Abstract

The relationship between autistic traits, stress, and anxiety experienced by the general population was investigated using an adult sample that evaluated the suitability of three theoretical models proposed by Green and Ben-Sasson. Participants completed online questionnaires that were analysed using structural equation modelling and partial correlation analyses. Of the models tested, the model that proposed SOR and stress as mediators of the relationship between autistic traits and anxiety was able to explain the variance in the data better than the other models. Based on these findings, we suggest that sensory neutral environments should be considered for the prevention and management of anxiety and stress symptoms for people in the general population with higher levels of autistic traits.
Autism Traits, Sensory Over-Responsivity, Anxiety, and Stress: A Test of Explanatory Models.

Sensory over-responsivity (SOR) refers to the subjective experience of sensory overload that would otherwise be untroublesome to those without SOR (Liss, Saulnier, Fein, & Kinsbourne, 2006; Robertson & Simmonds, 2013). For example, people with SOR may find noises or lights that are tolerable to most people to be unbearable. SOR has been observed in Autism Spectrum Disorder (ASD). SOR and other sensory difficulties have more recently been recognised as a key DSM criterion (Diagnostic and Statistical Manual of Mental Disorders [DSM–5]; American Psychiatric Association [APA], 2013) despite previous recognition as an associated feature of the condition (Roberston, 2012). Discomfort caused by SOR impacts on the everyday quality of life experienced by those diagnosed with ASD and avoiding sensory stimulation can consume much effort and time (Robertson, 2012). The recognition that symptoms of ASD fall along a spectrum suggests there are those who are not diagnosed with the disorder who experience associated symptomology at a much milder level (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001). This investigation will examine individuals from the general population who vary in these characteristics to determine whether or not there is an association in the general population between SOR and subclinical autistic traits, as well as with levels of anxiety and stress.

Sub-clinical autism traits

It has been long recognised that individuals with ASD and undiagnosed family members share several behavioural characteristics (Kanner, 1943; Bailey et al., 1995; Happé, Briskman, & Frith, 2001; Piven, 2001; Sucksmith, Roth, & Hoekstra, 2011, Landry & Chouinard, 2016), which suggests a broader autism phenotype resulting from an interaction between genes and the environment. Importantly, this genetic liability is known to extend beyond family members and is distributed throughout the general population (Gaugler et al., 2014). These observations have led to the development and use of a number of tools for assessing the degree of autistic traits in the general population (Landry & Chouinard, 2016). Some of these tools include the Autism Spectrum Quotient (AQ) questionnaire (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001), the Social Responsiveness Scale (SRS) (Constantino & Todd, 2003), and the Broader Autism Phenotype Questionnaire (BAPQ; Hurley, Losh, Parlier, Reznick, & Piven, 2007). These autistic traits often
include rigidity, aloofness, impaired executive functioning, and atypical social behaviour, among other expressions of ASD tendencies and experiences (Ingersoll & Hambrick, 2011). Evaluating the effects of subclinical autistic traits on cognition in the general population has become a focus of considerable research (Ruzich et al., 2015) given that these relationships often mirror behavioural characteristics in clinical populations (Landry & Chouinard, 2016). This provides an opportunity to evaluate explanatory models (such as those below) that are relevant to clinical ASD in a more accessible sample from the general population.

Landry and Chouinard (2016) identify multiple benefits to using the broader autism phenotype and autistic traits model, specifically: easier access to more variable populations; populations that are able to endure longer testing sessions; greater control of co-morbidities; comparisons between chronological and mental ages; and finally, researchers can more easily examine the developmental sequence implicated in ASD in greater isolation. The study of subclinical autism samples is quite common; take for example, Ruzich et al. (2015) who in their systematic review identified 73 papers that had administered the Autism Quotient to neurotypical adults.

**SOR in ASD**

Compared to other symptoms and co-morbid conditions within ASD, SOR is a more specific manifestation that may render detection of causal relationships within ASD more straightforward. The latest version of the DSM now specifies hypo or hyper-sensitivity to sensory stimuli as one of the possible diagnostic criteria for ASD (APA, 2013) – underscoring the increased recognition that atypical sensory processing is frequently associated with ASD. Studies report that 42 to 88% of people with ASD experience SOR (Baranek, 2006; Kientz & Dunn, 1997; Osterling & Dawson, 1994; Tomchek & Dunn, 2007). People with ASD may demonstrate SOR to auditory (Jones et al., 2009), visual (Baranek, 1999), tactile (Kern et al., 2001) olfactory (Bennetto, Kuschner, & Hyman, 2007), and / or gustatory (Cermak, Curtin, & Bandini, 2010) stimuli. These types of SOR can be uncomfortable and in many cases debilitating. Daily noises, such as those in restaurants, shopping centres, and traffic, may cause distress as well those from a number of household appliances (Kern et al., 2001). Bright lights have also been reported to cause a great deal of discomfort (Grinter et al., 2009). In terms of SOR to tactile stimulation, the feeling of certain textures and materials against the skin (e.g. tags and button) can also
be quite disconcerting to people with ASD (Kern et al., 2001; Wilbarger & Wilbarger, 1991). Indeed, there have been reports of the avoidance of hygiene practices due to discomfort, including bathing, teeth brushing, and hair washing (Kern et al., 2001; La Vecchia et al., 1997). Similarly, SOR to gustatory and olfactory stimuli can lead to the avoidance of certain foods, particularly those with strong tastes and textures (Kern et al., 2001). Whilst there has been significant research about SOR in people with ASD, there has been little research into the relationship between SOR and autistic traits in the typically developing population.

