Social anxiety in the eating disorders: A systematic review and meta-analysis

Jess Kerr-Gaffney[1] *, Amy Harrison[2], Kate Tchanturia[1, 3, 4]

[1]King's College London, Institute of Psychiatry, Psychology and Neuroscience, Psychological Medicine, London, UK

[2]University College London, Department of Psychology and Human Development, London, UK

[3]South London and Maudsley NHS Trust, National Eating Disorders Service, Psychological Medicine Clinical Academic Group, UK

[4]Ilia State University, Tbilisi, Georgia

*Address for correspondence: Jess Kerr-Gaffney, Section of Eating Disorders, 103 Denmark Hill, King's College London, London, SE5 8AZ. (Email: jess.kerr-gaffney@kcl.ac.uk)

Abstract

Social anxiety disorder (SAD) is one of the most common comorbid conditions in eating disorders (EDs). The aim of the current review and meta-analysis is to provide a qualitative summary of what is known about social anxiety (SA) in EDs, as well as to compare levels of SA in those with EDs and healthy controls (HCs). Electronic databases were systematically searched for studies using self-report measures of SA in ED populations. In total, 38 studies were identified, 12 of which were included in the meta-analyses. For both anorexia nervosa (AN) and bulimia nervosa (BN), there were significant differences between ED groups and HCs, with medium to large effect sizes. Findings from the qualitative review indicate that levels of SA are similar across the ED diagnoses, and SA improves with treatment in AN. In addition, high levels of SA are associated with more severe ED psychopathology, but not BMI. These findings add to the wider literature on socio-emotional functioning in EDs, and may have implications for treatment strategies.

Introduction

Eating disorders (EDs) are associated with high levels of psychiatric comorbidity (Blinder *et al.* 2006); a factor linked to poorer short- and long-term outcomes (Berkman *et al.* 2007; Vall & Wade, 2015). Anxiety disorders are common, with a lifetime prevalence of around 60% reported in both anorexia nervosa (AN) and bulimia nervosa (BN) (Bulik *et al.* 1997). In particular, social anxiety disorder (SAD; also known as social phobia) is consistently found to be the first or second most common comorbid anxiety disorder in EDs (Godart *et al.* 2000; Kaye *et al*. 2004; Swinbourne *et al*. 2012), with prevalence rates ranging from 16% to 88.2% in AN and 17% to 67.8% in BN (Swinbourne & Touyz, 2007). In comparison, lifetime prevalence of SAD in the general population is around 12% (Kessler *et al.* 2005). The association between SAD and EDs is also replicated in non-clinical populations, where disordered eating is positively associated with social anxiety (SA) levels (Gilbert & Meyer, 2003; Gadalla & Piran, 2008; Utschig *et al.* 2010; Ciarma & Mathew, 2017). High levels of SA in EDs may be part of a wider socio-emotional phenotype hypothesised to contribute to the development and maintenance of EDs (Treasure & Schmidt, 2013). For example, people with AN report having impoverished social networks and internalising problems in childhood, problems which are further accentuated by the ill state (Adambegan *et al.* 2012; Harrison *et al.* 2014; Westwood *et al.* 2016). Similarly, adolescents and young adults with ED show more insecure attachment styles (Dias *et al.* 2011), which are theorised to have lasting implications on emotion regulation, social processing, and self-evaluative processes (Gander *et al.* 2015).

Explanations for the link between EDs and SA have been proposed. Firstly, SA may be a risk factor for the development of an ED. For example, anxiety around how oneself appears to others may lead to an excessive interest in body weight and shape (Godart *et al.* 2000). Another possibility is that SA may be secondary to the ED, as a consequence of ED psychopathology or malnutrition. Studies examining the temporal relations between the two disorders lend some support to the former hypothesis, where it is consistently reported that SAD onset preceded the ED in the majority of those with both disorders (Bulik *et al.* 1997; Godart *et al.* 2000; Kaye *et al.* 2004; Swinbourne *et al.* 2012). However, such studies rely on retrospective accounts of age of onset, and are therefore subject to recall biases. Two prospective studies using representative samples provide conflicting results, and suggest that the relationship with SAD may differ as a function of ED diagnosis. Buckner *et al.* (2010) found that BN in adolescence significantly increased the risk of both SAD and panic disorder

in adulthood, however no anxiety or depressive disorder in adolescence predicted later BN. AN in adolescence did not increase the risk of any anxiety disorder or depression in adulthood, but adolescent obsessive compulsive disorder (OCD) predicted the development of AN in adulthood. In contrast, Ranta *et al.* (2017) found that both SAD and depression at age 15 predicted BN at age 17, however the relationship between SAD and BN was not significant after controlling for depression. Contrary to the previous study, neither AN or BN predicted later SAD, however this may be due to the far shorter follow-up period. Thus, evidence regarding the direction of causality is inconsistent.

A final explanation for the comorbidity is that SAD may share common vulnerability factors with ED, as has been found to be the case with OCD. In addition to AN being more common in probands of individuals with OCD (an effect that increases with degree of genetic relatedness), moderate genetic overlap between the two disorders has been reported in a large population-based twin sample (Cederlöf *et al.* 2015). Similarly, SAD occurs at significantly higher rates in first-degree relatives of probands with AN than those of healthy controls (HCs) (Strober *et al.* 2007). It may be that heritable vulnerability factors such as perfectionism partly explain the genetic overlap between anxiety disorders and EDs. For example, perfectionism, a trait that is elevated in both individuals with EDs and those with SAD (Antony *et al.* 1998; Lloyd *et al.* 2014) has been found to predict both SA and disordered eating in non-clinical women (Levinson & Rodebaugh, 2016). Perfectionism has also been shown to moderate the relationship between SA and bulimic symptoms specifically, where those with high SA and perfectionism showed the most bulimic symptoms (Silgado *et al.* 2010). Due to a lack of research on common vulnerability factors in clinical ED and SAD populations, no firm conclusions for the high levels of comorbidity can be drawn.

To date, only one review has examined comorbidity between EDs and SAD, within a general review of anxiety disorder comorbidity in EDs (Swinbourne & Touyz, 2007). However, this review (a) only provided categorical prevalence estimates of anxiety disorders in ED populations, and (b) did not examine whether SA differs across ED sub-types. Further, new studies have become available. Therefore, the aim of the current review and meta-analysis is to compare SA in EDs compared to HCs, and provide a qualitative synthesis of the literature, e.g., differences in SA between ED sub-types, the effects of treatment on SA, and associations between SA and factors such as body mass index (BMI) and ED psychopathology.

Method

The review and meta-analysis was conducted using the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement (Liberati *et al.* 2009).

Eligibility criteria

Studies using a quantitative measure of SA were included in the review. Inclusion criteria were: 1) at least one clinical ED sample; 2) means and standard deviations reported; 3) full article available in English; 4) published in a peer-reviewed journal. Studies measuring only related constructs (e.g., "secondary social phobia", "social appearance anxiety") were not included.

Information sources and search

The electronic databases PubMed, PsycInfo, SCOPUS, and Web of Science were searched independently by JKG and AH for papers up to February 2018. Search terms included social anxiety OR social phobia AND anorexia nervosa OR bulimia nervosa OR eating disorder OR binge eating disorder. No search limits were applied, except for in Web of Science, where results were filtered by the ED term for relevance.

Study selection

Screening and selection of articles is displayed in Figure 1. Where titles of papers appeared relevant, abstracts were screened to check eligibility. Full texts of potentially eligible studies were then retrieved. Studies that met all eligibility criteria but did not include a HC group were included in the qualitative review, whereas those that included a HC group were included in both the meta-analysis and the qualitative review. Where a study did not report means and standard deviations for SA scores, study authors were contacted. When no response was received, studies were excluded.

Data collection

The following information was extracted from each paper: number of participants in each group, diagnosis, mean SA score, SA measure used, age, BMI, illness duration, percentage of female participants, group matching technique, and recruitment source.

Risk of bias in individual studies

Risk of bias in individual studies was assessed by considering how certain methodological characteristics (participant recruitment source, group matching technique, and SA measure used) might have impacted the results of the studies.

Summary measure & data synthesis

The principle summary measure used in the meta-analysis was the difference in means and standard deviations for SA scores between ED and HCs. The meta-analyses were performed by pooling standardised effect sizes using a random effects model. Separate meta-analyses were performed for each ED sub-type, and studies that included more than one ED group (e.g., AN and BN) compared to HCs were included in each of the respective meta-analyses.

Statistical analysis

All analyses were performed using RStudio (R Core Team, 2017) using the metafor package (Viechtbauer, 2010). Cohen's d was used to estimate effect sizes and is reported with 95% confidence intervals (CIs). Effect sizes are interpreted using Cohen's (1988) definitions of small (0.2), medium (0.5), and large (0.8). Positive effect sizes indicate that the ED group had SA scores than HCs. Two AN studies included in the meta-analysis shared the same HC group, therefore a multivariate meta-analysis was conducted using the rma.mv command. Between study heterogeneity was calculated using Cochran's Q test. Where heterogeneity was found ($p < .05$), meta-regressions were performed using age and SA measure as moderators. BMI could not be used as a moderator due to missing data.

Risk of bias across studies

The presence of publication bias was assessed through visual inspection of funnel plots, where the absence of studies in the bottom right corner indicates publication bias. Symmetry of the funnel plots were formally assessed using Begg's rank correlation test (Begg & Mazumdar, 1994). Publication bias was also assessed using Rosenthal's fail-safe N (Rosenthal, 1979), which estimates the number of unpublished studies required to change the significant effect size into a non-significant one.

Results

Study selection

Thirty-eight studies were included in the review (Table 1). Studies that used the same sample (Duclos *et al.* 2014 and Courty *et al.* 2015; Hinrichsen *et al.* 2004a, 2004b, and 2007a) are combined and the data considered together. Fourteen studies included a HC group, however two could not be included in a meta-analysis and are discussed in the qualitative review. Of the 12 studies that could be included, ten included an AN group and five included a BN group. One of the BN studies reported scores for males and females separately (Gross *et al.*

1988), however males could not be included in the meta-analysis due to too few cases. Only one of the studies included in the meta-analysis provided mean SA scores for the different AN sub-types, therefore meta-analyses by sub-type could not be performed.

