Research in Autism Spectrum Disorders 52 (2018) 51–68

Contents lists available at ScienceDirect

Research in Autism Spectrum Disorders

journal homepage: www.elsevier.com/locate/rasd

Social anxiety in autism spectrum disorder: A systematic review

Debbie Spain[a,b,*], Jacqueline Sin[c], Kai B. Linder[a], Johanna McMahon[d], Francesca Happé[a]

[a] Institute of Psychiatry, Psychology & Neuroscience, King's College London, United Kingdom
[b] South London & Maudsley NHS Foundation Trust, United Kingdom
[c] St George's, University of London, United Kingdom
[d] Australian National University, Australia

ARTICLE INFO

Number of reviews completed is 3

Keywords:
Autism spectrum disorders (ASD)
Social skills
Social motivation
Social anxiety (SA)
Social phobia
Systematic review

ABSTRACT

Purpose: Social anxiety (SA) commonly co-occurs with autism spectrum disorders (ASD). It is conceivable that inherent socio-communication impairments, or their impact on social experiences, contribute to the development of SA.

Method: We undertook a systematic review to summarise English-language research about relationships between core ASD symptoms and SA in individuals with ASD.

Results: We searched five databases for studies published up until 28 July 2017. Of 1481 publications retrieved, 24 cross-sectional studies (described in 25 papers) met the inclusion criteria. Given methodological and clinical heterogeneity, data were synthesised narratively. SA, in individuals with ASD, was associated with poorer social skills and functioning, and reduced social motivation. There were associations between self-report SA and ASD measures, but a trend towards non-significant relationships between parent-ratings of these symptoms. Tentative evidence indicated that SA symptoms were not associated with restricted, repetitive behaviours or sensory sensitivities.

Conclusion: These findings support the notion that there are links between core ASD characteristics and SA. Further studies, employing qualitative and quantitative designs are needed to enhance understanding of causal, maintaining and protective mechanisms for SA in ASD.

Autism spectrum disorders (ASD) are common lifelong neurodevelopmental conditions, characterised by qualitative impairments in social communication and interaction, engagement in rituals and routines, and hypo- or hyper-sensory sensitivities (APA, 2013). It is widely accepted that many young people and adults with ASD experience anxiety. In part due to the heterogeneous profile, there is debate about whether anxiety is best conceptualised as being derived of, or co-morbid to, ASD (see Kerns & Kendall, 2012). In either instance, data from a range of epidemiological and clinical samples, employing a range of data collection methods, consistently indicate that individuals with ASD have high rates of anxiety disorders (see van Steensel & Heeman, 2017).

Social anxiety (SA), also known as social phobia, is especially common, with prevalence estimates reported to be as high as 50% (Bellini, 2004; Maddox & White, 2015; Spain et al., 2016); substantially higher than estimates of 7–13% cited for the non-ASD population (NICE, 2013a). Disparities in prevalence estimates across studies may be attributable to a number of reasons, including differences in sampling and selection criteria (e.g. epidemiological vs. clinical samples), methods of assessment (e.g. self- vs. clinician-rated measures, or use of one vs. multiple measures), diagnostic overshadowing (whereby co-morbid symptoms are wrongly

* Corresponding author at: MRC Social, Genetic and Developmental Psychiatry Centre, Institute of Psychiatry, Psychology & Neuroscience, King's College London, De Crespigny Park, London, SE5 8AF, United Kingdom.
E-mail address: debbie.spain@kcl.ac.uk (D. Spain).

https://doi.org/10.1016/j.rasd.2018.04.007
Received 2 November 2017; Received in revised form 13 April 2018; Accepted 17 April 2018

attributed to ASD alone), or impairments in cognitive functioning (e.g. in introspection) which render it difficult for individuals with ASD to describe their internal states.

Hallmark characteristics of SA include autonomic symptoms of anxiety manifesting in specific or general social situations, a fear of negative evaluation or judgement by others, and avoidance of or escape from cues that evoke anxiety (APA, 2013; WHO, 1992). In non-ASD individuals, SA symptoms often emerge during adolescence with wide-ranging and long-term consequences. Causal and maintaining mechanisms for SA in neurotypical individuals are considered to be multi-faceted. These primarily comprise psychosocial and environmental factors, potentially underpinned by a genetic or biological predisposition (see Clark, 2001; Clauss & Blackford, 2012; Fox & Kalin, 2014; Rapee & Heimberg, 1997). Psychological frameworks for SA indicate that this may develop and be maintained by some or all of the following factors: an inhibited temperament; adverse social experiences during formative years; overestimation of the threat associated with social situations; negative beliefs about the self, others or the world; biases in information, attention and emotion processing; negative imagery; and 'safety behaviours' such as avoidance, mental rehearsal and post-event processing, which indirectly reinforce anxiety over time (Clark, 2001; Rapee & Heimberg, 1997).

