. https://doi.org/10.1111/j.1600-0447.2010.01641.x

Qian, J., Hu, Q., Wan, Y., Li, T., Wu, M., Ren, Z., & Yu, D. (2013). Prevalence of eating disorders in the general population: A systematic review. *Shanghai Archives of Psychiatry*,

25, 212-223. https://doi.org/10.3969/j.issn.1002-0829.2013.04.003

Reas, D. L., & Grilo, C. M. (2008). Review and meta-analysis of pharmacotherapy for binge-eating disorder. *Obesity*, *16*, 2024-2038. https://doi.org/10.1038/oby.2008.333

Roberts, A., Cash, T. F., Feingold, A., & Johnson, B. T. (2006). Are black-white differences in females' body dissatisfaction decreasing? A meta-analytic review. *Journal of Consulting and Clinical Psychology*, *74*, 1121-1131. https://doi.org/10.1037/0022-006X.74.6.1121

Roberts, M. E., Tchanturia, K., Stahl, D., Southgate, L., & Treasure, J. (2007). A systematic review and meta-analysis of set-shifting ability in eating disorders. *Psychological Medicine*, *37*, 1075-1084. https://doi.org/10.1017/S0033291707009877

Rodino, I. S., Byrne, S. M., & Sanders, K. A. (2016). Eating disorders in the context of preconception care: Fertility specialists' knowledge, attitudes, and clinical practices. *Fertility and Sterility*, https://doi.org/10.1016/j.fertnstert.2016.10.036

Russell, C. J., & Keel, P. K. (2002). Homosexuality as a specific risk factor for eating disorders in men. *International Journal of Eating Disorders*, *31*, 300-306.

Sachs, K. V., Harnke, B., Mehler, P. S., & Krantz, M. J. (2016). Cardiovascular complications of anorexia nervosa: A systematic review. *International Journal of Eating Disorders*, *49*, 238-248. https://doi.org/10.1002/eat.22481

Saltzman, J. A., & Liechty, J. M. (2016). Family correlates of childhood binge eating: A systematic review. *Eating Behaviors*, *22*, 62-71. https://doi.org/10.1016/j.eatbeh.2016.03.027

Scherma, M., Fattore, L., Castelli, M. P., Fratta, W., & Fadda, P. (2014). The role of the endocannabinoid system in eating disorders: Neurochemical and behavioural preclinical evidence. *Current Pharmaceutical Design*, *20*, 2089-2099.

Seitz, J., Bühren, K., von Polier, G. G., Heussen, N., Herpertz-Dahlmann, B., & Konrad, K. (2014). Morphological changes in the brain of acutely ill and weight-recovered patients with anorexia nervosa. A meta-analysis and qualitative review. *Zeitschrift für Kinder- und Jungendsychiatrie und Psychotherapie*, *42*, 7-17; quiz 17. https://doi.org/10.1024/1422-4917/a000265

Shapiro, J. R., Berkman, N. D., Brownley, K. A., Sedway, J. A., Lohr, K. N., & Bulik, C. M. (2007). Bulimia nervosa treatment: a systematic review of randomized controlled trials. *International Journal of Eating Disorders*, *40*, 321-336. https://doi.org/10.1002/eat.20372

Sheehan, D. V., & Herman, B. K. (2015). The psychological and medical factors associated with untreated binge eating disorder. *Primary Care Companion CNS Disorders*, *17*, https://doi.org/10.4088/PCC.14r01732

Shoebridge, P., & Gowers, S. G. (2000). Parental high concern and adolescent-onset anorexia nervosa. A case-control study to investigate direction of causality. *British Journal of Psychiatry*, *176*, 132-137. https://doi.org/10.1192/bjp.176.2.132

Sim, L. A., Homme, J. H., Lteif, A. N., Vande Voort, J. L., Schak, K. M., & Ellingson, J. (2009). Family functioning and maternal distress in adolescent girls with anorexia nervosa. *International Journal of Eating Disorders*, *42*, 531-539. https://doi.org/10.1002/eat.20654

Skunde, M., Walther, S., Simon, J. J., Wu, M., Bendszus, M., Herzog, W., & Friederich, H. C. (2016). Neural signature of behavioural inhibition in women with bulimia nervosa. *Journal of Psychiatry and Neuroscience*, *41*, E69-78.

Smink, F. R., van Hoeken, D., & Hoek, H. W. (2012). Epidemiology of eating disorders: Incidence, prevalence and mortality rates. *Current Psychiatry Reports*, *14*, 406-414. https://doi.org/10.1007/s11920-012-0282-y

Steinglass, J. E., & Walsh, B. T. (2016). Neurobiological model of the persistence of anorexia nervosa. *Journal of Eating Disorders*, *4*, 19. https://doi.org/10.1186/s40337-016-0106-2

Steinhausen, H. C., Jakobsen, H., Helenius, D., Munk-Jørgensen, P., & Strober, M. (2015). A nation-wide study of the family aggregation and risk factors in anorexia nervosa over three generations. *International Journal of Eating Disorders*, *48*, 1-8. https://doi.org/10.1002/eat.22293

Steinhausen, H. C., & Weber, S. (2009). The outcome of bulimia nervosa: Findings from one-quarter century of research. *American Journal of Psychiatry*, *166*, 1331-1341. https://doi.org/10.1176/appi.ajp.2009.09040582

Stice, E., & Bohon, C. (2013). Eating Disorders. In T. P. H. S. P. Beauchaine (Ed.), *Child and Adolescent Psychopathology* (pp. 715-738). Hoboken NJ: Wiley.

Stice, E., Marti, C. N., & Rohde, P. (2013). Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-year prospective community study of young women. *Journal of Abnormal Psychology*, *122*, 445-457.

Stice, E., Cameron, R. P., Killen, J. D., Hayward, C., & Taylor, C. B. (1999). Naturalistic weight-reduction efforts prospectively predict growth in relative weight and onset of obesity among female adolescents. *Journal of Consulting and Clinical Psychology*, *67*, 967.

Stice, E., Marti, C. N., Shaw, H., & Jaconis, M. (2009). An 8-year longitudinal study of the natural history of threshold, subthreshold, and partial eating disorders from a community sample of adolescents. *Journal of Abnormal Psychology*, *118*, 587-597. https://doi.org/http://dx.doi.org/10.1037/a0016481

Stice, E., Presnell, K., & Spangler, D. (2002). Risk factors for binge eating onset in adolescent

girls: A 2-year prospective investigation. *Health Psychology*, *21*, 131-138. https://doi.org/10.1037//0278-6133.21.2.131

Strober, M., Freeman, R., Lampert, C., Diamond, J., & Kaye, W. (2000). Controlled family study of anorexia nervosa and bulimia nervosa: Evidence of shared liability and transmission of partial syndromes. *American Journal of Psychiatry*, *157*, 393-401.

Strober, M., & Humphrey, L. L. (1987). Familial contributions to the etiology and course of anorexia nervosa and bulimia. *Journal of Consulting and Clinical Psychology*, *55*, 654-659.

Stuhldreher, N., Konnopka, A., Wild, B., Herzog, W., Zipfel, S., Löwe, B., & König, H. H. (2012). Cost-of-illness studies and cost-effectiveness analyses in eating disorders: A systematic review. *International Journal of Eating Disorders*, *45*, 476-491. https://doi.org/10.1002/eat.20977

Suisman, J. L., Thompson, J. K., Keel, P. K., Burt, S. A., Neale, M., Boker, S., . . . Klump, K. L. (2014). Genetic and environmental influences on thin-ideal internalization across puberty and preadolescent, adolescent, and young adult development. *International Journal of Eating Disorders*, *47*, 773-783. https://doi.org/10.1002/eat.22321

Titova, O. E., Hjorth, O. C., Schiöth, H. B., & Brooks, S. J. (2013). Anorexia nervosa is linked to reduced brain structure in reward and somatosensory regions: a meta-analysis of VBM studies. *BMC Psychiatry*, *13*, 110. https://doi.org/10.1186/1471-244X-13-110

Trace, S. E., Baker, J. H., Peñas-Lledó, E., & Bulik, C. (2013). The genetics of eating disorders. *Annual Review of Clinical Psychology*, *9*, 589-620. https://doi.org/10.1146/annurev-clinpsy-050212-185546

Treasure, J., Murphy, T., Szmukler, G., Todd, G., Gavan, K., & Joyce, J. (2001). The experience of caregiving for severe mental illness: A comparison between anorexia nervosa and

psychosis. *Social Psychiatry and Psychiatric Epidemiology*, *36*, 343-347.

Treasure, J., Stein, D., & Maguire, S. (2015). Has the time come for a staging model to map the course of eating disorders from high risk to severe enduring illness? An examination of the evidence. *Early Intervention in Psychiatry*, *9*, 173-184. https://doi.org/10.1111/eip.12170

Vall, E., & Wade, T. D. (2015). Trail making task performance in inpatients with anorexia nervosa and bulimia nervosa. *European Eating Disorders Review*, *23*, 304-311. https://doi.org/10.1002/erv.2364

Van den Eynde, F., Giampietro, V., Simmons, A., Uher, R., Andrew, C. M., Harvey, P. O., . . . Schmidt, U. (2013). Brain responses to body image stimuli but not food are altered in women with bulimia nervosa. *BMC Psychiatry*, *13*, 302. https://doi.org/10.1186/1471-244X-13-302

Van den Eynde, F., Suda, M., Broadbent, H., Guillaume, S., Van den Eynde, M., Steiger, H., . . . Schmidt, U. (2012). Structural magnetic resonance imaging in eating disorders: A systematic review of voxel-based morphometry studies. *European Eating Disorders Review*, *20*, 94-105. https://doi.org/10.1002/erv.1163

Wagner, A., Simmons, A. N., Oberndorfer, T. A., Frank, G. K., McCurdy-McKinnon, D., Fudge, J. L., . . . Kaye, W. H. (2015). Altered sensitization patterns to sweet food stimuli in patients recovered from anorexia and bulimia nervosa. *Psychiatry Research*, *234*, 305-313. https://doi.org/10.1016/j.pscychresns.2015.10.010

Watson, H. J., Joyce, T., French, E., Willan, V., Kane, R. T., Tanner-Smith, E. E., . . . Egan, S. J. (2016). Prevention of eating disorders: A systematic review of randomized, controlled trials. *International Journal of Eating Disorders*, *49*, 833-862. https://doi.org/10.1002/eat.22577

Welch, E., Ghaderi, A., & Swenne, I. (2015). A comparison of clinical characteristics between adolescent males and females with eating disorders. *BMC Psychiatry*, *15*, 45. https://doi.org/10.1186/s12888-015-0419-8

Westmoreland, P., Krantz, M. J., & Mehler, P. S. (2016). Medical complications of anorexia nervosa and bulimia. *American Journal of Medicine*, *129*, 30-37. https://doi.org/10.1016/j.amjmed.2015.06.031

Weygandt, M., Schaefer, A., Schienle, A., & Haynes, J. D. (2012). Diagnosing different binge-eating disorders based on reward-related brain activation patterns. *Human Brain Mapping*, *33*, 2135-2146. https://doi.org/10.1002/hbm.21345

Whisman, M. A., Dementyeva, A., Baucom, D. H., & Bulik, C. M. (2012). Marital functioning and binge eating disorder in married women. *International Journal of Eating Disorders*, *45*, 385-389. https://doi.org/10.1002/eat.20935

Whitney, J., Murray, J., Gavan, K., Todd, G., Whitaker, W., & Treasure, J. (2005). Experience of caring for someone with anorexia nervosa: qualitative study. *British Journal of Psychiatry*, *187*, 444-449. https://doi.org/10.1192/bjp.187.5.444

Wierenga, C. E., Ely, A., Bischoff-Grethe, A., Bailer, U. F., Simmons, A. N., & Kaye, W. H. (2014). Are extremes of consumption in eating disorders related to an altered balance between reward and inhibition. *Frontiers in Behavioral Neuroscience*, *8*, 410. https://doi.org/10.3389/fnbeh.2014.00410

Wu, M., Hartmann, M., Skunde, M., Herzog, W., & Friederich, H. C. (2013). Inhibitory control in bulimic-type eating disorders: A systematic review and meta-analysis. *PLoS One*, *8*, e83412. https://doi.org/10.1371/journal.pone.0083412

Yager, J. (1982). Family issues in the pathogenesis of anorexia nervosa. *Psychosomatic*

Medicine, 44, 43-60.