**Anxiety in ASD**

Anxiety refers to symptoms such as restlessness, feeling on edge, being fatigued easily, difficulty concentrating, irritability, muscle tension, and apprehensive expectation (APA, 2013). Despite not being part of the major diagnostic criteria for ASD, elevated anxiety has been classified as an associated feature (APA, 2013). Similar to ASD, anxiety occurs on a continuum where individuals may experience higher levels than others (Cox, Endler, & Swinson, 1991). One major theory pertaining to anxiety is the presence of a multi-process model of cognitive vulnerability to anxiety, which relates to a dysfunction in any of the following processing models of anxiety – orientation, engagement, disengagement, avoidance, and interpretation (Ouimet, Gawronski, & Dozois, 2009). As these processes are typically interrupted in ASD, anxiety may be exacerbated in those who are already vulnerable (Ouimet et al., 2009).

Recent evidence has supported this claim by demonstrating that those diagnosed with ASD are at increased risk of anxiety disorders. When van Steensel, Bögels, and Perrin (2011) conducted a meta-analysis to determine the co-morbidity of different anxiety disorders in ASD, they found across 31 studies involving 2,121 young people that 39.6% of those diagnosed with ASD had at least one comorbid DSM-IV anxiety disorder.

**Stress in ASD**

Stress is a major area of interest within the field of ASD. Responses to stress in individuals diagnosed with ASD have been found to differ to responses in neurotypical individuals (Corbett, Mendoza, Abdullah, Wegelin, & Levine, 2006). Research has found that there are relationships between physiological and behavioral stress, and sensory sensitivity in autism (Corbett, Schupp, Levine, &
Mendoza, 2009). More recently, Corbett et al. (2016) investigated the impact of SOR on the physiological stress response in children with and without ASD and found that there were significant differences between the two groups. What has been less investigated is the causes of stress and whether or not stress is implicated in individuals with higher autistic traits in regard to SOR. It is important to note here that heightened sensitivity is a common response to stress in any susceptible individual, and for this research will be considered as such. However, it’s important to investigate what factors, and indeed what levels of these factors (such as autistic traits) impact upon an individual’s susceptibility to stress and how that interacts with the closely related construct, anxiety.

Models of anxiety and SOR in ASD

Emerging evidence of the association between SOR, anxiety, and stress in ASD has resulted in theoretical speculation regarding potential directional relationships. Green and Ben-Sasson (2010) proposed two explanatory models of the relationship between SOR, anxiety, stress, and ASD. In the first model, ASD produces anxiety and associated stress, which then results in SOR. This is referred to by Green and Ben-Sasson as the primary anxiety model. In the second model, ASD produces SOR, which in turn leads to perceptions of stress, which induces anxiety. This is referred to by Green and Ben-Sasson as the primary SOR model. They also consider the possibility that there is no direct causative relationship between SOR and anxiety, and that their presence is due to a common general risk factor. We refer to this possible situation in this study as the alternate hypothesis. In the next sections, we will introduce each of these models in more detail.

The primary anxiety model

The primary anxiety model is based on the premise that ASD is associated with elevated anxiety characterised by a state of hypervigilance (i.e. a heightened awareness to one’s surroundings) as part of the stress response (Green & Ben Sasson, 2010). Derryberry and Reed (2002) investigated the role of attentional bias in anxiety and demonstrated that an individual’s ability to regulate attention may be compromised in those with high trait anxiety. Further, participants with high anxiety traits experienced poorer attentional control when compared to participants with low anxiety traits. Poor attentional control was found to predict attentional bias, where participants with good control were better able to shift their attention away from threatening stimuli (Derryberry & Reed, 2002). A meta-analysis by Bar-
Haimm et al. (2007) including 172 studies relating to attentional bias found that those with anxiety traits were more likely to orient their attention to threats within the environment when compared to participants with low anxiety traits. Further, once participants’ attention was focused on a threat, participants with higher trait anxiety took significantly longer to reduce or shift their attention away from the threat when compared to participants with low trait anxiety. This model is particularly applicable to ASD populations due to the high co-morbidity of anxiety within the disorder (van Steensel, Bögels, & Perrin, 2011).

The primary SOR model

This model states that ASD produces SOR, which in turn produces stress, which then induces anxiety (Green & Ben-Sasson, 2010). It is a model based on learning theory – in particular classical conditioning. More specifically, SOR can result in an intense stress response to sensory stimuli, which may then induce states of anxiety after repeated pairing with an aversive stimulus, which may be more common in those diagnosed with ASD. For instance, if an individual is exposed to the loud noise of a popping balloon multiple times, they may eventually no longer require the loud noise associated with the popping of a balloon to elicit an anxiety response. The presence of the balloon alone may be enough to induce an anxiety response through the individual’s learned anticipation of that response. This may lead to a specific phobia, however, generalised anxiety may also be produced through context conditioning. As a result, the individual may learn to avoid certain locations or contexts where the aversive stimulus has occurred (e.g. avoiding parties, or carnivals). The uncontrollability of contexts that potentially involve the aversive stimuli may also contribute to states of anxiety (Green & Ben Sasson, 2010).

The alternate hypothesis

It is possible that SOR and anxiety are not directly causally related but are associated through a third variable such as overlapping diagnostic criteria. Specifically, SOR and anxiety may only appear to be correlated due to their shared diagnostic features (Green & Ben-Sasson, 2010). Both conditions are characterised by misperception of threat (Kallen et al., 2008) and hyper-activation of the HPA axis (e.g. Herman et al., 2003). The same measures are often used to quantify both anxiety and SOR. For example, cortisol levels have been used to assess both anxiety and SOR (Elnazerv & Baldwin, 2014;
Sindi, Fiocco, Juster, Pruessner, & Lupien, 2013). However, while cortisol levels can be useful for assessing whether or not an individual is in a state of stress, they cannot distinguish between SOR and anxiety. Furthermore, self-report measures of anxiety and SOR often include similar questions, which may inflate the relationship between these two factors (Green & Ben-Sasson, 2010). Thus, the relationship between anxiety and SOR may only be apparent due to the similarities in the measures as opposed to any true diagnostic overlap. Overall, this alternate hypothesis suggests that there is no causative relationship between SOR and anxiety, and that their common association with stress can explain the shared variability. This model provides a more simplistic interpretation of the relationships between SOR anxiety and ASD.

