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

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Review Article

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Abstract

Social anxiety disorder 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. 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, 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 body mass index. 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 17–67.8% in BN (Swinbourne & Touyz, 2007). In comparison, the 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

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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 the degree of genetic relatedness), the moderate genetic overlap between the two disorders has been reported in a large population-based twin sample (Cederlof *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 with 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 Fig. 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 al.l 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 and 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 with HCs were included in each of the respective meta-analyses.

Statistical analysis

All analyses were performed using R Studio (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 ($p < 0.05$), the 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

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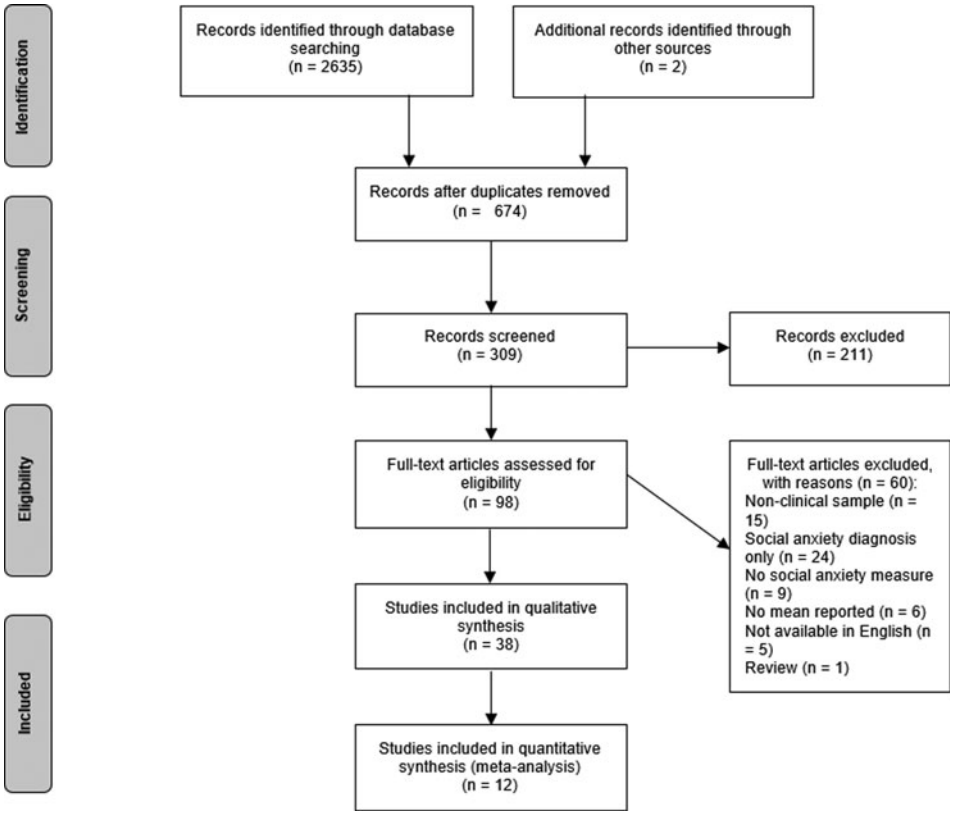

Fig. 1. Systematic review search process.

bottom right corner indicates publication bias. The symmetry of the funnel plots was 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 (Hinrichsen *et al.* 2004*a*, *b*; 2007*a*; Duclos *et al.* 2014; Courty *et al.* 2015) 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, 10 included an AN group and five included a BN group. One of the BN studies reported scores for males and females separately (Gross & Rosen, 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 and 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