Yao, S., Kuja-Halkola, R., Thornton, L. M., Runfola, C. D., D'Onofrio, B. M., Almqvist, C., . . . Bulik, C. M. (2016). Familial liability for eating disorders and suicide attempts: Evidence from a population registry in Sweden. *JAMA Psychiatry*, *73*, 284-291. https://doi.org/10.1001/jamapsychiatry.2015.2737

Yilmaz, Z., Hardaway, J. A., & Bulik, C. M. (2015). Genetics and Epigenetics of Eating Disorders. *Advances in Genomics and Genetics*, *5*, 131-150. https://doi.org/10.2147/AGG.S55776

Zerwas, S., Larsen, J. T., Petersen, L., Thornton, L. M., Mortensen, P. B., & Bulik, C. M. (2015). The incidence of eating disorders in a Danish register study: Associations with suicide risk and mortality. *Journal of Psychiatric Research*, *65*, 16-22. https://doi.org/10.1016/j.jpsychires.2015.03.003

Supplementary Table S3. Somatic and Psychiatric Comorbidities and Psychosocial Manifestations Associated with Eating Disorders

Outcome	Anorexia nervosa	Bulimia Nervosa	Binge-Eating Disorder	Source
Somatic manifestations and comorbidities				
Body composition				
Body fat content and distribution	El Ghoch, Calugi, Lamburghini, & Dalle Grave, 2014; Greco, Lenzi, & Migliaccio, 2016			SR; NR
Increased bone marrow fat	Donaldson & Gordon, 2015; Hardouin, Rharass, & Lucas, 2016; Scheller, Burr, MacDougald, & Cawthorn, 2016			NR
Bone mineral density	Osteopenia, osteoporosis and fractures (Donaldson & Gordon, 2015; El Ghoch et al., 2016; Greco et al., 2016; Misra, Golden, & Katzman, 2016; Misra & Klibanski, 2016; Robinson, Aldridge, Clark, Misra, & Micali, 2016; Schorr & Miller, 2017; Solmi, Veronese, Correll, et al., 2016; Thornton & Gordon, 2016; Westmoreland, Krantz, & Mehler, 2016)	Osteopenia (Robinson et al., 2016)		NR; SR; MA
Increased physical activity	Achamrah, Coëffier, & Déchelotte, 2016; Gümmer et al., 2015			SR
Brain				
Gray matter atrophy	Seitz et al., 2014; Titova, Hjorth, Schiöth, & Brooks, 2013; Westmoreland et al., 2016			SR; MA
White matter atrophy	Seitz et al., 2014; Titova et al., 2013;			NR;

Outcome	Anorexia nervosa	Bulimia Nervosa	Binge-Eating Disorder	Source
	Westmoreland et al., 2016			QR; MA
Cardiovascular system				
Atherosclerotic vascular disease	Sachs, Harnke, Mehler, & Krantz, 2016			SR
Coronary heart disease			Olguin et al., 2016	SR
Autonomic dysfunction/altered heart rate variability	Sachs et al., 2016	Increased variability (Peschel et al., 2016a, 2016b)	Decreased variability (Mitchell, 2016; Olguin et al., 2016)	SR
Decreased left ventricular mass	Sachs et al., 2016; Spaulding-Barclay, Stern, & Mehler, 2016			NR; SR
Decreased left ventricular end-diastolic and left ventricular end-systolic dimensions	Spaulding-Barclay et al., 2016			NR
Altered heart wall thicknesses	Spaulding-Barclay et al., 2016			NR
Lower cardiac output			Olguin et al., 2016	SR
Mitral valve prolapse	Sachs et al., 2016; Spaulding-Barclay et al., 2016			NR; SR
Myocardial fibrosis	Lamzabi et al., 2015; Sachs et al., 2016			SR; Autopsy
Cardiomyopathy		Westmoreland et al., 2016		NR
Sinus bradycardia	Sachs et al., 2016; Spaulding-Barclay et			NR; SR

Outcome	Anorexia nervosa	Bulimia Nervosa	Binge-Eating Disorder	Source
	al., 2016; Westmoreland et al., 2016			
Blood pressure	Hypotension (Sachs et al., 2016; Spaulding-Barclay et al., 2016)		Hypertension (Mitchell, 2016)	NR; SR
Postural orthostatic tachycardia syndrome	Sachs et al., 2016; Spaulding-Barclay et al., 2016			NR; SR
Cardiac arrhythmias		Westmoreland et al., 2016		NR
QT dispersion	Sachs et al., 2016; Spaulding-Barclay et al., 2016	Olguin et al., 2016		NR; SR
QT interval prolongation	Sachs et al., 2016; Spaulding-Barclay et al., 2016; Westmoreland et al., 2016	Olguin et al., 2016		NR; SR
Conduction delays	Sachs et al., 2016; Spaulding-Barclay et al., 2016			NR; SR
Junctional escape rhythms	Sachs et al., 2016; Spaulding-Barclay et al., 2016			NR; SR
Dysregulation of peripheral vasoconstriction/vasodilatation	Sachs et al., 2016			SR
Acrocyanosis	Sachs et al., 2016			SR
Arterial vasospasm	Sachs et al., 2016			SR
Dermatology				
Xerosis	Westmoreland et al., 2016			NR

Outcome	Anorexia nervosa	Bulimia Nervosa	Binge-Eating Disorder	Source
Lanugo hair	Westmoreland et al., 2016			NR
Hair thinning	Westmoreland et al., 2016			NR
Perniosis	Westmoreland et al., 2016			NR
Acne	Westmoreland et al., 2016			NR
Carotenoderma	Westmoreland et al., 2016			NR
Callus on back of hand (Russell's sign)		Mehler & Rylander, 2015		NR
Immunology				
Increased risk of infection	Dobner & Kaser, in press			SR
Hematology				
Anemia	Westmoreland et al., 2016			NR
Leukopenia	Westmoreland et al., 2016			NR
Thrombocytopenia	Westmoreland et al., 2016			NR
Dentistry				
Dental erosion	Hermont et al., 2014; Kisely, Baghaie, Lalloo, & Johnson, 2015	Hermont et al., 2014; Kisely et al., 2015; Westmoreland et al., 2016		NR; SR; MA
Decayed, missing and filled teeth or surfaces	Kisely et al., 2015	Kisely et al., 2015; Westmoreland et al., 2016		SR; MA

Outcome	Anorexia nervosa	Bulimia Nervosa	Binge-Eating Disorder	Source
Dry mouth	Kisely et al., 2015	Kisely et al., 2015		SR; MA
Endocrinology				
Hypogonadotropic hypogonadism with relative estrogen and androgen deficiency	Allaway, Southmayd, & De Souza, 2016; Donaldson & Gordon, 2015; Misra & Klibanski, 2016; Schorr & Miller, 2017; Westmoreland et al., 2016			NR
Growth hormone resistance with low insulin-like growth factors 1 (IGF-1)	Fazeli & Klibanski, 2014; Misra & Klibanski, 2016; Schorr & Miller, 2017			NR
Hyperaldosteronism		Westmoreland et al., 2016		NR
Hypercortisolemia	Donaldson & Gordon, 2015; Misra & Klibanski, 2016; Schorr & Miller, 2017; Westmoreland et al., 2016		Mitchell, 2016	NR
Thyroid function (Low T3 syndrome)	Donaldson & Gordon, 2015; Schorr & Miller, 2017			NR
Hypooxytocinemia	Romano, Tempesta, Micioni Di Bonaventura, & Gaetani, 2015; Rutigliano et al., 2016; Schorr & Miller, 2017			NR; SR; MA
Hypoleptinemia	Donaldson & Gordon, 2015; Schorr & Miller, 2017; Westmoreland et al., 2016			NR
Adiponectin	Elevated (Khalil & El Hachem, 2014; Scheller et al., 2016; Schorr & Miller, 2017)	Elevated, normal, decreased (Khalil & El Hachem, 2014)	Decreased (Khalil & El Hachem, 2014)	NR; SR

Outcome	Anorexia nervosa	Bulimia Nervosa	Binge-Eating Disorder	Source
Hypo- and hypervitaminosis	Veronese et al., 2015; Westmoreland et al., 2016			NR; MA
Gastrointestinal peptides				
Ghrelin	Increased (Misra & Klibanski, 2016; Müller & Tschöp, 2013; Schorr & Miller, 2017; Wittekind & Kluge, 2015)	Increased (Wittekind & Kluge, 2015)	Suppressed (Wittekind & Kluge, 2015)	NR; SR
Elevated peptide YY	Misra & Klibanski, 2016; Schorr & Miller, 2017			NR
Gastrointestinal symptoms				
Postprandial fullness	Sato & Fukudo, 2015			NR
Early satiety	Sato & Fukudo, 2015; Westmoreland et al., 2016			NR
Postprandial distress syndrome	Sato & Fukudo, 2015			NR
Altered esophageal motility	Sato & Fukudo, 2015; Weterle-Smolińska et al., 2015			NR
Inflammation of the esophagus		Mehler & Rylander, 2015		NR
Abdominal distention	Sato & Fukudo, 2015			NR
Abdominal pain	Sato & Fukudo, 2015			NR

Outcome	Anorexia nervosa	Bulimia Nervosa	Binge-Eating Disorder	Source
Gastric acid reflux		Westmoreland et al., 2016		NR
Gastric distension	Sato & Fukudo, 2015; Westmoreland et al., 2016			NR
Mallory-Weiss tears		Westmoreland et al., 2016		NR
Acute gastric dilatation	Westmoreland et al., 2016		Sato & Fukudo, 2015	NR
Gastric perforation			Sato & Fukudo, 2015	NR
Delayed gastric emptying	Norris et al., 2016; Westmoreland et al., 2016; Weterle-Smolińska et al., 2015			NR; SR
Superior mesenteric artery syndrome	Sato & Fukudo, 2015; Westmoreland et al., 2016			NR
Irritable bowel syndrome	Sato & Fukudo, 2015			NR
Functional constipation	Sato & Fukudo, 2015			NR
Intestinal dysbiosis	Kleiman et al., 2015			LRS
Celiac			Olguin et al., 2016	SR
Metabolism				
Diabetes Type 1			Olguin et al., 2016	SR
Diabetes Type 2		García-Mayor & García-Soidán, in press	García-Mayor & García-Soidán, in press; Olguin et al., 2016; Racicka & Bryńska, 2015	NR; SR
Electrolyte and acid-base	Mehler & Walsh, 2016	Mehler & Walsh, 2016;		SR

Outcome	Anorexia nervosa	Bulimia Nervosa	Binge-Eating Disorder	Source
abnormalities		Westmoreland et al., 2016		
Hyponatremia	Schorr & Miller, 2017			NR
Hypokalemia		Westmoreland et al., 2016		NR
Elevated liver transaminases	Westmoreland et al., 2016			NR
Metabolic syndrome			Mitchell, 2016	SR
Plasma tryptophan lowered during acute disease	Gauthier, Launay, Thiebaud, & Godart, 2015			SR
Nephrology				
Urinary urgency	Stheneur, Bergeron, & Lapeyraque, 2014			SR
Urinary lithiasis	Stheneur et al., 2014			SR
Nocturnal enuresis	Stheneur et al., 2014			SR
Neurology				
Epilepsy			Olguin et al., 2016	SR
Idiopathic intracranial hypertension			Olguin et al., 2016	SR
Obstetrics and gynecology				
Menstrual disturbances	Kimmel, Ferguson, Zerwas, Bulik, & Meltzer-Brody, 2016	Kimmel et al., 2016	Kimmel et al., 2016	SR
Infertility		Kimmel et al., 2016		SR

Outcome	Anorexia nervosa	Bulimia Nervosa	Binge-Eating Disorder	Source
Unplanned pregnancy	Kimmel et al., 2016	Kimmel et al., 2016		SR
Miscarriage	Kimmel et al., 2016	Kimmel et al., 2016	Kimmel et al., 2016	SR
Poor nutrition during pregnancy	Kimmel et al., 2016		Kimmel et al., 2016	SR
Hyperemesis gravidarum		Kimmel et al., 2016		SR
Sexual dysfunction	Kimmel et al., 2016	Kimmel et al., 2016	Kimmel et al., 2016	SR
Earlier cessation of breastfeeding	Kimmel et al., 2016			SR
Polycystic ovarian syndrome		Kimmel et al., 2016	Kimmel et al., 2016; Olguin et al., 2016	SR
Gynecologic cancers	Kimmel et al., 2016		Kimmel et al., 2016	SR
Otolaryngology (Ear, nose, and throat)				
Dysphagia	Westmoreland et al., 2016	Westmoreland et al., 2016		NR
Epistaxis		Westmoreland et al., 2016		NR
Lagophthalmos	Westmoreland et al., 2016			NR
Laryngitis		Mehler & Rylander, 2015		NR
Olfaction	Islam et al., 2015			SR
Parotid gland enlargement		Westmoreland et al., 2016		NR
Pharyngitis		Mehler & Rylander, 2015		NR

Outcome	Anorexia nervosa	Bulimia Nervosa	Binge-Eating Disorder	Source
Sialadenosis		Westmoreland et al., 2016		NR
Vocal cord inflammation		Westmoreland et al., 2016		NR
Psychiatric Comorbidities and Psychosocial Manifestations				
Attention deficit hyperactivity disorder	Nazar et al., 2016; Ptacek et al., 2016	Levin & Rawana, 2016; Nazar et al., 2016; Ptacek et al., 2016	Levin & Rawana, 2016; Nazar et al., 2016	NR, SR, MA
Adverse life experiences			Palmisano, Innamorati, & Vanderlinden, 2016	SR
Anxiety	Jenkins, Hoste, Meyer, & Blissett, 2011; Kezelman, Touyz, Hunt, & Rhodes, 2015	Jenkins et al., 2011	Jenkins et al., 2011	SR
Autistic traits	Westwood et al., 2016			SR, MA
Bipolar disorder		Álvarez Ruiz & Gutiérrez-Rojas, 2015	Álvarez Ruiz & Gutiérrez-Rojas, 2015	SR
Body image disturbances	Legenbauer, Thiemann, & Vocks, 2014	Legenbauer et al., 2014		NR
Childhood abuse		Caslini et al., 2016	Caslini et al., 2016; Palmisano et al., 2016	SR, MA
Depression	Abbate-Daga et al., 2015; Puccio, Fuller-Tyszkiewicz, Ong, & Krug, 2016	Puccio et al., 2016	Puccio et al., 2016	NR; SR, MA
Interpersonal problems	Jones, Lindekilde, Lübeck, & Clausen, 2015	Jones et al., 2015		SR
Non-suicidal self-injury	Cucchi et al., 2016	Cucchi et al., 2016		SR, MA
Perinatal depression and	Kimmel et al., 2016	Kimmel et al., 2016	Kimmel et al., 2016	SR

