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The Role of Anxiety Sensitivity in the Etiology of Anxiety and Cognitive Symptoms of Eating Disorders

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By

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ABSTRACT

The present study examined common genetic and environmental contributions to the etiology of anxiety sensitivity, cognitive symptoms of eating disorders, and anxiety severity in a community sample of adolescents. I leveraged data from the Twins Early Development Study, a longitudinal epidemiological study of mono- and dizygotic twins born in England and Wales between 1994 and 1996. At age 16, families of twins (*n*=5,111) were assessed for anxiety sensitivity, cognitive symptoms of eating disorders (i.e., felt fat, fear of gaining weight, weight- and shape-influences thoughts about self), and anxiety severity. Strong phenotypic correlations among the four cognitive symptoms of eating disorder items were underpinned by correlated genetic and person-specific environmental contributions to all 4 symptoms; correlated genetic factors contributed particularly strongly to commonality among items. Genetic influences contributed to between-person variance in anxiety sensitivity, cognitive symptoms of eating disorders, and anxiety severity. Person-specific environmental influences contributed to the remaining variance in anxiety sensitivity and cognitive symptoms of eating disorders, while shared and person-specific environmental influences contributed to remaining variance in anxiety severity. Common genetic and person-specific influences contributed to the correlated liability to anxiety sensitivity, cognitive symptoms of eating disorders, and anxiety severity but did not explain the phenotypic correlation of anxiety severity with cognitive symptoms of eating disorders. These results suggest that genetic and environmental influences contribute to the etiology of anxiety sensitivity, cognitive symptoms of eating disorders, and anxiety severity. Further, as compared to anxiety severity, anxiety sensitivity may play a particularly prominent role in the etiology of cognitive symptoms of eating disorders in adolescence.

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Introduction

Eating disorder symptoms

Eating disorder symptoms refer to core features of eating disorders, as described in the Diagnostic Statistical Manual of Mental Disorders, fifth edition (DSM-5), such as fear of gaining weight; the influence of weight and shape concern on self-evaluation; food restraint; and binge eating (American Psychiatric Association, 2013); as well as closely related constructs, such as body dissatisfaction, drive for thinness, and frequently feeling fat. Under this broad definition, eating disorder symptoms can also be organized into two main components: behavioural and cognitive symptoms. Behavioural symptoms include specific overt behaviours such as fasting, binge eating, and compensatory behaviours (e.g., vomiting, laxative use, exercising to offset caloric intake). Cognitive symptoms refer to eating- and weight-related thoughts and beliefs, such as body dissatisfaction; weight and shape concern; fear of gaining weight; and drive for thinness.

Cognitive symptoms of eating disorders are highly prevalent in community samples of both men and women (Paxton, 2000). Seventy percent of individuals in community samples, including 80% of women and 60% of men report some degree of body dissatisfaction (Griffiths et al., 2016). Likewise, 60% of women in community samples endorse eating, weight, or shape concern, while 79% report that weight and shape concerns influence thoughts about themselves (Gagne et al., 2012). Generally, cognitive symptoms of eating disorders appear to affect a larger proportion of women, but recent evidence suggests that cognitive symptoms of eating disorder measures, such as assessments of body dissatisfaction, may be more sensitive to the manifestation of eating disorder cognitions in women compared to men (Mitchison & Mond, 2015). For example, when broadened to include muscle dissatisfaction, up to 85% of men in

community samples endorse body dissatisfaction (Mitchison & Mond, 2015). In community samples, cognitive symptoms of eating disorders are also associated with impaired mental and physical health, quality of life, psychological distress (e.g., stress, anxiety symptoms, depressive symptoms), and impaired psychosocial functioning (Griffiths et al., 2016; Mitchison, et al., 2015; Wilson et al., 2013). Further, cognitive symptoms of eating disorders are associated with increased risk of engaging in eating disorder behaviours (Gonçalves & Gomes, 2012; Johnson & Wardle, 2005) and increased risk of developing a more severe clinical eating disorders (Keel & Forney, 2013; Killen et al., 1994; Stice et al., 2011; Zeiler et al., 2016).

Like adults, cognitive symptoms of eating disorders affect a large proportion of children and adolescents in the general population. For example, in community samples of adolescents between the ages of 11 to 22 years, 80% endorse some level of body dissatisfaction (Furnham et al., 2002). Further, some studies indicate that 50% of girls and 30% of boys between the ages of 10 to 18 years endorse some level of body image concern (e.g., weight, shape, or muscular concern), while 30% of girls and 11% of boys endorse feeling fat (Zeiler et al., 2016). Cognitive symptoms of eating disorders may also emerge as early as 5 years of age. In a community sample of 5-year-old girls, 9% stated that they were dissatisfied with their bodies and 21% stated that they were concerned with their weight (Davison et al., 2000). Although prevalent among children, cognitive symptoms of eating disorders, including body dissatisfaction and weight and shape concern, may increase during high school (Nowak et al., 2001), particularly for girls (Calzo et al., 2012; Hoffmann & Warschburger, 2017). In community samples of adolescents, cognitive symptoms of eating disorders are associated with impaired quality of life; internalizing and externalizing problems (e.g., depressive symptoms; Herpertz-Dahlmann et al., 2008; Zeiler et al., 2016); psychological distress (Johnson & Wardle, 2005); low self-esteem (Herpertz-

Dahlmann et al., 2008); and low body self-esteem (Gowers & Shore, 2001). Further, adolescents who endorse in cognitive symptoms of eating disorders are at higher risk of eating disorder behaviours, such as emotional eating and dietary restraint (Johnson & Wardle, 2005), and are at greater risk of developing eating disorders (Keel & Forney, 2013; Killen et al., 1994; Stice et al., 2011; Zeiler et al., 2016).

Adolescence is generally defined as the ages of 10 to 25 years old and is subdivided into stages including early adolescence (10 to 13 years), adolescence (14 to 17 years), and early adulthood (18 to 25 years; Curtis, 2015). Adolescence, which is characterized by substantial neural changes (Giedd, 2015), is a period of heightened risk for the emergence of psychopathology. Approximately 50% of individuals who experience a psychological disorder in their lifetime will experience it by the age of 14 years; 75% will experience it by the age of 24 years (Giedd, 2015). The prevalence of cognitive symptoms of eating disorders also increases over childhood into adolescence and remains prevalent across the lifespan (Runfola et al., 2013; McCabe & Ricciardelli, 2004). While cognitive symptoms of eating disorders are prevalent across the lifespan, they are more closely related to self-esteem and self-concept during adolescence and early adulthood compared to later adulthood. For example, body dissatisfaction had a greater influence on self-esteem and self-concept among adolescents than among older individuals (Tiggemann, 2004). Thus, adolescents may be at heightened risk for impairment related to cognitive symptoms of eating disorders.

Numerous risk factors for cognitive symptoms of eating disorders also emerge during the adolescent period and may contribute the increased prevalence of cognitive eating disorder symptoms during adolescence. For example, higher body mass index is associated with increased risk of cognitive symptoms of eating disorders (Gowers & Shore, 2001), especially among girls

between the ages of 8 to 13 years (Allen et al., 2008). Specifically, the increase in body fat from a mean of 8% to 22% that occurs during puberty among girls, may increase weight and shape sensitivity in this age group (Gowers & Shore, 2001). During adolescence, individuals also spend more time with peers and less time with family (Choukas-Bradley & Prinstein, 2014); peers become increasingly important influences (Myers & Crowther, 2009). Accordingly, peer groups and social media are both linked to elevated cognitive symptoms of eating disorders (Gowers & Shore, 2001). For example, spending time with peers who emphasize weight, eating, and shape concern is associated with elevated cognitive symptoms of eating disorders among adolescents (Myers & Crowther, 2009; Voelker et al., 2015). Similarly, social comparisons and the use of appearance-related social media (e.g., Instagram) are associated with elevated cognitive symptoms of eating disorders in adolescence (Jarman et al., 2021; Myers & Crowther, 2009). As most adolescent girls (75%) and boys (69%) report using at least one social media platform (Wilksch et al., 2020), social media use may be a particularly salient risk factor for eating disorder symptoms in this age group.

Research is critically needed to examine the etiology of cognitive eating disorder symptoms during adolescence. During adolescence, the prevalence of eating disorder symptoms peaks along with impairment related to eating disorder symptoms as compared to other periods across the lifespan. Thus, research efforts to identify environmental and biopsychosocial risk factors during this at-risk period may be particularly relevant to reduce widespread emerging biopsychosocial impairment due to eating disorder symptoms. Risk factors that contribute to the development of cognitive eating disorder symptoms may also change across the lifespan, such that identifying risk factors at specific developmental stages is important to clarify at critical developmental periods. Adolescence is a developmental period during which eating disorder

symptoms often emerge and when risk factors for eating disorders may contribute to both the development and maintenance of newly emerging eating disorder symptoms. Further, eating disorder symptoms and their risk factors may influence one another over time in a reciprocal relationship. For example, while anxiety symptoms often precede eating disorder symptoms, some research demonstrates that eating disorder symptoms during childhood and adolescence influence the development of anxiety over time (see Godart et al., 2002 for review). By clarifying the etiology of cognitive eating disorders symptoms during their emergence in adolescence, research may clarify and inform the associations of eating disorder symptoms with later psychopathology risk.

Comorbidity of Eating Disorder Symptoms with Anxiety

Community and epidemiological samples consistently document a strong correlation of eating disorder symptoms with both anxious distress and anxiety disorders among adults. Broadly, anxiety is associated with eating disorder symptoms. For example, general anxiety symptoms, stress (Fragkos & Frangos, 2013), trait anxiety (Davey & Chapman, 2009), and fear of social judgment (e.g., eating in public; Levinson et al., 2018) are associated with elevated cognitive and behavioural symptoms of eating disorders, including weight and shape concern, body dissatisfaction, food restraint, and binge eating.

Although there is limited research on the comorbidity of anxiety disorders with eating disorder symptoms in community samples, a large body of research documents a strong correlation of anxiety disorders with eating disorders in clinical samples (e.g., Godart et al., 2002), supporting the correlation of anxiety and anxiety syndromes with eating disorder symptoms found in the general population. Anxiety disorders are significantly more prevalent among patients with an eating disorder as compared to the general population (Swinbourne &

Touyz, 2007) and are consistently among the most prevalent comorbid disorder in individuals with an eating disorder (Ulfvebrand et al., 2015). For example, in a large sample that included 7,156 men and women, 70% of individuals who met diagnostic criteria for an eating disorder presented with a comorbid disorder, of which anxiety disorders were the most prevalent comorbid condition (Ulfvebrand et al., 2015). Similarly, among inpatient and outpatient women who met diagnostic criteria for an eating disorder (i.e., anorexia nervosa, bulimia nervosa, eating disorder not otherwise specified), 65% met criteria for a comorbid anxiety disorder (Swinbourne et al., 2012). Prior research on individual anxiety disorders documented similar rates of comorbidity with eating disorders. For example, Woodside and Staab (2006) estimated the prevalence rates of social anxiety disorder, specific phobia, post-traumatic stress disorder, panic disorder, and generalized anxiety disorder at 20%, 15%, 13%, and 10%, respectively, in individuals diagnosed with an eating disorder. Similar to evidence from non-clinical populations, while anxiety is broadly associated with eating disorders, as evidenced by the wide range of anxiety disorders comorbid with eating disorders, specific anxiety disorders, such as social anxiety disorder, may be particularly comorbid with eating disorders.

The presence of comorbid anxiety or a comorbid anxiety disorder may also be associated with increased severity and longer duration of the eating disorder in adults (Hughes, 2012; Swinbourne et al., 2012). Moreover, Spindler and Milos (2007) suggested that comorbid anxiety may be associated with elevated eating disorder symptom severity more strongly than concurrent depressive symptoms or symptoms of substance use disorders, which further indicates the relevance of comorbid anxiety for the development and prognosis of both eating disorders and eating disorder symptoms. However, empirical evidence is mixed; some studies document no difference in eating disorder symptom severity among those with and without a comorbid

anxiety disorder (e.g., Hughes., 2012). Further, most findings to date are based on clinical samples.

A growing body of evidence suggests that, when comorbid, the development of anxiety symptoms may precede the development of eating disorder symptoms. For example, fear that others may negatively evaluate one's appearance predicts later elevated eating disorder symptoms in community samples (Levinson & Rodebaugh, 2016). Godart and colleagues (2002) reviewed research from 1985 to 2001 to examine the chronology of anxiety disorders and anxiety symptoms with both eating disorders and eating disorder symptoms in community samples aged 16 to 65 years. They conclude that empirical findings varied widely between studies but were broadly consistent with the conclusion that anxiety disorders and symptoms may precede eating disorders and eating disorder symptoms (Godart et al., 2002). In line with this conclusion, when comorbid, the onset of an anxiety disorder often precedes the onset of an eating disorder in clinical samples. For example, among patients with comorbid anxiety and eating disorders, an anxiety disorder preceded the onset of an eating disorder in 69% of patients with a primary eating disorder and 71% of patients with a primary anxiety disorder (Swinbourne et al., 2012). Similarly, prospective reports among women with anorexia nervosa indicated that childhood overanxious disorder often preceded the onset of anorexia nervosa (Raney et al., 2008). However, similar to findings from community samples, empirical findings are mixed (see Swinbourne & Touyz, 2007 for review). It is possible that specific anxiety disorders and eating disorder symptoms are more closely related to one another than are other anxiety disorders. If so, heterogeneity among anxiety disorders may contribute to the mixed results in the current literature. For example, fear of negative evaluation, a symptom of social anxiety disorder, might increase susceptibility to upward social comparisons, internalization of the thin ideal, and

resulting body dissatisfaction. There is some evidence to support this. For example, in a network analysis of bridge symptoms between social anxiety disorder and eating disorder symptoms in clinical and non-clinical samples, embarrassment of eating and drinking in public and feeling nervous about one's appearance were identified as bridge symptoms between social anxiety disorder and eating disorder symptoms. This study illustrates the relative importance of specific symptoms in the comorbidity of anxiety disorders with eating disorder symptoms (Levinson et al., 2018).

Anxiety symptoms are also associated with eating disorder symptoms in community samples of children and adolescents (e.g., Herpertz-Dahlmann et al., 2008; Lee & Vaillancourt, 2019). For example, in children and adolescents aged 8 to 13 years, elevated anxiety symptoms were associated with elevated symptoms of bulimia nervosa (Rowe et al., 2002). Similarly, compared to those without any eating pathology, adolescent girls who presented with elevated eating disorder symptoms or a subclinical eating disorder were more likely to have symptoms of separation anxiety disorder and generalized anxiety disorder (Touchette et al., 2011). Thus, although a limited number of studies assessed the association of anxiety with eating disorder symptoms in community samples of children or adolescents, the evidence suggests an association of anxiety with eating disorder symptoms.

A large body of research in clinical samples of children and adolescents supports the broad association of eating disorders with anxiety disorders in children and adolescents. Anxiety disorders are consistently one of the most prevalent comorbid disorders among children and adolescents with eating disorders (e.g., Vardar & Erzengin, 2011). For example, over half of adolescents who present with an eating disorder also present with a comorbid anxiety disorder (Frtzsimmons-Craft et al., 2019). Moreover, in a sample of 10,123 adolescents aged 13 to 18

years, anorexia nervosa, bulimia nervosa, and binge eating disorder were more strongly associated with anxiety and mood disorders than with other psychological disorders (Swanson et al., 2011). Likewise, in a review of the association of anxiety disorders with anorexia nervosa specifically, 24% to 60% of children, adolescents, or adults with anorexia nervosa may also present with a comorbid anxiety disorder (Hughes., 2012). Because there were limited studies on children and adolescents, the study broadened its scope to include relevant studies in adult populations. Moreover, the review included both clinical and non-clinical participants, as well as a broad range of anxiety symptoms (Hughes., 2012). Therefore, the wide range in the prevalence of comorbid anxiety and anorexia nervosa reported was likely due to age, symptoms assessed, and severity of symptoms. Similarly, in a longitudinal study in children and adolescents, 56% of those who met criteria for anorexia nervosa also had a lifetime diagnosis of an anxiety disorder over 10 years of follow up assessment (Herpertz-Dahlmann et al., 2001). Regarding specific anxiety disorders, social anxiety disorder may be associated with bulimia nervosa and binge eating disorder particularly strongly (Spettigue et al., 2020). Together, this literature indicates a consistent correlation of anxiety symptoms and anxiety disorders with both eating disorders and eating disorder symptoms in children and adolescents.