Assessing the accuracy of the competing models within ASD populations.

Following the publication of these competing models, research has focused on assessing their accuracy. Green, Ben-Sasson, Soto and Carter (2012) examined the relationship between SOR and anxiety in 149 toddlers with ASD where they examined the changes in anxiety and SOR symptoms over one year. To assess bidirectional effects over time they used a cross-lag panel analysis. They found a moderate, positive correlation between SOR and anxiety. Overall, SOR predicted changes in anxiety over time, demonstrating support for the primary SOR model. Additionally, their results indicated that anxiety did not predict changes in SOR, a key feature of the primary anxiety model. Limitations of this study include its heavy reliance on parent report scales. Parents’ anxiety state may influence their ability to assess their child’s experiences independently (Krain & Kendall, 2000). Additionally, the parent scale for measuring sensory sensitivity in this study was the Infant Toddler Social and Emotional Assessment which has internal consistency reliability statistics below .60 (Carter, Briggs-Gowan, Jones, & Little, 2003). Despite this, the longitudinal design allowed for sophisticated cross-lag panel analyses that can test for bi-directional effects between variables. This study provides preliminary evidence for the primary SOR model in toddlers with ASD.

Lane, Reynolds, and Dumenci (2012) expanded on the work by Green et al. (2012) by further testing the primary SOR model. They focused on a wider group of children including those with attention-deficit/hyperactivity disorder, ASD and typical development. A merit of the Lane et al. study is that it used physiological measures in conjunction with parent report measures. Physiological
measures were used to supplement parents’ perceptions of their child’s SOR by measuring galvanic skin response to sensory arousal. Furthermore, cortisol samples were collected pre and post to supplement parent-report child anxiety measures. Lane et al. explored the relationship between SOR and anxiety in children with ASD using a path analysis. They tested whether SOR was a mediator of the relationship between ASD and anxiety, confirming this relationship and providing evidence for the primary SOR model. Although the researchers collected data from three groups (attention-deficit/hyperactivity disorder, ASD, and typically developing), there was no attempt to co-vary for condition, nor to compare the applicability of the model to the different conditions. Despite not testing all three of the models proposed by Green and Ben-Sasson, they did provide additional empirical support for the primary SOR model. Although, without comparing all models it is difficult to accept conclusive support about the explanatory ability of each competing model.

Mazurek et al. (2013) who found that self-reported anxiety was a strong predictor of SOR in children with an ASD have recently presented further evidence for SOR exacerbating anxiety. A limitation of their study is that it focused only on correlations and made no attempt to test for direction of the relationship or for mediation. Collectively, the evidence provided above outline strong support for the primary SOR model.

Collectively, the aforementioned studies carried out in ASD populations suggest converging support for what may be a function of autism traits in the general population; however, the degree to which certain symptoms apply in the general population is largely unknown. Nonetheless, Aron, Aron, and Davies (2005) have provided some evidence in favour of the primary anxiety model. Namely, Aron et al. showed that participants with high hyper-responsivity were more prone to experience anxiety when compared to hypo-responsivity participants, providing some evidence for SOR eliciting anxiety symptoms in adults. However, their study did not attempt to validate this effect within ASD or ASD trait populations, and merely postulated the relationship between anxiety and SOR.

*Assessing the accuracy of anxiety models in autistic traits*

Despite a growing literature on anxiety and SOR in ASD populations (e.g. Green, Ben-Sasson, Soto, & Carter, 2012; Lane, Reynolds, & Dumenci, 2012; Mazurek et al., 2013), little research has been carried out to understand SOR, anxiety, and autistic traits relationships in the general population. A
study by Robertson and Simmonds (2013) explored directly whether or not there was a correlation between autistic traits and SOR. To assess the relationship between autistic traits and SOR, they created an SOR questionnaire designed specifically for use with the general population. This allowed them to detect the subtler differences in sensory processing that are usually not detected using questionnaires designed for clinical SOR populations (Robertson & Simmonds, 2013). Along with a measure of autistic traits, they applied their newly developed measure to test whether or not a relationship exists between autistic traits and SOR in a sample of 212 participants recruited online. Results showed a significant strong positive correlation between autistic traits and frequency of SOR, providing evidence for the applicability of the primary SOR model in autistic traits (Robertson & Simmonds, 2013). Horder, Wilson, Mendez, and Murphy (2014) investigated whether or not anxiety correlated with SOR and autistic traits. They found that autistic traits positively correlated with anxiety symptoms and, using partial correlations, reported that anxiety partially explained the relationship between autistic traits and SOR. However, even after controlling for anxiety, the relationship between SOR and autistic traits remained stable, thus providing evidence against the primary anxiety model.

Aims and hypotheses

Our study builds on this existing literature by identifying how those in the general population experience SOR and its specific relationship with anxiety and stress. An analysis of how the specific models of SOR and anxiety apply in the general population, especially in those with high autistic traits, is novel and may have practical implications for reducing anxiety and stress in general population areas such as the workforce, schools and other environments that are susceptible to causing over-stimulation.

The present investigation expands on this initial work from these earlier studies by evaluating how theoretical models proposed by Green and Ben-Sasson (2010) can explain the variance in an adult sample drawn from the general population with different degrees of autistic traits.

Our aim was to evaluate the theoretical models proposed by Green and Ben-Sasson (2010) by quantifying the strength of associations between autistic traits, SOR, stress, and anxiety in an adult sample from the general population using SEM, which has never been examined before. In addition, we felt it was important to consider the alternate hypothesis that Green and Ben-Sasson also proposed. The following hypotheses were tested. Based on the primary anxiety model, it was hypothesised that
the relationship between autistic traits and SOR would be serially mediated by anxiety and stress, in a manner depicted in Figure 1a. Based on the primary SOR model, it was hypothesised that the relationship between autistic traits and anxiety would be serially mediated by SOR and stress, in the manner illustrated in Figure 1b. Based on the alternate hypothesis, it was predicted that the relationship between SOR and anxiety would be non-significant when the effects autistic traits and stress were controlled. For this hypothesis to be accepted, autistic traits and stress would need to account completely for the relationship between SOR and anxiety.