Outcome	Anorexia nervosa	Bulimia Nervosa	Binge-Eating Disorder	Source
anxiety				
Personality disorders	Martinussen et al., 2016; Rotella, Fioravanti, & Ricca, 2016	Martinussen et al., 2016; Rotella et al., 2016	Friborg et al., 2014; Gerlach, Loeber, & Herpertz, 2016; Rotella et al., 2016	SR, MA
Psychotic experiences	Kouidrat, Amad, Lalau, & Loas, 2014; McGrath et al., 2016			RCS; SR
Reduced quality of life	Ágh et al., 2016; Jenkins et al., 2011; Winkler et al., 2014	Ágh et al., 2016; Jenkins et al., 2011; Winkler et al., 2014	Ágh et al., 2016; Jenkins et al., 2011; Winkler et al., 2014	SR, MA
Schizophrenia	Kouidrat et al., 2014	Kouidrat et al., 2014	Kouidrat et al., 2014	SR
Sexual dysfunction	Castellini, Lelli, Ricca, & Maggi, 2016; Kimmel et al., 2016; Waldinger, 2015	Castellini et al., 2016; Kimmel et al., 2016; Waldinger, 2015	Castellini et al., 2016; Kimmel et al., 2016	NR, SR
Sexual trauma	Madowitz, Matheson, & Liang, 2015	Madowitz et al., 2015	Madowitz et al., 2015	SR
Shame/Guilt	Jenkins et al., 2011	Jenkins et al., 2011	Jenkins et al., 2011	SR
Sleep disturbances	Allison, Spaeth, & Hopkins, 2016	Allison et al., 2016	Allison et al., 2016; Olguin et al., 2016	NR, SR
Smoking		Solmi, Veronese, Sergi, et al., 2016	Solmi, Veronese, Sergi, et al., 2016	SR, MA
Substance use disorder	Cucchi et al., 2016	Cucchi et al., 2016	Schreiber, Odlaug, & Grant, 2013	SR, MA
Suicidality	Cucchi et al., 2016	Cucchi et al., 2016		SR, MA

NR: Narrative review; SR: Systematic review; MA: Meta-analysis; LRS: Longitudinal refeeding study; RCS: Retrospective cohort study

References for Supplementary Table S3

Abbate-Daga, G., Buzzichelli, S., Marzola, E., Aloi, M., Amianto, F., & Fassino, S. (2015). Does depression matter in neuropsychological performances in anorexia nervosa? A descriptive review. *International Journal of Eating Disorders*, *48*, 736–745. https://doi.org/10.1002/eat.22399

Achamrah, N., Coëffier, M., & Déchelotte, P. (2016). Physical activity in patients with anorexia nervosa. *Nutrition Reviews*, *74*, 301–311.

Ágh, T., Kovács, G., Supina, D., Pawaskar, M., Herman, B. K., Vokó, Z., & Sheehan, D. V. (2016). A systematic review of the health-related quality of life and economic burdens of anorexia nervosa, bulimia nervosa, and binge eating disorder. *Eating and Weight Disorders*, *21*, 353–364. https://doi.org/10.1093/nutrit/nuw001

Allaway, H. C. M., Southmayd, E. A., & De Souza, M. J. (2016). The physiology of functional hypothalamic amenorrhea associated with energy deficiency in exercising women and in women with anorexia nervosa. *Hormone Molecular Biology and Clinical Investigation*, *25*, 91–119. https://doi.org/10.1515/hmbci-2015-0053

Allison, K. C., Spaeth, A., & Hopkins, C. M. (2016). Sleep and eating disorders. *Current Psychiatry Reports*, *18*, 92. https://doi.org/10.1007/s11920-016-0728-8

Álvarez Ruiz, E. M., & Gutiérrez-Rojas, L. (2015). Comorbidity of bipolar disorder and eating disorders. *Revista de Psiquiatria y Salud Mental*, *8*, 232–241. https://doi.org/10.1016/j.jad.2004.11.008

Caslini, M., Bartoli, F., Crocamo, C., Dakanalis, A., Clerici, M., & Carrà, G. (2016). Disentangling the association between child abuse and eating disorders: A systematic review and meta-analysis. *Psychosomatic Medicine*, *78*, 79–90. https://doi.org/10.1097/PSY.0000000000000233

Castellini, G., Lelli, L., Ricca, V., & Maggi, M. (2016). Sexuality in eating disorders patients:

Etiological factors, sexual dysfunction and identity issues. A systematic review. *Hormone Molecular Biology and Clinical Investigation*, *25*, 71–90. https://doi.org/10.1515/hmbci-2015-0055

Cucchi, A., Ryan, D., Konstantakopoulos, G., Stroumpa, S., Kaçar, A. Ş., Renshaw, S., … Kravariti, E. (2016). Lifetime prevalence of non-suicidal self-injury in patients with eating disorders: A systematic review and meta-analysis. *Psychological Medicine*, *46*, 1345–1358. https://doi.org/10.1017/S0033291716000027

Dobner, J., & Kaser, S. (In Press). Body mass index and the risk of infection - from underweight to obesity. *Clinical Microbiology and Infection.* https://doi.org/10.1016/j.cmi.2017.02.013

Donaldson, A. A., & Gordon, C. M. (2015). Skeletal complications of eating disorders. *Metabolism: Clinical and Experimental*, *64*, 943–951. https://doi.org/10.1016/j.metabol.2015.06.007

El Ghoch, M., Calugi, S., Lamburghini, S., & Dalle Grave, R. (2014). Anorexia nervosa and body fat distribution: A systematic review. *Nutrients*, *6*, 3895–3912. https://doi.org/10.3390/nu6093895

El Ghoch, M., Gatti, D., Calugi, S., Viapiana, O., Bazzani, P. V., & Dalle Grave, R. (2016). The association between weight gain/restoration and bone mineral density in adolescents with anorexia nervosa: A systematic review. *Nutrients*, *8*, 769. https://doi.org/10.3390/nu8120769

Fazeli, P. K., & Klibanski, A. (2014). Determinants of GH resistance in malnutrition. *Journal of Endocrinology*, *220*, R57–65. https://doi.org/10.1530/JOE-13-0477

Friborg, O., Martinussen, M., Kaiser, S., Øvergård, K. T., Martinsen, E. W., Schmierer, P., & Rosenvinge, J. H. (2014). Personality disorders in eating disorder not otherwise specified and binge eating disorder: A meta-analysis of comorbidity studies. *Journal of Nervous*

and Mental Disease, *202*, 119–125. https://doi.org/10.1097/NMD.0000000000000080

García-Mayor, R. V., & García-Soidán, F. J. (In Press). Eating disorders in type 2 diabetic people: Brief review. *Diabetes & Metabolic Syndrome*. https://doi.org/10.1016/j.dsx.2016.08.004

Gauthier, C., Launay, J.-M., Thiebaud, M.-R., & Godart, N. (2015). The Impact of malnutrition on the peripheral serotoninergic system in anorexia nervosa: A systematic review. *Current Psychiatry Reviews*, *11*, 8–18. https://doi.org/10.2174/1573400510666140619211433

Gerlach, G., Loeber, S., & Herpertz, S. (2016). Personality disorders and obesity: A systematic review. *Obesity Reviews*, *17*, 691–723. https://doi.org/10.1111/obr.12415

Greco, E. A., Lenzi, A., & Migliaccio, S. (2016). The pathophysiological basis of bone tissue alterations associated with eating disorders. *Hormone Molecular Biology and Clinical Investigation, 28,* 121-132. https://doi.org/10.1515/hmbci-2016-0006

Gümmer, R., Giel, K. E., Schag, K., Resmark, G., Junne, F. P., Becker, S., … Teufel, M. (2015). High levels of physical activity in anorexia nervosa: A systematic review. *European Eating Disorders Review*, *23*, 333–344. https://doi.org/10.1002/erv.2377

Hardouin, P., Rharass, T., & Lucas, S. (2016). Bone marrow adipose tissue: To be or not to be a typical adipose tissue? *Frontiers in Endocrinology*, *7*, 85. https://doi.org/10.3389/fendo.2016.00085

Hermont, A. P., Oliveira, P. A. D., Martins, C. C., Paiva, S. M., Pordeus, I. A., & Auad, S. M. (2014). Tooth erosion and eating disorders: A systematic review and meta-analysis. *PloS One*, *9*, e111123. https://doi.org/10.1371/journal.pone.0111123

Islam, M. A., Fagundo, A. B., Arcelus, J., Agüera, Z., Jiménez-Murcia, S., Fernández-Real, J. M., … Fernandez-Aranda, F. (2015). Olfaction in eating disorders and abnormal eating behavior: A systematic review. *Frontiers in Psychology*, *6*, 1431. https://doi.org/

10.3389/fpsyg.2015.01431

Jenkins, P. E., Hoste, R. R., Meyer, C., & Blissett, J. M. (2011). Eating disorders and quality of life: A review of the literature. *Clinical Psychology Review*, *31*, 113–121. https://doi.org/10.1016/j.cpr.2010.08.003

Jones, A., Lindekilde, N., Lübeck, M., & Clausen, L. (2015). The association between interpersonal problems and treatment outcome in the eating disorders: A systematic review. *Nordic Journal of Psychiatry*, *69*, 563–573. https://doi.org/10.3109/08039488.2015.1019924

Kezelman, S., Touyz, S., Hunt, C., & Rhodes, P. (2015). Does anxiety improve during weight restoration in anorexia nervosa? A systematic review. *Journal of Eating Disorders*, *3*, 7. https://doi.org/10.1186/s40337-015-0046-2

Khalil, R. B., & El Hachem, C. (2014). Adiponectin in eating disorders. *Eating and Weight Disorders*, *19*, 3–10. https://doi.org/10.1007/s40519-013-0094-z

Kimmel, M. C., Ferguson, E. H., Zerwas, S., Bulik, C. M., & Meltzer-Brody, S. (2016). Obstetric and gynecologic problems associated with eating disorders. *International Journal of Eating Disorders*, *49*, 260–275. https://doi.org/10.1002/eat.22483

Kisely, S., Baghaie, H., Lalloo, R., & Johnson, N. W. (2015). Association between poor oral health and eating disorders: Systematic review and meta-analysis. *British Journal of Psychiatry*, *207*, 299–305. https://doi.org/10.1192/bjp.bp.114.156323

Kleiman, S. C., Watson, H. J., Bulik-Sullivan, E. C., Huh, E. Y., Tarantino, L. M., Bulik, C. M., & Carroll, I. M. (2015). The intestinal microbiota in acute anorexia nervosa and during renourishment: Relationship to depression, anxiety, and eating disorder psychopathology. *Psychosomatic Medicine*, *77*, 969–981. https://doi.org/10.1097/PSY.0000000000000247

Kouidrat, Y., Amad, A., Lalau, J.-D., & Loas, G. (2014). Eating disorders in schizophrenia:

Implications for research and management. *Schizophrenia Research and Treatment*, *2014*, 791573. https://doi.org/10.1155/2014/791573

Lamzabi, I., Syed, S., Reddy, V. B., Jain, R., Harbhajanka, A., & Arunkumar, P. (2015). Myocardial changes in a patient with anorexia nervosa: A case report and review of literature. *American Journal of Clinical Pathology*, *143*, 734–737. https://doi.org/10.1309/AJCP4PLFF1TTKENT

Legenbauer, T., Thiemann, P., & Vocks, S. (2014). Body image disturbance in children and adolescents with eating disorders. Current evidence and future directions. *Zeitschrift für Kinder- und Jugendpsychiatrie und Psychotherapie*, *42*, 51–59. https://doi.org/10.1024/1422-4917/a000269

Levin, R. L., & Rawana, J. S. (2016). Attention-deficit/hyperactivity disorder and eating disorders across the lifespan: A systematic review of the literature. *Clinical Psychology Review*, *50*, 22–36. https://doi.org/10.1016/j.cpr.2016.09.010

Madowitz, J., Matheson, B. E., & Liang, J. (2015). The relationship between eating disorders and sexual trauma. *Eating and Weight Disorders*, *20*, 281–293. https://doi.org/10.1007/s40519-015-0195-y.

