Published in final edited form as:
J Autism Dev Disord. 2012 June ; 42(6): 1112–1119. doi:10.1007/s10803-011-1361-3.

Anxiety and Sensory Over-Responsivity in Toddlers with Autism Spectrum Disorders: Bidirectional Effects Across Time

Shulamite A. Green,
Department of Psychology, University of California, Los Angeles, Los Angeles, CA, USA

Ayelet Ben-Sasson,
Department of Occupational Therapy, Faculty of Social Welfare and Health Sciences, University of Haifa, Mount Carmel, Haifa, Israel

Timothy W. Soto, and
Department of Psychology, University of Massachusetts, Boston, Boston, MA, USA

Alice S. Carter
Department of Psychology, University of Massachusetts, Boston, 100 Morrissey Boulevard, Boston, MA, USA

Alice S. Carter: alice.carter@umb.edu

Abstract

This report focuses on the emergence of and bidirectional effects between anxiety and sensory over-responsivity (SOR) in toddlers with autism spectrum disorders (ASD). Participants were 149 toddlers with ASD and their mothers, assessed at 2 annual time points. A cross-lag analysis showed that anxiety symptoms increased over time while SOR remained relatively stable. SOR positively predicted changes in anxiety over and above child age, autism symptom severity, NVDQ, and maternal anxiety, but anxiety did not predict changes in SOR. Results suggest that SOR emerges earlier than anxiety, and predicts later development of anxiety.

Keywords

Autism spectrum disorders; Anxiety; Sensory over-responsivity

Introduction

Autism spectrum disorders (ASD) are characterized by three symptom clusters: (1) impairments in communication, (2) impairments in social interaction, and (3) repetitive or stereotyped behaviors (American Psychiatric Association [DSM-IV-TR] 2000). In addition to these core symptoms, children with ASD often exhibit related symptoms or symptom clusters, which can significantly increase their functional impairment. One such symptom cluster is sensory over-responsivity (SOR), an often clinically impairing condition characterized by heightened and unusual reactivity to sensations (Ben-Sasson et al. 2010),

Correspondence to: Alice S. Carter, alice.carter@umb.edu.

which occurs more frequently among children with ASD (e.g., Baranek et al. 2006) than among typically developing children (e.g., Ben-Sasson et al. 2008). Anxiety disorders and symptoms are also elevated in the ASD population (Gadow et al. 2004; Kim et al. 2000; Muris et al. 1998; Weisbrot et al. 2005), and often co-occur with SOR (Ben-Sasson et al. 2009; Liss et al. 2006; Pfeiffer et al. 2005). However, the reason for the co-occurrence of SOR and anxiety is unknown, and there is little research on the early development of SOR and anxiety symptoms in young children with ASD. Examining the emergence of anxiety and SOR as well as their relationship over time in very young children with ASD can increase our understanding of potential reciprocal relationships between these two conditions (Green and Ben-Sasson 2010).

Although there is normative variation in sensory responsiveness, SOR is manifested by extreme or unusual negative reactions to sensory stimuli such as noisy or visually stimulating environments, seams in clothing, or being touched unexpectedly (Dunn 1999; Interdisciplinary Council on Developmental and Learning Disorders [ICDL] 2005; Lane 2002; Parham and Mailloux 2005; Reynolds and Lane 2008). Regulating sensory responses to the environment is an ability one develops early in life as a protective and discriminative mechanism (Dunn 1997). It has been hypothesized that children who over-respond to sensory stimuli have a low neurological threshold resulting in strong reactions to such stimuli with minimal input (Pfeiffer et al. 2005), reflecting a failure in achieving a balance between sensitization and habituation (Dunn 1997).

As a primary condition, SOR is considered a type of sensory modulation disorder (SMD) in which an individual presents with exaggerated, intense, and/or prolonged responses towards certain sensations relative to same-age peers (Miller et al. 2007), and has been described in children (Stagnitti et al. 1999) as well as adults (Kinnealey and Fuiek 1999). Reports of SOR rates in children with ASD vary, but studies indicate elevated rates of about 61% (Schoen et al. 2009) to 70% (Baranek et al. 2006) in children with ASD relative to rates of 10–17% in the general population (Ben-Sasson et al. 2009; Ben-Sasson et al. 2008). Moreover, SOR is impairing. SOR may be characterized by avoidance, anxiety, aggression and/or defiance, may reduce psychological well-being (Kinnealey and Fuiek 1999), and is associated with increased functional impairment in children with ASD, including lower levels of social and adaptive skills (Liss et al. 2006; Pfeiffer et al. 2005), negative emotionality (Ben-Sasson et al. 2009), and over-focused attention (Liss et al. 2006).