Study characteristics

Overall, reporting of study characteristics varied considerably. All studies provided information on the SA measure used, and in total, 12 different self-report questionnaires were used to measure SA. The most frequently used (n = 11) was the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987).

Studies included in the meta-analysis and qualitative review

Two of the 12 studies did not report the mean age of participants. Nine studies did not report the mean BMI or percentage of Ideal Body Weight (IBW) in at least one participant group, and 10 studies did not report mean illness duration of the ED group. Most studies only included female participants, however two studies included a small proportion of males in their ED group, and six included males in their HC group. Two studies did not report participants' gender in at least one group. ED groups were most often inpatients (n = 6). Three studies did not report the recruitment source for their control group, and one did not report this information for the ED group. Groups were matched on some characteristic in eight of the studies, most often sex.

Studies included in the qualitative review

All 26 studies reported the mean age of participants. Four studies did not report mean BMI or percentage IBW in at least one participant group, and half did not report mean illness duration. Again, most studies included exclusively female participants, however six studies included males. One study did not report participants' gender. ED groups were most often recruited from inpatient services (n = 8), but specialist ED or psychiatric services where it was ambiguous as to whether patients were outpatients or inpatients were also common (n = 7). In studies where SA was compared between groups (n = 13), groups were most often matched by sex (n = 8), however five studies did not report a group matching technique.

Synthesis of results & risk of bias

The random-effects model with a total sample size of 1859 participants (AN = 281, HC = 1578) revealed that those with AN had significantly higher levels of SA than HCs, with a large effect size (d = 1.65, [95% CI 1.03, 2.27] z = 5.20, p <.001) (Figure 2). The funnel plot

for the AN studies is displayed in Figure 3. There was no evidence of publication bias (Begg's test p = .216, Rosenthal's fail-safe N = 1033).

The random-effects model with a total sample size of 1031 participants (BN = 232, HC = 799) showed that those with BN had significantly higher levels of SA than HCs, with a medium effect size (d = 0.71, [95% CI 0.47, 0.95] z = 5.74, p <.001) (Figure 4). The funnel plot for the BN studies is displayed in Figure 5. There was no evidence of publication bias (Begg's test p = .817, Rosenthal's fail-safe N = 112).

Additional Analyses

There was evidence of significant heterogeneity in the AN studies Q(9) = 131.14, p <.001, therefore meta-regressions with age and SA measure as moderator variables were performed. BMI and illness duration could not be included as moderators due to studies not reporting this information. The moderators explained a significant amount of the variance, QM(4) = 32.56, p < .001. Age had a significant influence on the size of the effect (b = 0.12, [95% CI 0.03, 0.21], z = 2.53, p = .01), as did using the LSAS as a measure of SA (b = 1.64, [95% CI 0.78, 2.50], z = 3.73, p < .001). The test for residual heterogeneity was not significant, QE(4) = 7.96, p = .09. There was no evidence of heterogeneity in the BN studies Q(3) = 0.67, p = .87.

Qualitative Review

Differences between ED and HC

A few studies comparing ED groups to HCs could not be included in the meta-analysis due to there being too few comparisons. Otrovsky *et al.* (2013) examined SA scores in individuals with BED compared to overweight controls, finding that those with BED had significantly higher SA scores than controls. The second study examined SA scores in a mixed ED group compared to controls, finding that the ED group had significantly higher SA scores than HCs (Goddard & Treasure, 2013). This study also compared SA scores of parents of daughters with EDs to parents of HCs. Parents of daughters with EDs had higher SA scores than control parents, however effect sizes were small and not significant.

Differences between ED diagnoses

Of the seven studies that assessed differences between AN and BN, six found no difference in SA between groups (Bulik *et al.* 1991; Flament *et al.* 2001; Solano *et al.* 2005; Gilboa-Schechtman *et al*. 2006; Grabhorn *et al.* 2006; Obeid *et al.* 2013). The single study that

reported differences between ED groups found that individuals with AN-BP had significantly higher SA scores than AN-R and BN (Hinrichsen *et al.* 2003). Of the five studies that assessed differences between AN-R and AN-BP, four found no differences in SA across AN subtypes (Mattar *et al.* 2012a; Obeid *et al*. 2013; Duclos *et al.* 2014 and Courty *et al*. 2015; Abbate-Daga *et al.* 2015). As before, the single study that did find a difference was Hinrichsen *et al.* (2003).

One study examined whether patients that met eating disorder not otherwise specified (EDNOS) criteria in the DSM-IV but BN criteria in the DSM-5 differed from patients who met DSM-IV criteria for BN (MacDonald *et al.* 2014). SA scores did not differ between groups. Finally, a study by Schwalberg *et al.* (1992) examined differences between BN and BED, finding that the groups did not differ in SA scores. Overall, it seems that SA is similarly elevated across ED diagnostic groups.

Treatment effects and studies with recovered patients

Six studies examined change in SA over treatment, two of which involved adolescent patients with AN admitted to inpatient care. Mattar *et al.* (2012b) assessed 24 patients at admission and discharge (mean time in treatment = 3.2 months), in which time mean SA scores significantly improved. Neither intensity of weight loss or BMI at admission, discharge, or improvements in BMI during treatment were correlated with SA scores. The second study (Courty *et al*. 2015) assessed 60 patients in the second half of their inpatient admission (21 weeks on average). Patients were assessed at 6, 12, and 18 months. SA scores significantly decreased across time, with the largest reduction occurring between inclusion and 6-month follow-up. Further, SA levels were related to alexithymia across time, even after adjusting for depression, anxiety, and BMI.

The third study in adolescents with AN followed 29 outpatients receiving group cognitive behavioural therapy (G-CBT), assessing psychiatric, social, and emotional variables before, during (3 and 6 months), at the end of (9 months), and one year after completing treatment (Ohmann *et al.* 2013). Patients were split into groups based on outcomes. It was found that SA significantly improved by 9 months in patients with a good outcome (defined as attaining 25th BMI percentile and normal eating patterns), however SA did not improve in those with a poor or intermediate outcomes. Different from the aforementioned studies, the fourth treatment study (Abbate-Daga *et al.* 2015) examined 56 adult women with AN attending a day hospital service. The programme took a multidisciplinary approach with a focus on

psychodynamic psychotherapy, and patients were assessed at baseline, end of treatment (EOT; 6 months), and at follow up, 12 months after EOT. Significant reductions in SA scores were seen at EOT and follow-up. Unlike the inpatient studies, neither of these studies examined whether decreases in SA were due to improvements in BMI.

The final treatment study in AN was a randomised placebo-controlled trial examining the effects of intranasal oxytocin in 33 inpatients with AN (Russell *et al.* 2018). Contrary to predictions, there were no significant treatment, time, or treatment by time effects on SA scores, however EDE eating concern scores and cognitive rigidity were improved in the oxytocin group compared to placebo. The finding that SA scores did not improve over treatment in either group is at odds with the results of the former studies, however this might be due to the shorter follow up period (4-6 weeks). The final treatment study involved 29 young adult women with BED, who were randomly assigned to a cognitive-behavioural intervention ("Appetite Awareness Training") or a wait-list control group for 8 weeks (Allen & Craighead, 1999). It was found that SA scores reduced significantly in the intervention group compared to the control group. The intervention group also saw significant improvements in various measures of binge eating.

Finally, one study examined differences in SA scores between women with acute AN, women recovered from AN, and HCs (Schmelkin *et al.* 2017). Women recovered from AN scored significantly higher than HCs, but significantly lower than acute AN on the social fear, public fear, and social avoidance sub-scales of the LSAS. However, on the public avoidance sub-scale, those with AN scored higher than HCs and recovered AN, who did not differ from one another. Thus, while it seems that SA significantly improves with treatment in AN, those recovered from the disorder still experience high levels of SA compared to HCs. It also appears that improvements in SA in AN are not related to a specific treatment modality, although further studies with control groups are required to confirm this finding.

Associations with BMI

Six studies examined whether SA was associated with BMI and other clinical indicators of ED severity. Two of these studies involved AN patients only, both finding that current BMI was not associated with SA scores in inpatient women (Matter *et al.* 2012a; 2012b). In addition, Mattar *et al*. (2012a) found that SA scores were negatively correlated with blood albumin levels (an indicator of nutritional status), and positively correlated with age and duration of illness. In a sample of women with AN or BN, Bulik *et al.* (1991) found no

difference in SA scores when patients were split into underweight and normal weight groups. Similarly, in a mixed ED group (AN, BN, and EDNOS), SA was positively correlated with duration of illness and number of previous hospital admissions, but not current BMI (Goddard & Treasure, 2013). Further, those who were currently on medication had significantly higher SA than those not taking medication. Finally, in individuals with BED, higher SA was reported in those who became overweight as children, but was not correlated with current BMI (Sawaoka *et al.* 2012; Otrovsky *et al.* 2013). Thus, it seems that across the ED spectrum, SA is not related to BMI. Despite the lack of studies in this area, this finding suggests that high SA in those with EDs is not a result of malnutrition. Instead, those with a more severe illness may have higher levels of SA, as evidenced by the associations with longer duration of illness, more hospital admissions and medication use.

Associations with psychopathology

Consistent with the hypothesis that SA may be associated with a more severe illness, several studies have found positive associations between ED psychopathology and SA across the ED spectrum. In the same studies that found no association between SA and BMI in BED, significant positive relationships were found between SA and self-consciousness, depressive symptoms, Eating Disorder Examination (EDE) scores, weight, shape, and eating concerns, and binge eating severity (Sawaoka *et al.* 2012; Otrovsky *et al.* 2013). Similarly, in a mixed ED sample (AN, BN, or EDNOS), SA was significantly positively correlated with all eight Eating Disorder Inventory (EDI) subscales (drive for thinness, bulimia, body dissatisfaction, ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness, and maturity fears), as well as core beliefs about abandonment and emotional inhibition (Hinrichsen *et al.* 2004a; 2004b; 2007a). This latter finding was partially replicated in a larger sample by Hinrichsen *et al.* (2007b), who found that core beliefs regarding abandonment and defectiveness/shame (the belief that one is fundamentally flawed) explained almost a quarter of the variance in SA. Further, another study using the EDI on a sample of children and adolescents with AN found SA scores were positively associated with ineffectiveness and interpersonal distrust subscales, as well as trait anxiety (Schulze *et al.* 2009).