Martinussen, M., Friborg, O., Schmierer, P., Kaiser, S., Øvergård, K. T., Neunhoeffer, A.-L., … Rosenvinge, J. H. (2016). The comorbidity of personality disorders in eating disorders: A meta-analysis. *Eating and Weight Disorders,* 1-9. https://doi.org/10.1007/s40519-016-0345-x

McGrath, J. J., Saha, S., Al-Hamzawi, A., Andrade, L., Benjet, C., Bromet, E. J., … Kessler, R. C. (2016). The bidirectional associations between psychotic experiences and DSM-IV mental disorders. *American Journal of Psychiatry*, *173*, 997–1006. https://doi.org/10.1176/appi.ajp.2016.15101293

Mehler, P. S., & Rylander, M. (2015). Bulimia nervosa - medical complications. *Journal of*

Eating Disorders, *3*, 12. https://doi.org/10.1186/s40337-015-0044-4

Mehler, P. S., & Walsh, K. (2016). Electrolyte and acid-base abnormalities associated with purging behaviors. *International Journal of Eating Disorders*, *49*, 311–318. https://doi.org/10.1002/eat.22503

Misra, M., Golden, N. H., & Katzman, D. K. (2016). State of the art systematic review of bone disease in anorexia nervosa. *International Journal of Eating Disorders*, *49*, 276–292. https://doi.org/10.1002/eat.22451

Misra, M., & Klibanski, A. (2016). Anorexia nervosa and its associated endocrinopathy in young people. *Hormone Research in Pediatrics, 85*, 147-157. https://doi.org/10.1159/000443735

Mitchell, J. E. (2016). Medical comorbidity and medical complications associated with binge-eating disorder. *International Journal of Eating Disorders*, *49*, 319–323. https://doi.org/10.1002/eat.22452

Müller, T. D., & Tschöp, M. H. (2013). Ghrelin - a key pleiotropic hormone-regulating systemic energy metabolism. *Endocrine Development*, *25*, 91–100. https://doi.org/10.1159/000346590

Nazar, B. P., Bernardes, C., Peachey, G., Sergeant, J., Mattos, P., & Treasure, J. (2016). The risk of eating disorders comorbid with attention-deficit/hyperactivity disorder: A systematic review and meta-analysis. *International Journal of Eating Disorders*, *49*, 1045–1057. https://doi.org/10.1002/eat.22643

Norris, M. L., Harrison, M. E., Isserlin, L., Robinson, A., Feder, S., & Sampson, M. (2016). Gastrointestinal complications associated with anorexia nervosa: A systematic review. *International Journal of Eating Disorders*, *49*, 216–237. https://doi.org/10.1002/eat.22462

Olguin, P., Fuentes, M., Gabler, G., Guerdjikova, A. I., Keck, P. E., Jr., & McElroy, S. L.

(2016). Medical comorbidity of binge eating disorder. *Eating and Weight Disorders, 22,* 13-26. https://doi.org/10.1007/s40519-016-0313-5

Palmisano, G. L., Innamorati, M., & Vanderlinden, J. (2016). Life adverse experiences in relation with obesity and binge eating disorder: A systematic review. *Journal of Behavioral Addictions*, *5*, 11–31. https://doi.org/10.1556/2006.5.2016.018

Peschel, S. K. V., Feeling, N. R., Vögele, C., Kaess, M., Thayer, J. F., & Koenig, J. (2016a). A meta-analysis on resting state high-frequency heart rate variability in bulimia nervosa. *European Eating Disorders Review*, *24*, 355–365. https://doi.org/10.1002/erv.2454

Peschel, S. K. V., Feeling, N. R., Vögele, C., Kaess, M., Thayer, J. F., & Koenig, J. (2016b). A systematic review on heart rate variability in bulimia nervosa. *Neuroscience and Biobehavioral Reviews*, *63*, 78–97. https://doi.org/10.1016/j.neubiorev.2016.01.012

Ptacek, R., Stefano, G. B., Weissenberger, S., Akotia, D., Raboch, J., Papezova, H., … Goetz, M. (2016). Attention deficit hyperactivity disorder and disordered eating behaviors: Links, risks, and challenges faced. *Neuropsychiatric Disease and Treatment*, *12*, 571–579. https://doi.org/10.2147/NDT.S68763

Puccio, F., Fuller-Tyszkiewicz, M., Ong, D., & Krug, I. (2016). A systematic review and meta-analysis on the longitudinal relationship between eating pathology and depression. *International Journal of Eating Disorders*, *49*, 439–454. https://doi.org/10.1002/eat.22506

Racicka, E., & Bryńska, A. (2015). Eating disorders in children and adolescents with Type 1 and Type 2 Diabetes: Prevalence, risk factors, warning signs. *Psychiatria Polska*, *49*, 1017–1024. https://doi.org/10.12740/PP/39536

Robinson, L., Aldridge, V., Clark, E. M., Misra, M., & Micali, N. (2016). A systematic review and meta-analysis of the association between eating disorders and bone density. *Osteoporosis International, 27*, 1953-1966. https://doi.org/10.1007/s00198-015-3468-4

Romano, A., Tempesta, B., Micioni Di Bonaventura, M. V., & Gaetani, S. (2015). From autism to eating disorders and more: The role of oxytocin in neuropsychiatric disorders. *Frontiers in Neuroscience*, *9*, 497. https://doi.org/10.3389/fnins.2015.00497

Rotella, F., Fioravanti, G., & Ricca, V. (2016). Temperament and personality in eating disorders. *Current Opinion in Psychiatry*, *29*, 77–83. https://doi.org/10.1097/YCO.0000000000000212

Rutigliano, G., Rocchetti, M., Paloyelis, Y., Gilleen, J., Sardella, A., Cappucciati, M., … Fusar-Poli, P. (2016). Peripheral oxytocin and vasopressin: Biomarkers of psychiatric disorders? A comprehensive systematic review and preliminary meta-analysis. *Psychiatry Research*, *241*, 207–220. https://doi.org/10.1016/j.psychres.2016.04.117

Sachs, K. V., Harnke, B., Mehler, P. S., & Krantz, M. J. (2016). Cardiovascular complications of anorexia nervosa: A systematic review. *International Journal of Eating Disorders*, *49*, 238–248. https://doi.org/10.1002/eat.22481

Sato, Y., & Fukudo, S. (2015). Gastrointestinal symptoms and disorders in patients with eating disorders. *Clinical Journal of Gastroenterology*, *8*, 255–263. https://doi.org/10.1007/s12328-015-0611-x

Scheller, E. L., Burr, A. A., MacDougald, O. A., & Cawthorn, W. P. (2016). Inside out: Bone marrow adipose tissue as a source of circulating adiponectin. *Adipocyte*, *5*, 251–269. https://doi.org/10.1080/21623945.2016.1149269

Schorr, M., & Miller, K. K. (2017). The endocrine manifestations of anorexia nervosa: Mechanisms and management. *Nature Reviews. Endocrinology*, *13*, 174–186. https://doi.org/10.1038/nrendo.2016.175

Schreiber, L. R. N., Odlaug, B. L., & Grant, J. E. (2013). The overlap between binge eating disorder and substance use disorders: Diagnosis and neurobiology. *Journal of Behavioral Addictions*, *2*, 191–198. https://doi.org/10.1556/JBA.2.2013.015

Seitz, J., Bühren, K., von Polier, G. G., Heussen, N., Herpertz-Dahlmann, B., & Konrad, K. (2014). Morphological changes in the brain of acutely ill and weight-recovered patients with anorexia nervosa. A meta-analysis and qualitative review. *Zeitschrift für Kinder- und Jugendpsychiatrie und Psychotherapie*, *42*, 7–17. https://doi.org/10.1024/1422-4917/a000265

Solmi, M., Veronese, N., Correll, C. U., Favaro, A., Santonastaso, P., Caregaro, L., … Stubbs, B. (2016). Bone mineral density, osteoporosis, and fractures among people with eating disorders: A systematic review and meta-analysis. *Acta Psychiatrica Scandinavica*, *133*, 341–351. https://doi.org/10.1111/acps.12556

Solmi, M., Veronese, N., Sergi, G., Luchini, C., Favaro, A., Santonastaso, P., … Stubbs, B. (2016). The association between smoking prevalence and eating disorders: A systematic review and meta-analysis. *Addiction*, *111*, 1914–1922. https://doi.org/10.1111/add.13457

Spaulding-Barclay, M. A., Stern, J., & Mehler, P. S. (2016). Cardiac changes in anorexia nervosa. *Cardiology in the Young*, *26*, 623–628. https://doi.org/10.1017/S104795111500267X

Stheneur, C., Bergeron, S., & Lapeyraque, A.-L. (2014). Renal complications in anorexia nervosa. *Eating and Weight Disorders*, *19*, 455–460. https://doi.org/10.1007/s40519-014-0138-z

Thornton, D., & Gordon, C. M. (2016). Restrictive eating disorders and skeletal health in adolescent girls and young women. *Calcified Tissue International*. https://doi.org/10.1007/s00223-016-0164-0

Titova, O. E., Hjorth, O. C., Schiöth, H. B., & Brooks, S. J. (2013). Anorexia nervosa is linked to reduced brain structure in reward and somatosensory regions: A meta-analysis of VBM studies. *BMC Psychiatry*, *13*, 110. https://doi.org/10.1186/1471-244X-13-110

Veronese, N., Solmi, M., Rizza, W., Manzato, E., Sergi, G., Santonastaso, P., … Correll, C.

U. (2015). Vitamin D status in anorexia nervosa: A meta-analysis. *International Journal of Eating Disorders*, *48*, 803–813. https://doi.org/10.1002/eat.22370

Waldinger, M. D. (2015). Psychiatric disorders and sexual dysfunction. *Handbook of Clinical Neurology*, *130*, 469–489. https://doi.org/10.1016/B978-0-444-63247-0.00027-4

Westmoreland, P., Krantz, M. J., & Mehler, P. S. (2016). Medical complications of anorexia nervosa and bulimia. *American Journal of Medicine*, *129*, 30–37. https://doi.org/10.1097/01.yco.0000228768.79097.3e

Westwood, H., Eisler, I., Mandy, W., Leppanen, J., Treasure, J., & Tchanturia, K. (2016). Using the autism-spectrum quotient to measure autistic traits in anorexia nervosa: A systematic review and meta-analysis. *Journal of Autism and Developmental Disorders*, *46*, 964–977. https://doi.org/10.1007/s10803-015-2641-0

Weterle-Smolińska, K. A., Banasiuk, M., Dziekiewicz, M., Ciastoń, M., Jagielska, G., & Banaszkiewicz, A. (2015). Gastrointestinal motility disorders in patients with anorexia nervosa - a review of the literature. *Psychiatria Polska*, *49*, 721–729. https://doi.org/10.12740/PP/35482

Winkler, L. A.-D., Christiansen, E., Lichtenstein, M. B., Hansen, N. B., Bilenberg, N., & Støving, R. K. (2014). Quality of life in eating disorders: A meta-analysis. *Psychiatry Research*, *219*, 1–9. https://doi.org/10.1016/j.psychres.2014.05.002

Wittekind, D. A., & Kluge, M. (2015). Ghrelin in psychiatric disorders - A review. *Psychoneuroendocrinology*, *52*, 176–194. https://doi.org/10.1016/j.psyneuen.2014.11.013

Supplementary Table S4. Psychological and Neurocognitive Traits Associated with Eating Disorders

Trait	Anorexia Nervosa	Bulimia Nervosa	Binge-Eating Disorder	Source
Alexithymia	Pinna, Sanna, & Carpiniello, 2015			SR
Attachment insecurity	Gander, Sevecke, & Buchheim, 2015; Jewell et al., 2016; Tasca & Balfour, 2014	Gander et al., 2015; Tasca & Balfour, 2014	Gander et al., 2015; Tasca & Balfour, 2014	SR
Weak central coherence	Lang, Lopez, Stahl, Tchanturia, & Treasure, 2014	Lang et al., 2014		SR, MA
Attentional, information processing, and memory biases			Kittel, Brauhardt, & Hilbert, 2015; Voon, 2015	SR
Altered decision making	Guillaume et al., 2015; Mudan Wu et al., 2016	Guillaume et al., 2015; Mudan Wu et al., 2016	Voon, 2015; Mudan Wu et al., 2016	SR, MA
Emotion dysregulation	D'Agostino, Covanti, Rossi Monti, & Starcevic, 2017; Lavender et al., 2015; Oldershaw, Lavender, Sallis, Stahl, & Schmidt, 2015	D'Agostino et al., 2017; Lavender et al., 2015	Kittel et al., 2015; Nicholls, Devonport, & Blake, 2016; Schulte, Grilo, & Gearhardt, 2016	CR; NR; MA
Impulsivity		Pearson, Riley, Davis, & Smith, 2014	Schulte et al., 2016	
Impaired inhibitory control	Wu, Hartmann, Skunde, Herzog, & Friederich, 2013	Mudan Wu et al., 2013	Lavagnino, Arnone, Cao, Soares, & Selvaraj, 2016; Mudan Wu et al., 2013	SR, MA
Difficulties in recognition of emotions (mentalization)	Jewell et al., 2016			SR
Negative urgency		Culbert, Racine, & Klump, 2015; Pearson et al., 2014	Culbert et al., 2015	SR
High levels of perfectionism	Limburg, Watson, Hagger, & Egan, 2016; Rotella, Fioravanti, & Ricca, 2016	Limburg et al., 2016; Rotella et al., 2016		NR; MA
Obsessive-compulsive	Crane, Roberts, & Treasure, 2007	Pearson, Wonderlich, & Smith, 2015		SR