It is possible that additional risk factors, specifically those relating to and arising from core ASD characteristics, contribute to the development of SA in individuals with ASD. Inherent socio-communication impairments may affect interactions and relationships in several ways. Social motivation, behavioural inhibition and volition to initiate overtures can influence the number, frequency and range of social situations individuals engage in. Further, the nature of responses to others, and degree of cooperativeness and turn-taking may influence the extent to which these are sustained. Social skills deficits may derail interactions with others. Stereotyped and idiosyncratic speech or preferences for discussing circumscribed interests may affect the fluidity of conversation. Repetitive behaviours, such as hand mannerisms or stereotyped body movements, may appear odd. Together, these characteristics can increase susceptibility to social adversity, e.g. rejection, teasing or bullying (Schroeder, Cappadocia, Bebko, & Weiss, 2014), and thereby contribute to social withdrawal and isolation. Moreover, difficult social interactions can give rise to negative ways of thinking, including paranoia and rumination (Spain, Sin, & Freeman, 2016), negative thoughts (e.g. about being the 'odd one out' or different), and, ultimately, core beliefs (schema) pertaining to inadequacy and inferiority.

Sensory sensitivities to light, sound or sensations (e.g. heat) may prove distracting or anxiety-provoking in social settings. Similarly, aversions to very specific sensory stimuli (Lord, Rutter & Le Couteur, 1994), may give rise to anticipatory anxiety about meeting familiar or unfamiliar others. Both sensory sensitivities and aversions may lead to avoidance. While avoidance may initially manifest in relation to specific settings, such as one particular supermarket, we have found in our clinical experience that this can become generalised, e.g. to all shops. Finally, a tendency for adhering to rituals and routines may hamper engagement in some social opportunities, or be remarked upon negatively by others, further contributing to misunderstandings and avoidance.

Bi-directionally, SA can encourage individuals with ASD to withdraw further from social interaction, thereby resulting in fewer occasions to observe social norms and conventions. As a consequence, these individuals may be less able to augment their social knowledge and social skills *in vivo*. Importantly, data from intervention studies tentatively indicate that SA may in fact partly moderate the success of social skills interventions. That is, individuals with ASD and SA may attain less favourable outcomes from such interventions due to the impact of these co-occurring anxiety symptoms (see Maddox, Miyazaki, & White, 2016; Pellecchia et al., 2016, Spain, Blainey, & Vaillancourt, 2017).

The aim of the present review is to systematically gather together, for the first time, the empirical data regarding relationships between ASD symptomatology and SA in individuals with ASD across the lifespan. This may elucidate more fully causal and maintaining mechanisms for SA with implications for prevention, early intervention and the development of more targeted treatments. Our review sought to answer the following question: What relationships are there, if any, between ASD and SA symptoms?

1. Method

1.1. Search strategy

We searched five databases – the Cochrane Central Register of Controlled Trials (CENTRAL), PsycInfo, Medline, PubMed, and Web of Science – for studies published until 28 July 2017. Search terms were autis* – Asperger* – development* disorder* AND social* anx* – social* phobi*. *A priori* inclusion criteria were: 1) English-language articles, published in peer-reviewed journals describing empirical quantitative research; 2) about SA or social phobia, and associations with core ASD symptoms in any of the domains outlined by either the ICD-10 (1992) or DSM-4/5 (1994, 2013); and 3) in children, adolescents or adults diagnosed with any subtype of ASD, with or without a concurrent intellectual disability (ID), and irrespective as to whether participants had had or were receiving treatment at the time of research participation. We excluded studies reporting the prevalence of SA, but which did not measure relationships between this and ASD, and those examining associations between anxiety and other variables, but where no SA subscale data were provided.

1.2. Study selection

Fig. 1 provides an overview of study selection. The database searches initially yielded 1481 reports. Duplicates (n = 166) were removed. Two authors (DS & JS) independently screened 1315 titles and abstracts. Of these, 81 articles were retrieved for full text review. Following discussion, 56 of these were excluded for the following reasons: not an ASD sample (n = 5), review paper (n = 3), treatment study (n = 3), study focused on general anxiety rather than SA specifically, and we could not extrapolate SA data (n = 24), and study examined aspects of SA in ASD, but did not focus on associations or relationships between these symptoms (n = 21). We