Two studies examined SA and psychopathological variables in adolescent mixed ED samples (AN, BN, and EDNOS). Buchholz *et al.* (2007) demonstrated that SA was a unique predictor of body dissatisfaction, and was also significantly positively associated with “self-silencing” (keeping negative thoughts or feelings to oneself), while Obeid *et al.* (2013) found a negative association between SA and self-esteem. Finally, one study found different results based on

ED diagnosis. Hinrichsen *et al.* (2003) examined emotion coping strategies in women with EDs, reporting that while SA was associated with greater dissociation among those with AN-R, this was not the case for those with AN-BP, BN, or HCs. Instead, higher levels of SA in BN and HC women were associated with higher bulimic psychopathology. Thus, it can be seen that SA in EDs is associated with not only more severe ED symptoms, but also beliefs and behaviours regarding self-esteem and emotion regulation.

Associations with other comorbid symptoms

Two studies assessed differences in SA between those with and without comorbid symptoms. Solano *et al.* (2005) examined differences between women with AN and BN who did and did not engage in self-injurious behaviour (SIB). Interestingly, while there was no effect of diagnosis on SIB, it was found that those who self-injured had significantly higher ED psychopathology, SA scores, and body image disturbance than those who did not. The second study examined differences in SA in women with BN, who either did or did not have a comorbid compulsive buying (CB) disorder (Jiménez-Murcia *et al.* 2015). CB is not currently recognised by international diagnostic classifications, but shares similarities with other impulse-related disorders such as BN and gambling disorder. It was found that SA scores were higher in women with both BN and CB, compared to those with BN only. However, after adjusting for age, this difference was no longer significant. Thus, although the results of Solano *et al*. (2005) suggest that high SA in EDs may be associated with more severe illness features such as self-harm, this might not be the case for addictive-type disorders. More research is required to clarify the possible transdiagnostic nature of SA in EDs and other disorders.

Discussion

The aim of this review was to examine group differences in SA in EDs compared to HCs, and provide a qualitative synthesis of the literature. There were significant differences in SA scores between both AN (10 studies) and BN (5 studies) compared to HCs, with large and medium effect sizes respectively, indicating that those with AN or BN have significantly higher levels of SA than HCs. Only one study compared levels of SA in BED compared to HCs, finding that SA was also significantly elevated in individuals with BED compared to HCs. While there was no evidence of publication bias in either meta-analysis, there was significant heterogeneity across AN studies. Meta-regressions with age and SA measure as moderator variables revealed that these variables explained a significant amount of the

heterogeneity, such that use of the LSAS and older age of participants was associated with larger effect sizes. The association between the SA measure used by studies (namely the LSAS) and larger effect sizes has important implications for both research in this area and clinical practice. Self-report measures of SA have several advantages: they are quicker and easier to administer; and can provide an estimate of SA in those who do not meet full diagnostic criteria for SAD. However, the results from this meta-analysis suggest that SA in EDs may be over- or under-estimated, depending on the measure used. While cut-off scores on self-report measures have been established in groups with a diagnosis of SAD, there are outstanding questions regarding what can be considered a clinically significant level of SA in individuals with EDs. Identifying those with high SA may be useful when deciding on the type of treatment offered to patients. Further, while some scales measure SA unidimensionally, others provide sub-scores for different aspects of SA, such as fear, avoidance, and physiological arousal. Which of these types of measure would be most useful in ED populations is another question for future research. Physiological arousal as it relates to SA in EDs may be a particularly interesting domain to explore, given the reduced sensitivity to interoceptive signals reported in AN (Pollatos *et al.* 2008).

The association between age and higher SA scores was also found in a few AN studies not included in the meta-analysis (Zonnevylle-Bender *et al.* 2004; Mattar *et al.* 2012a). One explanation for this finding is that those with a longer illness duration may experience higher levels of SA, in agreement with the results of Goddard and Treasure (2013). This may indicate a more severe illness, as suggested by the positive association between SA and ED psychopathology (Hinrichsen *et al.* 2003; 2004b; Schulz *et al.* 2009; Sawaoka *et al*. 2012; Otrovsky *et al*. 2013). This finding has important implications for understanding the etiological link between EDs and SA. From a developmental perspective, it has been postulated that there is a social phenotype for those at risk of developing an ED, characterised by loneliness, shyness, internalising problems, inferiority and low social support in childhood (Fairburn *et al.* 1999; Krug *et al.* 2013; Treasure & Schmidt, 2013). Indeed, SAD mostly occurs before ED onset in individuals diagnosed with both disorders (Bulik *et al.* 1997; Godart *et al.* 2000; Kaye *et al.* 2004; Swinbourne *et al.* 2012), and may be exacerbated by the ill state. The finding that levels of SA in individuals recovered from AN lie between that of HCs and acutely ill individuals lends further support for this hypothesis (Shmelkin *et al.* 2017).

Importantly, the lack of any association between BMI and SA indicates that it is not the degree of malnutrition that exacerbates SA, but some other factor associated with the illness. One possible explanation concerns emotional avoidance. Many have theorised that ED psychopathology (for example, a focus on food and weight, restrictive behaviours, and binge eating) helps individuals avoid having to experience negative emotions and challenging interpersonal situations (Slade, 1982). It has been demonstrated that comorbid depressive and anxiety symptoms are associated with higher ED psychopathology in AN, and this relationship is almost fully mediated by emotional avoidance (Wildes, Ringham, & Marcus, 2010). Therefore, it could be the case that those with higher social anxiety avoid situations that may elicit high emotion through an intense focus on food and weight, therefore reinforcing and maintaining the disorder. Further studies examining the relationship between SA (rather than general anxiety symptoms) and emotional avoidance are required to test this hypothesis.

Clinical implications

Findings from the current review contribute to the broader literature on socio-emotional functioning in EDs, which have demonstrated problems in areas such as theory of mind (Bora & Kose, 2016), emotion expression and recognition (Caglar-Nazali *et al.* 2014; Davies *et al.* 2016), social anhedonia (Harrison *et al*. 2014), and alexithymia (Westwood *et al.* 2017a). Further, they contribute to a growing evidence base documenting high levels of autism spectrum disorder (ASD) traits in those with AN (Westwood *et al.* 2017b). Like those with EDs, individuals with ASD show high levels of SA, with over half of adolescents meeting clinical cut-offs on self-report measures (Kuusikko *et al.* 2008). Because of the high degree of symptom overlap between SAD and ASD, it is possible that the high levels of SA displayed in EDs are linked to ASD traits. While it may not be possible to delineate the unique contributions of SAD and ASD traits to the social difficulties seen in AN and other EDs, these difficulties may be useful targets for treatment. For example, there is evidence that social skills training in adolescents with EDs may improve self-esteem and social withdrawal (Lázaro *et al.* 2011). In addition, preliminary evidence suggests that Cognitive Remediation and Emotion Skills Training (CREST), an intervention designed to target emotion processing, decreases social anhedonia and alexithymia in adults with AN (Tchanturia *et al.* 2015). Future research into the influence of SA on outcomes and prognosis is warranted.

Limitations

Several limitations of this review should be noted. Firstly, a considerable number of the studies included did not report important participant characteristics, such as BMI. Therefore, BMI could not be entered as a moderator variable in the meta-analysis. A further limitation is that none of the studies included in this review examined SA in EDNOS or OSFED ("not otherwise specified" categories in the DSM-IV and DSM-5 respectively) compared to HCs. Considering such diagnoses make up a significant proportion of those with EDs (Allen *et al.* 2013; Fairweather-Schmidt & Wade, 2014), establishing whether these patients show similar social difficulties will have important implications for their treatment. Finally, the number of studies that could be included in the meta-analyses (especially for BN studies) was relatively few, since the majority of studies did not include a HC group.

Conclusions

Both AN and BN are characterised by high levels of SA, even in those who do not have a formal diagnosis of SAD. SA in these patients is associated with longer illness duration, older age, and higher ED psychopathology, suggesting that SA may be indicative of a more severe form of ED. Despite significant reductions following treatment, there is some evidence that SA remains elevated in those recovered from AN compared to HCs. Whether SA impacts on treatment adherence and outcomes has not yet been examined, but such research may be important in improving prognosis for EDs. Furthermore, research in this area may lead to new insights into common illness pathways and transdiagnostic factors for AN and other disorders, such as ASD or SAD.

Acknowledgements

JKG received financial support from the Economic and Social Research Council (ESRC), and would like to thank Jenni Leppanen for her assistance in using the statistical programme. KT would like to acknowledge financial support from MRC and MRF child and young adult mental health – the underpinning aetiology of self-harm and eating disorders and Swiss Anorexia Nervosa Foundation (grant 58-16).

Conflicts of interest

None.

References

Abbate-Daga G, Marzola E, De-Bacco C, Buzzichelli S, Brustolin A, Campisi S, Amianto F, Migliaretti G, Fassino S (2015). Day Hospital Treatment for Anorexia Nervosa: A 12-Month Follow-up Study. *European Eating Disorders Review* **23**, 390–398.

Adambegan M, Wagner G, Nader IW, Fernandez-Aranda F, Treasure J, Karwautz A (2012). Internalizing and externalizing behaviour problems in childhood contribute to the development of anorexia and bulimia nervosa - A study comparing sister pairs. *European Eating Disorders Review* **20**, 116–120.

Allen HN, Craighead LW (1999). Appetite Monitoring in the Treatment of Binge Eating Disorder. *Behavior Therapy* **30**, 253–272.

Allen KL, Byrne SM, Oddy WH, Crosby RD (2013). DSM–IV–TR and DSM-5 eating disorders in adolescents: Prevalence, stability, and psychosocial correlates in a population-based sample of male and female adolescents. *Journal of Abnormal Psychology* **122**, 720–732.