Trait	Anorexia Nervosa	Bulimia Nervosa	Binge-Eating Disorder	Source
personality traits				
Reward	Aberrant (O'Hara, Campbell, & Schmidt, 2015)		Dysfunction (Schulte et al., 2016)	NR
Difficulties with set shifting	Van Autreve & Vervaet, 2015; Wu et al., 2014	Wu et al., 2014	Wu et al., 2014	SR, MA
Low self-esteem	Jenkins, Hoste, Meyer, & Blissett, 2011	Jenkins et al., 2011	Jenkins et al., 2011	SR
Temperament	Increased persistence, harm avoidance (Atiye, Miettunen, & Raevuori-Helkamaa, 2015)	Increased persistence, novelty seeking, harm avoidance (Atiye et al., 2015)	Increased harm avoidance (Atiye et al., 2015)	MA

SR: Systematic review; NR: Narrative review; MA: Meta-analysis; CR: Conceptual review

References for Supplementary Table S4

Atiye, M., Miettunen, J., & Raevuori-Helkamaa, A. (2015). A meta-analysis of temperament in eating disorders. *European Eating Disorders Review*, *23*, 89–99. https://doi.org/10.1002/erv.2342

Crane, A. M., Roberts, M. E., & Treasure, J. (2007). Are obsessive-compulsive personality traits associated with a poor outcome in anorexia nervosa? A systematic review of randomized controlled trials and naturalistic outcome studies. *International Journal of Eating Disorders*, *40*, 581–588. https://doi.org/10.1002/eat.20419

Culbert, K. M., Racine, S. E., & Klump, K. L. (2015). Research Review: What we have learned about the causes of eating disorders - a synthesis of sociocultural, psychological, and biological research. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *56*, 1141–1164. https://doi.org/10.1111/jcpp.12441

D'Agostino, A., Covanti, S., Rossi Monti, M., & Starcevic, V. (2017). Reconsidering emotion dysregulation. *Psychiatric Quarterly, 88*, 1-19. https://doi.org/10.1007/s11126-017-9499-6

Gander, M., Sevecke, K., & Buchheim, A. (2015). Eating disorders in adolescence: Attachment issues from a developmental perspective. *Frontiers in Psychology*, *6*, 1136. https://doi.org/10.3389/fpsyg.2015.01136

Guillaume, S., Gorwood, P., Jollant, F., Van den Eynde, F., Courtet, P., & Richard-Devantoy, S. (2015). Impaired decision-making in symptomatic anorexia and bulimia nervosa patients: A meta-analysis. *Psychological Medicine*, *45*, 3377–3391. https://doi.org/10.1017/S003329171500152X.

Jenkins, P. E., Hoste, R. R., Meyer, C., & Blissett, J. M. (2011). Eating disorders and quality of life: A review of the literature. *Clinical Psychology Review*, *31*, 113–121. https://doi.org/10.1016/j.cpr.2010.08.003

Jewell, T., Collyer, H., Gardner, T., Tchanturia, K., Simic, M., Fonagy, P., & Eisler, I. (2016). Attachment and mentalization and their association with child and adolescent eating pathology: A systematic review. *International Journal of Eating Disorders*, *49*, 354–373. https://doi.org/10.1002/eat.22473

Kittel, R., Brauhardt, A., & Hilbert, A. (2015). Cognitive and emotional functioning in binge-eating disorder: A systematic review. *International Journal of Eating Disorders*, *48*, 535–554. https://doi.org/10.1002/eat.22419

Lang, K., Lopez, C., Stahl, D., Tchanturia, K., & Treasure, J. (2014). Central coherence in eating disorders: An updated systematic review and meta-analysis. *World Journal of Biological Psychiatry*, *15*, 586–598. https://doi.org/10.3109/15622975.2014.909606

Lavagnino, L., Arnone, D., Cao, B., Soares, J. C., & Selvaraj, S. (2016). Inhibitory control in obesity and binge eating disorder: A systematic review and meta-analysis of neurocognitive and neuroimaging studies. *Neuroscience and Biobehavioral Reviews*, *68*, 714–726. https://doi.org/10.1016/j.neubiorev.2016.06.041

Lavender, J. M., Wonderlich, S. A., Engel, S. G., Gordon, K. H., Kaye, W. H., & Mitchell, J. E. (2015). Dimensions of emotion dysregulation in anorexia nervosa and bulimia nervosa: A conceptual review of the empirical literature. *Clinical Psychology Review*, *40*, 111–122. https://doi.org/10.1016/j.cpr.2015.05.010

Limburg, K., Watson, H. J., Hagger, M. S., & Egan, S. J. (2016). The relationship between perfectionism and psychopathology: A meta-analysis. *Journal of Clinical Psychology*. https://doi.orh/10.1002/jclp.22435

Nicholls, W., Devonport, T. J., & Blake, M. (2016). The association between emotions and eating behaviour in an obese population with binge eating disorder. *Obesity Reviews*, *17*, 30–42. https://doi.org/10.1111/obr.12329

O'Hara, C. B., Campbell, I. C., & Schmidt, U. (2015). A reward-centered model of anorexia

nervosa: A focused narrative review of the neurological and psychophysiological literature. *Neuroscience and Biobehavioral Reviews*, *52*, 131–152. https://doi.org/10.1016/j.neubiorev.2015.02.012

Oldershaw, A., Lavender, T., Sallis, H., Stahl, D., & Schmidt, U. (2015). Emotion generation and regulation in anorexia nervosa: A systematic review and meta-analysis of self-report data. *Clinical Psychology Review*, *39*, 83–95. https://doi.org/10.1016/j.cpr.2015.04.005

Pearson, C. M., Riley, E. N., Davis, H. A., & Smith, G. T. (2014). Two pathways toward impulsive action: An integrative risk model for bulimic behavior in youth. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *55*, 852–864. https://doi.org/10.1111/jcpp.12214

Pearson, C. M., Wonderlich, S. A., & Smith, G. T. (2015). A risk and maintenance model for bulimia nervosa: From impulsive action to compulsive behavior. *Psychological Review*, *122*, 516–535. https://doi.org/10.1037/a0039268

Pinna, F., Sanna, L., & Carpiniello, B. (2015). Alexithymia in eating disorders: therapeutic implications. *Psychology Research and Behavior Management*, *8*, 1–15. https://doi.org/10.2147/PRBM.S52656

Rotella, F., Fioravanti, G., & Ricca, V. (2016). Temperament and personality in eating disorders. *Current Opinion in Psychiatry*, *29*, 77–83. https://doi.org/10.1097/YCO.0000000000000212

Schulte, E. M., Grilo, C. M., & Gearhardt, A. N. (2016). Shared and unique mechanisms underlying binge eating disorder and addictive disorders. *Clinical Psychology Review*, *44*, 125–139. https://doi.org/10.1016/j.cpr.2016.02.001

Tasca, G. A., & Balfour, L. (2014). Attachment and eating disorders: a review of current research. *International Journal of Eating Disorders*, *47*, 710–717. https://doi.org/10.1002/eat.22302

Van Autreve, S., & Vervaet, M. (2015). Are there differences in central coherence and set shifting across the subtypes of anorexia nervosa?: A systematic review. *Journal of Nervous and Mental Disease*, *203*, 774–780. https://doi.org/10.1097/NMD.0000000000000366

Voon, V. (2015). Cognitive biases in binge eating disorder: the hijacking of decision making. *CNS Spectrums*, *20*, 566–573. https://doi.org/10.1017/S1092852915000681

Wu, M., Brockmeyer, T., Hartmann, M., Skunde, M., Herzog, W., & Friederich, H.C. (2014). Set-shifting ability across the spectrum of eating disorders and in overweight and obesity: A systematic review and meta-analysis. *Psychological Medicine*, *44*, 3365–3385. https://doi.org/10.1017/S0033291714000294

Wu, M., Brockmeyer, T., Hartmann, M., Skunde, M., Herzog, W., & Friederich, H.-C. (2016). Reward-related decision making in eating and weight disorders: A systematic review and meta-analysis of the evidence from neuropsychological studies. *Neuroscience and Biobehavioral Reviews*, *61*, 177–196. https://doi.org/10.1016/j.neubiorev.2015.11.017

Wu, M., Hartmann, M., Skunde, M., Herzog, W., & Friederich, H.C. (2013). Inhibitory control in bulimic-type eating disorders: A systematic review and meta-analysis. *PloS One*, *8*(12), e83412. https://doi.org/10.1371/journal.pone.0083412

Supplementary Table S5. Summary of Psychological Treatments for Eating Disorders

	Anorexia Nervosa		Bulimia Nervosa		Binge-Eating Disorder	
Age Group	Tx	Source	Tx	Source	Tx	Source
Well Established Treatments						
Adolescents	**FBT**	Systematic reviews & meta-analyses (Couturier, Kimber, & Szatmari, 2013; Lock, 2015; Watson & Bulik, 2013) RCTs (Eisler et al., 2000; Eisler, Simic, Russell, & Dare, 2007; Lock et al., 2010; Lock, Agras, Bryson, & Kraemer, 2005; Robin et al., 1999; Russell, Szmukler, Dare, & Eisler, 1987)	**None**		**None**	
Adults	**None**		**CBT- I****	Systematic reviews & meta-analyses (Costa & Melnik, 2016; Grilo, Reas, & Mitchell, 2016; Hay, Bacaltchuk, Stefano, & Kashyap, 2009; Mitchell, Agras, & Wonderlich, 2007; Palavras, Hay, Filho, & Claudino, 2017; Shapiro et al., 2007; Wilson, Wilfley, Agras, & Bryson, 2010) RCTs (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000; Chen et al., 2003; Fairburn et al., 1995; Fairburn et al., 1991; Fairburn, Jones, Peveler, Hope, & O'Connor, 1993; Mitchell et al., 2011; Poulsen et al., 2014; Wonderlich et al., 2014)	**CBT-I**	Systematic reviews & meta-analyses (Brownley et al., 2016; Brownley, Berkman, Sedway, Lohr, & Bulik, 2007; Costa & Melnik, 2016; Palavras et al., 2017) RCTs (Dingemans, Spinhoven, & van Furth, 2007; Grilo, Masheb, Wilson, Gueorguieva, & White, 2011; Hilbert et al., 2012; Peterson, Mitchell, Crow, Crosby, & Wonderlich, 2009; Peterson, Mitchell, & Engbloom, 1998; Tasca et al., 2006)
			IPT	Reviews (Costa & Melnik, 2016; Hay et al., 2009) RCTs (Fairburn et al., 1995; Fairburn et al., 1993)	**IPT-G**	Systematic review (Wilson et al., 2010) RCTs (Hilbert et al., 2012; Wilfley et al., 2002)
Possibly Efficacious Treatments						
Adolescents	**FT-S** **IO**	RCTs (Agras et al., 2014; Godart et al., 2012) RCTs (Lock et al., 2010)	**CBT** **CBT-GSH**	RCTs (Stefini et al., 2017) RCTs (Schmidt et al., 2007)	**CBT-Int-GSH** **IPT**	RCTs (Jones et al., 2008) RCT (Tanofsky-Kraff et al., 2014) Pilot study (Tanofsky-Kraff et al., 2010)

Age Group	Anorexia Nervosa		Bulimia Nervosa		Binge-Eating Disorder	
	Tx	Source	Tx	Source	Tx	Source
Adolescents (cont.)						Case report & series (Tanofsky-Kraff et al., 2007; Tanofsky-Kraff, Shomaker, Young, & Wilfley, 2016)
			FBT	RCTs (le Grange, Crosby, Rathouz, & Leventhal, 2007; le Grange, Lock, Agras, Bryson, & Jo, 2015)		
			PD	RCTs (Stefini et al., 2017)		
Adults	**CBT-E-I**	RCTs (Dalle Grave, Calugi, Conti, Doll, & Fairburn, 2013; Zipfel et al., 2014)	**CBT-G**	RCTs (Chen et al., 2003; Katzman et al., 2010)	**BT*** **	RCTs (Grilo et al., 2011)
	FPT	RCTs (Wild et al., 2009; Zipfel et al., 2014)	**CBT-GSH**	RCTs (Bailer et al., 2004; Banasiak, Paxton, & Hay, 2005; Ghaderi & Scott, 2003)	**CBT-GSH**	RCTs (Carrard et al., 2011; Carter & Fairburn, 1998; Ghaderi & Scott, 2003; Grilo & Masheb, 2005)
	MANTRA	RCTs (Schmidt et al., 2012, 2015)	**CBT-Int**	RCTs (Mitchell et al., 2008; Sánchez-Ortiz et al., 2011)	**DBT**	RCTs (Telch, Agras, & Linehan, 2001)
				Pilot study (Shapiro et al., 2010)		Pilot study (Chen, Matthews, Allen, Kuo, & Linehan, 2008)
	SSCM	RCTs (Schmidt et al., 2012, 2015)	**ICAT**	RCTs (Wonderlich et al., 2014)	**DBT-G**	RCTs (Safer & Jo, 2010)
						Pilot study (Telch, Agras, & Linehan, 2000)
			PP	RCTs (Poulsen et al., 2014)		
Experimental Treatments						
Adolescents	**CBT-E- I**	Case series (Dalle Grave, Calugi, Doll, & Fairburn, 2013)	**CBT-E-I**	Case series (Lock, 2005)	**DBT**	Case series & reports (Safer, Couturier, & Lock, 2007; Salbach-Andrae, Bohnekamp, Pfeiffer, Lehmkuhl, & Miller, 2008)
	CT	RCTs (Lock et al., 2013) – no differences between clinical outcomes.	**DBT**	Pilot study (Fischer & Peterson, 2015)		
	CRT	Pilot studies (Herbrich et al., 2017; van Noort, Kraus, Pfeiffer, Lehmkuhl, & Kappel, 2016)	**SP-I**	RCTs (le Grange et al., 2007)		
		Case reports (Cwojdzińska, Markowska-Regulska, & Rybakowski, 2008; Giombini, Turton, Turco, Nesbitt, & Lask, 2016)				