Method

Participants

Prior to commencement of the study, approval was acquired from our university’s research ethics board. The current study utilised combined data from two waves of recruitment. Wave one was conducted from June to August of 2014, and wave two was conducted from June to August of 2015. The method for recruitment was consistent between the two waves. Participants were recruited through the circulation of posters throughout our university, community noticeboards, and local organisations and as such is a convenience sample. Additionally, electronic advertisements were circulated through online social networking platforms such as Facebook or e-mail. Due to a significantly larger number of female cases, we decided to use a subset of the female data that was collected over the two waves to equalise the amount of females and males in the final dataset. We randomly selected female cases to form the subsample using the random selection function in SPSS. T-tests were conducted on the scores of the major variables and found no statistically significant differences between the initial and final samples. The final sample utilised the combined data from both waves of recruitment and included 458 participants ($n = 229$ female; $M_{\text{age}} = 30.61$ years; $SD = 12.88$), age range: 18-76 years).

For further information on demographic data and differences across the two samples, see supplementary materials. Advertisements directed participants to the online questionnaires hosted by the Qualtrics website via a link (Qualtrics, Provo, UT, USA). Participants provided information relating to age, gender, education level, employment status, relationship status, and family member diagnosis of ASD. This was followed by the three counter-balanced questionnaires taking approximately 20
minutes to complete in total. Informed consent was obtained from all individual participants included in the study.

Materials

To quantify autistic traits, we administered the Broad Autism Phenotype Questionnaire (BAPQ; Hurley, Losh, Parlier, Reznick, & Piven, 2007). The BAPQ is a 36 item, self-report measure, divided into three subscales designed to assess the major aspects of the expression of ASD traits in non-autistic relatives of individuals with ASD. More recently, the BAPQ has been used in general population samples to detect autistic traits (Ingersoll, Hopwood, Wainer, & Donnellan, 2011). The BAPQ utilises a Likert design with item responses ranging from 1= “very rarely” to 6= “very often”, where extreme scores are indicative of greater autistic trait expression. Possible scores range from 36 to 216. Participants were instructed to keep in mind the following instructions whilst completing the questionnaire: (1) to consider interactions they had with people in general rather than special relationships immediate family members or their partner; (2) to consider their behaviour across their entire adult life rather than specific time periods or instances; and (3) to guess if they were unsure how to answer. The BAPQ has demonstrated internal consistency with Cronbach alpha coefficients ranging from .85-.95 among the subscales (aloof personality, rigid personality, and pragmatic language) (Hurley et al., 2007). Research using exploratory factor analysis has found that the factor loadings of the BAPQ adequately encompass the full range of autistic trait expression (Ingersoll et al., 2011).

To quantify SOR, we administered the Glasgow Sensory Questionnaire (GSQ; Robertson & Simmonds, 2013). The test consists of 42 self-report items that quantify both hyper- (SOR) and hypo-sensitivities (Sensory under-responsivity, (SUR)) in seven sensory areas: visual; auditory; gustatory; olfactory; tactile; vestibular and proprioceptive. Items are equally distributed among sensory domains consisting of three questions evaluating reported SOR and three determining SUR. Whilst the entire GSQ was administered, only the SOR subscale was analysed.

A Likert scale is used to determine how frequently certain sensory events are experienced with responses ranging from 0= “Never”, 1= “Rarely”, 2= “Sometimes”, 3= “Often”, and, 4= “Always”, and possible scores ranging from 0 to 168. Possible total scores range from 0 to 84 for each subscale and higher scores are indicative of greater abnormal sensory experiences. Participants were instructed to
consider their experiences or behaviours that occurred within the last 12 months. The GSQ has demonstrated good internal consistency with a Cronbach alpha coefficient of .94 and a split half reliability statistic of .93 (Robertson & Simmonds, 2013).

To quantify stress and anxiety, we administered the Depression, Anxiety, and Stress Scales 21 (DASS-21; Lovibond & Lovibond, 1995b). This test is a 21 item, self-report measure of depressive symptoms, anxiety, and stress. For the current study the entire DASS-21 was administered, however only the anxiety and stress subscales were analysed. The stress scale (Stress) was used to measure stress symptoms and the anxiety scale (Anxiety) was used to measure anxiety symptoms. Respondents were required to indicate how often they experienced each state within the last week. Items were scored using a Likert scale with responses ranging from 0 = “did not apply to me at all” to 3 = “applied to me very much, or most of the time”. Higher scores are indicative of more severe symptomology. Possible total scores range from 0 to 21 for each subscale. Several studies have reported good internal consistency with Cronbach alpha coefficients exceeding .88 for both Stress and Anxiety scales (Henry & Crawford, 2005). For all scales, high sensitivity and specificity percentages have been reported (Lovibond & Lovibond, 1995a).

**Statistical analysis**

Statistical analyses were performed using SPSS 22.0 for Windows. Pearson product moment correlations ($r$) were conducted between the two subscales of the GSQ (SOR and SUR), the anxiety scale of the DASS-21, and the BAPQ to measure the strength and direction of relationships between the variables. All subsequent analyses co-varied for gender given that gender differences are typically present in measures designed to quantify autistic traits. As previous studies have found gender differences when testing for autistic traits (Baron-Cohen et al., 2001, Constantino & Todd, 2003), we will use multiple group analysis, a form of structural invariance, to test for gender effects.