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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)	BSPS	25.03 (5.75)	16.31 (2.66)	7.8 (5.34)	100	Day hospital	NA
		6 months 26.42 (9.66)							
		12 months 22.81 (10.30)							
Allen & Craighead (1999)	11 BED (treatment)	Pre-treatment 23.09 (4.11)	FNE	21 (1.2)[a]	122.82 (22.86)% IBW	NR	100	University advertisement	Sex
		Post-treatment 20.0 (5.0)							
	9 BED (waitlist)	Pre-treatment 23.11 (9.68)			116.5 (21.98)% IBW	NR	100	University advertisement	
		Post-treatment 22.33 (8.66)							
Buchholz *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)	SIAS	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
		37.33 (12.49)	SPS						
		39.86 (11.05)	BFNE						
	15 young male adolescent ED	29.88 (12.24)	SIAS			NR	0		
		32.31 (13.13)	SPS						
		32.01 (12.88)	BFNE						
	444 older female adolescent ED	37.01 (13.06)	SIAS	16.74 (0.61)[a]		NR	100		
		44.41 (13.88)	SPS						
		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)	LSAS	16 (1.6)	16.9 (1.1)	1.38 (0.57)	100	Inpatient unit	NA
		6 months 33.1 (27.9)							
		12 months 33.2 (29.7)							
		18 months 27.2 (26.3)							
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-Schechtman *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 & Rosen (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		

(*Continued*)

Table 1. (*Continued.*)

Study	Sample	Mean (SD) SA score	Measure	Mean age (SD)	Mean BMI (SD)	Mean (yrs) illness duration (SD)	% female	Recruitment source	Groups matched by
	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.* (2004*a*); (2004*b*); (2007*a*)	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.* (2007*b*)	191 ED	27.4 (7.84)	BFNE	28.4 (8.62)	20.4 (6.77)	NR	100	Specialist ED service	NA
Jiménez-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.* (2012*a*)	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 al.* (2012*b*)	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 (0.5)	22.5 (0.4)	NR	100		
	28 HC	22.25 (15.68)		23.9 (0.8)	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		

(*Continued*)

Table 1. (*Continued.*)

Study	Sample	Mean (SD) SA score	Measure	Mean age (SD)	Mean BMI (SD)	Mean (yrs) illness duration (SD)	% female	Recruitment source	Groups matched by
Schulze *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 al.* (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 advertisements	
	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.

significantly higher levels of SA than HCs, with a large effect size [$d = 1.65$, (95% CI 1.03–2.27) $z = 5.20$, $p < 0.001$] (Fig. 2). The funnel plot for the AN studies is displayed in Fig. 3. There was no evidence of publication bias (Begg's test $p = 0.216$, 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) $z = 5.74$, $p < 0.001$] (Fig. 4). The funnel plot for the BN studies is displayed in Fig. 5. There was no evidence of publication bias (Begg's test $p = 0.817$, Rosenthal's fail-safe $N = 112$).

Additional analyses

There was evidence of significant heterogeneity in the AN studies $Q(9) = 131.14$, $p < 0.001$, 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, $QM(4) = 32.56$, $p < 0.001$. Age had a significant influence on the size of the effect [$b = 0.12$, (95% CI 0.03–0.21), $z = 2.53$, $p = 0.01$], as did using the LSAS as a measure of SA [$b = 1.64$, (95% CI 0.78–2.50), $z = 3.73$, $p < 0.001$]. The test for residual heterogeneity was not significant, $QE(4) = 7.96$, $p = 0.09$. There was no evidence of heterogeneity in the BN studies $Q(3) = 0.67$, $p = 0.87$.

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. Ostrovsky *et al.* (2013) examined SA scores in individuals with BED compared with 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 with 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.* 2012*a*; Obeid *et al.* 2013; Duclos *et al.* 2014; Abbate-Daga *et al.* 2015; Courty *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