Age Group	Anorexia Nervosa		Bulimia Nervosa		Binge-Eating Disorder	
	Tx	Source	Tx	Source	Tx	Source
Adolescents (cont.)	**CRT-G**	Pilot studies (Pretorius et al., 2012; Wood, Al-Khairulla, & Lask, 2011)				
	DBT	Case series (Salbach-Andrae et al., 2008)				
	ACT	Case series (Berman, Boutelle, & Crow, 2009)				
	C-TX	Open trial (Baucom et al., 2017; Watson & Bulik, 2013)	**DBT**	Pilot study (Chen et al., 2008)	**A-BBT**	Pilot study (Juarascio et al., 2017)
		Case report (Kirby, Fischer, Raney, Baucom, & Bulik, 2016)				
	CRT	Pilot Study (Mac Neil et al., 2016)				
Adults		Case reports & series (Abbate-Daga, Buzzichelli, Marzola, Amianto, & Fassino, 2012; Pitt, Lewis, Morgan, & Woodward, 2010; Tchanturia et al., 2008; Tchanturia, Davies, & Campbell, 2007)				
	CRT-G	Pilot study (Genders & Tchanturia, 2010)				

TX: Treatment; RCTs: Randomized Control Trials; Treatment Abbreviations: BT: Behavioral Therapy; CBT-E-I: Individual Enhanced Cognitive Behavioral Therapy; CBT-G: Group Cognitive Behavioral Therapy; CBT-GSH: Cognitive Behavioral Therapy - Guided Self Help; CBT-I: Individual Cognitive Behavioral Therapy; CBT-Int: Cognitive Behavioral Therapy Internet or Telemedicine; CBT-Int-GSH: Cognitive Behavioral Therapy Internet or Telemedicine Guided Self-Help; CRT: Cognitive Remediation Therapy ; CRT-G: Group Cognitive Remediation Therapy; CT: Cognitive Training; C-TX: Couples-Based Treatment; DBT: Dialectical Behavioral Therapy; DBT-G: Dialectical Behavioral Therapy; FPT: Focal Psychodynamic Therapy; FBT: Family Based Treatment; FT-S: Family Therapy - Systematic; ICAT: Integrative Cognitive- Affective Therapy; IPT: Interpersonal Psychotherapy; IO: Insight Oriented; MANTRA: Maudsley Model of Anorexia Nervosa Treatment for Adults; PP: Psychoanalytic Psychotherapy; PD: Psychodynamic Treatments; SP-I: Supportive Psychotherapy Individual; SSCM: Specialist Supportive Clinical Management

*Summary includes treatments for loss of control eating, a key symptom of binge eating found in children.

**Includes CBT-BN (Christopher G. Fairburn & Cooper, 1989; Latner & Wilson, 2000)

***Support for weight loss in patients with BED

References for Supplementary Table S5

Abbate-Daga, G., Buzzichelli, S., Marzola, E., Amianto, F., & Fassino, S. (2012). Effectiveness of cognitive remediation therapy (CRT) in anorexia nervosa: A case series. *Journal of Clinical and Experimental Neuropsychology*, *34*, 1009–1015. https://doi.org/10.1080/13803395.2012.704900

Agras, W. S., Lock, J., Brandt, H., Bryson, S. W., Dodge, E., Halmi, K. A., … Woodside, B. (2014). Comparison of 2 family therapies for adolescent anorexia nervosa: A randomized parallel trial. *JAMA Psychiatry*, *71*, 1279–1286. https://doi.org/10.1001/jamapsychiatry.2014

Agras, W. S., Walsh, T., Fairburn, C. G., Wilson, G. T., & Kraemer, H. C. (2000). A multicenter comparison of cognitive-behavioral therapy and interpersonal psychotherapy for bulimia nervosa. *Archives of General Psychiatry*, *57*, 459–466. https://doi.org/10.1001/archpsyc.57.5.459

Bailer, U., de Zwaan, M., Leisch, F., Strnad, A., Lennkh-Wolfsberg, C., El-Giamal, N., … Kasper, S. (2004). Guided self-help versus cognitive-behavioral group therapy in the treatment of bulimia nervosa. *International Journal of Eating Disorders*, *35*, 522–537. https://doi.org/10.1002/eat.20003

Banasiak, S. J., Paxton, S. J., & Hay, P. (2005). Guided self-help for bulimia nervosa in primary care: A randomized controlled trial. *Psychological Medicine*, *35*, 1283–1294. https://doi.org/10.1017/S0033291705004769

Baucom, D. H., Kirby, J. S., Fischer, M. S., Baucom, B. R., Hamer, R., & Bulik, C. M. (2017). Findings from a couple-based open trial for adult anorexia nervosa. *Journal of Family Psychology.* https://doi.org/10.1037/fam0000273

Berman, M., Boutelle, K., and Crow, S. (2009). A case series investigating acceptance and commitment therapy as a treatment for previously treated, unremitted patients with anorexia nervosa. *European Eating Disorders Review, 17*, 426-434. https://doi.org/10.1002/erv.962

Brownley, K. A., Berkman, N. D., Peat, C. M., Lohr, K. N., Cullen, K. E., Bann, C. M., & Bulik, C. M. (2016). Binge-eating disorder in adults: A systematic review and meta-analysis. *Annals of Internal Medicine*, *165*, 409–420. https://doi.org/10.7326/M15-2455

Brownley, K. A., Berkman, N. D., Sedway, J. A., Lohr, K. N., & Bulik, C. M. (2007). Binge eating disorder treatment: A systematic review of randomized controlled trials. *International Journal of Eating Disorders*, *40*, 337–348. https://doi.org/10.1002/eat.20370

Carrard, I., Crépin, C., Rouget, P., Lam, T., Golay, A., & Van der Linden, M. (2011). Randomised controlled trial of a guided self-help treatment on the internet for binge eating disorder. *Behaviour Research and Therapy*, *49*, 482–491. https://doi.org/10.1016/j.brat.2011.05.004

Carter, J. C., & Fairburn, C. G. (1998). Cognitive-behavioral self-help for binge eating disorder: A controlled effectiveness study. *Journal of Consulting and Clinical Psychology*, *66*, 616–623.

Chen, E., Touyz, S. W., Beumont, P. J. V., Fairburn, C. G., Griffiths, R., Butow, P., … Basten, C. (2003). Comparison of group and individual cognitive-behavioral therapy for patients with bulimia nervosa. *International Journal of Eating Disorders*, *33*, 241–54. https://doi.org/10.1002/eat.10137

Chen, E. Y., Matthews, L., Allen, C., Kuo, J. R., & Linehan, M. M. (2008). Dialectical behavior therapy for clients with binge-eating disorder or bulimia nervosa and borderline personality disorder. *International Journal of Eating Disorders*, *41*, 505–512. https://doi.org/10.1002/eat.20522

Costa, M. B., & Melnik, T. (2016). Effectiveness of psychosocial interventions in eating disorders: an overview of Cochrane systematic reviews. *Einstein*, *14*, 235–277. https://doi.org/10.1590/S1679-45082016RW3120

Couturier, J., Kimber, M., & Szatmari, P. (2013). Efficacy of family-based treatment for adolescents with eating disorders: A systematic review and meta-analysis. *International Journal of Eating Disorders*, *46*, 3–11. https://doi.org/10.1002/eat.22042

Cwojdzińska, A., Markowska-Regulska, K., & Rybakowski, F. (2008). Cognitive remediation therapy in adolescent anorexia nervosa--case report. *Psychiatria Polska*, *43*, 115–124.

Dalle Grave, R., Calugi, S., Conti, M., Doll, H., & Fairburn, C. G. (2013). Inpatient cognitive behaviour therapy for anorexia nervosa: A randomized controlled trial. *Psychotherapy and Psychosomatics*, *82*, 390–398. https://doi.org/10.1159/000350058

Dalle Grave, R., Calugi, S., Doll, H. A., & Fairburn, C. G. (2013). Enhanced cognitive behaviour therapy for adolescents with anorexia nervosa: An alternative to family therapy? *Behaviour Research and Therapy*, *51*, R9–R12. https://doi.org/10.1016/j.brat.2012.09.008

Dingemans, A. E., Spinhoven, P., & van Furth, E. F. (2007). Predictors and mediators of treatment outcome in patients with binge eating disorder. *Behaviour Research and Therapy*, *45*, 2551–2562. https://doi.org/10.1016/j.brat.2007.06.003

Eisler, I., Dare, C., Hodes, M., Russell, G., Dodge, E., & le Grange, D. (2000). Family therapy for adolescent anorexia nervosa: The results of a controlled comparison of two family interventions. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *41*, 727–736. https://doi.org/10.1111/1469-7610.00660

Eisler, I., Simic, M., Russell, G. F. M., & Dare, C. (2007). A randomised controlled treatment trial of two forms of family therapy in adolescent anorexia nervosa: A five-year follow-up. *Journal of Child Psychology and Psychiatry, 48*, 552–560. https://doi.org/10.1111/j.1469-7610.2007.01726.x

Fairburn, C. G., & Cooper, P. J. (1989). Eating disorders. In K. Hawton, P. M. Salkovskis, J. Kirk, & D. M. Clark (Eds.), *Cognitive Behaviour Therapy for Psychiatric Problems* (pp. 277–314). Oxford University Press.

Fairburn, C. G., Jones, R., Peveler, R. C., Carr, S. J., Solomon, R. A., O'Connor, M. E., … Hope, R. A. (1991). Three psychological treatments for bulimia nervosa: A comparative trial. *Archives of General Psychiatry*, *48*, 463–469.

Fairburn, C. G., Jones, R., Peveler, R. C., Hope, R. A., & O'Connor, M. (1993). Psychotherapy and bulimia nervosa: Longer-term effects of interpersonal psychotherapy, behavior therapy, and cognitive behavior therapy. *Archives of General Psychiatry*, *50*, 419–428.

Fairburn, C. G., Norman, P. A., Welch, S. L., O'Connor, M. E., Doll, H. A., & Peveler, R. C. (1995). A prospective study of outcome in bulimia nervosa and the long-term effects of three psychological treatments. *Archives of General Psychiatry*, *52*, 304–312.

Fischer, S., & Peterson, C. (2015). Dialectical behavior therapy for adolescent binge eating, purging, suicidal behavior, and non-suicidal self-injury: A pilot study. *Psychotherapy*, *52*, 78–92. https://doi.org/10.1037/a0036065

Genders, R., & Tchanturia, K. (2010). Cognitive remediation therapy (CRT) for anorexia in group format: A pilot study. *Eating and Weight Disorders*, *15*, e234–9. https://doi.org/10.1007/BF03325304

Ghaderi, A., & Scott, B. (2003). Pure and guided self-help for full and sub-threshold bulimia nervosa and binge eating disorder. *British Journal of Clinical Psychology*, *42*, 257–269. https://doi.org/10.1348/01446650360703375

Giombini, L., Turton, R., Turco, M., Nesbitt, S., & Lask, B. (2017). The use of cognitive remediation therapy on a child adolescent eating disorder unit: Patients and therapist perspectives. *Clinical Child Psychology and Psychiatry, 22*, 288-300. https://doi.org/10.1177/1359104516657859

Godart, N., Berthoz, S., Curt, F., Perdereau, F., Rein, Z., Wallier, J., … Jeammet, P. (2012). A randomized controlled trial of adjunctive family therapy and treatment as usual following inpatient treatment for anorexia nervosa adolescents. *PloS One*, *7*, e28249. https://doi.org/10.1371/journal.pone.0028249

Grilo, C. M., & Masheb, R. M. (2005). A randomized controlled comparison of guided self-help cognitive behavioral therapy and behavioral weight loss for binge eating disorder. *Behaviour Research and Therapy*, *43*, 1509–1525. https://doi.org/10.1016/j.brat.2004.11.010

Grilo, C. M., Masheb, R. M., Wilson, G. T., Gueorguieva, R., & White, M. A. (2011). Cognitive-behavioral therapy, behavioral weight loss, and sequential treatment for obese patients with binge-eating disorder: A randomized controlled trial. *Journal of Consulting and Clinical Psychology*, *79*, 675–685. https://doi.org/10.1037/a0025049

Grilo, C. M., Reas, D. L., & Mitchell, J. E. (2016). Combining pharmacological and psychological treatments for binge eating disorder: Current status, limitations, and future directions. *Current Psychiatry Reports*, *18*, 55. https://doi.org/10.1007/s11920-016-0696-z

Hay, P. P., Bacaltchuk, J., Stefano, S., & Kashyap, P. (2009). Psychological treatments for bulimia nervosa and binging. *Cochrane Database of Systematic Reviews*, *4*, CD000562. https://doi.org/10.1002/14651858.CD000562.pub3

Herbrich, L., Noort, B., Pfeiffer, E., Lehmkuhl, U., Winter, S., & Kappel, V. (2017). Follow-up assessment of cognitive remediation therapy in adolescent anorexia nervosa: A pilot study. *European Eating Disorders Review*, *25*, 104–113. https://doi.org/10.1002/erv.2501