Among children and adolescents, comorbid anxiety disorders may also be associated with elevated eating disorder symptom severity (Brand-Gothelf et al., 2014) and poorer treatment outcomes (Bryant-Waugh et al., 1988; Milos et al., 2002), although empirical support is mixed (e.g., Hughes, 2012; Swinbourne & Touyz, 2007). The impact of comorbid anxiety disorders on the effectiveness of standard treatments for eating disorders may depend on additional factors such as the age of onset of the anxiety or eating disorder; eating and anxiety disorder subtype; severity of anxiety and eating disorder symptoms; and gender or biological sex. Taken together,

further research is needed to clarify the role of comorbid anxiety disorders among children and adolescents with eating disorders on symptom severity and prognosis including response to first- and second-line treatments.

Like in adults, anxiety symptoms and anxiety disorders often precede the onset of eating disorders and eating disorder symptoms in children and adolescents. Longitudinal studies suggest that anxiety symptoms predict eating disorder symptoms, such as binge eating, fasting, vomiting, body dissatisfaction, and weight concern among adolescents (Lee & Vaillancourt, 2019; Lloyd et al., 2020; Schaumberg et al., 2019); Parkinson and colleagues (2012) documented similar findings in children. Anxiety symptoms may also predict both later eating disorder symptoms and the development clinical eating disorders. For example, Schaumberg et al. (2019) assessed anxiety symptoms and eating disorder symptoms at ages 10, 14, and 16 years. Worry and physical symptoms of anxiety at age 10 predicted eating disorder symptoms (e.g., binge eating, fasting, and weight concerns) at age 14. Similarly, worry predicted the onset of clinical eating disorders (e.g., anorexia nervosa and bulimia nervosa) at 16 years of age. These findings suggest that anxiety broadly predicts both eating disorder symptoms and eating disorders. Similarly, anxiety disorders may predict the onset of eating disorders among children and adolescents. For example, among adolescents who developed an eating disorder over a 2-year period, 22% were diagnosed with an anxiety disorder prior to being diagnosed with an eating disorder (Rojo-Moreno et al., 2015). Moreover, in the study by Rojo-Moreno and colleagues (2015), anxiety disorders were the most prevalent psychiatric comorbidity and the only disorder to precede the onset of an eating disorder.

The Biometrical Twin Design

Research to clarify genetic and environmental contributions to the etiology of eating disorder symptoms in adolescence in the general population has both theoretical and clinical implications. The current etiological conceptualization of eating disorder symptoms (e.g., body dissatisfaction, weight concern) emphasizes environmental factors including numerous studies on environmental risk factors (e.g., societal pressure to be thin) that contribute to the development of eating disorder symptoms and related distress (e.g., body dissatisfaction) (Keel et al., 2013). A focus on environmental risk factors for eating disorder symptoms and related distress may have direct implications for the development of prevention and intervention strategies. Instead, clarification of genetic contributions to eating disorder risk may inform the identification of biopsychological substrates through which eating disorders develop through childhood and adolescence (Cuthbert & Insel, 2013; Insel et al., 2010). The subsequent identification of such biopsychological substrates could, in turn, open entirely new avenues for targeted preventive intervention as well as pharmacological and psychological treatment of eating disorder symptoms and related distress. Separately, research to identify which symptoms are predominantly influenced by environmental contributions may guide the provision of existing prevention and intervention strategies based on engaging with one's environment (e.g., use of social media). Thus, the genetic and environmental contributions in the etiology of distinct eating disorder symptoms provide important clinical information.

Within community and epidemiological research, eating disorder symptom severity is conceptualized dimensionally such that eating disorder symptom severity is assessed both within and outside of participants with a diagnosed eating disorder. Research on the etiology of eating disorder symptom severity can help to further delineate the similarities and differences between

eating disorder symptoms in community participants and in individuals diagnosed with clinical eating disorders, particularly to clarify why eating disorder symptoms may be particularly prevalent in the general population. For example, the co-occurrence of anxiety and eating disorder symptoms is highly prevalent in the general population. Etiological research can further elucidate the nature of this relationship to clarify how putative psychological traits contribute to the co-occurrence of anxiety and eating disorder symptoms and, thereby, may give rise to multiple, comorbid syndromes. Following research to identify putative psychological traits that may explain the development of multiple, frequently comorbid syndromes, such traits could clarify novel targets for prevention and intervention strategies that would address a wide range of frequently comorbid mental health outcomes.

The biometrical twin design distinguishes between latent (i.e., unobserved) genetic and environmental factors that contribute to a given phenotype (i.e., an observed trait). For example, the model decomposes between-person variance in an eating disorder symptom (e.g., felt fat) into latent genetic and environmental influences to the given eating disorder symptom. Specifically, under the biometrical twin design, between-person variance in a given phenotype is partitioned into latent additive genetic influences (A), environmental influences shared between both twins in a family (shared environmental influences; C), and person-specific environmental influences (E). The model is computationally estimated using structural equation modeling (Neale & Cardon, 1992). Computationally, it is often assumed that A, C, and E influences are linear and additive, which means that the model assumes no interaction between genes and environment, though this assumption can be relaxed in research on more complex family and twin designs (Berger, 2005). Thus, the total between-person variance in a given phenotype (e.g.,

an eating disorder symptom) is equal to the sum of the variance in A, C, and E (Rabe-Hesketh et al., 2008).

Additive genetic influences reflect the additive contribution of specific genetic variation across multiple loci that contribute to a phenotype (Berger, 2005). Shared environmental influences reflect environmental factors that are shared by twins, such as the shared home environment. Person-specific environmental influences reflect environmental factors that are unique to each twin, such as the perception of the home environment, unique friend groups, or events that may happen disproportionately to one child (e.g., peer victimization). To estimate A, C, and E, the biometrical twin design leverages the difference in genetic relatedness between monozygotic and dizygotic twins. Specifically, monozygotic twins share 100% of their segregating genes while dizygotic twins share 50%; both mono- and dizygotic twins share 100% of the so-defined shared environment (C) and none of the so-defined person-specific environment (E).

Genetic and Environmental Contributions to the Etiology of Eating Disorder Symptoms

Prior research demonstrated that both genetic and environmental factors influence the etiology of eating disorder symptoms in adults. For example, studies using the biometrical twin design show that genetic influences contribute to between-person variance in myriad eating disorder symptoms including cognitive symptoms of eating disorders, such as eating concern (46%; Wade et al., 1998), shape concern (64%), and the influence of weight and shape concern on self-evaluation (50%; Spanos et al., 2010), and eating disorder behaviours, such as dietary restraint (32%; Wade et al., 1998), disordered eating (60%), binge eating (50% to 80%), and compensatory behaviours (e.g., vomiting; 30 to 50%; Builk et al., 1998; Mazzeo et al., 2010; Root et al., 2010). Person-specific environmental influences contribute the remaining between-

person variance in eating disorder symptoms while environmental influences shared between (i.e., common to) twins (e.g., parenting style) contribute little or not at all to the variance in eating disorder symptoms (Rutherford, et al., 1993; Wade et al., 1998, 1999). The genetic contribution to weight concern is somewhat less clear; some studies indicate genetic contributions to weight concern (Spanos et al., 2010) while other studies suggest that only shared and person-specific environmental influences contribute to between-person variance in weight concern (Mazzeo et al., 2010; Wade et al., 1998, 1999). Nonetheless, genetic influences consistently account for a significant and substantial proportion of the between-person variance in most eating disorder symptoms. Biometrical twin research in clinical samples further supports these findings. For example, genetic and person-specific environmental factors each contribute approximately 50% of the variance in bulimia nervosa, anorexia nervosa, and binge eating disorder, while shared environmental influences contribute very little (Bulik, et al., 2010; Javaras et al., 2008; Kendler et al., 1991; Kortegaard et al., 2001; Mazzeo et al., 2010; Wade et al., 1998).

Genetic factors also contribute increasingly to eating disorder symptoms throughout childhood and into adolescence. Klump et al. (2000a) examined the genetic and environmental contributions to eating disorder symptoms, including body dissatisfaction, weight concern, and shape concern in a sample of children and early adolescents aged 10 to 12 years and adolescents aged 16 to 18 years. Among children and early adolescents, genetic factors contributed to 49% of the variance in body dissatisfaction but did not contribute to the variance in weight preoccupation, weight concern, or shape concern, which was entirely attributed to environmental factors, including both shared and person-specific environmental influences (Klump et al., 2000a). In contrast, among adolescents, genetic factors accounted for variance in body

dissatisfaction (60%), weight concern (50%), and shape concern (52%). Person-specific environmental influences accounted for the remaining variance in all eating disorder symptoms in this age group (Klump et al., 2000a).

Taken together, genetic contributions to eating disorder symptoms contribute to stability across symptoms and time during adolescence and adulthood but likely not during childhood. Genetic contributions to eating disorder symptoms may change across childhood to adolescence as a function of puberty. For instance, puberty moderates the genetic contribution to eating disorder symptoms, which provides a possible mechanism through which genetic influences increase from childhood into adolescence. For example, genetic influences on eating disorder symptoms increase from 0% to 44% across pubertal development among adolescents (Klump et al, 2007). It should be noted that some studies identify genetic influences on eating disorder symptoms among children. For example, Silberg & Bulik (2005) report that genetic factors contributed to the variance in eating disorder symptoms (e.g., binge eating, weight concern, compensatory behaviours) in children between the ages of 8 to 13 years. Prior research studied a variety of age ranges, which may contribute to the discrepancy in findings. It is also possible that, in childhood, genetic factors contribute to some eating disorder symptoms, such as body dissatisfaction, but not others.

While findings among children are more variable, genetic factors are consistently shown to contribute to eating disorder symptoms in adolescence. Eating disorder symptoms are more strongly correlated between monozygotic, as compared to dizygotic, female adolescent twins, which indicates the presence of additive genetic influences (Klump et al., 2000b). Similarly, Waszczuk et al. (2019) used the biometrical twin design to assess the correlations between eating disorder symptoms over a period of 6 years in a community sample of adolescents aged 12 to 18

years at the start of the study. Genetic factors accounted for variance in drive for thinness (56%-61%), body dissatisfaction (58%-68%), and bulimic symptoms (31%- 39%) over the 6-year study period. Waszczuk et al. (2019) further report that genetic factors accounted for within-symptom stability and the co-occurrence of different eating disorders while environmental factors were disorder- and time-specific. When taken together, these findings demonstrate that genetic factors play an increasingly important role across childhood into adolescence, at which time they are consistently shown to contribute significantly to myriad eating disorder symptoms. Genetic factors also may account for the stability of eating disorder symptoms across time and the co-occurrence of eating disorder symptoms into the syndromes presently known as eating disorders (e.g., anorexia nervosa, bulimia nervosa, binge eating disorder).

Genetic and environmental contributions to clinical eating disorders (i.e., anorexia nervosa and bulimia nervosa) in childhood and adolescence support the findings from non-clinical samples. Rowe and colleagues (2002) examined the genetic and environmental influences that contribute to subclinical and clinical bulimia nervosa in 2,790 male and female twins aged 8 to 17 years. Genetic factors contributed to the variance in bulimia nervosa among male (40%), pre-menarche female (60%), and post menarche female participants (52%). The remaining variance in subclinical and clinical bulimia nervosa was attributed to person-specific environmental influences. Similarly, among 17-year-old female participants, additive genetic and person-specific influences accounted for 74% and 26% of the variance in anorexia nervosa, respectively (Klump et al., 2001). Together, this evidence suggests that, while environmental influences largely contribute to eating disorder symptoms among children, genetic influences may also contribute substantially to eating disorder symptoms, particularly in adolescence.

Genetic and Environmental Contributions to the Etiology of Anxiety

Similar to eating disorder symptoms, genetic and environmental factors contribute to individual differences in liability to elevated anxiety symptoms in adults. Depending on the population studied and assessment of anxiety symptoms, genetic influences explain between 30% to 50% of the between-person variance in a broad range of anxiety symptoms and related conditions (López-Solà et al., 2014; Nes et al., 2007; Nivard et al., 2015), including symptoms of social anxiety disorder (30% to 50%; see Scaini et al, 2014 for review), symptoms of obsessive-compulsive disorder (40%), symptoms of conversion disorder (30%), and general symptoms of stress and anxiety (30%; Nes et al., 2007). Common genetic factors also contribute broadly to multiple anxiety syndromes (e.g., Kendler et al., 1987), which indicates that the same or correlated genes elevate risk for multiple anxiety syndromes. For example, common genetic factors account for between 30% to 56% of the variance in a range of anxiety symptoms, including symptoms of panic disorder; generalized anxiety disorder; specific phobias; obsessive-compulsive disorder, which was previously classified as an anxiety disorder; and post-traumatic stress disorder, which was also previously classified as an anxiety disorder (Tambs et al., 2009). Common genetic factors also account for most of the genetic influence to anxiety symptoms with less influence from disorder specific genetic contributions (e.g., Tambs et al., 2009). In addition to genetic influences, person-specific environmental influences also contribute to anxiety symptoms while shared environmental influences contribute very little to between-person variance in anxiety symptoms in adults (Kendler et al., 1987; Nes et al., 2007; Nivard et al., 2015; Scaini et al., 2014; Tambs et al., 2009).

Findings from clinical populations provides further support for the genetic influence to anxiety symptoms in adults. Family studies indicate that anxiety disorders are more common

among individuals who have a family member with an anxiety disorder (Hettema et al., 2001). In line with these findings, as demonstrated by research that leverages the biometrical twin design, genetic influences contribute to between-person variance in a range of anxiety disorders, including generalized anxiety disorder, panic disorder, agoraphobia, and social anxiety disorder (Dellava et al., 2011; Hettema et al. 2005; Mackintosh et al., 2006). For example, Hettema et al. (2005) demonstrated that genetic factors contribute to generalized anxiety disorder (23%), panic disorder (28%), agoraphobia (36%), specific phobia (24%) and social phobia (10%). Similar to research on anxiety symptoms, common genetic factors contribute broadly to anxiety disorders. For example, Hettema et al. (2005) found that a common genetic factor contributed significantly to generalized anxiety disorder, panic disorder, agoraphobia, and social phobia, while anxiety disorder specific genetic factors contributed very little to each condition. Person-specific environmental influences accounted for most of the remaining variance in anxiety disorders, while shared environmental influences contributed very little. For example, in research by Hettema and colleagues (2005), person-specific environmental influences accounted for between 62% to 79% of the variance in the anxiety disorders. In both community and clinical samples, genetic factors are broadly associated with the development of both anxiety symptoms and disorders. Moreover, in both community and clinical samples, common genetic influences may account for the high comorbidity among anxiety disorders.