Antony MM, Purdon CL, Huta V, Swinson RP (1998). Dimensions of perfectionism across the anxiety disorders. *Behaviour Research and Therapy* **36**, 1143–1154.

Begg CB, Mazumdar M (1994). Operating characteristics of a rank correlation test for publication bias. *Biometrics* **50**, 1088–1101.

Berkman ND, Lohr KN, Bulik CM (2007). Outcomes of eating disorders: A systematic review of the literature. *International Journal of Eating Disorders* **40**, 293-309.

Blinder BJ, Cumella EJ, Sanathara VA (2006). Psychiatric comorbidities of female inpatients with eating disorders. *Psychosomatic Medicine* **68**, 454–462.

Bora E, Kose S (2016). Meta-analysis of theory of mind in anorexia nervosa and bulimia nervosa: A specific impairment of cognitive perspective taking in anorexia nervosa? *International Journal of Eating Disorders* **49,** 739–740.

Buchholz A, Henderson K, Hounsell A, Wagner A, Norris M, Spettigue W (2007). Self-silencing in a clinical sample of female adolescents with eating disorders. *Journal of the Canadian Academy of Child and Adolescent Psychiatry* **16**, 158–163.

Buckner JD, Silgado J, Lewinsohn PM (2010). Delineation of differential temporal relations between specific eating and anxiety disorders. *Journal of Psychiatric Research* **44**, 781–787.

Bulik CM, Beidel DC, Duchmann E, Weltzin TE, Kaye WH (1991). An analysis of social anxiety in anorexic, bulimic, social phobic, and control women. *Journal of Psychopathology and Behavioral Assessment* **13**, 199–211.

Bulik CM, Sullivan PF, Fear JL, Joyce PR (1997). Eating disorders and antecedent anxiety disorders: A controlled study. *Acta Psychiatrica Scandinavica* **96**, 101–107.

Caglar-Nazali HP, Corfield F, Cardi V, Ambwani S, Leppanen J, Olabintan O, Deriziotis S, Hadjimichalis A, Scognamiglio P, Eshkevari E, Micali N, Treasure J (2014). A systematic review and meta-analysis of "Systems for Social Processes" in eating disorders. *Neuroscience and Biobehavioral Reviews* **42**, 55–92.

Cederlof M, Thornton LM, Baker J, Lichtenstein P, Larsson H, Ruck C, Bulik CM, Mataix-Cols D (2015). Etiological overlap between obsessive-compulsive disorder and anorexia nervosa: A longitudinal cohort, multigenerational family and twin study. *World Psychiatry* **14**, 333–338

Ciarma JL, Mathew JM (2017). Social anxiety and disordered eating: The influence of stress reactivity and self-esteem. *Eating Behaviors* **26**, 177–181.

Cohen J (1988). Statistical Power Analysis for the Behavioral Sciences, 2nd edn. Laurence Erlbaum Associates: London

Courty A, Godart N, Lalanne C, Berthoz S (2015). Alexithymia, a compounding factor for eating and social avoidance symptoms in anorexia nervosa. *Comprehensive Psychiatry* **56**, 217–228.

Dakanalis A, Carrà G, Calogero R, Zanetti MA, Volpato C, Riva G, Clerici M, Cipresso P (2016). The social appearance anxiety scale in italian adolescent populations: Construct validation and group discrimination in community and clinical eating disorders samples. *Child Psychiatry and Human Development* **47**, 133-150.

Davies H, Wolz I, Leppanen J, Fernandez-Aranda F, Schmidt U, Tchanturia K (2016). Facial expression to emotional stimuli in non-psychotic disorders: A systematic review

and meta-analysis. *Neuroscience and Biobehavioral Reviews* **64**, 252–271.

Dias P, Soares I, Klein J, Cunha JP, Roisman GI (2011). Autonomic correlates of attachment insecurity in a sample of women with eating disorders. *Attachment & Human Development* **13**, 155-167.

Duclos J, Dorard G, Berthoz S, Curt F, Faucher S, Falissard B, Godart N (2014). Expressed emotion in anorexia nervosa: What is inside the "black box"? *Comprehensive Psychiatry* **55**, 71–79.

Fairburn CG, Cooper Z, Doll HA, Welch SL (1999). Risk factors for anorexia nervosa: Three integrated case-control comparisons. *Archives of General Psychiatry* **56**, 468–476.

Fairweather-Schmidt AK, Wade TD (2014). DSM-5 eating disorders and other specified eating and feeding disorders: Is there a meaningful differentiation? *International Journal of Eating Disorders* **47**, 524–533.

Flament MF, Godart NT, Fermanian J, Jeammet P (2001). Predictive factors of social disability in patients with eating disorders. *Eating and Weight Disorders* **6**, 99–106.

Gadalla T, Piran N (2008). Psychiatric comorbidity in women with disordered eating behavior: A national study. *Women & Health* **48**, 467–484.

Gander M, Seveck K, Buchheim A (2015). Eating disorders in adolescence: attachment issues from a developmental perspective. *Frontiers in Psychology* **6,** 1136.

Gilbert N, Meyer C (2003). Social anxiety and social comparison: Differential links with restrictive and bulimic attitudes among nonclinical women. *Eating Behaviors* **4**, 257–264.

Gilboa-Schechtman E, Avnon L, Zubery E, Jeczmien P (2006). Emotional processing in eating disorders: Specific impairment or general distress related deficiency? *Depression and Anxiety* **23**, 331–339.

Godart NT, Flament MF, Lecrubier Y, Jeammet P (2000). Anxiety disorders in anorexia nervosa and bulimia nervosa: Co-morbidity and chronology of appearance. *European Psychiatry* **15**, 38–45.

Goddard E, Treasure J (2013). Anxiety and social-emotional processing in eating disorders: examination of family trios. *Cognitive Therapy and Research* **37**, 890-904.

Grabhorn M, Stenner H, Stangier U, Kaufhold A (2006). Social anxiety in anorexia and bulimia nervosa: The mediating role of shame. *Clinical Psychology and Psychotherapy* **13**, 12–19.

Gross J, Rosen JC (1988). Bulimia in adolescents: Prevalence and psychosocial correlates. *International Journal of Eating Disorders* **7**, 51-61.

Harrison A, Mountford VA, Tchanturia K (2014). Social anhedonia and work and social functioning in the acute and recovered phases of eating disorders. *Psychiatry Research* **218**, 187–194.

Hinrichsen H, Sheffield A, Waller G (2007a). The role of parenting experiences in the development of social anxiety and agoraphobia in the eating disorders. *Eating Behaviours* **8**, 285-290.

Hinrichsen H, Waller G, Dhokia R (2007b). Core beliefs and social anxiety in the eating disorders. *Eating and Weight Disorders* **12**, e14–e18.

Hinrichsen H, Waller G, Emanuelli F (2004a). Social anxiety and agoraphobia in the eating disorders: Associations with core beliefs. *The Journal of Nervous and Mental Disease* **192**, 784-787.

Hinrichsen H, Waller G, van Gerko K (2004b). Social anxiety and agoraphobia in the eating disorders: Associations with eating attitudes and behaviours. *Eating Behaviours* **5,** 285-290.

Hinrichsen H, Wright F, Waller G, Meyer C (2003). Social anxiety and coping strategies in the eating disorders. *Eating Behaviors* **4**, 117–126.

Jiménez-Murcia S, Granero R, Moragas L, Steiger H, Israel M, Aymamí N, Gómez-Peña M, Sauchelli S, Agüera Z, Sánchez I, Riesco N, Penelo E, Menchón JM, Fernández-Aranda F (2015). Differences and similarities between bulimia nervosa, compulsive buying and gambling disorder. *European Eating Disorders Review* **23**, 126–132.

Kaye WH, Bulik CM, Thornton L, Barbarich N, Masters K (2004). Comorbidity of

anxiety disorders with anorexia and bulimia nervosa. *American Jounral of Psychiatry* **161**, 2215-2221.

Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry* **62**, 593-602.

Krug I, Penelo E, Fernandez-Aranda F, Anderluh M, Bellodi L, Cellini E, di Bernardo M, Granero R, Karwautz A, Nacmias B, Ricca V, Sorbi S, Tchanturia K, Wagner G, Collier D, Treasure J (2013). Low social interactions in eating disorder patients in childhood and adulthood: A multi-centre European case control study. *Journal of Health Psychology* **18**, 26-37.

Kuusikko S, Pollock-Wurman R, Jussila K, Carter AS, Mattila ML, Ebeling H, Pauls DL, Moilanen I. (2008). Social anxiety in high-functioning children and adolescents with autism and Asperger syndrome. *Journal of Autism and Developmental Disorders* **38**, 1697–1709.

Lázaro L, Font E, Moreno E, Calvo R, Vila M, Andrés-Perpiñá S, Canalda G, Martínez E, Castro-Fornieles J (2011). Effectiveness of self-esteem and social skills group therapy in adolescent eating disorder patients attending a day hospital treatment programme. *European Eating Disorders Review* **19**, 398-406.

Levinson CA, Rodebaugh TL (2016). Clarifying the prospective relationships between social anxiety and eating disorder symptoms and underlying vulnerabilities. *Appetite* **107**, 38–46.

Liberati A, Altman DG, Tetzlaff J, Mulrow C, Gøtzsche PC, Ioannidis JPA, Clarke M, Devereaux PJ, Kleijnen J, Moher D (2009). The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate health care interventions: Explanation and elaboration. *PLoS Medicine* **6**. e1000100

Liebowitz MR (1987). Social phobia. *Mod Probl Pharmacopsychiatry* **22**, 141–173.

Lloyd S, Yiend J, Schmidt U, Tchanturia K (2014). Perfectionism in anorexia nervosa: Novel performance based evidence. *PLoS ONE* **9**, e111697.

MacDonald DE, McFarlane TL, Olmsted MP (2014). “Diagnostic shift” from eating

disorder not otherwise specified to bulimia nervosa using DSM-5 criteria: A clinical comparison with DSM-IV bulimia. *Eating Behaviors* **15**, 60–62.