**Using full SEM to fit models**

In this study, a full structural equation model (SEM) approach using AMOS version 23 was used to analyse the primary anxiety and SOR models. Unlike standard path analysis where summative scores (constructs) form the variables of interest, SEM allows the actual measured variables to be included in the analysis with the constructs entering the model as latent variables. A major advantage
of this approach is a substantial increase in the degrees of freedom available for assessing overall model fit. For the models under consideration, all direct and indirect (mediated) effects among the constructs are included which, in standard path analysis, yields saturated models for which no meaningful measure of fit can be calculated (Byrne, 2016).

We used partial correlation analyses to test hypothesis 3 evaluating the Alternate hypothesis. This allows testing to see if the remaining variables remain significantly related after controlling for stress and autistic traits.

Results

Correlations amongst variables

A summary of means, Pearson correlation coefficients, standard deviations, and internal reliability statistics for the DASS-21, BAPQ, GSQ and their subscales is displayed in Table 1. This table indicates that all correlations were significant and the pattern of the correlational data (i.e. positive / negative) were in the expected directions.

The path coefficients and p-values for testing path significance, squared multiple correlations ($R^2$) values for endogenous variables and overall model fit measures of Models 1 and 2 are presented in Table 2. Note that both models include the same latent and measured variables and the same path structure and are therefore equivalent for the purpose of overall model fit (Kline, 2010). The only difference between the models is the direction of paths (arrows). In the primary anxiety model, the paths finish at SOR, as anxiety is the ‘primary (i.e. occurs first, before SOR) along the path. Whereas, in the primary SOR model, SOR is the ‘primary (i.e. occurs first, before anxiety) along the path.

Overall Model Fit Measures

The fit measures consisted of a root mean square error of approximation (RMSEA) of 0.071, a comparative fit index (CFI) of 0.676, a Tucker-Lewis index (TLI) of 0.666, a normed fit index (NFI) of 0.592, and a $\chi^2/df = 3.229$ (Figure 2). These fit measures indicate that the models overall do not fit the data well. For model fit to be considered acceptable, the RMSEA should be less than 0.06, the CFI, the TLI and the NFI should all be above 0.9 and $\chi^2/df$ should not exceed 3 although this measure tends to be overly sensitive to sample size (Schreiber, Nora, Stage, Barlow, & King, 2006).
A possible reason for this relatively poor model fit may reside in the autistic trait construct. Investigation of the measurement model for autistic traits reveals poor fit (RMSEA = 0.118, CFI = 0.495, TLI = 0.464, NFI = 0.459, $\chi^2/df = 7.107$) with many item path coefficients being non-significant. For a validated instrument, this is surprising and may indicate an issue with the validity of the scale when applied using the current approach. The BAPQ was developed by Hurley, Losh, Parlier, Reznick, and Piven (2007) using a sample of 86 parents of autistic children and 64 control parents with no reported history of autism in first degree relative. Hurley et al. (2007) concede some bias in the instrument due to the selectivity of their recruitment practices and recommend large-scale studies of the general population to address the issue. In the current study, we used a sample of 55 participants with a diagnosed autistic family member and 403 without. This represents a significant mismatch in sample balance compared to the Hurley et al. (2007) sample and may be a source of the lack of fit found in the autistic traits measurement model. Although the above discussion identifies an important limitation of this study, it does not prevent us from proceeding with analysis provided this limitation is kept in mind when interpreting results.

Since the fit of both models was relatively poor, we decided to examine their structural invariance with respect to gender and broader autism phenotype status. This was carried out using multiple group analysis in AMOS where models were compared using a chi-squared difference test (Kline, 2010). For the primary SOR and primary anxiety models, no significant differences in fit (Bonferroni adjusted p-values > 0.05) between the gender unconstrained model and the measurement weight or structural weight invariant models were observed. A similar approach was attempted using broader autism phenotype status as the grouping variable, however group sample size issues prevented model estimation. Thus, variation due to gender at least, does not provide an explanation for the poor fit of the models.

**Mediation Effects**

Mediation effects are calculated by multiplying the relevant path coefficients in Table 2. For example, in the primary anxiety model the mediated effect of autistic traits via anxiety and stress on SOR is $0.317 \times 0.694 \times 0.370 + 0.317 \times 0.270 + 0.033 \times 0.370 = 0.179$. Testing the significance of an indirect effect is carried out within AMOS using a bias corrected bootstrap approach (Cheung &
Lau, 2008). Table 2 contains all estimated indirect effects and associated $p$-values for the primary anxiety and SOR models. Except for the Anxiety-Stress-SOR pathway which is significant at the $p < 0.05$ level all other mediation effects were significant at the $p < 0.01$ level.

**Primary Anxiety Model**

The primary anxiety model predicts that the relationship between autistic traits and SOR would be mediated by anxiety and stress. In the presence of mediation, this reduces to a zero autistic traits-SOR direct path coefficient. From Table 2, this coefficient is 0.058 with a $p$-value of 0.007 which leads to the conclusion that autistic traits and SOR are not fully mediated by anxiety and stress. The total effect of autistic traits on SOR is $0.058 + 0.179 = 0.237$ and so the direct effect represents 24.5% of this total effect. Thus, autistic traits and SOR are only partially mediated by anxiety and stress and the hypothesis is clearly rejected.

**Primary SOR Model**

The primary SOR model predicts that the relationship between scores on BAPQ and Anxiety would be mediated by SOR and stress. In the presence of mediation, this reduces to a zero autistic traits-Anxiety direct path coefficient. Figure 2, this coefficient is 0.001 with a $p$-value of 0.957 which leads to the conclusion that autistic traits and Anxiety are fully mediated by SOR and stress. Thus, autistic traits and Anxiety are mediated by SOR and stress and the hypothesis is confirmed.

**The Alternate Hypothesis**

The hypothesis relating to the alternate model predicted that the correlation between Anxiety and SOR would become non-significant after controlling for BAPQ scores, gender, and Stress in a partial correlation analysis. The relationship between anxiety and SOR was then subjected to a first-order partial correlation in order to explore the relationship controlling for the effects of stress and autistic traits. The original correlation between anxiety and SOR was $r = .83, p < .01$. After controlling for the effects of autistic traits and stress, the relationship remained significant, $r = .51, p < .01$, indicating that a relationship between anxiety and SOR exists above and beyond the effects of stress and autistic traits, but that the relationship is lessened; that is, stress and autistic traits affects both anxiety and SOR and is closely related to the two.