SOR has been linked to anxiety in children with ASD (Ben-Sasson et al. 2009; Liss et al. 2006; Pfeiffer et al. 2005), another co-morbid clinical condition that is elevated in the ASD population (Gadow et al. 2004; Kim et al. 2000; Muris et al. 1998; Weisbrot et al. 2005). Anxiety is also quite impairing for children with ASD; it is related to greater deficits in social competence (Bellini 2004; Pfeiffer et al. 2005; Sukhodolsky et al. 2008) and functional academics (Pfeiffer et al. 2005), as well as higher levels of externalizing behaviors (Kim et al. 2000). Thus, while ASD is often a severely disabling disorder on its own, both SOR and anxiety may cause even greater deficits, with potential implications for prognosis. Examination of the relationship between SOR and anxiety early in development therefore has potential implications for the treatment of children with ASD.

One potential explanation for the association between anxiety and SOR is overlapping genetic risk. For example, it is possible that parental anxiety or depression might contribute genetic risk to both anxiety and SOR, thus partially accounting for the correlation between anxiety and SOR. Furthermore, most of the studies on SOR and ASD are based upon maternal report. Maternal affect, such as anxiety or depression, may reduce the ability to accurately recall a child's behavior. This hypothesis is supported by evidence indicating that maternal depression and anxiety are associated with increased reporting of emotional and behavioral symptoms in children (Briggs-Gowan et al. 1996). Thus, in the current study we included maternal symptoms of depression and anxiety as covariates in our model to control partially for both genetic contribution and potential response biases that could account for the relationship between anxiety and SOR in children with ASD.

In previous work describing the sensory profile of the current sample at the first study assessment, we found that 56% of the sample showed high frequencies of SOR behaviors relative to existing norms for typical children. In addition, there was a subgroup of toddlers at Time 1 with extreme SOR scores who also had higher levels of anxiety relative to the other subgroups. However, overall the sample at Time 1 showed low levels of anxiety (Ben-Sasson et al. 2007, 2008). This work provoked questions regarding the course of this relation over time and whether the pattern of association might intensify if children's anxiety symptoms increased over time. Specifically, studying the emergence of each condition in young children as well as simultaneously examining whether each one predicts change in the other can help us understand the direction of effect, given that it is as yet unclear what reciprocal patterns (if any) exist between anxiety and SOR (Green and Ben-Sasson 2010). Furthermore, studying predictors of the development of each is important to prevention and early intervention for children with ASD.

Therefore, the purpose of this study was to examine the association between anxiety and SOR symptoms in young children with ASD, specifically: (1) to examine correlations between symptoms of anxiety and SOR (2) to examine changes in anxiety and SOR symptoms across one year and (3) to investigate the extent to which anxiety predicts changes in SOR and SOR predict changes in anxiety over a 1-year time interval. A cross-lag panel analysis was used to test for these potential bidirectional effects.

Methods

Participants

Participants were 149 toddlers (mean age = 28.3 months, SD = 5.5) who were enrolled in a longitudinal study of developmental trajectories of young children and their families (for additional details see Carter et al. 2007). All children had been diagnosed with autism or Pervasive Developmental Disorder—Not Otherwise Specified (PDD-NOS). Diagnoses were assigned or confirmed using the Autism Diagnostic Interview-Revised (ADI-R; Lord et al. 1994), the Autism Diagnostic Observation Schedule-Generic (ADOS-G; Lord et al. 2000), and a clinical psychologist's clinical impression. One hundred and eighteen (79.2%) of the children were boys and 31 girls (20.8%). The sample was very diverse with respect to autism severity, cognitive functioning and adaptive behavior (see Table 1). One hundred and twelve (75.2%) of participants met or exceeded the ADOS social-communication cut-off for

Autism, the remaining 37 (24.8%) met ADOS criteria for ASD. The participants were predominantly White (85.2%) and upper- to upper-middle class. Some mothers had a graduate level degree (24.2%), a majority had two or more years of college education (58.9%), 16.2% had high school degrees or had completed vocational training programs and 1 participant did not report her education level. Almost all mothers were married or living with their partner (91.9%). Children were excluded from the study if they had been diagnosed with a known genetic disorder such as Fragile X, a medical disorder that could impact cognitive functioning (e.g., lead poisoning), or a physically handicapping condition.

Procedures

Participating families were recruited through early intervention providers, physicians specializing in the diagnosis of ASD, local conferences, and events for families of children with ASD. Families with children between 18 and 33 months who had a diagnosis of an ASD or were suspected of having a diagnosis of ASD were invited to participate in the study. Families completed two visits in each assessment year: a child visit, which took place in a laboratory setting, and a parent interview, which was completed in either an office setting or the parents' home. Parents were also asked to complete a questionnaire booklet which included among other measures the Infant-Toddler Social and Emotional Assessment ITSEA; Carter and Briggs-Gowan 2005) Beck Anxiety Inventory (BAI; Beck et al. 1988), and the Center for Epidemiological Studies—Depression Scale (CES-D; Radloff 1977). Families were followed annually with the same child assessments, interviews, and questionnaires.