Mattar L, Huas C, Godart N (2012a). Relationship between Affective symptoms and malnutrition severity in severe anorexia nervosa. *PLoS ONE 7*, e49380.

Mattar L, Thiébaud MR, Huas C, Cebula C, Godart N (2012b). Depression, anxiety and obsessive-compulsive symptoms in relation to nutritional status and outcome in severe anorexia nervosa. *Psychiatry Research* **200**, 513–517.

McFarlane T, MacDonald DE, Trottier K, Olmsted MP (2015). The effectiveness of an individualized form of day hospital treatment, *Eating Disorders* **23**, 191-205.

Melfsen S, Walitza S, Warnke A (2006). The extent of social anxiety in combination with mental disorders. *European Child and Adolescent Psychiatry* **15**, 111-117.

Obeid N, Buchholz A, Boerner KE, Henderson KA, Norris M (2013). Self-esteem and social anxiety in an adolescent female eating disorder population: Age and diagnostic effects. *Eating Disorders* **21**, 140–153.

Ohmann S, Popow C, Wurzer M, Karwautz A, Sackl-Pammer P, Schuch B (2013). Emotional aspects of anorexia nervosa: Results of prospective naturalistic cognitive behavioral group therapy. *Neuropsychiatrie* **27**, 119–128.

Ostrovsky NW, Swencionis C, Wylie-Rosett J, Isasi CR (2013). Social anxiety and disordered overeating: An association among overweight and obese individuals. *Eating Behaviors* **14**, 145–148.

Pollatos O, Kurz AL, Albrecht J, Schreder T, Kleemann AM, Schöpf V, Kopietz R, Wiesmann M, Schandry R (2008). Reduced perception of bodily signals in anorexia nervosa. *Eating Behaviours* **9**, 381-388.

Ranta K, Väänänen J, Fröjd S, Isomaa R, Kaltiala-Heino R, Marttunen M (2017). Social phobia, depression and eating disorders during middle adolescence: Longitudinal associations and treatment seeking. *Nordic Journal of Psychiatry* **71**, 605-613.

Rosenthal **R** (1979). The “file drawer problem” and tolerance for null results. *Psychological Bulletin* **86**, 638–641

RStudio Team (2015). RStudio: Integrated Development for R. RStudio, Inc., Boston, Available at: <http://www.rstudio.com/>

Russell J, Maguire S, Hunt GE, Kesby A, Suraev A, Stuart J, Booth J, McGregor IS (2018). Intranasal oxytocin in the treatment of anorexia nervosa: Randomized controlled trial during re-feeding. *Psychoneuroendocrinology* **87**, 83–92.

Sawaoka T, Barnes RD, Blomquist KK, Masheb RM, Grilo CM (2012). Social anxiety and self-consciousness in binge eating disorder: Associations with eating disorder psychopathology, *Comprehensive Psychiatry* **53**, 740–745.

Schmelkin C, Plessow F, Thomas JJ, Gray EK, Marengi DA, Pulumo R, Silva L, Miller KK, Hadjikhani N, Franko DL, Eddy KT, Lawson EA (2017). Low oxytocin levels are related to alexithymia in anorexia nervosa. *International Journal of Eating Disorders* **50**, 1332–1338.

Schneier FR, Kimeldorf MB, Choo TH, Steinglass JE, Wall MM, Fyer AJ, Simpson HB (2016). Attention bias in adults with anorexia nervosa, obsessive-compulsive disorder, and social anxiety disorder. *Journal of Psychiatric Research* **79**, 61-69.

Schulze UME, Calame S, Keller F, Mehler-Wex C (2009). Trait anxiety in children and adolescents with anorexia nervosa. *Eating and Weight Disorders* **14**, e163–e168.

Schwalberg MD, Barlow DH, Alger SA, Howard LJ (1992). Comparison of bulimics, obese binge eaters, social phobics, and individuals with panic disorder on comorbidity across DSM-III—R anxiety disorders. *Journal of Abnormal Psychology* **101**, 675–681.

Silgado J, Timpano KR, Buckner JD, Schmidt NB (2010). Social anxiety and bulimic behaviors: The moderating role of perfectionism. *Cognitive Therapy and Research* **34**, 487–492.

Slade P (1982). Towards a functional analysis of anorexia nervosa and bulimia nervosa. *British Journal of Clinical Psychology* **21**, 167-179.

Solano R, Ferna F, Vallejo J, Aitken A, Lo C (2005). Self-injurious behaviour in people with eating disorders, *European Eating Disorders Review* **10**, 3–10.

Steinglass JE, Lempert KM, Choo TH, Kimeldorf MB, Wall M, Walsh BT, Fyer AJ, Schneier FR, Simpson HB (2017). Temporal discounting across three psychiatric

disorders: Anorexia nervosa, obsessive compulsive disorder, and social anxiety disorder. *Depression and Anxiety* **34,** 463-470.

Steinman SA, Ahmari SE, Choo TH, Kimeldorf MB, Feit R, Loh S, Risbrough V, Geyer MA, Steinglass JE, Wall M, Schneier FR, Fyer AJ, Simpson HB (2016). Prepulse inhibition deficits only in females with obsessive-compulsive disorder. *Depression and Anxiety* **33,** 238-246.

Striegel-Moore RH, Silberstein LR, Rodin J (1993). The social self in bulimia nervosa: Public self-consciousness, social anxiety, and perceived fraudulence. *Journal of Abnormal Psychology* **102**, 297-303.

Strober M, Freeman R, Lampert C, Diamond J (2007). The association of anxiety disorders and obsessive compulsive personality disorder with anorexia nervosa: Evidence from a family study with discussion of nosological and neurodevelopmental implications. *International Journal of Eating Disorders* **41**, 174–179.

Swinbourne J, Hunt C, Abbott M, Russell J, St Clare T, Touyz S (2012). The comorbidity between eating disorders and anxiety disorders: Prevalence in an eating disorder sample and anxiety disorder sample. *Australian & New Zealand Journal of Psychiatry* **46**, 118–131.

Swinbourne J, Touyz S (2007). The co-morbidity of eating disorders and anxiety disorders: A review. *European Eating Disorders Review* **15**, 215–221.

Tchanturia K, Doris E, Mountford V, Fleming C (2015). Cognitive Remediation and Emotion Skills Training (CREST) for anorexia nervosa in individual format: Self-reported outcomes. *BMC Psychiatry* **15**, 53.

Treasure J, Schmidt U (2013). The cognitive-interpersonal maintenance model of anorexia nervosa revisited: A summary of the evidence for cognitive, socio-emotional and interpersonal predisposing and perpetuating factors. *Journal of Eating Disorders* **1**, 13.

Utschig AC, Presnell K, Madeley MC, Smits JAJ (2010). An investigation of the relationship between fear of negative evaluation and bulimic psychopathology. *Eating Behaviors* **11**, 231–238.

Vall E, Wade TD (2015). Predictors of treatment outcome in individuals with eating

disorders: A systematic review and meta-analysis. *International Journal of Eating Disorders* **48**, 946-971.

Viechtbauer W (2010). Conducting meta-analyses in R with the metafor package. *Journal of Statistical Software* **36,** 3.

Westwood H, Kerr-Gaffney J, Stahl D, Tchanturia K (2017a). Alexithymia in eating disorders: Systematic review and meta-analyses of studies using the Toronto Alexithymia Scale. *Journal of Psychosomatic Research* **99**, 66–81.

Westwood H, Lawrence V, Fleming C, Tchanturia K (2016). Exploration of friendship experiences, before and after illness onset in females with anorexia nervosa: A qualitative study. *PLOS ONE* **11**, e0163528.

Westwood H, Mandy W, Tchanturia K (2017b). Clinical evaluation of autistic symptoms in women with anorexia nervosa. *Molecular Autism* **8**, 12.

Wildes JE, Ringham RM, Marcus MD (2010). Emotion avoidance in patients with anorexia nervosa: Initial test of a functional model. *International Journal of Eating Disorders* **43**, 398-404.

Zonnevylle-Bender MJS, van Goozen SHM, Cohen-Kettenis PT, **Jansen LMC, van Elburg A, van Engeland H** (2005). Adolescent anorexia nervosa patients have a discrepancy between neurophysiological responses and self-reported emotional arousal to psychosocial stress. *Psychiatry Research* **135**, 45-52.

Zonnevylle-Bender MJS, van Goozen SHM, Cohen-Kettenis PT, van Elburg A, De Wildt M, Stevelmans E, van Engeland H (2004). Emotional functioning in anorexia nervosa patients: Adolescents compared to adults. *Depression and Anxiety* **19**, 35–42.

Table 1. *Characteristics of studies.*

Study	Sample	Mean (SD) SA score	Measure	Mean age (SD)	Mean BMI (SD)	Mean (yrs) illness duration (SD)	% female	Recruitment source	Groups matched by
Abbate Daga *et al.* (2015)	56 AN	Baseline 37.16 (12.66) 6 months 26.42 (9.66) 12 months 22.81 (10.30)	BSPS	25.03 (5.75)	16.31 (2.66)	7.8 (5.34)	100	Day hospital	NA
Allen & Craighead (1999)	11 BED (treatment)	Pre-treatment 23.09 (4.11) Post-treatment 20.0 (5.0)	FNE	21 (1.2)[a]	122.82 (22.86)% IBW	NR	100	University advertisement	Sex
	9 BED (waitlist)	Pre-treatment 23.11 (9.68) Post-treatment 22.33 (8.66)			116.5 (21.98)% IBW	NR	100	University advertisement	
Bucholz *et al.* (2007)	149 ED	15.02 (7.17)	MASC social anxiety subscale	15.65 (1.17)	NR	NR	100	Tertiary care childrens hospital	NA
Bulik *et al.* (1991)	23 AN	86.2 (26.4)	SPAI difference scores	20.3 (8.3)	NR	NR	100	Inpatient unit	Sex
	54 BN	84.9 (27.9)		22.4 (6.0)	NR	NR	100	Inpatient unit	
	43 SAD	98.7 (35.3)		36.3 (9.6)	NR	NR	100	Social phobia clinic	
	50 HC	54.6 (35.9)		18.5 (1.2)	NR	NA	100	University undergraduates	
Dakanalis *et al.* (2016)	189 young female adolescent ED	30.29 (12.61) 37.33 (12.49) 39.86 (11.05)	SIAS SPS BFNE	12.59 (0.70)[a]	AN = 15.52 (1.24), BN = 21.18 (2.07), EDNOS = 20.05 (3.54)	NR	100	Specialist child and adolescent ED service	NR
	15 young male adolescent ED	29.88 (12.24) 32.31 (13.13) 32.01 (12.88)	SIAS SPS BFNE			NR	0		
	444 older female adolescent ED	37.01 (13.06) 44.41 (13.88)	SIAS SPS	16.74 (0.61)[a]		NR	100		