**Discussion**
The present investigation aimed to determine which of three models proposed by Green and Ben-Sasson (2010) accounts best for relationship between SOR, Anxiety and autistic traits in the adult general population. The models that were tested consisted of: 1) the primary anxiety model, 2) the primary SOR model, and 3) the alternate hypothesis. We used structural equation modelling and partial correlation analyses to test these models. It was determined that the second model explained the variance in the data better than the first and third models. In the ensuing discussion, we will comment on the overall model fit as well as discuss how well each model explained the variance in the data and how the results obtained with previous studies examining ASD compared with the results from this study. We will then end by discussing practical implications and suggest avenues for future research.

**Overall model fit**

The fit measures indicate that the models overall did not fit the data well. To confirm that the weakness in overall fit was not due to gender effects or the autistic trait construct, we examined the data’s structural invariance with respect to gender and broader autism phenotype status. As there were no significant differences in the model fit after controlling for gender, gender, at least, does not provide an explanation for the relatively poor fit of the model. We also attempted to investigate the impact of broader autism phenotype status on model fit, however group sample size issues prevented model estimation. Being unable to test for the impact of the autistic trait construct is indeed a limitation, however it does not necessarily indicate that it is contributing to the weakness in overall fit. While the overall fit is an important limitation of the current study, it did not prevent us from continuing with the individual models, and that is why we have applied a conservative approach when interpreting the results of the individual models.

**Primary anxiety model**

The primary anxiety model predicts that the relationship between BAPQ and SOR would be mediated by anxiety and stress. This hypothesis was not supported, as the direct effect remained significant after the inclusion of anxiety and stress into the model, although it is important to note that the magnitude of this significance was quite low. Although there has been some previous support for the primary anxiety model (Aron et al., 2005), the present investigation only found partial support for this model as the relationship between autistic traits and SOR remained significant after accounting for
anxiety and stress. The results found by this study are similar to those reported by Horder et al. (2014), who found the relationship between autistic traits and SOR remained stable, even after controlling for anxiety. Therefore, it may be concluded for this study, that the primary anxiety model proposed by Green and Ben-Sasson (2012) was not supported.

Primary SOR model

The primary SOR model predicts that the relationship between scores on BAPQ and Anxiety would be mediated by SOR and stress. This hypothesis was supported as the relationship between autistic traits and anxiety became non-significant after accounting for the effects of SOR and stress. These findings support previous attempts by Green et al. (2012) to assess the directional relationship between anxiety and SOR in children with ASD. Using longitudinal data, Green et al. (2012) found that SOR produced increased anxiety over time and that anxiety did not produce SOR. In addition, this study corroborates the findings of Lane et al. (2012) who, using path analyses, found preliminary evidence that SOR may have a causal relationship with anxiety. The current study also builds on the work by Horder et al. (2014), who found correlational evidence of a relationship between anxiety, SOR, and autistic traits. Taken together, Green and Ben-Sasson’s (2010) primary SOR model provides a strong account for explaining the relationships between autistic traits, SOR and anxiety in the adult general population.

The alternate hypothesis

The alternate hypothesis predicts that the correlation between anxiety and SOR would become non-significant after controlling for BAPQ scores and stress. This hypothesis was partially supported as the relationship was reduced from a significant correlation representing a large effect size \( r = .83 \) to a significant correlation representing a moderate effect size \( r = .51 \) according to Cohen (1988). This indicates that, although the relationship between anxiety and SOR remained significant, controlling for autistic traits and stress reduced the strength of the relationship. For this hypothesis to be accepted, autistic traits and stress would need to account completely for the relationship between SOR and anxiety. This is consistent with the alternate hypothesis proposed by Green and Ben-Sasson (2010) predicting that the relationship between anxiety and SOR can be partly explained by their common relationship with stress. While the strength of the relationship did reduce, it remained significant, which
indicates that the correlation between the two variables cannot be completely explained by their common association with stress. The level of shared variability detected is expected due to how heightened sensitivity typically leads to a trivial amount of stress in susceptible individuals. Therefore, there is some support for the relationship between anxiety and SOR, which may be explained by the crossover of diagnostic symptomology, this may explain the variance not accounted for by the accepted Model 2.

Comparison to existing literature.

Green, Ben-Sasson, Soto, and Carter (2012) examined changes in anxiety and SOR symptoms over one year in 149 toddlers with ASD that provided preliminary evidence for the primary SOR model and is in agreement with the results obtained in the present investigation. Green et al. (2012) relied on parents’ reports of their child’s behaviours where the current utilised a general population sample that overcame these limitations by allowing the individual to report their own experiences rather than relying on parents’ reports.

Further research by Lane et al. (2012) expanded on the work by Green et al. (2012) and found evidence for the primary SOR model in children with attention-deficit/hyperactivity disorder, ASD, and typically developing children. Similarly, to the current study, Lane et al. (2012) explored these relationships using path analysis. The difference between path analysis and SEM is that path analysis assumes all variables are measured without error and SEM uses latent variables to account for measurement error (Kline, 2010). Despite not testing all three of the models proposed by Green and Ben-Sasson, they did provide additional empirical support for the primary SOR model, which corroborates well with our findings. Our results also agree with those of Mazurek et al. (2013) who found that self-reported anxiety was a strong predictor of SOR in children with an ASD.