The child session included the Autism Diagnostic Observation Schedule-Generic (ADOS-G; Lord et al. 2000) and the Mullen Scales of Early Learning (Mullen 1995). During the parent visit the Autism Diagnostic Interview-Revised (ADI-R; Lord, et al. 1994) and the Vineland Adaptive Behavior Scales (VABS; Sparrow et al. 1984) were administered. Parents were compensated $50 for participating in the larger study. All children were assessed for the first time between 18 and 33 months (Time 1) and for the second time one year later (Time 2).

Measures

Autism Diagnostic Interview-Revised (ADI-R; Lord et al. 1994)—The ADI-R is an investigator-based, semi-structured informant interview for the diagnosis of ASD. Current research supports the use of the ADI-R with children with a mental age of less than 18 months (Lord et al. 2006; Risi et al. 2006). Items are coded on a zero to three scale, with zero indicating no ASD-specific atypical behavior present, and three indicating extremely atypical behavior. Consistent with Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; APA 1994) and ICD-10 diagnostic guidelines, the algorithm focuses on three areas: communication, social, and restricted and repetitive behaviors. Established cut-off scores for each area have been shown to adequately discriminate autistic individuals from a mental-age matched non-autistic comparison group of participants with language impairment and/or mental retardation (Lord et al. 1994). The measure yields acceptable internal consistency for each subscale: Social, alpha of .95; Restricted and Repetitive Behaviors, alpha of .69; Verbal alpha of .85; Communication alpha of .84. Diagnostic research criteria proposed by the Collaborative Programs of Excellence in Autism for young

children were employed (Lord et al. 2006; Risi et al. 2006). Specifically, a classification of autism spectrum disorder was given to individuals who met criteria for autism on either the Social or Communication domains and were within two points on the other. A score of three or greater on the restricted and repetitive behaviors domain was required for a diagnosis of autism, but was not required for a diagnosis of PDD-NOS.

Autism Diagnostic Observation Schedule-Generic (ADOS-G; Lord et al. 2000)—The ADOS-G is a semi-structured, interactive observation designed to assess social and communicative functioning in individuals who may have an ASD. One of four appropriate developmental modules is used, based on the child's language level. At Time 1, children in this study were administered either Module 1 (preverbal or single words; n = 142) or Module 2 (phrase speech; n = 7). The assessment involves a variety of social presses. Established cut-off scores are used to differentiate autism, autism spectrum disorder, and non-autism spectrum participants. Inter-rater reliability on the ADOS-G was .90.

Mullen Scales of Early Learning (Mullen 1995)—The Mullen Scales of Early Learning provide an assessment of cognitive functioning. For this study, raw scores on the Visual Reception and Fine Motor Skills scales were standardized and summed to derive a measure of nonverbal developmental functioning (NVDQ) at each assessment point. Similarly, raw scores on the Receptive and Expressive Language scales were used to derive a measure of verbal developmental functioning (VDQ). Each developmental quotient score is derived based on the child's age, and has a mean of 100 and a standard deviation of 15. The Mullen has good psychometric properties: Internal consistency is .91 for the composite score, and ranges in value from .75 to .83. for the domain scores The 1- to 2-week test–retest reliability ranged in value from .71 to .96. (Mullen 1995).

The Infant Toddler Social and Emotional Assessment (ITSEA; Carter and Briggs-Gowan 2005)—Child anxiety and sensory sensitivity were measured using mothers' report on General Anxiety (e.g., Seems nervous, tense, or fearful; Feels sick when nervous or upset; Worries about own body) and Sensory Sensitivity (e.g., Won't touch some objects because of how they feel; Is bothered by loud noises or bright lights; Is bothered by certain odors) scales of the ITSEA at Times 1 and 2. This is a parent report measure of social-emotional and behavioral problems and competencies in infants and toddlers. Parents use a 3-point response format to rate their child's behavior in the past month from 0 'not true/rarely' to 2 'very true/often'. The ITSEA has strong psychometric properties (Carter et al. 2003). Internal consistency in a community sample was .71 for General Anxiety and .63 for sensory sensitivity. Internal consistency for the current sample was .59 for General Anxiety and .71 for Sensory Sensitivity.