		45.55 (10.55)	BFNE						
	55 older male adolescent ED	36.65 (13.32)	SIAS			NR	0		
		34.35 (12.96)	SPS						
		34.69 (11.11)	BFNE						
Duclos *et al.* (2014) and Courty *et al.* (2015)	60 AN	Baseline 48.3 (31.3) 6 months 33.1 (27.9) 12 months 33.2 (29.7) 18 months 27.2 (26.3)	LSAS	16 (1.6)	16.9 (1.1)	1.38 (0.57)	100	Inpatient unit	NA
Flament *et al.* (2001)	29 AN-R	50 (10.30)	LSAS	17.9 (4.3)	NR	2 (3)	93.1	Inpatient unit	NR
	34 BN	54 (16.51)		26.6 (6.5)	NR	7 (6)	100	Outpatient clinic	
Gilboa-Schectman *et al.* (2006)	20 AN	73.05 (37.28)	LSAS	16.60 (2.48)	NR	NR	100	Outpatient clinic	Age, sex, education
	20 BN	58.60 (42.48)		19.65 (5.01)	NR	NR	100	Outpatient clinic	
	20 HC	33.90 (18.67)		19.65 (5.01)	NR	NA	100	Community advertisement	
Goddard & Treasure (2013)	63 ED	61.6 (31.6)	LSAS	21.8 (5.5)	16.8 (2.5)	4.0 (2.0-7.6)[b]	100	ED clinics	Age, sex, education, IQ
	50 HC	29.8 (17.8)		21.5 (5.9)	21.1 (2.0)	NA	100	University advertisement	
Grabhorn *et al.* (2006)	30 AN	33.5 (12.9)	SIAS	25.5 (7.7)	NR	NR	100	Inpatient clinic referrals	Sex
		29.3 (11.3)	SPS						
	30 BN	39.4 (12.9)	SIAS	24.9 (6.8)	NR	NR	100		
		35.4 (16.2)	SPS						
	30 Depression	30.6 (14.9)	SIAS	41.1 (10.9)	NR	NR	100		
		21.3 (13.0)	SPS						
	30 Anxiety	23.6 (14.5)	SIAS	36.9 (12.8)	NR	NR	100		
		19.5 (18.1)	SPS						
Gross *et al.* (1988)	65 BN (female)	9.49 (7.03)	SADS	NR	NR	NR	100	Public schools	Sex
	612 HC (female)	6.98 (6.14)		NR	NR	NA	100		
	8 BN (male)	15.29 (5.9)		NR	NR	NR	0		
	645 HC (male)	7.69 (4.5)		NR	NR	NA	0		

Hinrichsen *et al*. (2003)	21 AN-R	23.5 (8.68)	FNE	25.7 (9.06)	NR	NR	100	Specialist ED service	Sex
	34 AN-BP	26.7 (4.32)		28.0 (6.79)	NR	NR	100		
	59 BN	23.6 (6.72)		26.9 (6.56)	NR	NR	100		
	50 HC	18.5 (6.97)		19.8 (0.86)	NR	NA	100	University undergraduates	
Hinrichsen *et al.* (2004a); (2004b); (2007a)	70 ED	88.41 (35.10)	SPAI social phobia subscale	27.9 (8.76)	22.9 (11.53)	NR	100	Specialist ED service referrals (outpatient)	NA
Hinrichsen *et al.* (2007b)	191 ED	27.4 (7.84)	BFNE	28.4 (8.62)	20.4 (6.77)	NR	100	Specialist ED service	NA
Jimenez-Murcia *et al*. (2015)	50 BN	13.7 (8.50)	SADS	28.1 (8.2)	NR	NR	100	Psychiatric department referrals	Sex
	49 BN + compulsive buying	15.7 (7.64)		26.9 (9.1)	NR	NR	100		
MacDonald *et al*. (2014)	171 BN	30.26 (14.66)	SPIN	26.2 (8.2)	22.7 (5.33)	9.2 (8.2)	96.5	Day hospital	NA
Mattar *et al.* (2012a)	155 AN	57.73 (15.85)	LSAS	20.90 (6.16)	14.43 (1.46)	4.29 (4.71)	100	Inpatient unit	NA
Mattar *et a*l. (2012b)	24 AN-R	Baseline 47.05 (28.30)	LSAS	16.38 (1.93)	13.84 (1.26)	0.98 (0.82)	100	Inpatient unit	NA
		EOT 24.95 (26.91)							
McFarlane *et al.* (2015)	299 ED	31.5 (15.1)	SPIN	26.0 (7.8)	17.1 (1.0)	8.3 (7.3)	97	Day hospital	NR
	130 ED	33.9 (15.5)		30.9 (11.1)	17.0 (1.0)	14.4 (11.2)	96.8		
Melfsen *et al.* (2006)	48 AN	15.35 (9.25)	SPAI-C	NR	NR	NR	NR	Child and adolescent psychiatric departments	NR
	31 SAD	29.59 (9.79)		12.19 (2.59)	NR	NR	58.1		
	7 AS	20.77 (13.77)		15.71 (2.5)	NR	NR	28.6		
	1197 HC	12.51 (7.87)		12.51 (2.05)	NR	NA	51.5	NR	
Obeid *et al.* (2013)	182 AN-R or EDNOS-R	14.49 (7.32)[c], 14.2 (7.19)[d]	MASC social anxiety subscale	15.6 (1.39)	17.02 (2.27)[c], 17.05 (2.14)[d]	NR	100	Children's tertiary care facility	Sex
	99 AN-BP or EDNOS-BP	15.21 (6.04)[c], 16.14 (6.94)[d]			20.55 (3.50)[c], 19.94 (3.71)[d]	NR	100		

	63 BN	19.89 (5.71)[c], 14.08 (6.78)[d]			22.02 (2.62)[c], 22.30 (3.79)[d]	NR	100		
Ostrovsky *et al*. (2013)	29 BED	50.3[e]	SPIN	36.0 (12.8)[a]	33.7 (6.7)[a]	NR	86.8[a]	Online and university advertisements	NR
	202 HC	32.6[e]				NA			
Ohmann *et al.* (2013)[f]	29 AN	Baseline 24.1 (9.1)	SIAS	14.3[e]	15.7 (1.3)	0.6[e]	100	Inpatient unit	NA
		Baseline 15.4 (9.9)	SPS						
		9 months 26.7 (14.3)	SIAS		17.8 (1.7)				
		9 months 14.6 (15.0)	SPS						
Russell *et al.* (2018)	16 AN (oxytocin)	Baseline 59.4 (29.1)	LSAS	22.4 (3.6)	16.61 (1.77)	NR	100	Inpatient unit	Sex
		Follow-up 59.4 (28.9)			18.00 (1.86)				
	17 AN (placebo)	Baseline 63.1 (24.4)		23.5 (10.2)	16.75 (1.36)	NR	100		
		Follow-up 58.4 (27.3)			18.10 (1.29)				
Sawaoka *et al.* (2012)	113 BED	15.13 (4.51)	SCS social anxiety subscale	45.03 (8.30)	37.1 (7.3)	NR	77.9	Newspaper advertisements	NA
Schmelkin *et al*. (2017)	19 AN	61.95 (30.53)	LSAS	25.1 (1.7)	17.7 (0.2)	NR	100	Community	Sex
	23 AN-WR	36.74 (19.15)		22.9 (.5)	22.5 (0.4)	NR	100		
	28 HC	22.25 (15.68)		23.9 (.8)	22.6 (0.3)	NA	100		
Schneier *et al.* (2016)	30 AN	54.1 (26.1)	LSAS	26.9(7.5)	NR	NR	97	Media notices and referrals from health professionals	NR
	43 SAD	76.4 (19.8)		29.9 (7.5)	NR	NR	53		
	50 OCD	24.2 (16.7)		29.2 (5.9)	NR	NR	50		
	74 HC	11.5 (8.0)		28.9 (7.6)	NR	NA	51		
Schultz *et al.* (2009)	23 AN	17.4 (9.7)	SPAI-C	14.69 (1.54)	14.7 (1.58)	NR	100	Inpatients at a child and adolescent psychiatric department	NR
	145 PC	16.52 (10.77)		13.29 (2.86)	NR	NR	NR	NR	
	1197 HC	12.51 (7.87)		12.51 (2.05)	NR	NA	51.5	NR	