The results of the current study are in contrast with results obtained by Aron et al. (2005) who found that participants with high hyper-responsivity were more prone to experience anxiety when compared to hypo-responsivity participants. Whilst they provided some evidence for the primary anxiety model, their study did not attempt to validate this effect within ASD or ASD trait populations, and merely postulated the relationship between anxiety and SOR.
Our study built on this existing literature by identifying how those in the general population experience SOR and its specific relationship with anxiety and stress. An analysis of how the specific models of SOR and anxiety apply in the general population, especially in those with high autistic traits, is novel and may have practical implications for reducing anxiety and stress in general population areas such as the workforce, schools and other environments that are susceptible to causing over-stimulation.

Practical implications for ASD and autistic traits

Together, this research has implications for individuals diagnosed with ASD and for members of the general population who experience high autistic traits. Research investigating SOR in clinical ASD may be restricted to using smaller samples, as access to people with ASD is limited when compared to accessing neurotypical individuals (Landry & Chouinard, 2016). Accessing large samples is important in detecting small effect sizes, where recruiting from the general population allows for access to larger samples. If autistic traits are used as a model to understand ASD (Landry & Chouinard, 2016) then the results of the present investigation are suggestive that a substantial proportion of co-morbid anxiety in ASD is driven by SOR. In light of our findings, we recommended the development of techniques and interventions to alter the individual’s level of sensitivity or to reduce extreme sensory environments.

There are some techniques currently available for individuals diagnosed with ASD that aim to alter sensitivity to different sensory modalities. For example, Sensory Integration Therapy aims at exposing an individual systematically to different kinds of sensory experiences, like, hair brushing, wearing a weighted vest, sitting on an inflated ball, riding on a scooter (Lang et al., 2012). It is thought that exposure to sensations can reduce an individual’s hyper-responsivity over time as they accustom to the sensory experiences (Lang et al., 2012). Alternatively, alterations could be made to the environment; for example, the use of neutral tones for wall colourings, weighted blankets during sleep, and certain fabrics for clothing (Arbesman & Lieberman (2010). The research from this current study indicates that individuals with high autistic traits from the general population could also benefit from these applications.

Recently there has been a growing interest surrounding the potential negative outcomes associated with high autistic traits; however, interest has yet to expand to SOR. Evidence provided by
the current study suggests that there may be a causal relationship between SOR and anxiety in those with high autistic traits, which may have implications for many people. In particular, school (Sheild, Greenland, & Dockrell, 2010) and workplace environments (Stiff, 2012) may benefit by reducing extreme sensory environments in an attempt to reduce autistic traits-induced anxiety.

The recent popularity of open plan learning environments in schools that encourage a ‘community’ vibe within the classroom may cause those with high levels of autistic traits to experience elevated anxiety (Sheild, Greenland, & Dockrell, 2010). While there are many personality types that may benefit from open interactive classrooms, the results of the current study indicate that they may not be beneficial for those with high autistic traits. Therefore, those with high autistic traits should be considered when schools adopt open plan learning environments. The inclusion of sensory neutral areas such as private, quite spaces, should be provided for those students who experience anxiety due to sensory over-responsivity (Sheild, Greenland, & Dockrell, 2010).

The findings of this study also have implications for the workplace. Workplace environments that are loud and utilize bright fluorescent lighting may increase anxiety levels in workers who have higher autistic traits. Anxiety has been linked to decreased productivity in the workplace (Haslam, Atkinson, Brown, & Haslam, 2005). Therefore, the adoption of subtler lighting, neutral wall colourings, and measures to reduce noise may assist in increasing productivity, whilst making the work environment less anxiety inducing for workers with higher autistic traits (Stiff, 2012).

The benefits of less extreme sensory environments have been demonstrated recently, where cognitive functioning was found to be improved in sensory neutral, natural environments compared to sensory extreme, urban environments (Berman, Jonides, & Kaplan, 2008). The findings of the current study support these conclusions by indicating a stable relationship between SOR and anxiety. As a large portion of the population may experience autistic trait-related SOR (Robertson, 2012), the adoption of sensory neutral areas (e.g. parks) within cities should be encouraged to reduce anxiety, which may be caused by sensory extreme environments associated with inner city living.

Limitations of the study

In agreement with the Horder et al. (2014), Robertson and Simmonds (2013) as well as the Green et al. (2012) studies, our results provide evidence that the autistic traits produces SOR, which
results in heightened levels of stress and anxiety. Our study is the first to examine the relationship between SOR, anxiety, stress, and autistic traits through structural equation modelling. Nonetheless, future experiments would be useful to corroborate certain aspects of our findings, particularly concerning causation.

A major limitation of the current study is the different statistical approach used for the alternate hypothesis compared to those used to measure the primary SOR and primary anxiety models. This is in part due to the theoretical nature of the alternate hypothesis and its ability to fit clearly into the other models. In our study, we have provided one possible interpretation of Green and Ben-Sasson’s (2010) theoretical models and we acknowledge that it is possible there will be other interpretations. Whilst we have attempted to do this as accurately as possible, due to the complex nature of autism spectrum disorders there may be some other factors that affect the etiology of these symptoms, especially in the less researched broader autism phenotype. We recommend that future experiments consider models that can incorporate all of Green and Ben-Sasson’s (2010) models equally in a way that allows for similar statistical methods.