The Center for Epidemiological Studies—Depression Scale (CES-D; Radloff 1977)—The 20-item CES-D scale assesses an individual's depressive symptoms. Items are rated on a 4-point scale based on the frequency with which the item has been experienced in the previous week. Scores are then summed to create an overall depression rating. A score of 16 or higher signifies the risk of clinical depression. The CES-D has an established alpha of .85 in a general community sample. Internal consistency for the current sample was .88.

Beck Anxiety Inventory (BAI)—The BAI (Beck et al. 1988) is a widely-used self-report measure of physiological and cognitive symptoms of anxiety. This measure was used as an indicator of the parent's anxiety on a continuous scale. The respondent is asked to rate the degree to which he or she has "been bothered by these feelings" (e.g., nervous; fear of losing control) in the past week. The BAI has 21 items with answers given on a 4-point scale of 0 (not at all) to 3 (severely); scores of 10 or above are considered indicative of above average anxiety. Internal consistency (alpha) is .92 and 1-week test–retest correlation is .75 (Beck et al. 1988). Internal consistency in this sample was .86.

Results

Descriptives

Mean scores for all covariates and main study variables are displayed in Table 1. A paired-samples t-test was used to examine change over time in child anxiety and SOR symptoms. Mean anxiety scores increased significantly from Time 1 to Time 2, $t(148) = -3.00$, $p = .003$ but mean SOR scores were stable across time, $t(148) = -1.10$, $p = .275$. The percent of children over the clinical cutoff (approximately 10th percentile) for these subscales showed a similar pattern: 8.3 and 13.5% were over the cutoff for anxiety and 22.6 and 23.6% were over the cutoff for SOR at Time 1 and Time 2, respectively.

Correlations Among Variables

SOR and anxiety were significantly correlated both at Time 1 ($r = .52$, $p < .001$) and Time 2 ($r = .60$, $p < .001$). Prior to testing primary hypotheses regarding associations between anxiety and sensory sensitivity symptoms, relations between anxiety and SOR symptoms with child age, verbal developmental quotient (VDQ), nonverbal developmental quotient (NVDQ), autism social-communication symptom severity on the ADOS, and mother's depression and anxiety were examined to determine inclusion as covariates in cross-lag models. Child age was correlated with ITSEA anxiety scores at Time 1 ($r = .20$, $p = .017$). Child ADOS severity was correlated with both ITSEA anxiety and SOR scores negatively: anxiety at Time 1 ($r = -.21$, $p = .01$) and Time 2 ($r = -.22$, $p = .01$), and sensory scores at Time 1 ($r = -.22$, $p = .01$) and Time 2 ($r = -.16$, $p = .048$). Child NVDQ was correlated with ITSEA anxiety scores at Time 1 ($r = .16$, $p = .04$) and Time 2 ($r = .29$, $p < .001$). Mothers' depression scores at Time 1 were correlated with ITSEA anxiety scores at Time 2 ($r = .20$, $p = .02$), and ITSEA sensory scores at Time 2 ($r = .21$, $p = .01$). Mothers' anxiety scores were correlated with ITSEA anxiety scores at Time 2 ($r = .23$, $p = .005$), and with ITSEA sensory scores at Time 1 ($r = .24$, $p = .002$) and Time 2 ($r = .28$, $p = .001$). Thus, child age, NVDQ, and autism socialcommunication symptom severity, as well as mothers' depression and anxiety scores, were tested as covariates in the cross-lag models. To be conservative, covariates that were significant at $p < .1$ were included in the final model.

Cross-Lag Analyses

Cross-lagged panel analyses were conducted in Mplus to examine the bidirectional effects between child anxiety and SOR between Time 1 and Time 2. This analysis simultaneously examined the two predictions of interest (Time 1 anxiety predicting Time 2 SOR, controlling for Time 1 SOR, and Time 1 SOR predicting Time 2 anxiety, controlling for

Time 1 anxiety). In this type of analysis, both dependent variables (anxiety and SOR) are entered into the model and allowed to correlate, so it is more conservative than a traditional regression approach, as it accounts for multicollinearity between the two dependent variables.

Of the covariates tested, only child NVDQ and mother's anxiety scores were significant in predicting either anxiety or SOR symptoms at $p < .10$, so only these covariates were retained in the final model.

Figure 1 shows the cross-lagged panel analysis. Model fit was extremely good (RMSEA < .001; TLI = 1.00). There was high stability across the two time points for anxiety ($B = .64$, $p < .001$) and for SOR ($B = .55$, $p < .001$). Child NVDQ at Time 1 significantly predicted change in anxiety ($B = .003$, $p = .002$) but not change in SOR ($B = .001$, $p = .632$). Mother's anxiety at Time 1 significantly predicted change in anxiety ($B = .007$, $p = .011$) but only marginally predicted change in SOR ($B = .008$, $p = .059$). There was a significant cross-lagged effect from Time 1 SOR to Time 2 anxiety (controlling for Time 1 anxiety; $B = .11$, $p = .026$), but Time 1 anxiety did not predict Time 2 SOR (was not significant ($B = .17$, $p = .214$). These results suggest that SOR predicts change in anxiety across toddlerhood, but anxiety does not predict change in SOR.