Schwalberg *et al.* (1992)	20 BN	14.7 (5.9)	SCS social anxiety subscale	26.35 (6.44)	104.5 (12.5)% IBW	NR	100	ED clinics	Sex
	20 BED	15.1 (5.6)		41.18 (7.66)	157.9 (31.8)% IBW	NR	100		
	20 SAD	19.6 (3.7)		34.70 (9.74)	NR	NR	100	Anxiety disorder clinics	
	20 PD	13.9 (6.1)		31.50 (6.90)	NR	NA	100		
Solano *et al*. (2005)	35 AN & BN (SI)	19.41 (6.79)	SADS	22.31 (4.46)	18.85 (4.12)	4.83 (3.69)	100	Outpatient clinic	Sex
	74 AN & BN (no SI)	15.09 (9.05)		23.24 (6.08)	19.43 (4.70)	5.82 (5.74)	100		
Steinglass *et a*l. (2017)	27 AN	52.44 (23.07)	LSAS	27.7 (7.5)	17.5 (1.0)	NR	100	Inpatient unit	Age, sex, ethnicity
	44 SAD	75.72 (20.05)		30.0 (4)	23.9 (6.3)	NR	57	Outpatient clinic	
	50 OCD	24.18 (16.66)		29.2 (5.8)	24.6 (5.3)	NR	48	Outpatient clinic	
	75 HC	11.39 (7.96)		29.0 (7.6)	24.1 (4.4)	NA	52	NR	
Steinman *et al.* (2016)	26 AN	47.81 (20.14)	LSAS	26.93 (7.67)	NR	NR	100	Inpatients	NR
	37 SAD	75.00 (19.32)		28.54 (6.66)	NR	NR	59	Media and referrals from health professionals	
	45 OCD	23.04 (17.03)		28.80 (5.89)	NR	NR	47		
	62 HC	12.42 (8.01)		27.60 (6.50)	NR	NA	53		
Striegel-Moore *et al.* (1993)	34 BN	19.71 (5.39)	SCS social anxiety subscale	23.36 (5.8)[a]	21.64 (2.76)	6.8 (4.15)	100	ED clinics	Age, sex, ethnicity, BMI
	33 sub-clinical ED	19.24 (4.49)			22.47 (2.92)	NA	100	University undergraduates and newspaper advertisments	
	67 HC	15.01 (5.0)			22.08 (2.87)	NA	100		
Zonnevylle-Bender *et al.* (2004)	23 adult AN	108.6 (41.4)	SPAI social phobia subscale	21.3 (3.1)	15.7 (1.4)	NR	NR	Inpatient eating disorder clinic	NR
	48 adolescent AN	80 (31.4)		15.5 (1.1)	14.8 (3.3)	NR	NR	Inpatients or outpatients at a child and adolescent eating disorder clinic	
Zonnevylle-Bender *et al*. (2005)	10 AN-R	103.6 (37.6)	SPAI social phobia subscale	15.5 (1.8)	16.2 (1.2)	0.92	100	NR	Age, sex, education
	22 HC	68.09 (25.1)		14.9 (1.1)	NR	NA	100	High school	

AN, anorexia nervosa; AN-BP, anorexia nervosa binge-purge sub-type; AN-R, anorexia nervosa restricting sub-type; AN-WR, anorexia nervosa weight restored; AS, Asperger's syndrome; BED, binge eating disorder; BFNE, Brief Fear of Negative Evaluation scale; BMI, body mass index; BN, bulimia nervosa; BSPS, brief social phobia scale; ED, eating disorder; EDNOS, eating disorder not otherwise specified; FNE, Fear of Negative Evaluation scale; HC, healthy control; IBW, ideal body weight; IQ, intelligence quotient; LSAS, Liebowitz Social Anxiety Scale; MASC, Multidimensional Anxiety Scale for Children; NA, not applicable; NR, not reported; OCD, obsessive compulsive disorder; PC, psychiatric control; PD, panic disorder; SAD, social anxiety disorder; SADS, Social Avoidance and Distress Scale; SA, social anxiety; SCS, Self-Consciousness Scale; SD, standard deviation; SI, self-injury; SIAS, Social Interaction Anxiety Scale; SPAI, Social Phobia & Anxiety Inventory; SPAI-C, Social Phobia & Anxiety Inventory for Children; SPIN, Social Phobia Inventory; SPS, Social Phobia Scale

[a]Values reported for groups combined

[b]Median and inter-quartile range

[c]Mid-adolescents

[d]Late-adolescents

[e]No SD reported

[f]Four additional follow-up periods included in paper excluded here for brevity

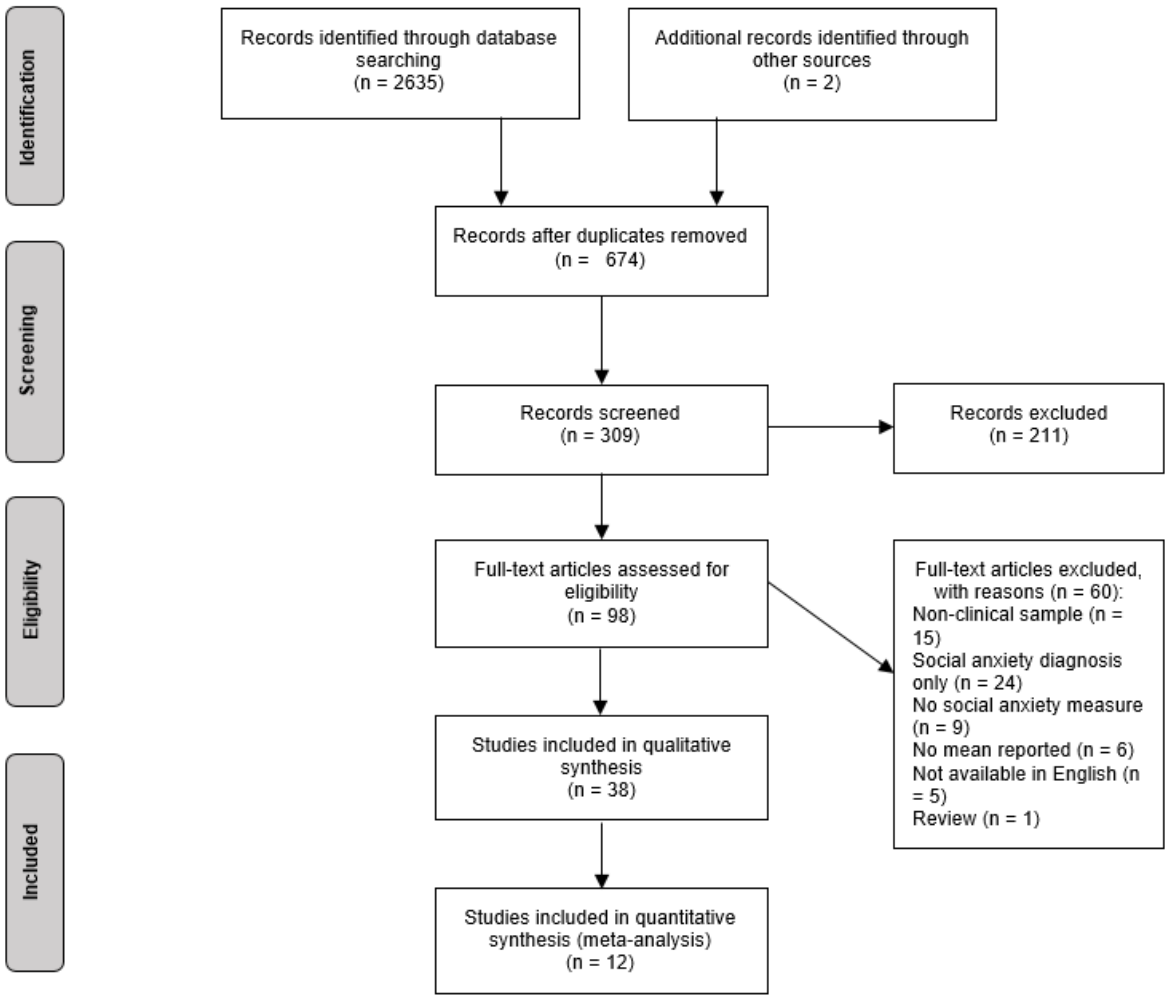

Figure 1. Systematic review search process.

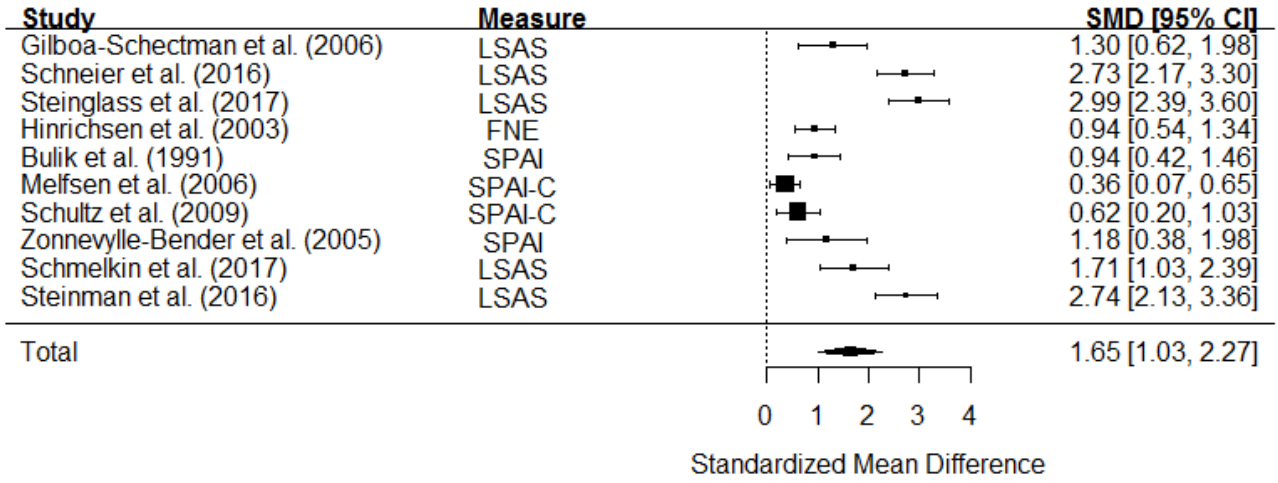

Figure 2. Forest plot of standardized mean effect size for differences (SMD) between anorexia nervosa (AN) and healthy controls (HC). CI, confidence interval; FNE, Fear of Negative Evaluation scale; LSAS, Liebowitz Social Anxiety Scale; SPAI, Social Phobia & Anxiety Inventory; SPAI-C, Social Phobia & Anxiety Inventory for Children