This study utilised self-report measures, which are vulnerable to selective recall, telescoping, limited insight, misattribution, and can rarely be independently verified (Stone et al., 2000). These biases can either diminish or exacerbate one’s perception of their actual anxiety levels (Barbosa, Tannock, & Manassis, 2002). For example, high anxiety may have crossover with questions on the BAPQ such as “I avoid public places”, and this may have accounted for high correlations between the two scales. Moreover, there is arguably some overlap of the content in the GSQ-SOR subscale and the BAPQ. This means that the variance detected in both anxiety and stress may be caused by autistic traits, indicating a potential issue of common input. It will be important for future studies to repeat the same procedures in a more controlled environment and obtain objective measures using electrophysiological/physiological measures to shed light on the difference between autistic and non-autistic groups regarding the stress reaction and anxiety. These additional methods would also allow for a closer examination of the alternate hypothesis to determine more clearly the degree of variance explained by overlapping stress in SOR and anxiety.
Although sampling bias is a consideration for this form of recruitment, we implemented the following strategies to control and reduce this bias as far as practically possible. The current method of recruitment is consistent with other correlational research in this field (see Horder et al., 2014) and responses on the BAPQ and GSQ are consistent with other studies looking at the general population. To negate the bias whereby individuals interested in autism specifically seek out autism related studies, our recruitment posters referred to this study as a “personality questionnaire” with no mention of autism at all. Furthermore, autistic traits items were included in the questionnaire amongst a battery of other unspecified personality characteristics. These strategies will have reduced an autism-related self-selection bias, although the authors recognise that certain demographic groups (i.e. female, young) are more likely to respond to university and social media-based recruitment (Correa, Hinsley & De Zuniga, 2010). We attempted to control for such a bias by employing a sex-balanced sample, in that we included only a random a sub-set of females from the initial sample. Importantly, t-tests conducted found that there were no significant differences in the scores between the initial and final sample of females. The remaining age-related sampling bias may be in fact be beneficial in detecting this specific phenomenon as young adults represent a segment of the population who typically have healthy brains. This works in our favour, as noise creating, extraneous variables are reduced, such as co-morbid conditions and age-related factors like cognitive decline.

Moreover, we used a cross-sectional design, which does not allow one to infer causation to the same degree as longitudinal studies (Preacher & Hayes, 2008). This is particularly important when considering the results of this study, as the primary anxiety model and primary SOR model imply a causative serial effect. The results of this study are limited to making inferences as to which factor (SOR or Anxiety) has a stronger indirect effect for deducing possible causal relationships. Thus, we recommend that future research in this area use similar longitudinal procedures to Green et al. (2012) to test the models and establish causation in those with higher autistic traits from the general population. A further limitation of this study is that if focused specifically on the models put forward by Green and Ben-Sasson (2010), this means that not all possible partial correlations or models were directly tested. Other, more under-explored models, could potentially better explain the relationships between autistic traits, SOR, anxiety and stress. In addition to this point, a limitation of assessing autistic traits in the
general population within Green and Ben-Sasson’s framework is that their models were originally based on research pertaining to clinical ASD in children. While the approach used in the current study to utilize a BAP population is a valid (Landry & Chouinard, 2016), it is important to note that there may be a discontinuity between the linearly observed pattern of ASD-like behaviours commonly observed in a clinical context and the pattern of those same behaviours within the general population. Further, it is likely that these models potentially oversimplify the presentation of autistic traits in the general population, as multiple personality and environmental factors may also contribute to the development of both anxiety and SOR.

Taken together, the next stage in terms of providing further evidence for the models proposed by Green and Ben-Sasson (2010) may occur in several ways. Further support of the primary SOR model could be undertaken by utilising physiological measures of anxiety and SOR provide stronger reliability to the model. These, incorporated with an experimental or longitudinal design, would provide stronger evidence for the true relationship between anxiety, SOR, stress and autistic traits in the general population.

Conclusion

The current study contributes to the understanding of the relationships between SOR, stress and anxiety in regard to autistic traits in a nonclinical sample. The findings provide support for the theoretical model of this relationship in which SOR associated with autistic traits leads to stress and anxiety as proposed by Green and Ben-Sasson (2010). Findings suggest a role for the introduction of sensory neutral environments within schools, workplaces, and other extreme sensory environments to reduce anxiety symptoms often associated with high autistic traits. However, longitudinal studies are required to examine possible sources of causality more directly. Further research into the negative outcomes associated with autistic traits, in particular SOR and anxiety, is important as sensory problems can have serious and widespread negative effects on an individual's life and wellbeing. It is suggested that the models produced by Green and Ben-Sasson be further refined through the introduction of other influential factors on the relationship between SOR anxiety and autistic traits.
Compliance with Ethical Standards

Funding: This study was funded by a La Trobe Understanding Disease Research Focus Area grant.

Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

References


Kanner L (1943), Autistic disturbance of affective contact. *Nerv Child* 2, 217-250


Table 1: Correlations among major variables.

<table>
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<tr>
<th>Measure</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>Sample M</th>
<th>Sample SD</th>
<th>GP M</th>
<th>GP SD</th>
<th>AS M</th>
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<td></td>
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<td></td>
<td>76.53</td>
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<td></td>
<td></td>
<td>38.31</td>
<td>17.32</td>
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<td>*</td>
<td>*</td>
<td>*</td>
</tr>
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<td>3. SUR</td>
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<td>(-0.91)</td>
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<td></td>
<td></td>
<td></td>
<td>38.37</td>
<td>16.22</td>
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<td></td>
<td></td>
<td></td>
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<td>.81*</td>
<td>.78*</td>
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<td>.69*</td>
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<td>.66*</td>
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*Numbers within the parentheses are alpha reliabilities *p < .001, GP = general population (DASS-21: Crawford, Cayley, Lovibond, Wilson, & Hartley, 2011; GSQ full scale: Roberston et al., 2013; BAPQ: Hurley et al., 2007). ASD = autism spectrum population (DASS-21: Zimmerman, Ownsworth, O'Donovan, Roberts, & Gullo, 2016; GSQ full scale: Tavassoli, Miller, Schoen, Nielsen, & Baron-Cohen, 2014; BAPQ: Hurley et al., 2007). SOR and SUR not available for general population or autism-spectrum population, DASS-21 total score not available for autism-spectrum population.
Figure 1. Interpretation of Green and Ben-Sasson (2010) models of SOR and anxiety: (a) Primary Anxiety Model; (b) Primary SOR Model.
Figure 2. Primary Anxiety and SOR model diagrams with key values included. Note that measured variables and error terms are excluded for clarity. Model Fit Measures RMSEA = 0.071; CFI = 0.676; TLI = 0.666; NFI = 0.592; $\chi^2$/df = 3.229.