Genetic influences also play a significant and substantial role in the etiology of anxiety symptoms or anxiety disorders in children and adolescents. Genetic contributions to anxiety symptoms are evident beginning at a young age. In a large sample of 4-year-old twins, genetic influences accounted for 39% to 64% of the variance in anxiety symptoms including general distress, fears, obsessive-compulsive behaviours, shyness, and inhibition (Eley et al., 2003). The

genetic contributions to anxiety symptoms among older children are comparable and range from 20% to 60% depending on the specific anxiety syndrome assessed. For example, among children aged 7 to 9 years, genetic influences accounted for 40% to 60% of the variance in negative affect, fear, and symptoms of social anxiety disorder (Hallett et al., 2009). Similarly, in 9- to 14-year-old children, genetic influences accounted for between 18% to 35% of the variance in anxiety symptoms, including symptoms of generalized anxiety disorder, panic disorder, social anxiety disorder, and separation anxiety disorder (Sawyers et al., 2019). Genetic factors also contribute to anxiety symptoms in adolescence in a similar manner to that seen in childhood. For example, genetic influences accounted for 50% of the variance in trait anxiety among children and adolescents aged 9 to 19 years (Chen et al., 2015). Thus, across childhood and adolescence, genetic factors contribute to the variance in anxiety symptoms and related traits to a similar extent.

Environmental influences also contribute to child and adolescent liability to anxiety. However, the contribution of shared and person-specific environmental influences may change over development. Shared environmental influences contribute to the variance in anxiety symptoms particularly among younger children (Eley et al., 2003) while only person-specific environmental influences contribute to variance in anxiety symptoms among older children and adolescents (Chen et al., 2015; Hallett et al., 2009; Sawyers et al., 2019). At younger ages, children have fewer opportunities to experience person-specific stressful events, which may lead to a relatively larger contribution of the shared environment in the etiology of anxiety symptoms. However, as children age, they may be exposed to more person-specific environmental influences (e.g., peer victimization, social support), which positively or negatively influence the development of anxiety symptoms. In support of this notion, longitudinal research from

childhood to adulthood demonstrates that while stability in anxiety-related behaviour is largely attributed to genetic influences, environmental influences are age-dependent (Nivard et al., 2015; Patterson et al., 2018). Thus, the environmental contributions to anxiety symptoms change across time while genetic influences remain stable.

Similar to adulthood, in childhood and adolescence, common genetic factors contribute to the liability to a broad range of anxiety symptoms and related behaviour. For example, in children between the ages of 7 to 9 years, correlations among the genetic contributions to negative affect, fear, and social anxiety range from .13 to .38 (Hallett et al., 2009). Similarly, in children and adolescents aged 9 to 14 years, common genetic factors contributed between 5% and 14% of the variance in a range of fear and anxiety symptoms (Sawyers et al., 2019). These studies suggest that common genetic factors contribute to symptoms of diverse anxiety syndromes. However, while common genetic factors contribute to a broad range of anxiety symptoms, they may have a greater influence on certain anxiety syndromes as compared to others. Anxiety symptoms broadly construed share the strongest genetic correlations with symptoms of diverse anxiety syndromes (Eley et al., 2003; Sawyers et al., 2019). For example, genetic correlations are highest between general distress and symptoms of all other anxiety syndromes, including separation anxiety disorder, shyness, inhibition, and fears (Eley et al., 2003). Common or correlated environmental factors also contribute to the shared liability to symptoms of multiple anxiety syndromes. Prior research demonstrates moderate to high correlations of shared environmental influences to multiple anxiety syndromes and related symptoms among children (Eley et al., 2003; Hallett et al., 2009). Together, this evidence suggests that common genetic and environmental factors contribute both to the liability to a

range of anxiety-related symptoms and behaviours and to their frequent co-occurrence with one another.

Prior research also demonstrates that genetic influences contribute to clinical anxiety *disorders* among children and adolescents. In a meta-analysis that included studies using the bivariate twin design to examine the heritability of separation anxiety disorder in children, additive genetic effects accounted for approximately 40% of the variance in separation anxiety disorder (Scaini et al., 2012). Likewise, Bolton et al. (2006) examined the heritability of specific phobia among 6-year-old twins. Genetic influences accounted for 60% of the variance in specific phobia. Genetic factors influence anxiety disorders in adolescence to a similar extent. For example, genetic factors contributed 25% of the variance in generalized anxiety disorder and between 40% to 50% of the variance in separation anxiety disorder in adolescents between the ages of 12 to 19 years (Ehringer et al., 2006). Similar to anxiety symptoms in community samples of children and adolescents, shared environmental influences play a larger role in anxiety disorders among younger children (Bolton et al., 2006), while person-specific environmental factors account for the environmental influences among older children and adolescents (Bolton et al., 2006; Ehringer et al., 2006; Scaini et al., 2012).

Common Etiology of Anxiety and Eating Disorder Symptoms

The high and consistently demonstrated comorbidity of anxiety and eating disorders suggests that they may share common etiological influences. Common genetic factors contribute broadly to symptoms of multiple anxiety syndromes (e.g., Rappaport et al., 2020). It is possible that common genetic factors also contribute broadly to anxiety and eating disorder symptoms, which would represent shared etiological influences and partially explain the evident high comorbidity between the two syndrome clusters. Family and twin studies, including those that

employ the biometrical twin design, help to elucidate etiological influences common to both anxiety and eating disorder symptoms. In adults, anxiety and eating disorders and symptoms co-occur in families (Lilenfeld, 1998). For example, anxiety disorders, including generalized anxiety disorder, panic disorder, agoraphobia, and social anxiety disorders are significantly more prevalent among family members of individuals with an eating disorder (Lilenfeld, 1998; Wade et al., 2004).

Biometrical analysis of data from twins further supports this notion. In adults, genetic contributions to anxiety symptoms are significantly correlated with genetic contributions to a range of eating disorder symptoms, including caloric restraint, emotional eating, and external eating (Song et al., 2019). Further, genetic contributions to anxiety may be correlated with genetic contributions to later eating disorder symptoms, which suggests that genetic factors common to both anxiety and eating disorder symptoms are stable over time (Song et al., 2019). For clinically assessed eating disorders, the genetic contribution to anorexia nervosa may be moderately correlated with the genetic contribution to generalized anxiety disorder (Dellava et al., 2011). Similarly, common genetic factors contribute to the variance in bulimia nervosa, panic disorder, and specific phobia (Kendler et al., 1995). Common person-specific environmental influences also contribute to the shared liability to anxiety and eating disorder symptoms. For example, Song et al. (2019) demonstrated significant correlations among person-specific environmental contributions to anxiety symptoms and eating disorder symptoms. Similarly, Dellava et al. (2011) report moderately correlated person-specific contributions to both generalized anxiety disorder and anorexia nervosa. Thus, both common genetic and person-specific environmental influences contribute to the shared liability of anxiety with eating disorders and symptoms among adults.

The notion of genetic pleiotropy (i.e., that a common set of genes may influence multiple disorders) is well founded in research on psychopathology in children and adolescents including research on anxiety and eating disorder symptoms. Among children and adolescents aged 8 to 17 years, common genetic factors contributed to the liability of early and late onset of symptoms related to overanxious disorder, major depressive disorder, separation anxiety disorder, eating disorder symptoms, and eating disorders (Silberg & Bulik, 2005). These findings suggest a common etiology of anxiety and eating disorder symptoms. Specifically, these findings suggest that common genetic and environmental influences may contribute to the liability to both anxiety and eating disorder symptoms, which could partially explain consistent evidence of their co-occurrence.

Etiology of Psychological Mechanisms that Influence Both Anxiety and Eating Disorder Symptoms

Putative psychological traits may elevate risk for both anxiety and eating disorder symptoms, in which case such common traits may partially account for their shared genetic or environmental etiology and, therein, their frequent co-occurrence (e.g., comorbidity). For example, neuroticism and perfectionism have both been implicated in a range of anxiety and eating disorder syndromes and share common genetic and environmental influences with anxiety and eating disorder syndromes (e.g., Hettema et al., 2006). In adults and adolescents, neuroticism is strongly correlated phenotypically with both anxiety disorders and symptoms (see Gottschalk & Domschke, 2017 for review). A meta-analysis of the correlation between the genetic contributions to neuroticism and the genetic contributions to multiple psychopathologies found consistent, strong genetic correlations of neuroticism with anxiety disorders (viz., r =.86), which

reflects the strongest genetic correlation of neuroticism with a psychological disorder identified to date (Gottschalk & Domschke, 2017).

Among children and adolescents, common genetic as well as shared and person-specific environmental influences have been implicated in the strong phenotypic correlation of neuroticism with anxiety. Past research indicates that common genetic factors more strongly contribute to the phenotypic correlation of neuroticism with anxiety symptoms than do common environmental factors (Hansell et al., 2012). Thus, common genetic liability to neuroticism and anxiety symptoms manifest across childhood, adolescence, and adulthood. Similarly, neuroticism is broadly associated with eating disorders and symptoms. In children and adolescents, correlations of the genetic contributions to neuroticism with the genetic contributions to eating disorder symptoms, including both eating cognitions and behaviours, range from .37 to .49; the remaining covariance of neuroticism with eating disorder symptoms is explained by common person-specific environmental influences (Culbert et al., 2015). Together, this research suggests that neuroticism shares common genetic influences with both anxiety and eating disorder symptoms. One potential explanation for these findings is that neuroticism may be a core trait that increases liability to both anxiety and eating disorder symptoms, which would account for the high comorbidity between anxiety and eating disorder symptoms. Further research is required to test this hypothesis, most notably longitudinal research to understand how neuroticism and symptoms of anxiety and eating disorders interact over time.

Similarly, perfectionism is strongly correlated phenotypically with anxiety, eating disorders, and eating disorder symptoms. Perfectionism is correlated with anxiety symptoms in adults, adolescents, and children; higher perfectionism is positively correlated with elevated anxiety symptoms (Kawamura et al., 2001; Morgan-Lowes et al., 2019). In young adults,

common genetic influences contribute to both perfectionism and anxiety, which suggests common genetic etiology and the possibility that perfectionism serves as a distal risk factor for the development of anxiety symptoms. Moser et al. (2012) examined the genetic and environmental correlations of individual components of perfectionism (e.g., concern over mistakes and doubts about action) with anxiety (e.g., trait anxiety and anxiety symptoms). Common (i.e., correlated) genetic influences primarily contributed to the phenotypic correlations of components of maladaptive perfectionism with anxiety. Common shared environmental factors also contributed to the correlation between perfectionism and anxiety, but to a lesser extent (Moser et al., 2012).

Perfectionism is also associated with eating disorder symptoms among adults and adolescents (see Culbert et al., 2015 for review). For example, as compared to adolescents who endorse adaptive-perfectionism or non-perfectionism, adolescents who endorsed maladaptive perfectionism report more severe eating disorder symptoms (Boone et al., 2010). Etiological research has further delineated the correlation of perfectionism with eating disorder symptoms. Among young adults and adolescents, genetic correlations of perfectionism with eating disorder symptoms are moderate to strong, while environmental correlations are weak, which indicates that genetic influences largely account for the phenotypic correlations of perfectionism with eating disorder symptoms (Wade & Bulik, 2007; Spanos 2012).

Anxiety Sensitivity

Anxiety sensitivity is a psychological trait that may contribute to the common genetic and environmental etiology of both anxiety and eating disorder symptoms. Anxiety sensitivity is conceptualized in extant literature as a heightened interoceptive awareness of, and attention to, bodily sensations, such that they are perceived as harmful or catastrophic (Taylor, 1999).

Theoretically, anxiety sensitivity may predispose one to worsened anxiety symptom severity and anxiety disorders (e.g., panic disorder) as the cognitive fear of anxiety may increase subsequent anxiety both acutely and about anticipated experiences of anxiety or fear (e.g., about anticipated panic attacks; Reiss, 1987).

Anxiety sensitivity is measured using self-report, commonly on the Anxiety Sensitivity Index, although alternative versions of the Anxiety Sensitivity Index (e.g., the Anxiety Sensitivity Index-3, the Anxiety Sensitivity Index-Revised) are also used. The factor structure of anxiety sensitivity is inconsistent across adult or child and adolescent studies. Generally, anxiety sensitivity is structured hierarchically with one higher order factor (i.e., anxiety sensitivity) and two- (Schmidt & Joiner, 2002), three- (Rodriguez et al., 2004; Zinbarg et al., 1997, 1999), or four- (Deacon et al., 2003) lower order factors. Among children and adolescents, anxiety sensitivity is measured using an adaptation of the Anxiety Sensitivity Index for children and adolescents, the Child Anxiety Sensitivity Index. Like in adult studies, the factor structure of anxiety sensitivity in children and adolescents is inconsistent across studies. In a systematic review that included 50 studies including children and adolescents under the age of 18 years, results indicated that most studies supported two-, three-, or four- factor solutions for the Child Anxiety Sensitivity Index. Overall, the hierarchical three factor structure, which included physical, cognitive, and social concerns secondary to a total index of overall anxiety sensitivity was the most robust factor structure (Francis et al., 2019). Regardless, controversy remains over the specific nature and structure of subfactors of anxiety sensitivity in children, adolescents, and adults. However, consistently, the presence and interpretation of total anxiety sensitivity as a highest-order, overall severity of anxiety severity is robust in research with child, adolescent, and adult samples.

Despite its theoretical association with anxiety disorders and symptom severity, empirical research on anxiety sensitivity suggests that it may be broadly associated with psychopathology. Among adults, anxiety sensitivity is strongly related to anxiety symptoms and disorders, including panic disorder; agoraphobia; social anxiety and social phobia; specific phobia; generalized anxiety; and post-traumatic stress disorder *(*Naragon-Gainey., 2010). However, higher anxiety sensitivity is also reported in individuals with obsessive-compulsive, depressive (Naragon-Gainey., 2010), personality (Gratz et al., 2007), eating (Anestis et al., 2007), and substance use (East et al., 2007) disorders and symptoms, as well as hypochondriasis (Taylor, 1999), pain (Markfelder & Pauli, 2020), and suicidal ideation (see Stanley et al., 2018 for review). Anxiety sensitivity is also associated with greater functional impairment (Lebowitz et al., 2012), which provides further evidence for its broad implications in psychopathology. Anxiety sensitivity may also be associated with poorer outcomes of psychotherapy treatment; in patients with obsessive-compulsive disorder, higher anxiety sensitivity at baseline predicted higher symptom severity immediately following treatment (Blakey et al., 2017). Together, these findings suggest that anxiety sensitivity is broadly implicated in psychopathology and is associated with poorer psychosocial functioning, suicidal ideation, and poorer treatment outcomes.

Similarly, among children and adolescents, anxiety sensitivity is associated with anxiety symptoms (see Noël & Francis, 2011 for review), including health anxiety (Wright et al., 2016) in non-clinical samples. Likewise, among children and adolescents, anxiety sensitivity is associated with clinical anxiety disorders, including specific phobia, agoraphobia, and separation anxiety, generalized anxiety, and panic disorders (see Noël & Francis, 2011 for review). However, anxiety sensitivity is also associated more broadly with psychopathology risk. For

example, higher anxiety sensitivity is also reported in individuals with post-traumatic stress disorder (Noël & Francis, 2011), obsessive-compulsive, depressive (Noël & Francis, 2011), eating (Fairweather-Schmidt & Wade, 2020), substance use (Guillot et al., 2020), and conversion (Yilmaz et al., 2016) symptoms and disorders. Anxiety sensitivity is relatively stable over time in adolescence; adolescents with higher anxiety sensitivity are more likely to experience anxiety-related symptoms (Allan et al., 2016; Weems et al., 2002). Anxiety sensitivity may also increase risk for anxiety and depressive disorders in children and adolescents. For example, in a longitudinal study of 848 adolescents aged 15 to 21 years, higher anxiety sensitivity at baseline predicted higher anxiety, depression, and stress symptom severity at a two-year follow-up (Qi et al., 2021). Moreover, the stronger association of anxiety sensitivity with anxiety in adolescence as compared to childhood (Noël & Francis, 2011) may indicate that although children experience anxiety sensitivity, they have not yet reached a period of greatest risk for the emergence of anxiety syndromes. This provides some evidence that anxiety sensitivity may precede and thus be involved in the development of anxiety disorders. Together, this evidence suggests that anxiety sensitivity may be broadly related to psychopathology risk, particularly for internalizing psychopathology.