Hilbert, A., Bishop, M. E., Stein, R. I., Tanofsky-Kraff, M., Swenson, A. K., Welch, R. R., & Wilfley, D. E. (2012). Long-term efficacy of psychological treatments for binge eating disorder. *British Journal of Psychiatry*, *200*, 232–237. https://doi.org/10.1192/bjp.bp.110.089664

Jones, M., Luce, K. H., Osborne, M. I., Taylor, K., Cunning, D., Doyle, A. C., … Taylor, C. B. (2008). Randomized, controlled trial of an internet-facilitated intervention for reducing binge eating and overweight in adolescents. *Pediatrics*, *121*, 453–462. https://doi.org/10.1542/peds.2007-1173

Juarascio, A. S., Manasse, S. M., Espel, H. M., Schumacher, L. M., Kerrigan, S., & Forman, E. M. (2017). A pilot study of an acceptance-based behavioral treatment for binge eating disorder. *Journal of Contextual Behavioral Science*, *6,* 1-7. https://doi.org/10.1016/j.jcbs.2016.12.003

Katzman, M. A., Bara-Carril, N., Rabe-Hesketh, S., Schmidt, U., Troop, N., & Treasure, J. (2010). A randomized controlled two-stage trial in the treatment of bulimia nervosa, comparing CBT versus motivational enhancement in Phase 1 followed by group versus individual CBT in Phase 2. *Psychosomatic Medicine*, *72*, 656–663. https://doi.org/10.1097/PSY.0b013e3181ec5373

Kirby, J. S., Fischer, M. S., Raney, T. J., Baucom, D. H., & Bulik, C. M. (2016). Couple-based interventions in the treatment of adult anorexia nervosa: A brief case example of UCAN. *Psychotherapy*, *53*, 241–250. https://doi.org/10.1037/pst0000053

Latner, J. D., & Wilson, G. T. (2000). Cognitive-behavioral therapy and nutritional counseling in the treatment of bulimia nervosa and binge eating. *Eating Behaviors*, *1*, 3–21. https://doi.org/10.1016/S1471-0153(00)00008-8

le Grange, D., Crosby, R. D., Rathouz, P. J., & Leventhal, B. L. (2007). A randomized controlled comparison of family-based treatment and supportive psychotherapy for adolescent bulimia nervosa. *Archives of General Psychiatry*, *64*, 1049-1056. https://doi.org/1049–1056. 10.1001/archpsyc.64.9.1049

le Grange, D., Lock, J., Agras, W. S., Bryson, S. W., & Jo, B. (2015). Randomized clinical trial of family-based treatment and cognitive-behavioral therapy for adolescent bulimia nervosa. *Journal of the American Academy of Child and Adolescent Psychiatry*, *54*, 886–94.e2. https://doi.org/10.1016/j.jaac.2015.08.008

Lock, J. (2005). Adjusting cognitive behavior therapy for adolescents with bulimia nervosa: Results of case series. *American Journal of Psychotherapy*, *59*, 267–281.

Lock, J. (2015). An update on evidence-based psychosocial treatments for eating disorders in children and adolescents. *Journal of Clinical Child and Adolescent Psychology*, *44*, 707–721. https://doi.org/10.1080/15374416.2014.971458

Lock, J., Agras, W. S., Bryson, S., & Kraemer, H. C. (2005). A comparison of short- and long-term family therapy for adolescent anorexia nervosa. *Journal of the American Academy of Child and Adolescent Psychiatry*, *44*, 632–639. https://doi.org/10.1097/01.chi.0000161647.82775.0a

Lock, J., Agras, W. S., le Grange, D., Couturier, J., Safer, D., & Bryson, S. W. (2013). Do end of treatment assessments predict outcome at follow-up in eating disorders? *International Journal of Eating Disorders*, *46*, 771–778. https://doi.org/10.1097/01.chi.0000161647.82775.0a

Lock, J., le Grange, D., Agras, W. S., Moye, A., Bryson, S. W., & Jo, B. (2010). Randomized clinical trial comparing family-based treatment with adolescent-focused individual therapy for adolescents with anorexia nervosa. *Archives of General Psychiatry*, *67*, 1025–1032. https://doi.org/10.1001/archgenpsychiatry.2010.128

Mac Neil, B. A., Nadkarni, P., Leung, P., Stubbs, L., O'Brien, C., Singh, M., & Leduc, S. (2016). Cognitive remediation therapy, eh! An exploratory study at a Canadian adult eating disorders clinic. *Canadian Journal of Counselling and Psychotherapy*, *50*, 180.

Mitchell, J. E., Agras, S., Crow, S., Halmi, K., Fairburn, C. G., Bryson, S., & Kraemer, H. (2011). Stepped care and cognitive–behavioural therapy for bulimia nervosa: Randomised trial. *British Journal of Psychiatry*, *198*, 391–397. https://doi.org/10.1192/bjp.bp.110.082172

Mitchell, J. E., Agras, S., & Wonderlich, S. (2007). Treatment of bulimia nervosa: Where are we and where are we going? *International Journal of Eating Disorders*, *40*, 95–101. https://doi.org/10.1002/eat.20343

Mitchell, J. E., Crosby, R. D., Wonderlich, S. A., Crow, S., Lancaster, K., Simonich, H., … Cook Myers, T. (2008). A randomized trial comparing the efficacy of cognitive–behavioral therapy for bulimia nervosa delivered via telemedicine versus face-to-face. *Behaviour Research and Therapy*, *46*, 581–592. https://doi.org/10.1016/j.brat.2008.02.004

Palavras, M. A., Hay, P., Filho, C. A. D. S., & Claudino, A. (2017). The efficacy of psychological therapies in reducing weight and binge eating in people with bulimia nervosa and binge eating disorder who are overweight or obese-a critical synthesis and meta-analyses. *Nutrients*, *9,* E299. https://doi.org/10.3390/nu9030299

Peterson, C. B., Mitchell, J. E., Crow, S. J., Crosby, R. D., & Wonderlich, S. A. (2009). The efficacy of self-help group treatment and therapist-led group treatment for binge eating disorder. *American Journal of Psychiatry*, *166*, 1347–1354. https://doi.org/10.1176/appi.ajp.2009.09030345

Peterson, C. B., Mitchell, J. E., & Engbloom, S. (1998). Group cognitive-behavioral treatment of binge eating disorder: A comparison of therapist-led versus self-help formats. *International Journal of Eating Disorders*. *24*, 125-136. https://doi.org/10.1002/(SICI)1098-108X(199809)24:2<125::AID-EAT2>3.0.CO;2-G

Pitt, S., Lewis, R., Morgan, S., & Woodward, D. (2010). Cognitive remediation therapy in an outpatient setting: A case series. *Eating and Weight Disorders*, *15*, e281–6. https://doi.org/10.1007/BF03325310

Poulsen, S., Lunn, S., Daniel, S. I. F., Folke, S., Mathiesen, B. B., Katznelson, H., & Fairburn, C. G. (2014). A randomized controlled trial of psychoanalytic psychotherapy or cognitive-behavioral therapy for bulimia nervosa. *American Journal of Psychiatry*, *171*, 109–116. https://doi.org/ 10.1176/appi.ajp.2013

Pretorius, N., Dimmer, M., Power, E., Eisler, I., Simic, M., & Tchanturia, K. (2012). Evaluation of a cognitive remediation therapy group for adolescents with anorexia nervosa: Pilot study. *European Eating Disorders Review*, *20*, 321–325. https://doi.org/ 10.1002/erv.2176

Robin, A. L., Siegel, P. T., Moye, A. W., Gilroy, M., Dennis, A. B., & Sikand, A. (1999). A controlled comparison of family versus individual therapy for adolescents with anorexia nervosa. *Journal of the American Academy of Child and Adolescent Psychiatry*, *38*, 1482–1489. https://doi.org/10.1097/00004583-199912000-00008

Russell, G. F., Szmukler, G. I., Dare, C., & Eisler, I. (1987). An evaluation of family therapy in anorexia nervosa and bulimia nervosa. *Archives of General Psychiatry*, *44*, 1047–1056.

Safer, D. L., Couturier, J. L., & Lock, J. (2007). Dialectical behavior therapy modified for adolescent binge-eating disorder: A case report. *Cognitive and Behavioral Practice*, *14*, 157–167. https://doi.org/10.1016/j.cbpra.2006.06.001

Safer, D. L., & Jo, B. (2010). Outcome from a randomized controlled trial of group therapy for binge eating disorder: Comparing dialectical behavior therapy adapted for binge eating to an active comparison group therapy. *Behavior Therapy*, *41*, 106–120. https://doi.org/10.1016/j.beth.2009.01.006

Salbach-Andrae, H., Bohnekamp, I., Pfeiffer, E., Lehmkuhl, U., & Miller, A. L. (2008). Dialectical behavior therapy of anorexia and bulimia nervosa among adolescents: A case series. *Cognitive and Behavioral Practice*, *15*, 415–425. https://doi.org/10.1016/j.cbpra.2008.04.001

Sánchez-Ortiz, V. C., Munro, C., Stahl, D., House, J., Startup, H., Treasure, J., … Schmidt, U. (2011). A randomized controlled trial of internet-based cognitive-behavioural therapy for bulimia nervosa or related disorders in a student population. *Psychological Medicine*, *41*, 407–417. https://doi.org/10.1017/S0033291710000711

Schmidt, U., Lee, S., Beecham, J., Perkins, S., Treasure, J., Yi, I., … Eisler, I. (2007). A randomized controlled trial of family therapy and cognitive behavior therapy guided self-care for adolescents with bulimia nervosa and related disorders. *American Journal of Psychiatry*, *164*, 591–598. https://doi.org/10.1176/ajp.2007.164.4.591

Schmidt, U., Magill, N., Renwick, B., Keyes, A., Kenyon, M., Dejong, H., … Others. (2015). The Maudsley outpatient study of treatments for anorexia nervosa and related conditions (MOSAIC): Comparison of the Maudsley model of anorexia nervosa treatment for adults (MANTRA) with specialist supportive clinical management (SSCM) in outpatients with broadly defined anorexia nervosa: A randomized controlled trial. *Journal of Consulting and Clinical Psychology*, *83*, 796. https://doi.org/10.1037/ccp0000019

Schmidt, U., Oldershaw, A., Jichi, F., Sternheim, L., Startup, H., McIntosh, V., … Treasure, J. (2012). Out-patient psychological therapies for adults with anorexia nervosa: Randomised controlled trial. *British Journal of Psychiatry*, *201*, 392–399. https://doi.org/10.1192/bjp.bp.112.112078

Shapiro, J. R., Bauer, S., Andrews, E., Pisetsky, E., Bulik-Sullivan, B., Hamer, R. M., & Bulik, C. M. (2010). Mobile therapy: Use of text-messaging in the treatment of bulimia nervosa. *International Journal of Eating Disorders*, *43*, 513–519. https://doi.org/10.1002/eat.20744

Shapiro, J. R., Berkman, N. D., Brownley, K. A., Sedway, J. A., Lohr, K. N., & Bulik, C. M. (2007). Bulimia nervosa treatment: A systematic review of randomized controlled trials. *International Journal of Eating Disorders*, *40*, 321–336. https://doi.org/10.1002/eat.20372

Stefini, A., Salzer, S., Reich, G., Horn, H., Winkelmann, K., Bents, H., … Kronmüller, K.-T. (2017). Cognitive-behavioral and psychodynamic therapy in female adolescents with bulimia nervosa: A randomized controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, *56*, 329–335. https://doi.org/10.1016/j.jaac.2017.01.019

Tanofsky-Kraff, M., Shomaker, L. B., Wilfley, D. E., Young, J. F., Sbrocco, T., Stephens, M., … Yanovski, J. A. (2014). Targeted prevention of excess weight gain and eating disorders in high-risk adolescent girls: A randomized controlled trial. *American Journal of Clinical Nutrition*, *100*, 1010–1018. https://doi.org/10.3945/ajcn.114.092536

Tanofsky-Kraff, M., Shomaker, L. B., Young, J. F., & Wilfley, D. E. (2016). Interpersonal psychotherapy for the prevention of excess weight gain and eating disorders: A brief case study. *Psychotherapy*, *53*, 188–194. https://doi.org/10.1037/pst0000051

Tanofsky-Kraff, M., Wilfley, D. E., Young, J. F., Mufson, L., Yanovski, S. Z., Glasofer, D. R., & Salaita, C. G. (2007). Preventing excessive weight gain in adolescents: Interpersonal psychotherapy for binge eating. *Obesity*, *15*, 1345–1355. https://doi.org/10.1038/oby.2007.162

Tanofsky-Kraff, M., Wilfley, D. E., Young, J. F., Mufson, L., Yanovski, S. Z., Glasofer, D. R., … Schvey, N. A. (2010). A pilot study of interpersonal psychotherapy for preventing excess weight gain in adolescent girls at-risk for obesity. *International Journal of Eating Disorders*, *43*, 701–706. https://doi.org/10.1002/eat.20773

Tasca, D. G. A., Ritchie, K., Conrad, G., Balfour, L., Gayton, J., Lybanon, V., & Bissada, H. (2006). Attachment scales predict outcome in a randomized controlled trial of two group therapies for binge eating disorder: An aptitude by treatment interaction. *Psychotherapy Research*, *16*, 106–121. https://doi.org/10.1080/10503300500090928