Discussion

The purpose of this study was to examine relations between anxiety and SOR symptoms in young children with ASD across one year of early childhood (i.e., from 18–33 months to 30–45 months). SOR remained relatively stable over time, but anxiety significantly increased. More children showed clinically concerning elevations in SOR relative to those who showed clinically concerning elevations in anxiety, particularly in Year 1. Child NVDQ at Time 1 significantly positively predicted increases in anxiety, which is consistent with previous studies showing higher rates of anxiety in higher-functioning children with ASD (e.g., Sukhodolsky et al. 2008). While VDQ predicted changes in anxiety when entered into the model on its own, it did not predict over and above NVDQ, suggesting that it is overall developmental level rather than verbal ability that is related to the development of anxiety. A higher developmental level may lead to greater awareness of the environment, and thus greater understanding and anticipation of potentially aversive events.

None of the covariates included predicted change over time in SOR scores. This may be partially due to the fact that SOR was relatively stable over this one-year time period. Covariates such as autism severity and developmental level might be predictive of changes in SOR over a greater period of time. Alternatively, it is possible that social-communication symptoms and NVDQ are not important predictors of SOR. Rather, SOR may be related to a different underlying neuropathology (e.g., amygdala or thalamus dysfunction; Green and Ben-Sasson 2010; Hardan et al. 2008; Hitoglou et al. 2010). While child social-communication symptom severity and mothers' depression scores were all correlated with child anxiety scores at Time 1, they did not predict increases in anxiety. This is likely due to the high correlation between the autism severity scores and child NVDQ, and between mothers' depression and anxiety scores. The ASD diagnostic domain of stereotyped

behaviors was not included in the model as stereotyped behaviors may serve as a coping mechanism to regulate arousal or affective state (such as anxiety). The direct overlap and perhaps causal relationship of stereotyped behaviors with anxiety and/or SOR is left for future inquiry.

Using a cross-lag model, we simultaneously tested SOR as a predictor of change in anxiety and anxiety as a predictor of change in SOR. Results indicated that SOR positively predicted increases in anxiety, while anxiety did not predict changes in SOR. These results suggest that SOR emerges earlier than anxiety, and support the theory that SOR may increase the risk of developing anxiety or exacerbate a predisposition for anxiety. For example, an over reaction to the sensory stimulus may generalize to a whole environment or situation through context conditioning, thus contributing to hypervigilance and anxiety (Green and Ben-Sasson 2010). Both conditions might also stem from a common underlying neuropathology such as amygdala hyperactivity; in that case these results would suggest that SOR is a developmentally earlier manifestation of amygdala hyperactivity, while anxiety is a later manifestation.

The stability of SOR over a one-year period is in line with data from non-clinical samples (e.g., Dunn 2002; Ben-Sasson et al. 2010) indicating persisting levels of SOR across early childhood. Additionally, findings from this study suggest that clinical levels of SOR are also stable across time, as about 23% of the current sample showed clinically concerning levels of SOR at both time points. There is contradicting evidence from cross-sectional ASD studies showing increases (Talay-Ongan and Wood 2000) and decreases in SOR (Kern et al. 2007) over an older and wider age period. Discrepancies in design, measures, and age range greatly limit comparability.

Mothers' anxiety at Time 1 was also positively predictive of increases in anxiety over and above NVDQ, age, and autism severity. Maternal anxiety can be situational, provoked by the challenges of raising a child with autism, dealing with a new diagnosis and negotiating services. In addition, maternal anxiety can be a trait that the mother brings into parenthood.

This study was unique in that it took a longitudinal approach to examining the bidirectional relationship between anxiety and SOR. However, this study did have some limitations. Both anxiety and SOR were measured by mothers' report on the ITSEA. It is possible that parents are better able to recognize their children's anxiety as their children age and become better able to communicate their anxiety. Thus; the findings that anxiety increased over time could be partially explained by the fact that anxiety was measured through parent report.

Demographic risk may contribute to the presentation of stress in young children and their mothers independent of ASD. Since this sample consisted of a fairly demographically homogeneous and low risk group of families it was not possible to analyze the role of socioeconomic status and race in the course of symptoms of children with ASD and SOR. There is some evidence suggesting elevated risk for the combination of SOR and externalizing rather than internalizing behaviors in children from lower socioeconomic status (Ben-Sasson et al. 2009); however future research is needed to examine this issue in ASD.