Although anxiety sensitivity is correlated with trait anxiety and other fear-related measures (e.g., neuroticism, distress tolerance, etc.), a large body of evidence demonstrates that anxiety sensitivity is a distinct construct (Bernstein et al., 2008; Peterson & Heilbronner, 1987; Sexton et al., 2003). For example, anxiety sensitivity is only moderately correlated with trait anxiety (Peterson & Heilbronner, 1987; Sandin et al., 2001). Similarly, the shared variance of anxiety sensitivity with trait anxiety and other fear-related measures, such as neuroticism, does not exceed 50% (Taylor, 1999). Factor analytic investigations provide further evidence for the

distinction between anxiety sensitivity and trait anxiety. Empirically, trait anxiety items loaded onto a trait anxiety factor while anxiety sensitivity items loaded onto a distinct, yet correlated anxiety sensitivity factor (Peterson & Heilbronner, 1987; Sandin et al., 2001; Taylor et al., 1991). When considering anxiety sensitivity and trait anxiety in relation to anxiety disorders, anxiety sensitivity also accounts for additional variance in anxiety disorders above and beyond that accounted for by trait anxiety (McNally, 1989). Together, this evidence suggests that, although anxiety sensitivity and trait anxiety are moderately correlated with one another, they are distinct constructs. Similarly, child and adolescent research demonstrated that, among children and adolescents with anxiety disorders, both trait anxiety and anxiety sensitivity account for unique proportions of the variance in anxiety-related symptoms (Muris et al., 2001). Moreover, anxiety sensitivity moderates the association of trait anxiety with other psychopathology, such as substance use severity (Comeau et al., 2001). Together these studies suggest that, although correlated with trait anxiety, anxiety sensitivity is a distinct construct present in childhood and adolescence that uniquely influences anxiety symptoms and related psychopathology.

Anxiety Sensitivity and Eating Disorder Symptoms

Conceptually, anxiety sensitivity may be associated with eating disorder symptoms in multiple ways. First, anxiety sensitivity may be theoretically extended to include heightened sensitivity to interoceptive sensations related to eating and digestive cues (e.g., fullness). Thus, elevated anxiety sensitivity may increase the likelihood of associating eating and digestive cues with negative outcomes (e.g., weight gain, negative social consequences) and interpreting them as harmful or dangerous. This could subsequently result in frequent negative thoughts related to eating and body weight, as well as disordered eating behaviours to ameliorate negative affect (e.g., fasting). Second, anxiety sensitivity may increase demand to regulate negative affect (i.e.,

distress), which may be experienced both more frequently and as more aversive. Theoretically, elevated anxiety sensitivity indicates that an individual is more aware of interoceptive cues for distress, which may produce more subsequent distress and produce distress more frequently. Therefore, an individual with elevated anxiety sensitivity may feel a need to downregulate distress more frequently both because distress is more frequently experienced and because distress is more frequently experienced as intensely aversive. Consistent with theoretical foundations for eating disordered behaviours (e.g., Hearon et al., 2014; Kauffman et al., 2019), an individual might use disordered eating (e.g., binge eating, compensatory behaviour, caloric restriction) as an affect regulation strategy. If so, then elevated anxiety sensitivity may increase the frequency and severity of cues to employ this strategy to downregulate distress. To date, few studies have examined the specific association of anxiety sensitivity with eating disorder symptoms in adults. In community samples, anxiety sensitivity predicts subsequent eating disorder symptoms, including weight and shape concern (Davey & Chapman, 2009); dysregulated eating (Anestis et al., 2007; Anestis et al., 2008); and binge eating (DeBoer et al., 2012). The association of anxiety sensitivity with a drive for thinness is more variable; some studies demonstrate that elevated anxiety sensitivity is associated with elevated drive for thinness (Davey & Chapman, 2009), while others find no association between anxiety sensitivity and drive for thinness Anestis et al., 2007; 2008). When considering specific subfactors of anxiety sensitivity, fear of cognitive concerns may be specifically associated with eating disorder cognitions and behaviours in community samples (Fulton et al., 2012). Anxiety sensitivity is also associated with eating disorder symptoms in clinical samples. To our knowledge, in the only study to date to examine the influence of anxiety sensitivity on eating disorder symptoms in a

clinical population, Anestis et al. (2008) demonstrated that anxiety sensitivity predicted both symptoms of bulimia nervosa and higher drive for thinness.

Anxiety sensitivity may also be associated with eating disorder symptoms in children and adolescents. Recently, Fairweather-Schmidt and Wade (2020) assessed the association of anxiety sensitivity with eating disorder symptoms in children and adolescents aged 12 to 15 years. Findings from this study corroborate findings in adults and preliminary research in children and adolescents; correlations of anxiety sensitivity with eating disorder symptoms, including weight, shape and eating concern, and dietary restraint were moderate. Similarly, in adolescents with severe eating disorders, anxiety sensitivity predicted elevated eating disorder symptoms (Espel-Huynh et al., 2019). Thus far, evidence implicates anxiety sensitivity in eating disorder symptomatology, but putative mechanisms through which anxiety sensitivity and eating disorder symptomatology are related are unclear.

Etiology of Anxiety Sensitivity

Like anxiety and eating disorder symptoms, genetic and environmental factors contribute to anxiety sensitivity in adults. Using the biometrical twin design, Stein et al. (1999) assessed the genetic and environmental contributions to anxiety sensitivity as measured by the Anxiety Sensitivity Index. Additive genetic factors contributed to 45% of the variance in anxiety sensitivity while person-specific environmental influences accounted for the remaining variance. While well-established among women (Jang et al., 1999; Stein et al., 1999; Taylor et al., 2008), heritability estimates may be more variable among men. For example, Jang et al. (1999) examined anxiety sensitivity among monozygotic and dizygotic twins; genetic factors contributed 37% to 48% of the variance in anxiety sensitivity among women but only environmental factors, including shared and person-specific environmental factors, contributed

to anxiety sensitivity among men. Prior research also examined the influence of anxiety sensitivity severity on the heritability of anxiety sensitivity. In women, extant evidence is contradictory such that some report that genetic influences account for approximately half of the variance in anxiety sensitivity regardless of the severity (e.g., Stein et al., 1999) while others suggest that the heritability of anxiety sensitivity increases with severity (e.g., Taylor et al., 2008). Research is still needed to clarify the non-linear contribution of genetic and environmental influences to anxiety sensitivity severity in men. Nonetheless, regardless of severity, genetic influences contributed moderately to anxiety sensitivity. When taken together, anxiety sensitivity is influenced by genetic factors among women, while further research is needed in men.

The environmental and genetic contributions to anxiety sensitivity have also been estimated in children and adolescents using the biometrical twin design. In a large community sample of 8-year-old children, including male and female participants, genetic influences accounted for approximately 37% of the variance in anxiety sensitivity while the remaining variance in anxiety sensitivity was accounted for by person-specific environmental influences (Eley et al., 2007). In adolescents aged 12 to 19 years, genetic influences accounted for 60% of the variance in anxiety sensitivity; the remaining variance was attributed to shared and person-specific environmental factors (Brown et al., 2012). Family studies further assess the correlation of a phenotype between parents and their offspring to inform estimates of heritability. In children, family studies indicate that parent anxiety sensitivity is associated with child anxiety sensitivity among female but not male children (Tsao et al., 2005), which suggests that anxiety sensitivity may be particularly heritable among female children. However, subsequent research using the biometrical twin design demonstrates that anxiety sensitivity is heritable in both female

and male children with no significant differences as a function of biological sex (Brown et al., 2012; Zavos et al., 2012). Anxiety sensitivity is also temporally stable across childhood and adolescence; biometrical twin research suggests that this stability may be attributable to stable genetic influences while environmental influences are time-specific and contribute to variability in the presentation of anxiety sensitivity over time (Zavos et al., 2012).

Genetic and Environmental Contributions to Anxiety Sensitivity and Eating Disorder Symptoms

Anxiety sensitivity is phenotypically correlated with both anxiety severity and cognitive eating disorder symptoms. Further, correlated genetic and environmental influences contribute to anxiety sensitivity and cognitive symptoms of eating disorders, as well as to anxiety sensitivity and anxiety severity. Therefore, it is possible that common genetic or environmental influences contribute to all three constructs. If so, correlated genetic and environmental contributions could explain why anxiety sensitivity is phenotypically correlated with both anxiety and eating disorder symptoms. However, few studies have empirically examined the correlations among genetic contributions to anxiety sensitivity, eating disorder symptoms, and anxiety severity. In children and adolescents, correlated genetic and environmental influences contribute to the phenotypic correlation of anxiety sensitivity with anxiety. For example, Hettema et al. (2020) reported a large phenotypic correlation of anxiety sensitivity with anxiety, to which both correlated genetic and person-specific environmental influences contributed. To date, one research study by Fairweather-Schmidt and Wade (2020) examined correlated genetic and environmental influences between anxiety sensitivity and disordered eating in children or adolescents. The study included adolescent twins aged 12 to 15 years and assessed eating disorder symptoms using the eating disorder inventory, which generates an overall eating disorder symptom severity

score based on the severity of eating, weight and shape concern, and dietary restraint. Genetic influences accounted for approximately 50% of the variance in eating disorder symptoms and 42% of the variance in anxiety sensitivity. Person-specific environmental influences accounted for the remaining variance in both eating disorder symptoms and anxiety sensitivity. Further, Fairweather-Schmidt and Wade (2000) reported that anxiety sensitivity and eating disorder symptoms shared common genetic and person-specific environmental influences such that 25% of the eating disorder symptoms phenotype was derived from anxiety sensitivity, including correlated additive genetic and shared environmental influences. This study provides preliminary evidence for the role of anxiety sensitivity in eating disorder symptoms among children and adolescents. Further, the findings indicate that the phenotypic correlation of anxiety sensitivity with eating disorder symptoms may be attributable to common (i.e., correlated) genetic and environmental etiological influences.

The Present Study

Eating disorder symptoms are highly prevalent in adolescence and are associated with impaired quality of life and psychosocial impairment including low self-esteem and internalizing and externalizing syndromes (Zeiler et al., 2016). Prior research in community samples of adolescents indicated that anxiety symptoms are frequently comorbid with eating disorder symptoms (Fitzsimmons-Craft et al., 2019; Hughes., 2012; Touchette et al., 2011). For example, anxious distress and anxiety-related traits are strongly correlated with eating disorder symptom severity in adolescence (Bulik et al., 2006; Touchette et al., 2011). Moreover, longitudinal research suggests that anxiety may precede and contribute to the later development of eating disorder symptoms (Lee & Vaillancourt, 2019; Parkinson et al., 2012). Research to clarify the genetic and environmental contributions to the etiology of eating disorder cognitions during

adolescence will add to our current understanding of the development of eating disorder cognitions. A focus on both environmental and genetic risk factors may promote the identification of biopsychological substrates through which eating disorder cognitions develop during adolescence. This may then inform future prevention and intervention strategies for adolescents.

Prior research leveraged the biometrical twin design to document the contribution of correlated genetic and environmental influences to both anxiety and eating disorder symptoms in adults (Dellava et al., 2011; Song et al., 2019), children, and adolescents (e.g., Silberg & Bulik, 2005). Given the high concordance between anxiety and eating disorder symptoms (Fitzsimmons-Craft et al., 2019), putative anxiety and fear-related traits (e.g., anxiety sensitivity) may broadly increase one's liability to symptoms of both anxiety and eating disorders. Prior research implicated anxiety sensitivity in myriad psychopathology including anxiety disorders, symptoms of multiple anxiety syndromes, and, more recently, eating disorders and eating disorder symptoms. Preliminary research also demonstrated that the genetic and environmental contributions to anxiety sensitivity may be correlated with the genetic and environmental contributions to both anxiety and eating disorder symptoms in adolescence.

Despite the high concordance of anxiety with eating disorder symptoms, and the potential role of anxiety sensitivity as a risk factor for both anxiety and eating disorder symptoms, there have been few studies that assessed this relationship from an etiological perspective. Therefore, the overarching goal of the present study was to examine the common genetic and environmental influences that contribute to anxiety sensitivity, eating disorder symptoms, and anxiety severity in a community sample of adolescents. Within this overarching goal, the present study had multiple aims. First, I hypothesized that a latent variable, i.e., cognitive symptoms of eating

disorders, would best explain the correlation between the four measured items of eating disorder cognitions, including fear of gaining weight, feeling fat, shape influences thoughts about oneself, and weight influences thoughts about oneself. Second, I hypothesized that genetic influences would contribute significantly to latent variance in cognitive eating disorder symptom severity. Third, I hypothesized that genetic influences to anxiety sensitivity, cognitive eating disorder symptoms, and anxiety severity would be correlated with one another, which may indicate that anxiety sensitivity functions as an underlying trait to contribute to both cognitive symptoms of eating disorders and anxiety severity.

The present study leveraged data from the Twins Early Development Study (TEDS), a longitudinal epidemiological study of pairs of monozygotic and dizygotic twins born in England and Wales between 1994 and 1996. TEDS included 13 waves of data. Twins were first recruited at 18 months of age; the most recent assessment wave occurred when twins were 21 years old. Twin births were identified through birth records and families were contacted. From these, 16,810 twin pairs indicated that they were interested in participating in the study; at first contact, 13,694 pairs of twins provided data. The number of twin pairs enrolled in the study decreased slightly in early childhood (n=10,150), middle childhood (n=8,819), and adolescence (n=8,697). To promote mental health research, data are shared freely with researchers around the world. The present study used data from age 16 when twins self-reported anxiety sensitivity, via the Child Anxiety Sensitivity Index, and eating disorder symptoms, via four items from the Eating Disorder Diagnostic Scale. Parents reported adolescent anxiety severity dimensionally via the Anxiety Related Behaviours Questionnaire. Initially, a structural equation model leveraged the differential genetic relatedness of monozygotic and dizygotic twins to estimate additive genetic; shared environmental; and unique, person-specific environmental contributions to a latent

composite of cognitive symptoms of eating disorders indexed by the four eating disorder cognition items. Secondly, anxiety severity and anxiety sensitivity were added to the model of cognitive symptoms of eating disorders to estimate i) their phenotypic correlation with cognitive symptoms of eating disorders and ii) the contribution of correlated environmental and genetic influences to the phenotypic correlation of cognitive symptoms of eating disorders with anxiety sensitivity and anxiety severity. The present study clarifies the development of cognitive symptoms of eating disorders and anxiety in adolescence to, ultimately, examine the etiology of cognitive symptoms of eating disorders, which presents a well-documented, substantial impediment to the well-being of a large number of adolescents.

Methods

Participants

The present study used data collected when twins were 16 years old (i.e., twins were assessed between February 2011 and June 2012). In total, 5,111 twin pairs provided usable data at this assessment wave, of which 55.3% twins were female and 64.4% pairs were monozygotic twins. At this assessment wave, 10,868 pairs of twins were initially contacted, of which 93.5% were Caucasian. A total of 5,144 twin pairs provided data; 33 twin pairs were excluded because information on twin zygosity was unavailable. At first contact, when twins were 18 months old, the sample included Caucasians (91.7%), females (50.1%), employed mothers (43.1%), and employed fathers (91.7%), which is representative of the general population from which the sample was recruited (Walker et al., 2001). Similar percentages remained throughout the study. Twin zygosity was assessed by DNA test or a well-validated parent-report questionnaire, which is 95% accurate when compared to DNA assay of zygosity (Price et al., 2000).