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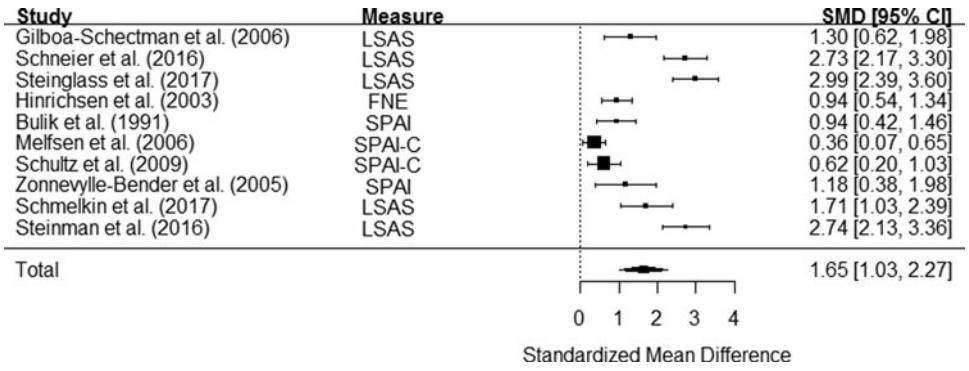

Fig. 2. Forest plot of standardised mean effect size for differences (SMD) between anorexia nervosa (AN) and healthy controls (HC). CI, confidence interval; FNE, Fear of Negative Evaluation scale; LSAS, Liebowitz Social Anxiety Scale; SPAI, Social Phobia & Anxiety Inventory; SPAI-C, Social Phobia & Anxiety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

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.* (2012*b*) 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

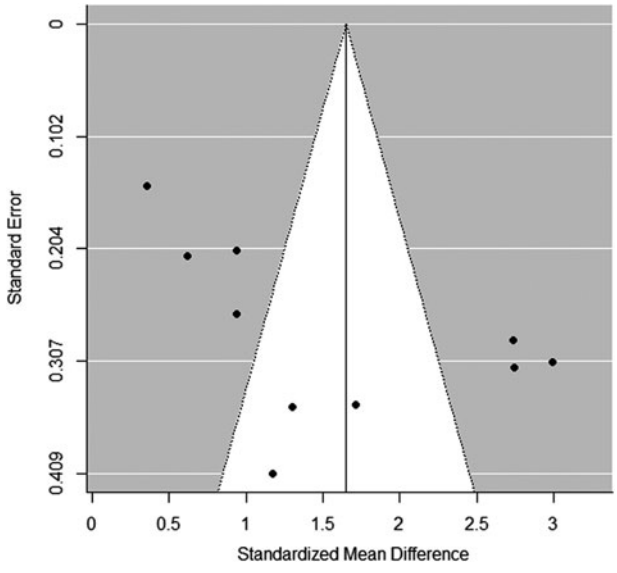

Fig. 3. Funnel plot of anorexia nervosa (AN) studies included in the meta-analysis to assess publication bias.

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 1 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 with 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 with 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

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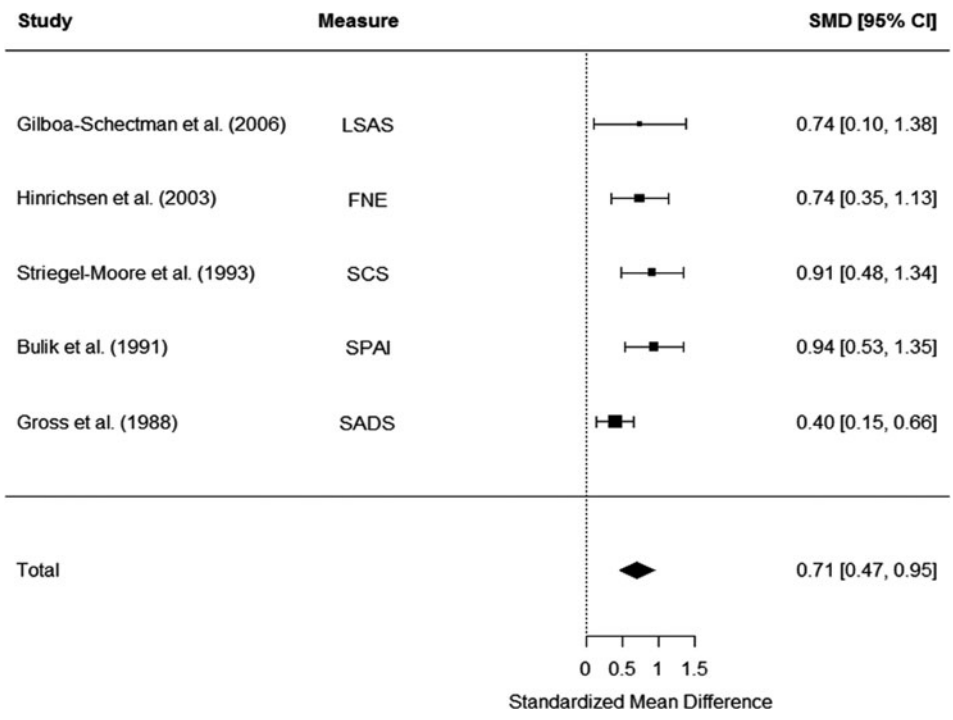