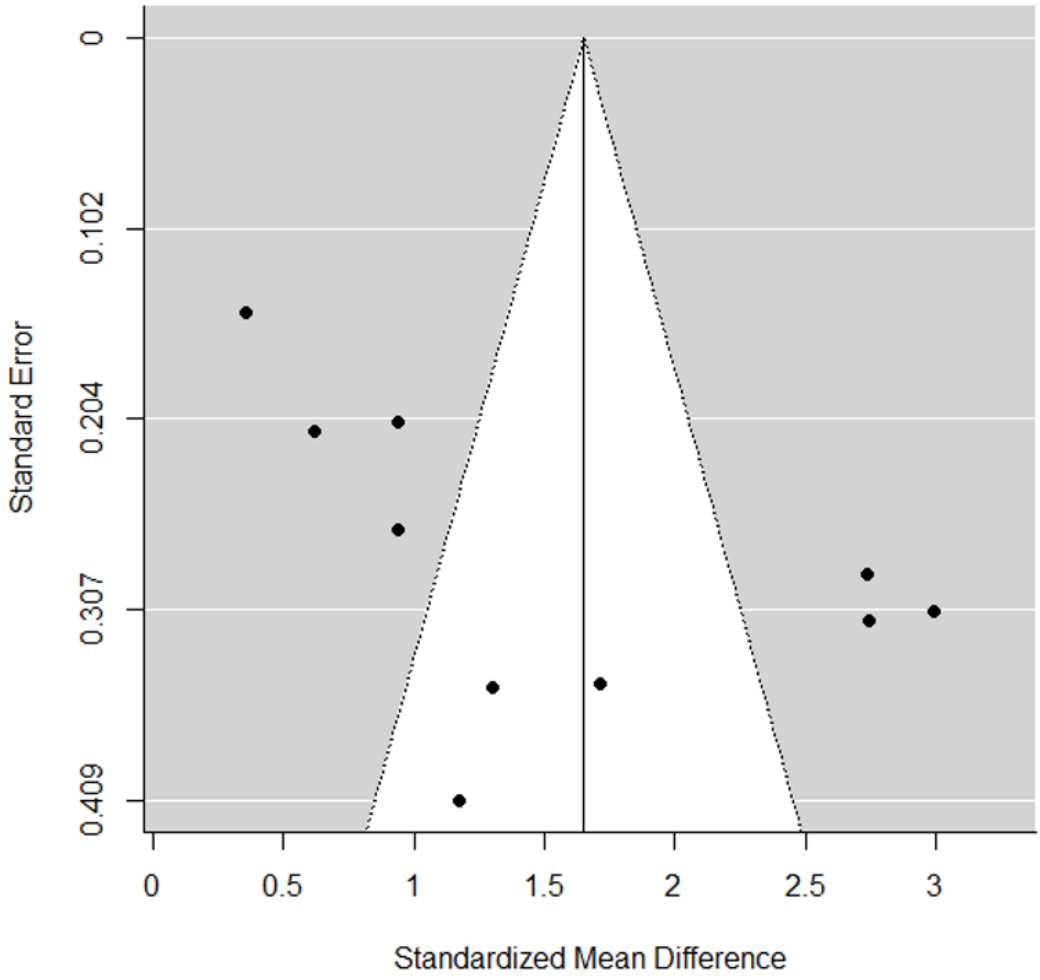

Figure 3. Funnel plot of anorexia nervosa (AN) studies included in the meta-analysis to assess publication bias.

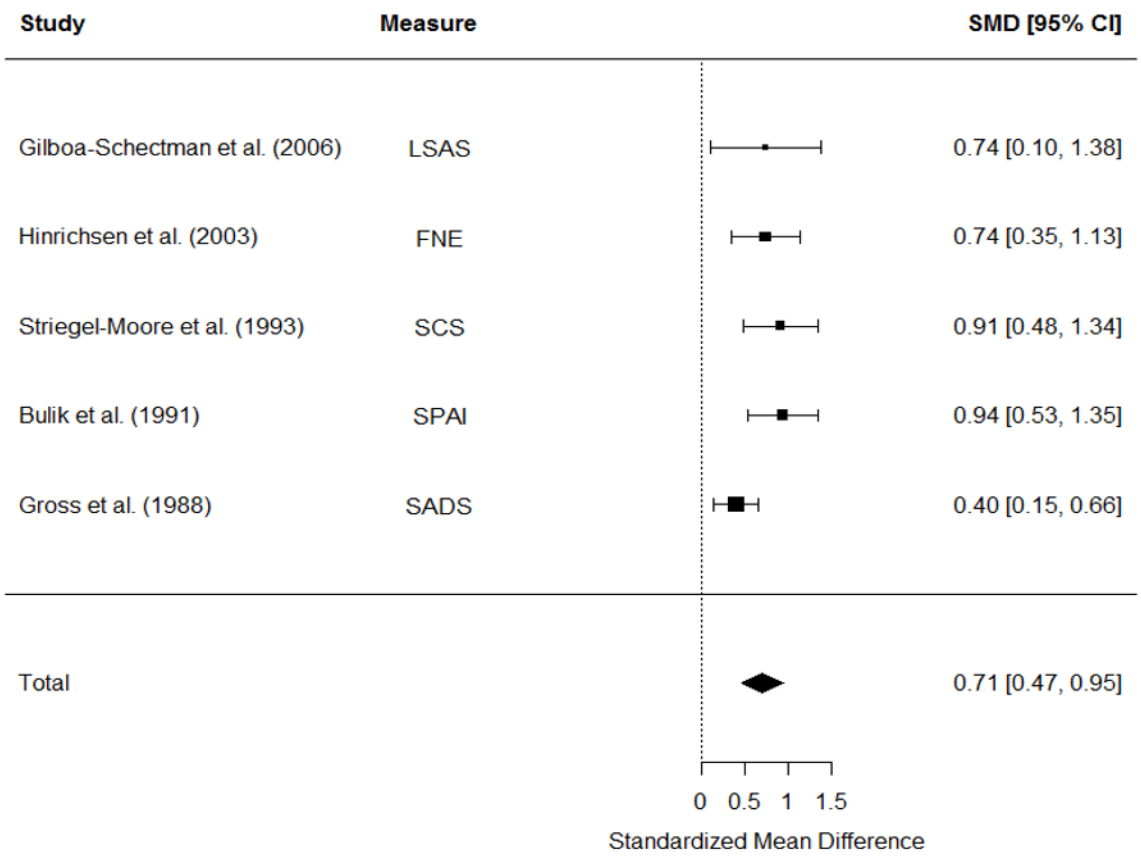

Figure 4. Forest plot of standardized mean effect size for differences (SMD) between bulimia nervosa (BN) and healthy controls (HC). CI, confidence interval; FNE, Fear of Negative Evaluation scale; LSAS, Liebowitz Social Anxiety Scale; SADS, Social Anxiety and Distress Scale; SCS, Self-Consciousness Scale; SPAI, Social Phobia & Anxiety Inventory

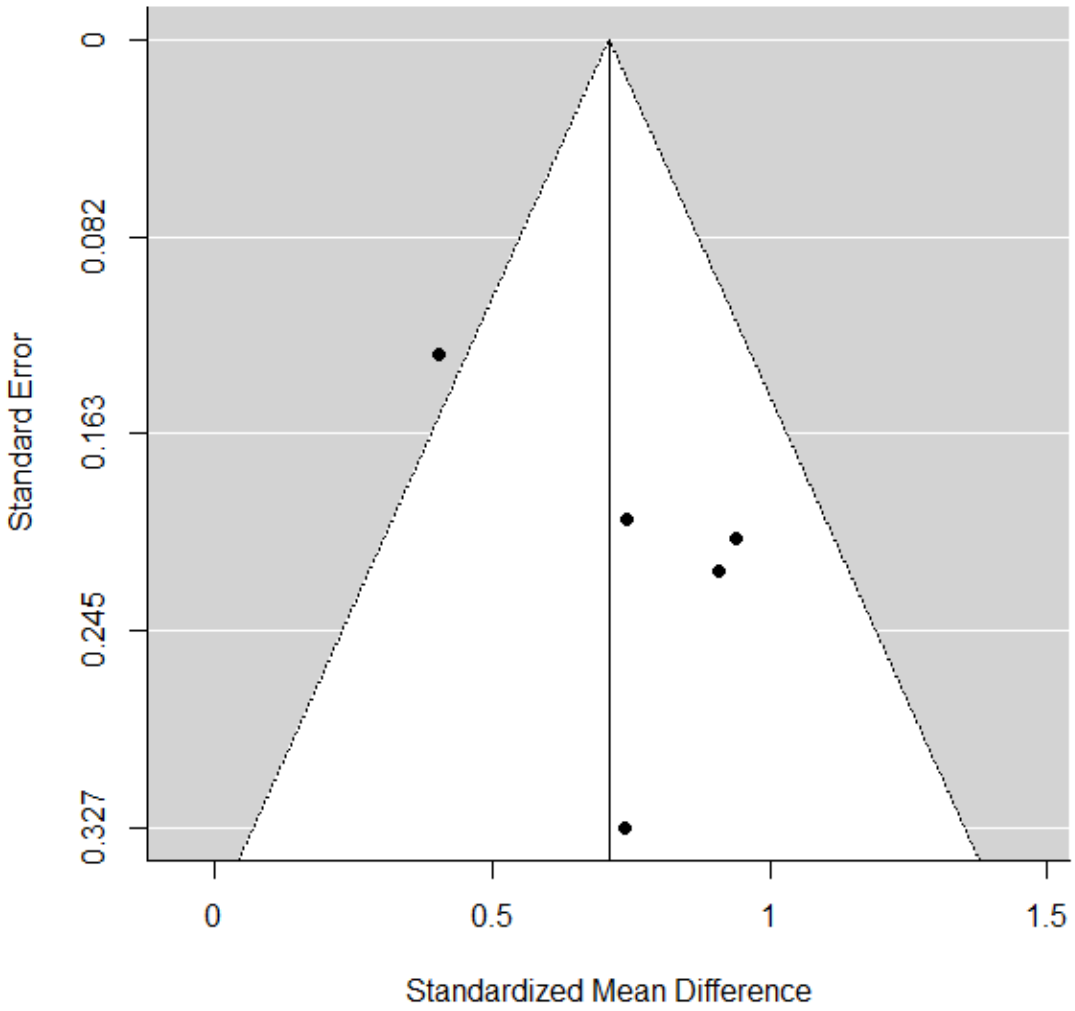

Figure 5. Funnel plot of bulimia nervosa (BN) studies included in the meta-analysis to assess publication bias.