Measures

Cognitive Symptoms of Eating Disorders

To assess cognitive symptoms of eating disorders, twins self-reported four items from the Eating Disorder Diagnostic Scale (Stice et al., 2000), which measures eating disorder symptoms over the past 6 months. The questions were specifically “Have you felt fat?”, “Have you had a definite fear that you might gain weight or become fat?”, “Has your weight influenced how you think about yourself as a person?”, and “Has your shape influenced how you think about yourself as a person?”. Each item was measured on a 7-point Likert scale ranging from “not at all” (1) to “extremely” (7). Rather than computing a total score for cognitive symptoms of eating disorders using the four Eating Disorder Diagnostic Scale items, cognitive eating disorder symptom severity was treated as a latent factor represented by variance shared between the four Eating Disorder Diagnostic Scale items. The full Eating Disorder Diagnostic Scale is comprised of 22 self-report items. Given time constraints of a multifaceted epidemiologic study, only four items from the scale were assessed during the 16-year-old assessment wave. However, these items cover central features of eating disorder cognitive symptoms indicated in the DSM-5 (APA, 2013). Prior research demonstrates strong reliability and validity of the full Eating Disorder Diagnostic Scale, including good criterion validity, test-retest reliability, and internal consistency (Stice et al., 2000).

Anxiety Sensitivity

To assess anxiety sensitivity, twin participants completed the Child Anxiety Sensitivity Index, a version of the Anxiety Sensitivity Index (Peterson, 1991) modified for children. The Child Anxiety Sensitivity Index is comprised of 18 self-reported items thatparticipants rate on a three-point Likert scale (0=none, 1=some, 2=a lot). The total anxiety sensitivity score is the sum

of all 18 items. Prior research supports strong psychometric properties of the Child Anxiety Sensitivity Index to assess anxiety sensitivity in children and adolescents. For example, Silverman and colleagues report good test-retest reliability in non-clinical and clinical samples as well as good interitem reliability in clinical and nonclinical samples (Silverman et al.,1991, 2003). The present study agrees with prior research in documenting high intraitem reliability (polychoric Cronbach $\alpha = 0.92$) and evidence that the scale is unifactorial (polychoric McDonald's $\Omega_{hierarchical} = 0.64$) at least as a higher-order factor of multiple subfactors (polychoric McDonald's $\Omega_{total} = 0.94$).

Anxiety Severity

Adolescent anxiety was assessed via parent report on the Anxiety Related Behaviour Questionnaire, which includes 19 items rated on a 3-point Likert scale (0= Not true, 1=Quite true, 2=Very true) to assess adolescent anxiety symptoms over the past 6 months. The total Anxiety Related Behaviour Questionnaire score was computed as the sum of all 19 items. While prior research reports varying factor structures of the Anxiety Related Behaviour Questionnaire as a function of child age (Eley et al., 2003; Hallett et al., 2009), high interitem reliability for the full scale is stable across samples and ages assessed (Eley et al., 2003; Hallett et al., 2009), which provides evidence for a consistent, reliable unifactorial structure. The present study agrees with prior research in documenting high intraitem reliability (polychoric Cronbach $\alpha = 0.92$) and evidence that the scale may be unifactorial (polychoric McDonald's $\Omega_{hierarchical} = 0.70$) at least as a higher-order factor of multiple subfactors (polychoric McDonald's $\Omega_{total} = 0.94$). Moreover, it was outside the scope of the present paper to establish the factor structure of the Anxiety Related Behaviour Questionnaire at age 16.

Data Analytic Plan

The analyses were conducted using the biometrical twin model in a structural equation modelling framework (see Evans et al., 2002 for review). The biometrical twin model leverages the difference in genetic concordance between monozygotic and dizygotic twins, who share 100% and 50% of their segregating genes, respectively. Thus, the variance in one trait (e.g., cognitive symptoms of eating disorders) or covariance between two traits (e.g., anxiety sensitivity and cognitive eating disorder symptom severity) can be decomposed into additive genetic, shared-, and person-specific- environmental influences (see Neale & Cardon, 1992 for review). Specifically, I aimed to estimate the contribution of additive genetic (A) factors to a latent composite of cognitive symptoms of eating disorders indexed by the four cognitive symptoms of eating disorder items, as well as influences of environment shared between twins within a family (i.e., shared environmental influences; C) and environmental influences specific to each child (i.e., person-specific environmental influences; E; e.g., Kendler et al., 1992). Analyses then extended this model to estimate the phenotypic correlation of cognitive eating disorder symptom severity with anxiety sensitivity and anxiety severity as well as the role of correlated genetic, shared environmental, and person-specific environmental contributions common to cognitive eating disorder symptom severity, anxiety sensitivity, and anxiety severity to explain their well-documented phenotypic correlation.

Assumptions

The biometrical model of twin relatedness rests on several assumptions. First, genetic, shared environmental, and person-specific environmental factors are assumed to be linear and mutually exclusive, such that there are no interactions between genetic and environmental (shared and person-specific) contributions to each phenotype (Maes, 2005). Second, the

biometrical twin model is founded on the equal environment assumption, which states that the influence of the shared environment on each phenotype is shared to the same extent for monozygotic and dizygotic twins (Kendler et al., 1993). Several studies demonstrate that the equal environment assumption holds for psychiatric traits in twins (see Roysamb & Tambs, 2016 for review; see also Cronk et al., 2002; Kendler et al., 1993). However, some contradictory evidence does exist. For example, Hettema et al. (1995) found the equal environment assumption was violated in adult female twins with bulimia nervosa, such that similarity in physical appearance significantly influenced concordance among twins. However, when broadly defined bulimia nervosa was included, the equal environment assumption was no longer violated (Hettema et al., 1995). In further support of the validity of the equal environment assumption in research on eating disorder symptoms, Klump and colleagues (2000b) report that the equal environment assumption are not violated for cognitive and behavioural eating disorder symptoms among a community sample of adolescents. Third, it is assumed that twins do not influence each other, such that there are no environmental effects due directly to one's twin (Maes, 2005). For example, it is assumed that neuroticism in one twin does not lead to behaviours that evoke greater neuroticism in the other twin. Finally, it is assumed that the phenotype of interest is equivalent across twin order, such that twins who are arbitrarily designated as twin one do not systematically differ from twins who are arbitrarily designated as twin two. Similarly, it is assumed that the phenotype of interest is equivalent across twin zygosity, such that monozygotic twins do not systematically differ from dizygotic twins.

The Structure of Genetic and Environmental Contributions to Cognitive Eating Disorders Symptom Severity

The biometrical twin model was used to estimate the contributions of genetic, shared environmental, and person-specific environmental influences on each of the four cognitive symptoms of eating disorder items. In an initial model, the total variance for each of the four cognitive symptoms of eating disorder items were decomposed into variance explained by genetic, shared environmental, and person-specific environmental factors. Next, I estimated the within-person phenotypic correlations among the four cognitive symptoms of eating disorder items: felt fat, fear of gaining weight, weight influences thoughts about oneself, and shape influences thoughts about oneself. Then, I estimated the correlations among the genetic, shared environmental, and person-specific environmental contributions to all four cognitive symptoms of eating disorder items.

I then fit a latent factor extension to the biometrical twin model with two latent factors to model cognitive eating disorders symptom severity and a possible residual correlation between weight and shape influences thoughts about oneself. The four cognitive symptoms of eating disorder items were allowed to freely correlate with the first latent factor, cognitive eating disorder symptom severity. To statistically identify the second factor, its association with the item, “felt fat” was constrained to 0; the remaining variables were allowed to freely correlate with the second factor. Model fit was assessed by comparing the two-latent factor model to the original model in which all four items were intercorrelated using the chi-square difference test and Akaike Information Criterion (AIC) comparative fit index. These extensions to the biometrical twin model also facilitated partitioning the total variance in each latent factor (e.g., latent cognitive eating disorder symptom severity) into that explained by the contribution of

additive genetic, shared environmental, and person-specific environmental influences. Similarly, the residual variance for each of the four cognitive symptoms of eating disorder items was also decomposed into variance explained by genetic, shared environmental, and person-specific environmental influences.

Association of Latent Cognitive Eating Disorder Symptom Severity with Anxiety Sensitivity and Anxiety Severity

A Cholesky decomposition of the additive genetic, shared environmental, and person-specific environmental matrices in the latent-factor biometrical twin model was used to estimate the proportion of variance in anxiety sensitivity, latent cognitive eating disorders symptom severity, and anxiety severity attributed to genetic and environmental (i.e., shared and person-specific environment) influences. Next, I estimated the phenotypic correlations among anxiety sensitivity, latent cognitive eating disorder symptom severity, and anxiety severity. I also estimated the correlations among the genetic and environmental contributions to anxiety sensitivity, latent cognitive eating disorder symptom severity, and anxiety sensitivity.

Items were treated as ordinal to test assumptions underlying the biometrical twin model and for regression of cognitive symptoms of eating disorders on age and sex. For all other analyses (e.g., estimation of phenotypic, genetic, and environmental correlations), analysis of items as ordinal proved infeasible with the substantial computational resources available. Therefore, variables were treated as ordinal whenever possible and continuous when necessary. Verhulst and Neale (2021) recently noted that analysis of ordinal data as continuous may overestimate the contribution of variance attributable to person-specific environmental contributions and underestimate variance attributable to genetic and shared environmental contributions. However, I note that, in their analyses, the difference between polychoric (i.e.,

ordinal) and Pearson (i.e., continuous) correlations would be considered a small effect (i.e., r=0.10), especially as the number of ordinal categories increases (Verhulst & Neale, 2021).

Results

Assumptions Underlying the Biometrical Statistical Model

The biometrical model of twin relatedness assumes that twin order (i.e., the order in which twins are arbitrarily labelled twin one or twin two) does not systematically influence their score on the underlying trait (e.g., each cognitive symptom of eating disorder item). Similarly, while monozygotic twins may be more similar to one another than are dizygotic twins, the biometrical model assumes that monozygotic twins will not systematically score higher or lower on each trait in question (Neale & Cardon, 1992). I tested both assumptions by first fitting a saturated model in which the item thresholds for twin 1 were allowed to differ from the item thresholds for twin 2 and item thresholds for monozygotic twins were allowed to differ from item thresholds for dizygotic twins. As described above, items were treated as ordinal whenever computationally feasible. Specifically, tests of assumptions that underlie the biometrical twin model examined item thresholds rather than item means and variances, which were constrained to 0 and 1, respectively. I then fit two constrained threshold models for 1) twin order, in which the item thresholds for twin 1 were constrained to equal the item thresholds for twin 2, and 2) twin zygosity, in which the item thresholds for monozygotic twins were constrained to equal the item thresholds for dizygotic twins. To test model fit for each of the constrained threshold models, I compared each model to the saturated model (i.e., in which all item thresholds are estimated and allowed to differ from one another) using the chi-square difference test and comparison of model AIC.

For each of the four cognitive symptoms of eating disorder items, the model in which item thresholds were constrained across twin order (i.e., in which thresholds for twin 1 were constrained to be equal to item thresholds for twin 2) was not a significantly worse fit than the unconstrained model (see Table 1). Similarly, for each of the four cognitive symptoms of eating disorder items, the model in which item thresholds were constrained across zygosity (i.e., in which monozygotic twins were constrained to have equal item thresholds as dizygotic twins) was not a significantly worse fit than the unconstrained model (see Table 1). Thus, the necessary assumptions are met that item thresholds are comparable across twins arbitrarily identified as twin 1 or twin 2 and between monozygotic and dizygotic twins for the four cognitive symptoms of eating disorder items.

Table 1

Evaluation of assumptions in statistical genetics: Effects of twin order and zygosity on item thresholds for each eating disorder cognition item

Felt fat						
Model	χ^2	df	AIC	$\Delta \chi^2$	Δ df	p
Fully saturated	7967.54	2204	8019.54			
Item Thresholds Constrained Across Twin Order	7977.35	2216	8005.35	9.81	12	.63
Item Thresholds Constrained Across Zygosity	7983.18	2222	7999.17	15.63	18	.62

Fear of gaining weight						
Model	χ^2	df	AIC	$\Delta \chi^2$	Δ df	p
Fully saturated	7663.15	2205	7715.14			
Item Thresholds Constrained Across Twin Order	7668.58	2217	7696.58	5.43	12	.94
Item Thresholds Constrained Across Zygosity	7671.31	2223	7687.31	8.16	18	.98

Weight influences thoughts about oneself						
Model	χ^2	df	AIC	$\Delta \chi^2$	Δ df	p
Fully saturated	539.40	2204	7591.40			
Item Thresholds Constrained Across Twin Order	7550.59	2216	7578.59	11.19	12	.51
Item Thresholds Constrained Across Zygosity	7555.02	2222	7571.02	15.62	18	.62

Shape influences thoughts about oneself						
Model	χ^2	df	AIC	$\Delta \chi^2$	Δ df	p
Fully saturated	7888.64	2200	7940.64			
Item Thresholds Constrained Across Twin Order	7903.96	2212	7931.96	15.32	12	.22
Item Thresholds Constrained Across Zygosity	7913.55	2218	7929.55	24.90	18	.13

To account for possible associations of age and sex with cognitive symptoms of eating disorders, age and sex were regressed onto each of the four items. Age was not significantly associated with any of the cognitive symptoms of eating disorders: felt fat, *b* = *-.26, 95%CI* = -6.74, 1.46, p = .21; fear of gaining weight, *b* = *-.09,* 95%*CI* = 5.11, 3.26, p = .66; weight

influences thoughts about oneself, *b* = *-.005,* 95%*CI* = -4.86, 3.87, p = .82; and shape influences thoughts about oneself, *b* = .008, 95%CI= -4.27, 4.44, p = .97. However, sex was significantly and strongly associated with each of the four cognitive symptoms of eating disorder items: felt fat, *b* = *-1.90*, 95%CI = -2.14, -1.81, p = 2.8 x 10^{-106}; fear of gaining weight, *b*=-1.96, 95%CI = -2.13, -1.79, p = 6.0 x 10^{-98}; weight influences thoughts about oneself, *b*=-1.44, 95%CI = -1.62, -1.26, p = 4.9 x 10^{-52}; and shape influences thoughts about oneself, *b*=-1.18, 95%CI = -1.36, -1.01, p = 3.7 x 10^{-37}. To adjust for their potential contribution to cognitive symptoms of eating disorders, as well as other constructs under investigation (e.g., anxiety severity), age and sex were included as covariates in all subsequent models.

The Structure of Genetic and Environmental Contributions to Cognitive Eating Disorder Symptom Severity

The mean score for each of the four cognitive eating disorder symptoms, including felt fat, fear of gaining weight, weight-, and shape- influences thoughts about self are presented in Table 2. Additive genetic influences contributed substantially to three out of the four indices of cognitive symptoms of eating disorders: felt fat (a^2=0.56, 95%CI [0.48, 0.62]), fear of gaining weight (a^2=0.47, 95%CI [0.34, 0.53]), and weight influences thoughts about oneself (a^2=0.33, 95%CI [0.14, 0.47]). Additive genetic influences contributed somewhat to shape influences thoughts about oneself (a^2=0.16, 95%CI [0, 0.36]), which were also influenced by shared environmental contributions (c^2=0.19, 95%CI [0.04, 0.33]), and person-specific contributions (e^2=0.65, 95%CI [0.57, 0.74]). Person-specific environmental influences contributed to the remaining variance in all other indices of cognitive symptoms of eating disorders, namely: felt fat (e^2=0.44, 95%CI[0.34, 0.50]), fear of gaining weight (e^2=0.53, 95%CI[0.48, 0.60]), and weight influences thoughts about oneself (e^2=0.60, 95%CI[0.53, 0.68]), whereas shared

environmental influences did not contribute to these three cognitive symptoms of eating disorders: felt fat (c^2=0, 95%CI[0.000003, 0.05]), fear of gaining weight (c^2=0, 95%CI[0.03, 0.09]), and weight influences thoughts about oneself (c^2=0.08, 95%CI[0.000002, 0.22]).