It was not possible to use the Sensory Profile (Dunn 1999) as the children ranged in age from 18 months to 45 months, and thus different children received different versions of this measure. In previous work with this sample, Sensory Profile data at Time 1 suggested a much higher rate of SOR (Ben-Sasson et al. 2007) thus the ITSEA may underestimate SOR. Especially because findings from this study suggest that SOR emerges at a young age in children with ASD, there is a need for a consistent measure of SOR that can be used across childhood. In addition, this study included a 1-year follow-up of anxiety and SOR, but these conditions likely develop and change throughout the lifespan, and may interact with or exacerbate each other over time. Future studies should examine the relationship between anxiety and SOR over a longer period of time, especially given elevated rates of anxiety in adolescents and young adults with ASD (Lecavalier 2006).

These findings have implications for intervention with young children with ASD. Regardless of whether anxiety emerges later than SOR or parents are not able to recognize anxiety in very young children, it appears that SOR predicts the development of anxiety in children with ASD. Families of young children with ASD and SOR may benefit from psychoeducation about the development of anxiety and preventative interventions for anxiety. Likewise, because SOR and anxiety are so highly correlated, and especially if SOR exacerbates anxiety, older children with ASD may benefit from the combination of interventions for SOR and anxiety. Such an approach has not yet been examined, but evidence that sensory treatments (e.g., deep pressure or other types of regular sensory input) may also reduce anxiety (Edelson et al. 1999; Pfeiffer and Kinnealey 2006) suggests that an integrated approach may be beneficial.

Acknowledgments

This research was conducted as part of the Studies to Advance Autism Research and Treatment (STAART) center at Boston University. Funding was provided by National Institute of Mental Health grant U54 MH 66398 (Helen Tager-Flusberg, Center Principal Investigator, Alice S. Carter, Project Principal Investigator) as well as grants from the National Alliance for Autism Research (Alice S. Carter, Principal Investigator) and the Boston University General Clinical Research Center. We are grateful to the families of the children in this study, whose participation in our project inspires this work and makes it possible.

References

American Psychiatric Association. Diagnostic and statistical manual of mental disorders (DSM-IV). Washington, DC: American Psychiatric Association; 1994.

American Psychiatric Association. Diagnostic and statistical manual of mental disorders (DSM-IV-TR). 4. Washington, DC: American Psychiatric Association; 2000.

Baranek GT, David FJ, Poe MD, Stone WL, Watson LR. Sensory experience questionnaire: Discriminating sensory features in young children with autism, developmental delays, and typical development. Journal of Child Psychology and Psychiatry. 2006; 47(6):591–601. [PubMed: 16712636]

Beck AT, Epstein N, Brown G, Steer RA. An inventory for measuring clinical anxiety: Psychometric properties. Journal of Consulting and Clinical Psychology. 1988; 56(6):893–897. [PubMed: 3204199]

Bellini S. Social skill deficits and anxiety in high-functioning adolescents with autism spectrum disorders. Focus on Autism and Other Developmental Disabilities. 2004; 19(2):78–86.

Ben-Sasson A, Carter AS, Briggs-Gowan MJ. Prevalence and correlates of sensory over-responsivity from infancy to elementary school. Journal of Abnormal Child Psychology. 2009; 37(5):705–716. [PubMed: 19153827]

Ben-Sasson A, Carter AS, Briggs-Gowan MJ. The development of sensory over-responsivity from infancy to elementary school. Journal of Abnormal Child Psychology. 2010; 38(8):1193–1202. [PubMed: 20623174]

Ben-Sasson A, Cermak SA, Orsmond GI, Carter AS, Kadlec MB, Dunn W. Extreme sensory modulation behaviors in toddlers with autism. American Journal of Occupational Therapy. 2007; 61(5):584–592. [PubMed: 17944296]

Ben-Sasson A, Cermak SA, Orsmond GI, Tager-Flusberg H, Kadlec MB, Carter AS. Sensory clusters of toddlers with autism spectrum disorders: Differences in affective symptoms. Journal of Child Psychology and Psychiatry. 2008; 49(8):817–825. [PubMed: 18498344]

Briggs-Gowan MJ, Carter AS, Schwab-Stone M. Discrepancies among mother, child, and teacher reports: Examining the contributions of maternal depression and anxiety. Journal of Abnormal Child Psychology. 1996; 24(6):749–765. [PubMed: 8970908]

Carter AS, Black D, Tewani S, Connolly C, Kadlec MB, Tager-Flusberg H. Sex differences in toddlers with autism spectrum disorders. Journal of Autism and Developmental Disorders. 2007; 37(1):86–97. [PubMed: 17216333]

Carter, AS.; Briggs-Gowan, MJ. The infant-toddler social and emotional assessment (ITSEA) manual. San Antonio, TX: Harcourt Assessment; 2005.