Fig. 4. Forest plot of standardized mean effect size for differences (SMD) between bulimia nervosa (BN) and healthy controls (HC). CI, confidence interval; FNE, Fear of Negative Evaluation scale; LSAS, Liebowitz Social Anxiety Scale; SADS, Social Anxiety and Distress Scale; SCS, Self-Consciousness Scale;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

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 with 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 (Mattar *et al.* 2012*a*, *b*). In addition, Mattar *et al.* (2012*a*) 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; Ostrovsky *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.

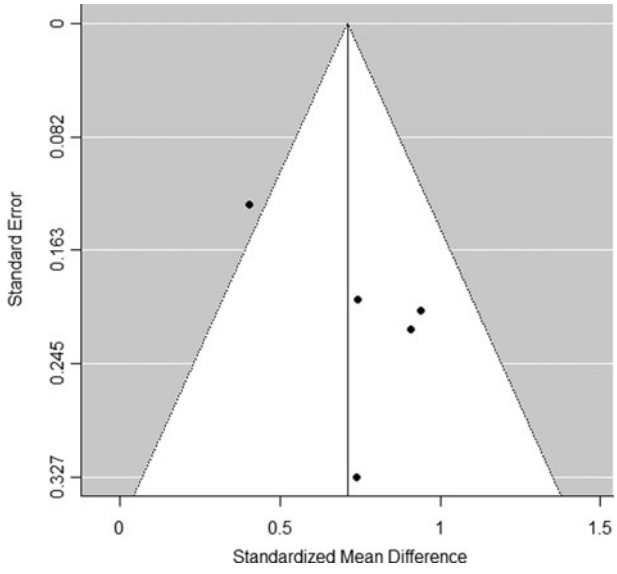

Fig. 5. Funnel plot of bulimia nervosa (BN) studies included in the meta-analysis to assess publication bias.

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; Ostrovsky *et al.* 2013). Similarly, in a mixed ED sample (AN, BN, or EDNOS), SA was significantly positively correlated with

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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.* 2004*a*, *b*; 2007*a*). This latter finding was partially replicated in a larger sample by Hinrichsen *et al.* (2007*b*), 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 with 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 with HCs, and provide a qualitative synthesis of the literature. There were significant differences in SA scores between both AN (10 studies) and BN (five studies) compared with 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 with HCs, finding that SA was also significantly elevated in individuals with BED compared with 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.* 2012*a*). 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 & Treasure (2013). This may indicate a more severe illness, as suggested by the positive association between SA and ED psychopathology (Hinrichsen *et al.* 2003, 2004*b*; Schulze *et al.* 2009; Sawaoka *et al.* 2012; Ostrovsky *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 (Schmelkin *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 *et al.*

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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.* 2017*a*). Further, they contribute to a growing evidence base documenting high levels of autism spectrum disorder (ASD) traits in those with AN (Westwood *et al.* 2017*b*). 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 with 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 with 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.

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Declaration of interest. None.

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