Table 2

The Mean and standard deviations for anxiety sensitivity, cognitive eating disorder symptoms, and anxiety severity, as a function of sex

Item	Total	Female	Male
Anxiety sensitivity	3.60	9.47	5.99
Eating disorder cognitive symptoms			
Felt fat	2.29	3.06	1.12
Fear of gaining weight	2.14	2.92	0.94
Weight influences oneself	2.04	2.62	1.16
Shape influences oneself	2.12	2.59	1.39
Anxiety severity	7.93	4.20	2.84

The within-person phenotypic correlations of the four cognitive symptoms of eating disorders indicate strong correlations among these four cognitive eating disorder symptoms, including of felt fat with fear of gaining weight (*r*=0.75, 95%CI[0.74, 0.77]), weight- (*r*=0.66, 95%CI[0.63, 0.68]), and shape- (*r*=0.59, 95%CI[0.62, 0.74]) influences thoughts about oneself; fear of gaining weight with both weight (*r*=0.67, 95%CI[0.65, 0.69]) and shape (*r*=0.62, 95%CI[0.60, 0.65]) influences thoughts about oneself; and of weight- with shape- influences thoughts about oneself (*r*=0.86, 95%CI[0.84, 0.87]). Table 3 provides the correlations among the additive genetic (rA), shared environmental (rC), and person-specific environmental (rE) contributions to all four cognitive symptoms of eating disorder items. The correlations among additive genetic and person-specific environmental contributions to each cognitive symptom of eating disorder item were notably strong. The correlations among shared environmental contributions to all four cognitive symptoms of eating disorder items should be considered in

light of generally weak contributions of shared environmental factors to three out of four items (see above), which may inflate the empirical correlation by dividing the covariance between any two given items by component variances that approach 0. Evidence of correlated genetic and person-specific environmental contributions to cognitive symptoms of eating disorder items agrees with strong phenotypic correlations among all four items to indicate a latent factor (e.g., cognitive eating disorder symptom severity) through which common genetic and person-specific environmental factors influence individual cognitive eating disorder symptoms. Because of these findings, I next tested model fit for a latent factor model, in which an overall latent factor is indexed by the four cognitive eating disorder symptoms.

Table 3

Additive genetic (rA), shared environmental (rC) and person-specific environmental (rE) correlations among eating disorder cognition items

		Estimate [95%CI]		
rA	Felt fat	Fear of gaining weight	Weight influences oneself	Shape influences oneself
Felt fat	1			
Fear of gaining weight	.83 [.72, .94]	1		
Weight influences oneself	.94 [.76, 1.25]	.94 [.75, 1.22]	1	
Shape influences oneself	1.06 [.72, 326]	1.00 [.67, 303]	.99 [.76, 214]	1
rC	Felt fat	Fear of gaining weight	Weight influences oneself	Shape influences oneself
Felt fat	1			
Fear of gaining weight	8168 [.35, $7.17x10^4$]	1		
Weight influences oneself	19.73 [-$.22x10^5$,$7.43x10^4$]	22.44 [-$1.22x10^5$, $9.03x10^4$]	1	
Shape influences oneself	62.05 [-$1.20x10^5$, 190]	93.63 [-$1.09x10^5$, 220]	1.07 [.75, 181]	1
rE	Felt fat	Fear of gaining weight	Weight influences oneself	Shape influences oneself
Felt fat	1			
Fear of gaining weight	.63 [.58, .69]	1		
Weight influences oneself	.48 [.40, .54]	.52 [.45, .58]	1	
Shape influences oneself	.42 [.34, .50]	.49 [.41, .55]	.80 [.76, .83]	1

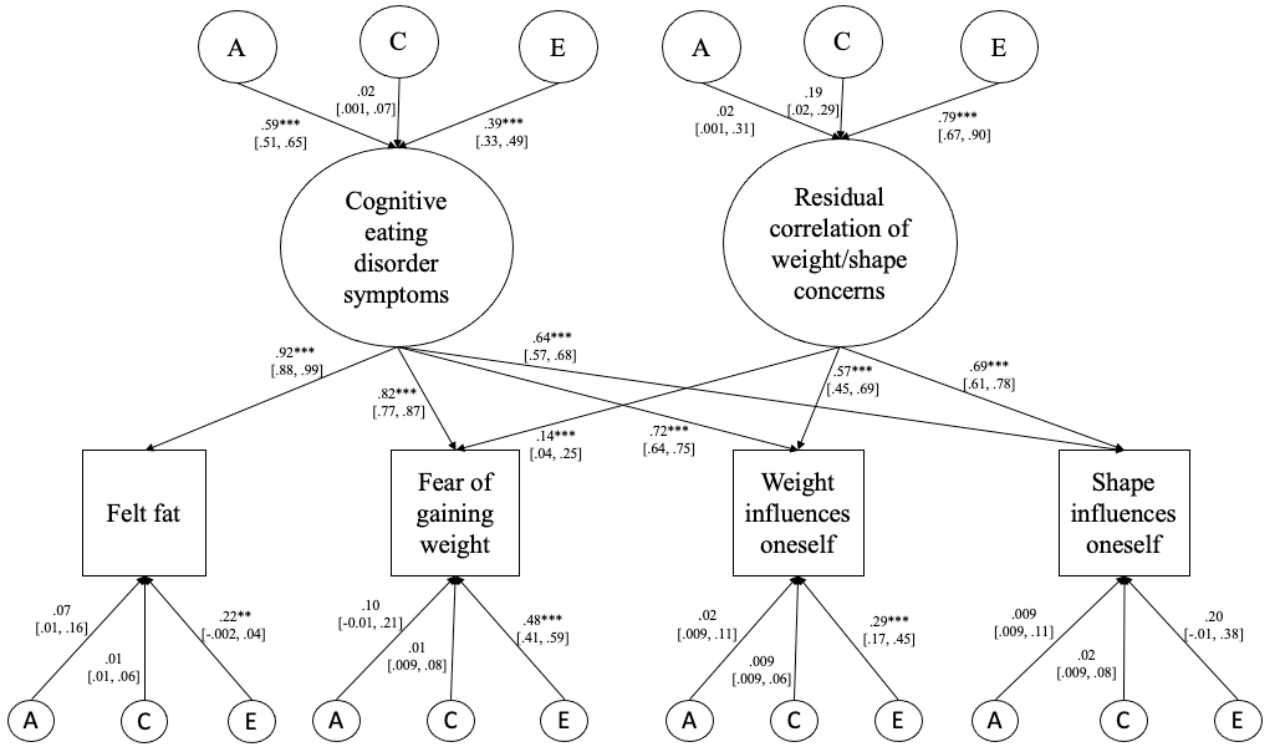
Specifically, I next fit a latent factor extension to the biometrical twin model with two latent factors to model latent cognitive eating disorder symptom severity and a possible residual correlation between the two items that index whether weight and shape influence thoughts about oneself, given their high conceptual similarity (see Figure 1). Specifically, latent cognitive eating disorder symptom severity was indexed by all four cognitive eating disorder symptoms (i.e., felt fat, fear of gaining weight, and weight- and shape- influences thoughts about oneself). This model was nested within the original biometrical model for the four cognitive eating disorder symptoms, in which all four symptoms were intercorrelated. Hence, model fit for this latent factor model was assessed through comparison against the first model, namely via the chi-square difference test and comparison of model AIC. The initial model, $\chi^2(8827) = 30700.11$, AIC = 30787.11, and the two-factor model, $\chi^2(8834) = 30709.83$, AIC = 30783.83, did not differ significantly from one another, $\Delta\chi^2(7) = 9.73$, $p = .20$. Since the fit of both models were comparable and because the two-factor model was more parsimonious, as indicated by a lower AIC value, I concluded that the two-factor model is a better fit to the data.

Felt fat; fear of gaining weight; and weight- and shape- influences thoughts about oneself, loaded significantly onto the latent factor that indexes cognitive eating disorder symptom severity. The cognitive eating disorder symptoms: fear of gaining weight and weight- and shape-influences thoughts about oneself also loaded significantly onto a second latent factor, although only the magnitude of weight- and shape-influences thoughts about oneself were high, which indicates that the second latent factor primarily reflected a residual correlation between these two conceptually similar items. The latent factor extension of the biometrical twin model also simultaneously decomposed the total variance in both latent factors into additive genetic, shared environmental, and person-specific environmental contributions to each latent factor. Additive

genetic (a^2=0.59, 95%CI[0.51, 0.65] and person-specific environmental influences (e^2=0.39, 95%CI[0.33, 0.49]) contributed significantly to variance in cognitive eating disorder symptom severity, which was not significantly influenced by shared environmental contributions (c^2=0.02, 95%CI [0.001, 0.07]). Person-specific (e^2=0.79, 95%CI[0.67, 0.90]) and shared (c^2=0.19, 95%CI[0.02, 0.29]) environmental influences, but not additive genetic influences (a^2=0.02, 95%CI[0.001, 0.31]) also contributed significantly to the variance in the residual correlation of weight and shape concern, which accounted for the residual correlation of weight- and shape-influence thoughts about oneself. All remaining residual variance in the four cognitive eating disorder symptoms was also partitioned into additive genetic, shared environmental, and person-specific environmental contributions. Only person-specific environmental influences contributed significantly to symptom residual variance (see Figure 1).

Figure 1

Factor loadings and additive genetic (A), shared environmental (C), and person-specific environmental (C) contributions to latent factors indexed by 4 cognitive eating disorder cognition symptoms and residual item variances.



Note. *p<.05. **p< .01. ***p< .001

Association of Cognitive Eating Disorder Symptom Severity with Anxiety Sensitivity and Anxiety Severity

Phenotypically, anxiety sensitivity was significantly correlated with cognitive eating disorder symptom severity, the residual correlation of weight and shape concern, and anxiety severity (see Table 4). Anxiety severity was also significantly correlated with cognitive eating disorder symptom severity and the residual correlation of weight and shape concern. Within this extension of the latent factor biometrical twin model, one can partition the total variance of anxiety sensitivity, cognitive eating disorder symptom severity, the residual correlation of weight

and shape concern, and anxiety severity into additive genetic, shared environmental, and person-specific environmental contributions (see Table 5). Additive genetic and person-specific environmental influences contributed to all the variance in anxiety sensitivity and cognitive eating disorder symptom severity. Additive genetic, shared environmental, and person-specific environmental influences contributed to the variance in anxiety severity. Only environmental influences, both shared and person-specific environmental influences, contributed to the variance in the residual correlation of weight and shape concern.

Table 4

Phenotypic correlations among anxiety sensitivity (AS), cognitive eating disorder symptom severity (CS), residual correlation of weight and shape concern (RC), and anxiety severity (Anx)

	Estimate [95%CI]			
Construct	AS	CS	RC	Anx
AS	1			
CS	.223 [.216, .234]***	1		
RC	.258 [.255, .271]***	.082 [.056, .089]*	1	
Anx	.206 [.206, .212]***	.098 [.060, .143]***	.109 [.098, .137]***	1

Note. *p<.05. **p<.01. ***p<.001.

Table 5

Additive genetic (A), shared environmental (C), and person-specific environmental (E) contributions to anxiety sensitivity (AS), cognitive eating disorder symptom severity (CS), residual correlation of weight and shape (RC), and anxiety severity (Anx)

Construct	A [95%CI]	C [95%CI]	E [95%CI]
AS	.404 [.396, .409]	$3.15x10^{-8}$ [$3.15x10^{-8}$, $2.05 x10^{-6}$]	.596 [.591, .598]
CS	.597 [.580, .608]	.00652 [.00549, .00809]	.397 [.386, .397]
RC	.036[.030, .041]	.179 [.169, .179]	.786 [.787, .823]
Anx	.389 [.374, .389]	.263 [.258, .267]	.348 [.341, .350]

The biometrical twin model also facilitates estimation of the correlations among genetic and environmental (i.e., shared and person-specific environment) contributions to anxiety sensitivity, cognitive eating disorder symptom severity, the residual correlation of weight and shape concern, and anxiety severity. As hypothesized, genetic influences that contributed to liability to anxiety sensitivity were significantly, moderately correlated with genetic influences that contributed to liability to cognitive eating disorder symptom severity and anxiety severity (see Table 6). There were no significant correlations among shared environmental influences to anxiety sensitivity, cognitive eating disorder symptom severity, the residual correlation of weight and shape concern, and anxiety severity, which may be due to low contributions of shared environmental influences to these phenotypes overall. Person-specific environmental influences to anxiety sensitivity were significantly, but modestly, correlated with person-specific environmental influences to cognitive eating disorder symptom severity, the residual correlation of weight and shape concern, and anxiety severity (see Table 6). Notably, cognitive eating disorder symptom severity was phenotypically correlated with elevated anxiety severity but contrary to hypotheses, there was no evidence of a correlation between the genetic and environmental contributions to cognitive eating disorder symptom severity and anxiety severity.

Table 6

Additive genetic (rA), shared environmental (rC), and person-specific environmental (rE) correlations among anxiety sensitivity (AS), cognitive eating disorder symptom severity (CS), residual correlation of weight and shape concern (RC), and anxiety severity (Anx) – Estimate [95% CI]

rA	AS	EC	RC	Anx
AS	1			
EC	.267 [.231, .281]**	1		
RC	1.00 [.999, 1.00]	.267 [.253, .291]***	1	
Anx	.393 [.393, .410]***	.065 [.0267, .076]	.388 [.285, .405]	1
rC	AS	EC	RC	Anx
AS	1			
EC	-.646 [-.889, .516]	1		
RC	-.984 [-.999, -.981]	.635 [.304, .989]	1	
Anx	-.165 [-.181, -.159]	.854 [.266, .992]	.140 [.090, .154]	1
rE	AS	EC	RC	Anx
AS	1			
EC	.188 [.171, .196]***	1		
RC	.201 [.181, .284]***	.038 [.033, .041]***	1	
Anx	.110 [.104, .115]***	.086 [.083, .131]	.062 [.075, .081]	1

Note. *p<.05. **p<.01. ***p<.001.

Discussion

The overarching goal of the present study was to examine the contribution of anxiety sensitivity to the etiology of eating disorder symptom severity. Within this broad goal, the present study had multiple specific aims. First, I estimated the genetic, shared environmental, and person-specific environmental contributions to unique cognitive eating disorder symptoms. Genetic influences contributed substantially to feeling fat and fear of gaining weight and moderately to weight and shape influences thoughts about oneself. Second, I estimated the contribution of additive genetic and environmental influences to a latent composite of cognitive eating disorder symptom severity indexed by the four symptoms assessed. Genetic and person-

specific environmental influences contributed broadly to the liability to cognitive eating disorder symptom severity, although genetic influences contributed particularly strongly to cross-symptom stability. Third, I estimated the genetic, shared environmental, and person-specific environmental contributions to the liability to anxiety sensitivity and anxiety severity as well as cognitive eating disorder symptom severity. Genetic and person-specific environmental influences contributed to the liability to anxiety sensitivity and cognitive eating disorder symptom severity, while genetic, and shared and person-specific environmental influences contributed to the liability to anxiety severity. Finally, I examined phenotypic correlations among anxiety sensitivity, cognitive symptoms of eating disorders, and anxiety severity. Anxiety sensitivity was significantly correlated with cognitive eating disorder symptoms and anxiety severity, as was anxiety severity with cognitive eating disorder symptoms. I then decomposed the variance among anxiety sensitivity, anxiety severity, and cognitive eating disorder symptom severity into component genetic and environmental influences to examine genetic and environmental contributions to these phenotypic correlations. I found correlated genetic and person-specific environmental contributions to the phenotypic correlations of anxiety sensitivity with cognitive eating disorder symptom severity and anxiety severity. In contrast, neither correlated genetic nor environmental influences contributed to the phenotypic correlation of anxiety severity with cognitive eating disorder symptom severity. Together, these results suggest that as compared to anxiety severity, anxiety sensitivity may play a more substantial role in the etiology of cognitive symptoms of eating disorders.