Carter AS, Briggs-Gowan MJ, Jones SM, Little TD. The infant-toddler social and emotional assessment (ITSEA): Factor structure, reliability, and validity. Journal of Abnormal Child Psychology. 2003; 31:495–514. [PubMed: 14561058]

Dunn W. The impact of sensory processing abilities on the daily lives of young children and their families: A conceptual model. Infants and Young Children. 1997; 9(4):23–35.

Dunn, W. Sensory profile user's manual. San Antonio, TX: Psychological Corporation; 1999.

Dunn, W. The infant/toddler sensory profile: User's manual. New York: Psychological Corporation; 2002.

Edelson SM, Edelson MG, Kerr DCR, Grandin T. Behavioral and physiological effects of deep pressure on individuals with autism: A pilot study investigating the efficacy of Grandin's 'Hug Machine'. American Journal of Occupational Therapy. 1999; 53:145–152. [PubMed: 10200837]

Gadow KD, DeVincent CJ, Pomeroy J, Azizian A. Psychiatric symptoms in preschool children with PDD and clinic and comparison samples. Journal of Autism and Developmental Disorders. 2004; 34(4):379–393. [PubMed: 15449514]

Green SA, Ben-Sasson A. Anxiety disorders and sensory over-responsivity in children with autism spectrum disorders: Is there a causal relationship? Journal of Autism and Developmental Disorders. 2010; 40(12):1495–1504. [PubMed: 20383658]

Hardan AY, Minshew NJ, Melhem NM, Srihari S, Jo B, Bansal R, et al. An MRI and proton spectroscopy study of the thalamus in children with autism. Psychiatry Research: Neuroimaging. 2008; 163(2):97–105.

Hitoglou M, Ververi A, Antoniadis A, Zafeiriou D. Childhood autism and auditory system abnormalities. Pediatric Neurology. 2010; 42(5):309–314. [PubMed: 20399382]

Interdisciplinary Council on Developmental, Learning Disorders. Diagnostic manual for infancy and early childhood. Bethesda: ICDL; 2005.

Kern JK, Trivedi MH, Grannemann BD, Garver CR, Johnson DG, Andrews AA, et al. Sensory correlations in autism. Autism. 2007; 11(2):123–134. [PubMed: 17353213]

Kim JA, Szatmari P, Bryson SE, Streiner DL, Wilson FJ. The prevalence of anxiety and mood problems among children with autism and Asperger syndrome. Autism. 2000; 4(2):117–132.

Kinnealey M, Fuiek M. The relationship between sensory defensiveness, anxiety, depression and perception of pain in adults. Occupational Therapy International. 1999; 6(3):195–206.

Lane, SJ. Sensory modulation. In: Bundy, AC.; Lane, SJ.; Murray, EA., editors. Sensory integration: Theory and practice. 2. Philadelphia: FA. Davis; 2002. p. 101-122.

Lecavalier L. Behavioral and emotional problems in young people with pervasive developmental disorders: Relative prevalence, effects of subject characteristics, and empirical classification. Journal of Autism and Developmental Disorders. 2006; 36(8):1101–1114. [PubMed: 16897387]

Liss M, Saulnier C, Fein D, Kinsbourne M. Sensory and attention abnormalities in autistic spectrum disorders. Autism. 2006; 10(2):155–172. [PubMed: 16613865]

Lord C, Risi S, DiLavore PS, Shulman C, Thurm A, Pickles A. Autism from 2 to 9 years of age. Archives of General Psychiatry. 2006; 63:694–701. [PubMed: 16754843]

Lord C, Risi S, Lambrecht L, Cook EH, Leventhal BL, DiLavore PC, et al. The autism diagnostic observation schedule-generic: A standard measure of social and communication deficits associated with the spectrum of autism. Journal of Autism and Developmental Disorders. 2000; 30(3):205–223. [PubMed: 11055457]

Lord C, Rutter M, Couteur A. Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. Journal of Autism and Developmental Disorders. 1994; 24(5):659–685. [PubMed: 7814313]

Miller L, Anzalone M, Lane S, Cermak S, Osten E. Concept evolution in sensory integration: A proposed nosology for diagnosis. American Journal of Occupational Therapy. 2007; 61:135–140. [PubMed: 17436834]

Mullen, EM. Mullen scales of early learning. Los Angeles: Western Psychological Corp; 1995. (AGS Ed.)

