| **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. 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. 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. Somatic, psychosocial, and psychological manifestations and comorbidities of eating disorders may be difficult to detect. | 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 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. | | Cross-sectional investigations of providers’ abilities to detect eating disorders in pediatric, primary care, and obstetrics and gynecology settings.  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. 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.** | | | | |
| * 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. | |  |
| * 1. Prototypical family interaction patterns that exist premorbidly among families with eating disorders have not been identified. | 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, 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). | | Longitudinal epidemiological studies to more rigorously assess parental and family factors associated with eating risk.  Recent systematic reviews call for additional longitudinal investigations (Larsen, Strandberg-Larsen, Micali, & Andersen, 2015; Saltzman & Liechty, 2016). |
| * 1. 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. |
| * 1. 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.** | | | | |
| * 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. | |  |
| * 1. 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 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). | | Case-control studies examining discrepancy in food and medical expenses.  Studies examining the long-term financial burden of eating disorders over time. |
| * 1. 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. |
| * 1. 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.** | | | | |
| * 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. | |
| * 1. 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. | |
| * 1. 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).  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. | Systematic reviews and meta-analyses that empirically summarize altered response to food and exercise related experiences in those with eating disorders.  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. | |
| * 1. 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. | |
| * 1. 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. | |
| * 1. 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. | |
| * 1. 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.** | | | | |
| * 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 (Núñez-Navarro, et al., 2012; Welch, Ghaderi, & Swenne, 2015). | | Epidemiological studies in large population-based registers applying DSM5 criteria globally to assess global distribution and region-specific risk factors. |
| * 1. 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). Late onset eating disorders are associated with less severe symptomatology (Bueno et al., 2014). | | Longitudinal examination of eating disorder development during midlife and later life. |
| * 1. 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. |
| * 1. 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. |
| * 1. 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).  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). Transgender people also experience greater body dissatisfaction compared to cisgender peers (Jones, Haycraft, Murjan, & Arcelus, 2016; Witcomb et al., 2015). | | Longitudinal studies of adolescent sexual and gender identity development and eating disorder risk.  Further examination of the relationship between eating disorder risk and gender identity. |
| * 1. 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.** | | | | |
| * 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. |
| * 1. 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.** | | | | |
| * 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). | |  |
| * 1. 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. |
| * 1. 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. |
| * 1. 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.** | | | | |
| * 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. |
| * 1. Many cases of eating disorders are sporadic, meaning there is no known family member who suffers from an eating disorder. | 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 limited in that eating disorder history among relatives may not be fully known or accurately captured. | | Further examination of eating disorder history among relatives. |
| * 1. 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, & 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. | | Aim for a clinician’s toolbox that includes psychological and pharmacological interventions that are effective for a range of eating disorders in diverse populations. |
| 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](https://en.wikipedia.org/wiki/Positron_emission_tomography); RCT: Randomized-controlled trial

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