Etiology of Cognitive Eating Disorder Symptoms

Latent genetic factors contributed to all cognitive eating disorder symptoms; between-person genetic variation may explain 16% to 60% of between-person variation in the presence

and severity of cognitive symptoms of eating disorders. Latent person-specific environmental factors contributed the remaining variance in all cognitive eating disorder symptoms except for variance in shape concerns about oneself, which was also influenced by environmental factors shared between twins. Evidence of genetic and person-specific environmental contributions to the specific cognitive eating disorder symptoms under investigation here (e.g., feeling fat) is in line with previous findings, which consistently demonstrate that genetic and person-specific environmental influences account for between-person variance in severity of a range of cognitive eating disorder symptoms, including weight preoccupation, body dissatisfaction, and drive for thinness, among community samples of adolescents (e.g., Klump et al., 2000a; Waszczuk et al., 2019).

I further demonstrated that the etiology of the items that assessed whether weight or shape influences thoughts about oneself, differed somewhat from the etiology of the items that assessed feeling fat or fear of gaining weight. A relatively smaller proportion of the variance in weight and shape influences thoughts about oneself could be attributed to genetic influences; instead, a larger proportion of variance in symptom severity could be attributed to environmental influences either those specific to each adolescent or those shared between twins within a family. For example, only 16% of the variance in the symptom that shape concern about oneself could be attributed to latent genetic influences; the remaining variance was accounted for by environmental influences. To our knowledge, this is the first study to examine the contribution of latent genetic and environmental factors in the etiology of the symptoms that weight and shape unduly influence one's thoughts about self in a community sample of adolescents. The present findings accord with prior research of young adults, which also demonstrates a relatively smaller contribution of genetic influences and larger contribution of person-specific environmental

influences to weight and shape concern as compared to other eating disorder symptoms (Mazzeo et al., 2010).

The present study demonstrated that shared environmental influences (i.e., environmental influences common to both twins within a family) may contribute to the etiology of the symptom that shape influences thoughts about oneself. However, this finding contrasts with previous research among community samples of adults, which found that only genetic and person-specific environmental influences contributed to the variance in concern about one's weight and shape (Mazzeo et al, 2010; Spanos et al., 2010). Thus, the present finding that shared environmental influences contributed to between-person variance in severity of an eating disorder symptom in adolescence is novel. Recent longitudinal research implicates shared environmental contributions in cognitive symptoms of eating disorders during childhood, which may decrease through adolescence and into adulthood (Waszczuk et al., 2019). Taken together, the present study suggests that environmental influences contribute to shape concern across childhood and adolescence, while eating disorder symptoms with a stronger genetic component are no longer influenced by environmental factors into adolescence. It is possible that as compared to cognitive symptoms of eating disorders with a higher genetic and lower environmental component, shared environment continues to influence liability to shape concerns into adolescence.

Phenotypic and Etiologic Structure of Cognitive Symptoms of Eating Disorders

Phenotypic correlations between the four cognitive symptoms of eating disorders were consistently high, which supports our a priori hypothesis that all four symptoms represent a higher order, latent factor, which I conceptualized as cognitive eating disorder symptom severity. In further support of this hypothesis, I found that the four cognitive symptoms of eating disorders shared common genetic and environmental etiological influences. Specifically, the correlations

among the genetic contributions to each of the four cognitive symptoms of eating disorder items were high while the correlations among the person-specific environmental contributions to each of the four cognitive eating disorder symptoms were moderate to high. Shared environmental factors did not contribute to between-person variance in most cognitive eating disorder symptoms. Hence, the correlations among shared environmental contributions to each of the four cognitive eating disorder symptoms were uninterpretable. Overall, these findings indicate that the strong phenotypic correlations among the four cognitive symptoms of eating disorder items were underpinned by common genetic and person-specific environmental influences, although genetic factors contributed particularly strongly to the commonality (i.e., phenotypic correlations) among items.

The strong phenotypic correlations among the four cognitive eating disorder symptoms, underpinned by common genetic and environmental influences, suggested that all four symptoms comprised a measure of the same latent construct. To confirm this hypothesis, I fit a latent factor model within the biometrical twin model such that all correlations among genetic and environmental contributions to all four cognitive eating disorder symptoms were accounted for by genetic and environmental influences to two latent factors. The first factor represented latent cognitive eating disorder symptom severity and accounted for most of the variance in cognitive eating disorder symptoms. A second factor, labelled the residual correlation of weight and shape concerns, accounted for the remaining, residual covariance between symptoms that assessed whether one's weight and shape influences thoughts about oneself. This model proved to be more parsimonious and no worse fit to a correlated model in which the genetic and environmental contributions to all four cognitive eating disorder symptoms were allowed to freely covary with one another. Hence, this theoretically-specified model of latent cognitive

symptoms of eating disorders was supported by the data including a residual latent correlation of the unduly influences of weight and shape concerns on thoughts about oneself.

The biometrical twin model further allowed me to decompose variance in both latent factors to determine the contribution of genetic and environmental influences to the etiology of latent cognitive symptoms of eating disorders and residual correlation of weight and shape concerns. Genetic and environmental contributions to latent factors represent genetic or environmental influences common to all items that load onto that factor. For example, genetic contributions to the latent factor of cognitive eating disorder symptom severity represents genetic contributions common to all four cognitive eating disorder symptoms. For this reason, I will describe the genetic and environmental contributions to latent factors as common genetic or common environmental influences to clarify that each reflect common contributions to multiple symptoms. Moreover, after accounting for common genetic and common environmental influences, there may be remaining genetic and environmental contributions to between-person variance in individual cognitive symptoms of eating disorders (e.g., feeling fat). This remaining genetic and environmental variance will be referred to as unique genetic and unique environmental influences, as it is the genetic and environmental influences unique to a specific cognitive eating disorder symptom.

Genetic and person-specific environmental influences accounted for 60% and 40% of the between-person variance in latent cognitive eating disorder symptom severity, respectively. This is in line with previous findings, which demonstrate that eating disorder cognitions, including weight preoccupation, drive for thinness, and body dissatisfaction are influenced by genetic (50% to 60%) and person-specific environmental (40% to 50%) factors among community samples of adolescents (Klump et al., 2000a; 200b, Waszczuk et al., 2019). The present study

used a particularly broad range of items, including feeling fat, fear of gaining weight, and weight and shape influences thoughts about oneself. Thus, our findings extend past research on body dissatisfaction, drive for thinness, and weight preoccupation among adolescents to show that a broader range of cognitive symptoms of eating disorders are influenced by both genetic and environmental factors in adolescence. After identifying that genetic contributions to cognitive symptoms of eating disorders are highly correlated, I parsed common genetic contributions to latent cognitive eating disorder symptom severity from residual, unique genetic contributions to individual cognitive eating disorder symptoms. Common genetic influences contributed significantly to cognitive eating disorder symptom severity. After accounting for common genetic influences, unique, residual genetic influences did not significantly contribute to any of the cognitive eating disorder symptoms. These findings indicated that genetic contributions broadly increased liability for cognitive eating disorder symptom severity and accounted for the continuity across cognitive symptoms. These findings extend previous etiological research on cognitive eating disorder symptoms among adolescents (Waszczuk et al., 2019), which demonstrates that genetic factors lead to a generalized vulnerability to eating disorder symptoms, including body dissatisfaction, drive for thinness and bulimic symptoms. Whereas past research has focused on select cognitive and behavioural symptoms of eating disorders, the present study demonstrates that genetic contributions broadly increase liability to a wide range of cognitive eating disorder symptoms in adolescence, which suggests that diversified cognitive eating disorder symptoms develop through common genetic risk.

Common person-specific environmental influences contributed significantly to latent cognitive eating disorder symptom severity. Specifically, common person-specific environmental influences accounted for approximately 39% of the between-person variance in latent cognitive

eating disorder symptom severity. This suggests that person-specific environmental factors also broadly influence liability to cognitive eating disorder symptoms. Unique person-specific environmental influences also contributed significantly to each cognitive eating disorder symptom, except for shape influences thoughts about oneself. Overall, these results suggest that while some person-specific environmental influences broadly increase liability to latent cognitive eating disorder symptom severity, others can also contribute to variability in the presentation of cognitive eating disorder symptoms. These findings are in line with previous studies, which suggest that while some person-specific environmental influences contribute to cross-symptom stability, others influence variability in the unique presentation of cognitive symptoms of eating disorders (Waszczuk et al., 2019). Common person-specific environmental influences also contributed 79% of between-person variance in the residual correlation of weight and shape influences thoughts about oneself, which suggests that there exists a set of environmental influences that increase liability to both weight and shape influences thoughts about oneself but that do not contribute to other cognitive eating disorder symptoms. These findings are consistent with Mazzeo et al. (2010), who demonstrated that the liability to weight and shape concern is more strongly influenced by environmental influences as compared to other eating disorder cognitions.

Similar to past research among community samples of adolescents (e.g., Klump et al., 2000a; Waszczuk et al., 2019) and adults (Rutherford, et al., 1993; Wade et al., 1998, 1999**),** latent shared environmental influences did not contribute significantly to cognitive symptoms of eating disorders. While shared environmental influences (i.e., environmental experiences shared between both twins in a family) may play a more prominent role in the development of cognitive symptoms of eating disorders among children, by adolescence, the influence of shared

environmental contributions may be replaced by more impactful or recent person-specific influences. Notably, adolescence is marked by a period in which less time is spent with the family and an increasing amount of time is spent with peers (e.g., Choukas-Bradley & Prinstein, 2014), which provides more opportunity for person-specific experiences and decreasing likelihood that twins will share common environmental influences that may contribute to psychological development or psychiatric distress.

Etiology of Anxiety Sensitivity and Anxiety Severity

Genetic influences contributed to 40% of the between-person variance in anxiety sensitivity; the remaining between-person variance was attributable to person-specific environmental influences. This is in line with previous research, which also showed that latent genetic influences account for approximately 40% of the variance in anxiety sensitivity while the remaining variance was attributable to person-specific environmental influences in community samples of adolescents including the large genetic epidemiologic sample used in the present study when assessed at a different age (Brown et al., 2012; Zavos et al., 2012).

Similarly, when assessed at age 16 years, latent genetic influences accounted for approximately 39% of between-person variance in anxiety symptom severity. This is also in accordance with past research that indicates the contribution of genetic influences to anxiety symptoms in community samples of adolescents (Eley et al., 1999; Chen et al., 2015; Nivard et al., 2015). In the present study, both person-specific and shared, familial environmental influences also contributed to between-person variance in anxiety symptom severity. The contribution of person-specific environmental influences is consistently demonstrated in prior research (e.g., Eley et al., 1999; Chen et al., 2015; Scaini et al., 2014; Zheng et al., 2016). Evidence in the present study that shared environmental influences also contribute to anxiety

symptom severity agrees with some prior research in community samples of adolescents (Eley et al., 1999; Chen et al., 2015; Scaini et al., 2014; Zheng et al., 2016), although findings have been mixed (Nivard et al., 2015; Sawyers et al., 2019). Factors such as the age of participants at assessment, the specific anxiety symptoms measured, and informant assessed (e.g., parents vs. adolescents) may contribute to inconsistent estimates in prior research. The influence of shared environmental contributions may also change over development. Shared environmental contributions may play a more significant role in anxiety in childhood as compared to adolescence and adulthood, such that the influence of shared environmental factors on anxiety symptoms may differ between younger as compared to older adolescents (Patterson et al., 2018). Discrete anxiety symptoms may also demonstrate distinct etiological influences. For example, consistent with the present findings, Patterson et al. (2018) reported that environmental, rather than genetic influences, contribute to variability in symptom expression. Regarding prior research on the genetic and environmental contributions to the etiology of anxiety symptoms, while some research focuses broadly on anxiety symptoms or related traits (e.g., trait anxiety), other research has examined more specific anxiety symptoms (e.g., social anxiety). Therefore, discrepant findings in prior research may be related to different measures used or different symptoms assessed.

Phenotypic and Etiologic Associations of Cognitive Eating Disorder Symptom Severity with Anxiety Sensitivity and Anxiety Severity

Phenotypically, cognitive eating disorder symptom severity was correlated with anxiety sensitivity and anxiety severity, which were correlated with one another as expected based on

theory and prior research. The latent factor that indexed the residual correlation of weight and shape concern was also phenotypically correlated with anxiety sensitivity, cognitive eating disorder symptom severity, and anxiety severity. These findings suggest that anxiety severity is broadly associated with elevated cognitive eating disorder symptoms and agree with existing research in community samples of adolescents, which demonstrate that symptoms of anxiety are concurrently (Johnson & Wardle, 2005; Trompeter et al., 2018; Touchette et al., 2011) and prospectively (Levinson & Rodebaugh, 2016; Schaumberg et al., 2019) associated with elevated cognitive symptoms of eating disorders among adolescents. While past research supports the present findings, most prior studies focused on female participants (e.g., Johnson & Wardle, 2005; Touchette et al., 2011) or assessed symptoms of social anxiety disorder (e.g., Levinson & Rodebaugh, 2016; Trompeter et al., 2018). The present findings build on prior literature to demonstrate that a broad range of anxiety symptoms are correlated with cognitive eating disorder symptom severity in a large genetic epidemiological sample of both male and female adolescents.

I also demonstrated that anxiety sensitivity was significantly correlated phenotypically with anxiety severity, which is supported by prior research that demonstrates a strong concurrent (Wright et al., 2016) and prospective (Allan et al., 2016; Ho et al., 2018; Qi et al., 2021; Waszczuk et al., 2013) association of anxiety sensitivity with anxiety symptoms in community samples of adolescents. As hypothesized, I also demonstrated that anxiety sensitivity may be phenotypically correlated with cognitive eating disorder symptom severity as well as with the residual correlation of weight and shape concern, which suggests that anxiety sensitivity is broadly associated with eating disorder psychopathology. These findings accord with previous research that also demonstrates an association of anxiety sensitivity with eating disorder

symptoms in both community (Fairweather-Schmidt & Wade., 2020) and clinical (Espel-Huynh et al., 2019) samples of adolescents. While anxiety sensitivity has been traditionally conceptualized as a risk factor for anxiety symptoms, the present findings contribute to a small but growing body of evidence that, among adolescents, anxiety sensitivity is associated with broad psychopathology in addition to anxiety disorders (Naragon-Gainey, 2010).

Genetic and person-specific environmental contributions to anxiety sensitivity were significantly correlated with the genetic and person-specific environmental contributions to both anxiety severity and cognitive eating disorder symptom severity. However, notably, I did not find evidence that the genetic or environmental contributions to anxiety severity were correlated with the genetic or environmental contributions to cognitive eating disorder symptom severity. This addresses our hypothesis that the genetic and environmental correlations of anxiety sensitivity with anxiety severity and cognitive eating disorder symptom severity are stronger than the genetic and environmental correlations of anxiety severity with cognitive eating disorder symptom severity. The present findings further suggest that anxiety sensitivity is etiologically similar to both cognitive eating disorder symptoms and anxiety severity. This suggests that anxiety sensitivity may contribute to genetic and environmental risk to both cognitive eating disorder symptoms and anxiety severity, which implicates anxiety sensitivity as a broad risk factor that underlies multiple psychopathological conditions. The present findings, therefore, implicate anxiety sensitivity as a contributing factor to the phenotypic correlation of anxiety severity with cognitive eating disorder symptom severity through its influence on the development of both anxiety severity and cognitive symptoms of eating disorders. The findings from the present study are in accordance with a growing body of research that implicates anxiety sensitivity as an underlying cognitive bias in the development of psychopathology (Qi et al.,

2021), including anxiety (Sexton et al., 2003; Schmidt et al., 2010; Waszczuk et al., 2013; Wauthia et al., 2019) and eating disorder symptoms (Anestis et al., 2007, 2008; Davey & Chapman, 2009; Fairweather et al., 2020). This also contrasts with previous findings from a community sample of adolescents (Silberg & Bulik., 2005), a clinical sample of female adolescents (Keel et al., 2005), and a clinical sample of adults (Kendler et al., 1995), which documented correlated genetic and person-specific contributions to anxiety and eating disorders (e.g., overanxious and separation anxiety disorder).