Muris P, Steerneman P, Merckelbach H, Holdrinet I, Meesters C. Comorbid anxiety symptoms in children with pervasive developmental disorders. Journal of Anxiety Disorders. 1998; 12(4):387–393. [PubMed: 9699121]

Parham, LD.; Mailloux, Z. Sensory integration. In: Case-Smith, J., editor. Occupational therapy for children. 5. St. Louis, MO: Elsevier Mosby; 2005. p. 356-409.(Original work published 1985)

Pfeiffer B, Kinnealey M. Treatment of sensory defensiveness in adults. Occupational Therapy International. 2006; 10(3):175–184. [PubMed: 12900790]

Pfeiffer B, Kinnealey M, Reed C, Herzberg G. Sensory modulation and affective disorders in children and adolescents with Asperger's disorder. The American Journal of Occupational Therapy. 2005; 59:335–345. [PubMed: 15969281]

Radloff LS. The CES-D scale: A self-report depression scale for research in the general population. Applied Psychological Measurement. 1977; 1:385–401.

Reynolds S, Lane SJ. Diagnostic validity of sensory over-responsivity: A review of the literature and case reports. Journal of Autism and Developmental Disorders. 2008; 38(3):516–529. [PubMed: 17917804]

Risi S, Lord C, Gotham K, Corsello C, Chrysler C, Szatmari P, et al. Combining information from multiple sources in the diagnosis of autism spectrum disorders. Journal of the American Academy of Child and Adolescent Psychiatry. 2006; 45(9):1094–1103. [PubMed: 16926617]

Schoen SA, Miller LJ, Brett-Green BA, Nielsen DM. Physiological and behavioral differences in sensory processing: A comparison of children with autism spectrum disorder and sensory modulation disorder. Frontiers in Integrative Neuroscience. 2009; 3(29):1–11. [PubMed: 19225578]

Sparrow, S.; Balla, D.; Cicchetti, D. Vineland adaptive behavior scales. Circle Pines, MN: American Guidance Service; 1984.

Stagnitti K, Raison P, Ryan P. Sensory defensiveness syndrome: A paediatric perspective and case study. Australian Occupational Therapy Journal. 1999; 46(4):175–187.

Sukhodolsky DG, Scahill L, Gadow KD, Arnold LE, Aman MG, McDougle CJ, et al. Parent-rated anxiety symptoms in children with pervasive developmental disorders: Frequency and association with core autism symptoms and cognitive functioning. Journal of Abnormal Child Psychology. 2008; 36:117–128. [PubMed: 17674186]

Talay-Ongan A, Wood K. Unusual sensory sensitivities in autism: A possible crossroads. International Journal of Disability, Development and Education. 2000; 47(2):201–212.

Weisbrot DM, Gadow KD, DeVincent CJ, Pomeroy J. The presentation of anxiety in children with pervasive developmental disorders. Journal of Child and Adolescent Psychopharmacology. 2005; 15(3):477–496. [PubMed: 16092912]

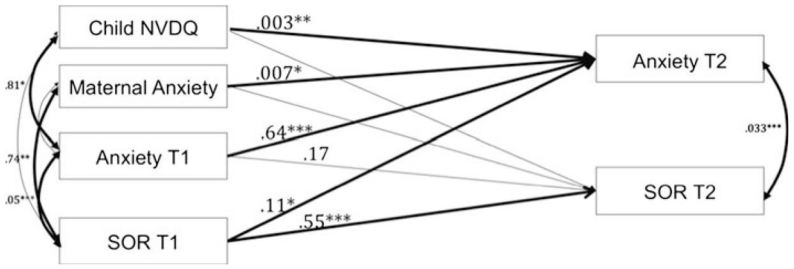

Fig. 1.

Cross-lagged panel analysis model predicting anxiety and SOR at Time 2 from Time 1. Significant pathways are indicated by *bold lines*

Table 1

Descriptive statistics for demographic and main study variables at Time 1 and (where applicable) Time 2

	Time 1 [mean (SD)]	Time 2 [mean (SD)]
Age	28.25 (3.99)	–
ADOS severity (social + communication standardized)	54.58 (18.08)	–
AODS severity (social + communication) − Module 1 only (n = 142)[a]	14.63 (3.83)	–
AODS severity (social + communication) − Module 2 only (n = 7)[b]	13.29 (3.45)	–
Mullen NVDQ	78.10 (18.06)	–
Mullen VDQ	58.62 (25.15)	
Mullen composite	67.72 (17.04)	–
ITSEA anxiety	.31 (.24)	.37 (.30)
ITSEA sensory sensitivity (SOR)	.53 (.43)	.57 (.43)
Mother's BAI score (cutoff for moderate anxiety is 22)	6.87 (6.53)	–
Mother's CESD score (clinical cutoff of 16)	13.69 (10.10)	–

[a] Cutoff scores for a diagnosis of autism = 12; for a diagnosis of ASD = 7

[b] Cutoff scores for a diagnosis of autism = 2; for a diagnosis of ASD = 8