Tchanturia, K., Davies, H., & Campbell, I. C. (2007). Cognitive remediation therapy for patients with anorexia nervosa: Preliminary findings. *Annals of General Psychiatry*, *6*, 14. https://doi.org/ 10.1186/1744-859X-6-14

Tchanturia, K., Davies, H., Lopez, C., Schmidt, U., Treasure, J., & Wykes, T. (2008). Neuropsychological task performance before and after cognitive remediation in anorexia nervosa: A pilot case-series. *Psychological Medicine*, *38*, 1371–1373. https://doi.org/10.1017/S0033291708003796

Telch, C. F., Agras, W. S., & Linehan, M. M. (2000). Group dialectical behavior therapy for binge-eating disorder: A preliminary, uncontrolled trial. *Behavior Therapy*, *31*, 569–582. https://doi.org/10.1016/S0005-7894(00)80031-3

Telch, C. F., Agras, W. S., & Linehan, M. M. (2001). Dialectical behavior therapy for binge eating disorder. *Journal of Consulting and Clinical Psychology*, *69*, 1061–1065. http://doi.org/10.1037/0022-006X.69.6.1061

van Noort, B. M., Kraus, M. K. A., Pfeiffer, E., Lehmkuhl, U., & Kappel, V. (2016). Neuropsychological and behavioural short-term effects of cognitive remediation therapy in adolescent anorexia nervosa: A pilot study. *European Eating Disorders Review*, *24*, 69–74. https://doi.org/ 10.1002/erv.2383

Watson, H. J., & Bulik, C. M. (2013). Update on the treatment of anorexia nervosa: Review of clinical trials, practice guidelines and emerging interventions. *Psychological Medicine*, *43*, 2477–2500. https://doi.org/10.1017/S0033291712002620

Wild, B., Friederich, H.-C., Gross, G., Teufel, M., Herzog, W., Giel, K. E., … Zipfel, S. (2009). The ANTOP study: Focal psychodynamic psychotherapy, cognitive-behavioural therapy, and treatment-as-usual in outpatients with anorexia nervosa--a randomized controlled trial. *Trials*, *10*, 23. https://doi.org/10.1186/1745-6215-10-23

Wilfley, D. E., Welch, R. R., Stein, R. I., Spurrell, E. B., Cohen, L. R., Saelens, B. E., … Matt, G. E. (2002). A randomized comparison of group cognitive-behavioral therapy and group interpersonal psychotherapy for the treatment of overweight individuals with binge-eating disorder. *Archives of General Psychiatry*, *59*, 713–721. 10.1001/archpsyc.59.8.713

Wilson, G. T., Wilfley, D. E., Agras, W. S., & Bryson, S. W. (2010). Psychological treatments of binge eating disorder. *Archives of General Psychiatry*, *67*, 94–101. https://doi.org/10.1001/archgenpsychiatry.2009.170

Wonderlich, S. A., Peterson, C. B., Crosby, R. D., Smith, T. L., Klein, M. H., Mitchell, J. E., & Crow, S. J. (2014). A randomized controlled comparison of integrative cognitive-affective therapy (ICAT) and enhanced cognitive-behavioral therapy (CBT-E) for bulimia nervosa. *Psychological Medicine*, *44*, 543–553. https://doi.org/10.1017/S0033291713001098

Wood, L., Al-Khairulla, H., & Lask, B. (2011). Group cognitive remediation therapy for adolescents with anorexia nervosa. *Clinical Child Psychology and Psychiatry*, *16*, 225–231. https://doi.org/10.1177/1359104511404750

Zipfel, S., Wild, B., Groß, G., Friederich, H.-C., Teufel, M., Schellberg, D., … Herzog, W. (2014). Focal psychodynamic therapy, cognitive behaviour therapy, and optimised treatment as usual in outpatients with anorexia nervosa (ANTOP study): Randomised controlled trial. *The Lancet*, *383*, 127–137. https://doi.org/10.1016/S0140-6736(13)61746-8

Supplementary Table S6. Medications for the Treatment of Eating Disorders.

	Anorexia Nervosa	Bulimia Nervosa	Binge-Eating Disorder	Source
Approved				
	None (Campbell & Peebles, 2014; McElroy, Guerdjikova, Mori, & Keck, 2015; Miniati et al., 2016)	Fluoxetine (only US) (McElroy et al., 2015)	Lisdexamfetamine (Comiran, Kessler, Fröehlich, & Limberger, 2016; Grilo, Reas, & Mitchell, 2016; Guerdjikova, Mori, Casuto, & McElroy, 2016; Reas & Grilo, 2014, 2015)	Narrative review, systematic review
Under Investigation/Published				
	Olanzapine (weight gain), dronabinol (weight gain), estrogen (improvement of anxiety) (McElroy et al., 2015)		Bupropion (mild weight loss), baclofen (reduction of binge-eating), chromium (improvement of glucose regulation), naloxone (reduction of time spent binge-eating) (McElroy et al., 2015)	Systematic review

References for Supplementary Table S6

Campbell, K., & Peebles, R. (2014). Eating disorders in children and adolescents: State of the art review. *Pediatrics*, *134*, 582–592. https://doi.org/10.1542/peds.2014-0194

Comiran, E., Kessler, F. H., Fröehlich, P. E., & Limberger, R. P. (2016). Lisdexamfetamine: A pharmacokinetic review. *European Journal of Pharmaceutical Sciences*, *89*, 172–179. https://doi.org/10.1016/j.ejps.2016.04.026

Grilo, C. M., Reas, D. L., & Mitchell, J. E. (2016). Combining pharmacological and psychological treatments for binge eating disorder: current status, limitations, and future directions. *Current Psychiatry Reports*, *18*, 55. https://doi.org/10.1007/s11920-016-0696-z

Guerdjikova, A. I., Mori, N., Casuto, L. S., & McElroy, S. L. (2016). Novel pharmacologic treatment in acute binge eating disorder - role of lisdexamfetamine. *Neuropsychiatric Disease and Treatment*, *12*, 833–841. https://doi.org/10.2147/NDT.S80881

McElroy, S. L., Guerdjikova, A. I., Mori, N., & Keck, P. E., Jr. (2015). Psychopharmacologic treatment of eating disorders: Emerging findings. *Current Psychiatry Reports*, *17*, 35. https://doi.org/10.1007/s11920-015-0573-1

Miniati, M., Mauri, M., Ciberti, A., Mariani, M. G., Marazziti, D., & Dell'Osso, L. (2016). Psychopharmacological options for adult patients with anorexia nervosa. *CNS Spectrums*, *21*, 134–142. https://doi.org/10.1017/S1092852914000790

Reas, D. L., & Grilo, C. M. (2014). Current and emerging drug treatments for binge eating disorder. *Expert Opinion on Emerging Drugs*, *19*, 99–142. https://doi.org/10.1517/14728214.2014.879291

Reas, D. L., & Grilo, C. M. (2015). Pharmacological treatment of binge eating disorder: Update review and synthesis. *Expert Opinion on Pharmacotherapy*, *16*, 1463–1478. https://doi.org/10.1517/14656566.2015.1053465

Supplementary Table S7. Brain Circuitry Regions Involved in the Regulation of Feeding and Eating Identified Through Optogenetics and Chemogenetics.

Brain Region	Nuclei involved in feeding and eating	Source
Hypothalamic nuclei	Arcuate nucleus	Aponte, Atasoy, & Sternson, 2011; Betley, Cao, Ritola, & Sternson, 2013; Denis et al., 2015; Kim et al., 2015; Krashes et al., 2011
	Paraventricular nucleus	Aponte, Atasoy, & Sternson, 2011; Betley, Cao, Ritola, & Sternson, 2013; Denis et al., 2015; Kim et al., 2015; Krashes et al., 2011
	Lateral hypothalamus through the ventral tegmental area	Barbano, Wang, Morales, Wise, 2016; Betley et al., 2013; Broberger, Johansen, Schalling, & Hökfelt, 1997; Jennings et al., 2015; Nieh et al., 2015; Nilsson et al., 2011; Nilsson, Lindfors, Schalling, Hökfelt, & Johansen, 2013; Stuber & Wise, 2016
Hindbrain	Parabrachial nucleus	Carter et al., 2013; Wu, Clark, & Palmiter, 2012
	Nucleus of the solitary tract	Wang et al., 2015; Wu et al., 2012
Amygdala	Amygdala central nucleus	Cai, Haubensak, Anthony, & Anderson, 2014; Carter, Soden, Zweifel, & Palmiter, 2013

References for Supplementary Table S7

Aponte, Y., Atasoy, D., & Sternson, S. M. (2011). AGRP neurons are sufficient to orchestrate feeding behavior rapidly and without training. *Nature Neuroscience*, *14*, 351-355. https://doi.org/10.1038/nn.2739

Barbano, M. F., Wang, H. L., Morales, M., & Wise, R. A. (2016). Feeding and reward are differentially induced by activating GABAergic lateral hypothalamic projections to VTA. *Journal of Neuroscience*, *36*, 2975-2985. https://doi.org/10.1523/JNEUROSCI.3799-15.2016

Betley, J. N., Cao, Z. F., Ritola, K. D., & Sternson, S. M. (2013). Parallel, redundant circuit organization for homeostatic control of feeding behavior. *Cell*, *155*, 1337-1350. https://doi.org/10.1016/j.cell.2013.11.002

Broberger, C., Johansen, J., Schalling, M., & Hökfelt, T. (1997). Hypothalamic neurohistochemistry of the murine anorexia (anx/anx) mutation: Altered processing of neuropeptide Y in the arcuate nucleus. *Journal of Comparative Neurology*, *387*, 124-135. https://doi.org/10.1002/(SICI)1096-9861(19971013)387:1<124::AID-CNE10>3.0.CO;2-U

Cai, H., Haubensak, W., Anthony, T. E., & Anderson, D. J. (2014). Central amygdala PKC-δ(+) neurons mediate the influence of multiple anorexigenic signals. *Nature Neuroscience*, *17*, 1240-1248. https://doi.org/10.1038/nn.3767

Carter, M. E., Soden, M. E., Zweifel, L. S., & Palmiter, R. D. (2013). Genetic identification of a neural circuit that suppresses appetite. *Nature*, *503*, 111-114. https://doi.org/10.1038/nature12596

Denis, R. G., Joly-Amado, A., Webber, E., Langlet, F., Schaeffer, M., Padilla, S. L., . . . Luquet, S. (2015). Palatability can drive feeding independent of AgRP neurons. *Cell Metabolism*, *22*, 646-657. https://doi.org/10.1016/j.cmet.2015.07.011

Jennings, J. H., Ung, R. L., Resendez, S. L., Stamatakis, A. M., Taylor, J. G., Huang, J., . . . Stuber, G. D. (2015). Visualizing hypothalamic network dynamics for appetitive and consummatory behaviors. *Cell*, *160*, 516-527. https://doi.org/10.1016/j.cell.2014.12.026

Kim, E. R., Wu, Z., Sun, H., Xu, Y., Mangieri, L. R., Xu, Y., & Tong, Q. (2015). Hypothalamic Non-AgRP, Non-POMC GABAergic neurons are required for postweaning feeding and NPY hyperphagia. *Journal of Neuroscience*, *35*, 10440-10450. https://doi.org/10.1523/JNEUROSCI.1110-15.2015

Krashes, M. J., Koda, S., Ye, C., Rogan, S. C., Adams, A. C., Cusher, D. S., . . . Lowell, B. B. (2011). Rapid, reversible activation of AgRP neurons drives feeding behavior in mice. *Journal of Clinical Investigation*, *121*, 1424-1428. https://doi.org/10.1172/JCI46229

Nieh, E. H., Matthews, G. A., Allsop, S. A., Presbrey, K. N., Leppla, C. A., Wichmann, R., ... & Tye, K. M. (2015). Decoding neural circuits that control compulsive sucrose seeking. *Cell*, *160*, 528-54. https://doi.org/10.1016/j.cell.2015.01.003

Nilsson, I. A., Lindfors, C., Schalling, M., Hökfelt, T., & Johansen, J. E. (2013). Anorexia and hypothalamic degeneration. *Vitamin Hormones*, *92*, 27-60. https://doi.org/10.1016/B978-0-12-410473-0.00002-7

Nilsson, I. A., Thams, S., Lindfors, C., Bergstrand, A., Cullheim, S., Hökfelt, T., & Johansen, J. E. (2011). Evidence of hypothalamic degeneration in the anorectic anx/anx mouse. *Glia*, *59*, 45-57. https://doi.org/10.1002/glia.21075

Stuber, G. D., & Wise, R. A. (2016). Lateral hypothalamic circuits for feeding and reward. *Nature Neuroscience*, *19*, 198-205. https://doi.org/10.1038/nn.4220

Wu, Q., Clark, M. S., & Palmiter, R. D. (2012). Deciphering a neuronal circuit that mediates appetite. *Nature*, *483*, 594-597. https://doi.org/10.1038/nature10899

Wang, X. F., Liu, J. J., Xia, J., Liu, J., Mirabella, V., & Pang, Z. P. (2015). Endogenous glucagon-like peptide-1 suppresses high-fat food intake by reducing synaptic drive onto mesolimbic dopamine neurons. *Cell Reports*, *12*, 726-733. https://doi.org/10.1016/j.celrep.2015.06.062