The present study differed from prior research on the common genetic and environmental contributions to anxiety and cognitive eating disorder symptoms in several important ways, which may have accounted for the low genetic and environmental correlations of anxiety symptoms with cognitive eating disorder symptom severity. First, I assessed cognitive eating disorder symptoms rather than eating disorder symptoms broadly defined (i.e., including disordered eating) or clinically-assessed eating disorders (e.g., anorexia nervosa). This assessment captures the association of those eating disorder symptoms theoretically implicated in anxiety sensitivity but does not assess possible genetic or environmental correlations of anxiety symptom severity with behavioural symptoms of eating disorders (e.g., binge eating, food restraint, etc.). Second, to enhance generalizability of the present study, I also assessed anxiety symptoms broadly rather than focusing on symptoms of a particular anxiety syndrome (e.g., social anxiety disorder). Given prior research on specific anxiety syndromes (e.g., Silberg & Bulik., 2005), it is possible that specific anxiety symptoms (e.g., separation anxiety disorder symptoms) share common genetic and environmental influences with symptoms of eating disorders not present for broad anxiety severity. The influence of anxiety syndrome studied may be especially relevant for the discrepancy in results pertaining to environmental contributions to

anxiety severity and cognitive eating disorder symptoms. Longitudinal and cross-symptom research demonstrates that while genetic factors contribute to stability of anxiety syndromes (Nivard et al., 2015; Patterson et al., 2018) and eating disorder symptoms (Klump et al., 2000a; Waszczuk et al., 2019), environmental influences tend to be symptom- and time-specific. Thus, findings related to common environmental influences across anxiety and eating disorder symptoms may be dependent on the specific symptoms being measured.

Beyond associations with cognitive eating disorder symptoms, the present results demonstrated significant correlations between the genetic and environmental contributions to anxiety sensitivity and anxiety severity, which suggests that the phenotypic correlation of anxiety sensitivity with anxiety symptoms may be underpinned by common genetic and person-specific environmental etiology. This evidence agrees with previous family and twin research that identified correlated genetic and person-specific environmental contributions to anxiety sensitivity and anxiety symptoms in community samples of children (Eley et al., 2007; Waszczuk et al., 2013; Zavos et al., 2012). The present study extends prior research to demonstrate the correlation between genetic and environmental contributions to anxiety sensitivity and anxiety severity in adolescence. Evidence that common genetic and environmental contributions influence both anxiety sensitivity and anxiety severity is particularly noteworthy given the strong genetic and person-specific environmental influences to anxiety sensitivity and anxiety severity (Zavos et al., 2012) and the consistently high phenotypic association of anxiety sensitivity with anxiety symptoms (e.g., Ho et al., 2018; Qi et al., 2021).

Similarly, the present results demonstrated that common genetic and person-specific environmental contributions influenced anxiety sensitivity and cognitive eating disorder symptom severity. These results agree with one recent study that identified correlated genetic

and environmental contributions to both anxiety sensitivity and eating disorder symptoms in a community sample of female adolescents (Fairweather-Schmidt & Wade., 2020). Fairweather-Schmidt and Wade (2020) examined the association of anxiety sensitivity with eating disorder symptoms in a sample of 699 female adolescents in Australia. Adolescents were between 16 to 18 years old, were predominantly Caucasian, and had a mean socioeconomic status that was slightly above the local mean. Eating disorder symptoms were assessed using the Eating Disorder Examination, which derives a total eating disorder symptom score from questions related to weight-, shape-, and eating- concern, and dietary restraint. Fairweather-Schmidt and Wade (2020) demonstrated a moderate phenotypic correlation between anxiety sensitivity and eating disorder symptoms. Further, they found a moderate genetic correlation, as well as a moderate person-specific environmental correlation, between anxiety sensitivity and eating disorder symptoms. These results contribute to a growing body of research supporting the association between anxiety sensitivity and eating disorder symptoms (Anestis et al., 2007, 2008) and provide preliminary evidence that supports the shared etiology of anxiety sensitivity and eating disorder symptoms. More specifically, these findings demonstrated that both common genetic and person-specific environmental influences contribute broadly to the liability of anxiety sensitivity and eating disorder symptoms.

The present study extends the findings of Fairweather-Schmidt and Wade (2020) in multiple important ways. First, whereas Fairweather and Wade (2020) demonstrated genetic and person-specific environmental correlations between anxiety sensitivity and eating disorder symptoms broadly (i.e., behavioural and cognitive symptoms of eating disorders), I demonstrated genetic and person-specific environmental correlations between anxiety sensitivity and cognitive symptoms of eating disorders, specifically. Behavioural and cognitive symptoms of eating

disorders represent distinct constructs which may differ in their etiology. For instance, cognitive symptoms of eating disorders often precede (Gonçalves & Gomes, 2012) and persist following the remission of (Heatherton et al., 1997) behavioural symptoms of eating disorders. Therefore, it is important to distinguish between the two when considering their etiology. The present study also focused on adolescents who were 16 years of age, rather than adolescents of a broader age range. Past research demonstrates that the etiology of eating disorder symptoms changes across childhood and adolescence (Klump et al., 2000a), thus it may be particularly important to use a narrow age range when assessing the etiology of cognitive symptoms of eating disorders during adolescence. The present study included 5,111 twin pairs from socioeconomic status backgrounds and ethnic backgrounds which comprise a sample representative of the general population in the U.K. at the time that the sample was recruited (see Haworth et al., 2013). Moreover, the present study included both male and female twin participants. This is an important contribution to the current literature, as most eating disorder symptom research is conducted in females, despite the high prevalence of eating disorder cognitions among males (Hoffman et al., 2018; McCabe & Ricciardelli, 2004). Taken together, the present findings extend the current literature by demonstrating that anxiety sensitivity shares common etiology specifically with cognitive symptoms of eating disorders. I found this to be true in a large, representative sample of 16-year-olds in the U.K. The prevalence of cognitive symptoms eating disorders is similar across Western societies, including the U.K. and Canada. Therefore, considering the large number of participants included in this study it is likely that these findings are generalizable to the Canadian population.

The present results demonstrate that genetic and person-specific environmental factors contribute to cognitive eating disorder symptom severity, anxiety sensitivity, and anxiety

severity. Moreover, the present results suggest that common genetic factors may underpin the phenotypic correlations of anxiety sensitivity with anxiety severity and cognitive eating disorder symptom severity. However, there was no evidence that the genetic contribution to anxiety severity was correlated with the genetic contribution to cognitive eating disorder symptom severity. These results support our hypothesis that the genetic contribution to the cognitive bias reflected in anxiety sensitivity is also associated with increased liability to both anxiety severity and cognitive eating disorder symptoms among adolescents in the general population. Likewise, the person-specific environmental contribution to anxiety sensitivity was correlated with the person-specific contributions to anxiety severity and cognitive eating disorder symptoms, which suggests that experiences that influence anxiety sensitivity also influence both anxiety severity and cognitive eating disorder symptoms. Evidence that anxiety sensitivity, but not anxiety severity, shared genetic and person-specific environmental influences with cognitive eating disorder symptom severity further supports our hypothesis that mutual associations with anxiety sensitivity may account for the comorbidity of anxiety symptom severity with cognitive eating disorder symptom severity.

These findings demonstrate that anxiety sensitivity shares common etiology with cognitive eating disorder symptom severity and anxiety severity, which suggests that anxiety sensitivity may function as an underlying cognitive bias that contributes to the development of cognitive eating disorder symptoms and anxiety severity among adolescents in the general population. Anxiety sensitivity is conceptualized as a fear of body-related cues associated with anxiety and theoretically, may be extended to include the fear of body-related cues associated with negative outcomes related to weight and shape concern (e.g., feeling fat). Anxiety sensitivity also increases one’s attention towards body related cues. For example, elevated

anxiety sensitivity is associated with body vigilance in non-clinical samples of adults (Rosa Esteve & Camacho, 2008). A heightened fear and awareness of body-related cues associated with weight, shape, and eating concern (e.g., stomach distension) may increase the frequency and intensity with which one negatively experiences their own body, leading to overall maladaptive thoughts related to their body, weight, and shape. There is some research to support this association. For example, internal body awareness is concurrently associated with elevated body dissatisfaction in community samples of women and men (Bekker et al., 2002). Thus, elevated anxiety sensitivity may increase one's risk to negatively evaluate weight and eating related cues and intensify a negative evaluation of their body, rendering them at greater risk of engaging in eating disorder cognitions and behaviours. However, further research is required to fully understand the mechanisms through which anxiety sensitivity influences cognitive eating disorder symptoms.

Finally, I found that both genetic and person-specific environmental influences contributed to anxiety sensitivity, cognitive eating disorder symptom severity, and anxiety severity. This suggests that while there are certain genes that increase one's vulnerability to anxiety sensitivity, cognitive eating disorder symptom severity, and anxiety severity, some person-specific environmental influences also play a role. Certain environmental risk factors have been implicated in both the development of anxiety sensitivity and eating disorder symptoms. For example, exposure to parental rejection during childhood is associated with elevated anxiety sensitivity (Scher & Stein, 2003) and eating disorder symptom severity (Vandewalle et al., 2017). Similarly, some specific genes may be implicated in the development of both anxiety sensitivity and eating disorder symptoms. For example, the short variant (s-allele) in the promotor of the serotonin transporter gene (5-HTTLRP) is less transcriptionally

efficient and is associated to increased reactivity to stress. This polymorphism in the serotonin transporter gene has been shown to interact with certain environmental stressors (e.g., parental rejection) to predict the development of anxiety sensitivity (Stein et al., 2008) and eating disorder symptoms (Akkermann et al., 2011). Future research is needed to further identify common genetic and environmental influences to anxiety sensitivity and eating disorder symptom severity. Future research is also needed to determine whether genetic and environmental influences common to both anxiety sensitivity and eating disorder symptoms may be associated with psychopathology because of a mediated influence via anxiety sensitivity.

Limitations

Findings in the present study should be considered in light of several limitations. First, anxiety severity was measured using parent report, while anxiety sensitivity and cognitive eating disorder symptoms were both measured using adolescent self-report. Parent-reported anxiety symptoms are modestly correlated with adolescent self-reported anxiety symptoms (Muris et al., 2003). For this reason, parent-reported anxiety severity may partially explain the relatively weak phenotypic correlations of anxiety severity with anxiety sensitivity or cognitive eating disorder symptom severity. However, prior research documents the validity of the Anxiety Related Behaviour Questionnaire as a parent-report assessment of child anxiety severity (Eley et al., 2003; Hallett et al., 2009). Thus, the present results may be considered a lower bound estimate of the phenotypic correlation of anxiety severity with anxiety sensitivity or cognitive eating disorder symptom severity. Parent report would not necessarily influence correlations of genetic and environmental contributions to anxiety severity with genetic and environmental contributions to anxiety sensitivity or cognitive eating disorder symptom severity. Within the biometrical family and twin models, correlated genetic and environmental contributions are

estimated by comparing the correlation of two phenotypes (e.g., anxiety severity and anxiety sensitivity) between monozygotic and dizygotic twins. The assessment method (e.g., parent-report) may impact one's score but would not necessarily lead to higher concordance between monozygotic, as compared to dizygotic, twins. Therefore, it would not necessarily alter the estimated genetic or environmental contributions to each phenotype. Similarly, while the assessment method may impact the correlation between two given phenotypes (e.g., anxiety severity and anxiety sensitivity), it would not necessarily lead to a lower correlation between the two phenotypes in dizygotic twins as compared to monozygotic twins. Therefore, the assessment method may impact the phenotypic correlation between two phenotypes but not necessarily the correlation between their genetic contributions. For example, the present study documents statistically significant correlations of genetic and environmental contributions to anxiety sensitivity and anxiety severity despite assessment of anxiety sensitivity via adolescent self-report and assessment of anxiety severity via parent-report.

Second, I did not examine possible qualitative and quantitative effects of biological sex on the etiology of cognitive eating disorder symptom severity. Rather, I included sex as a covariate in analyses to account for the higher severity of cognitive eating disorder symptoms among female participants. While eating disorder symptoms may be somewhat more prevalent or severe among female participants, symptom etiology, course, and outcomes do not necessarily differ as a function of participant biological sex or gender. Past family and twin biometrical studies demonstrate that the etiology of eating disorder symptoms do not differ between male and female adolescents (Baker et al., 2009), or young adults (Reichborn-Kjennerud et al., 2003). For example, Baker et al. (2009) demonstrated that among adolescents between the ages of 15 to

17 years, genetic influences that contributed to drive for thinness and body dissatisfaction are comparable between males and females.

Third, this study was conducted using cross-sectional twin data, such that anxiety sensitivity, cognitive eating disorders symptoms, and anxiety severity were each measured when twins were 16 years old. Thus, while our findings may suggest that anxiety sensitivity functions as an underlying cognitive bias to increase liability to cognitive eating disorder symptom severity and anxiety severity, longitudinal research is needed to further test this theoretical model. For example, it is possible that cognitive eating disorder symptoms precede anxiety sensitivity, that the two constructs develop concurrently, or that they share a bidirectional relationship in which anxiety sensitivity and cognitive eating disorder symptoms are influenced by one another. However, current research implicates anxiety sensitivity as an underlying cognitive bias which contributes to the development of symptoms of anxiety, and more broadly, psychopathology (Naragon-Gainey, 2010; Noël & Francis, 2011; Qi et al., 2021). For instance, in support of its role as a developmentally significant underlying cognitive bias, anxiety sensitivity is broadly associated with symptoms of psychopathology (Noël & Francis, 2011).

Conclusion

In conclusion, the present study demonstrates that cognitive eating disorder symptoms represent a range of eating and weight-related cognitions in adolescence, which comprise a latent factor of eating disorder symptom severity. I also found strong correlations among the genetic contributions, and moderate correlations among the person-specific environmental contributions, to all four cognitive eating disorder symptoms, which is consistent with a latent factor through which common genetic and environmental contributions influence multiple symptoms. When fit as a latent factor, common genetic influences accounted for the majority of between-person

variance in a latent variable that indexed cognitive eating disorder symptom severity, which suggests that genetic influences contribute to commonality across cognitive eating disorder symptoms.

Both anxiety sensitivity and cognitive eating disorder symptom severity were influenced by genetic and person-specific environmental influences, though anxiety severity was influenced by genetic and person-specific and shared environmental influences. Phenotypically, anxiety sensitivity was correlated with elevated cognitive eating disorder symptom severity and anxiety severity, which was also correlated with elevated cognitive eating disorder symptom severity. Genetic influences that contributed to anxiety sensitivity were correlated with genetic influences that contributed to cognitive eating disorder symptom severity and anxiety severity. Likewise, person-specific environmental influences that contributed to anxiety sensitivity were correlated with those that contributed to cognitive eating disorder symptom severity and anxiety severity. However, contrary to study hypotheses, genetic and person-specific environmental contributions to anxiety severity were not correlated with genetic or person-specific contributions to latent cognitive eating disorder symptom severity.

Together, these findings indicate that, as compared to anxiety severity, the genetic and person-specific influences that contribute to anxiety sensitivity are more strongly correlated with the genetic and person-specific environmental influences that contribute to latent cognitive eating disorder symptom severity. These findings support our overarching theory that anxiety sensitivity contributes both genetic and environmental risk to anxiety and cognitive eating disorder symptoms. Further, the findings indicate that anxiety sensitivity may function as an underlying trait to increase liability to both cognitive eating disorder symptoms and anxiety

severity, which may partially contribute to the high comorbidity between anxiety severity and eating disorder symptoms in the general population.

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