Published in final edited form as:

J Abnorm Child Psychol. 2018 April ; 46(3): 557–567. doi:10.1007/s10802-017-0300-y.

Parent-offspring transmission of internalizing and sensory over-responsivity symptoms in adolescence

Carol A. Van Hulle^{a,d}, Kathryn Lemery-Chalfant^b, and H. Hill Goldsmith^{a,c}

^aWaisman Center, University of Wisconsin-Madison, Madison, WI

^bDepartment of Psychology, Arizona State University, Tempe, AZ

^cDepartment of Psychology, University of Wisconsin-Madison, Madison, WI

Abstract

Reactions to sensory experiences are an overlooked correlate of affective regulation, despite the importance of bodily states on psychological processes. Children who display sensory over-responsivity (i.e., adverse reactions to typical sensations) are at greater risk for developing affective disorders. We extended this literature to adolescents and their middle-aged parents. Participants in a birth record-based study of families of adolescent twins ($N=506$ families; 1,012 adolescents; 53% female) completed a subset of items from the Adult Sensory Profile. We derived adolescent self-reported internalizing disorder symptoms and parent affective diagnoses from structured diagnostic interviews. Structural equation models tested the relationship between parent sensory over-responsivity symptoms and affective diagnoses and their adolescent offspring's sensory over-responsivity and internalizing symptoms. Adolescent sensory over-responsivity symptoms were correlated with internalizing disorder symptoms. Parents with a diagnosis of anxiety or depression (mothers only) reported more frequent SOR symptoms than parents without a diagnosis. Parent depression was significantly related to adolescent sensory over-responsivity symptoms, over and above parent sensory over-responsivity symptoms ($\beta=.26$, $p<.001$ for mothers; $\beta=.13$, $p=.004$ for fathers). Father alcohol abuse/dependency also predicted offspring sensory over-responsivity symptoms.

Offspring of parents with affective disorders were at additional risk for sensory dysregulation via parents' influence on offspring internalizing problems.

Keywords

adolescent; parents; anxiety; sensory over-responsivity

Most people can name at least one typical sensation they find unpleasant; think woolly socks or scraping metal. While annoying, these sensations rarely derail most people for long.

^dCorresponding author: Carol Van Hulle, Waisman Center, University of Wisconsin-Madison, 1500 Highland Ave, Madison, WI 53706, cavanhulle@wisc.edu, 608-265-2674.

Conflict of Interest: The authors declare that they have no conflict of interest.

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. Informed consent: Informed consent was obtained from all individual participants included in the study.

However, for some individuals, the sensations of everyday life can evoke extreme adverse reactions, potentially disrupting daily activities and social interactions (Johnson-Ecker & Parham, 2000; Koenig & Rudney, 2010). Such individuals may have an underlying condition called sensory over-responsivity, a common form of sensory processing disorder (Miller, Anzalone, Lane, Cermak, & Osten, 2007). Among children, sensory over-responsivity symptoms are related to depression and anxiety (Carter, Ben-Sasson, & Briggs-Gowan, 2011; Miller, Nielsen, & Schoen, 2012). We examined the relationship between sensory over-responsivity and depression/anxiety in adolescent twins and their middle-aged parents.

Definition and Background

Sensory processing disorders (SPD) involve both hypo- and hyper-responses to typical sensations, as well as sensation seeking (Dunn, 1994; Miller et al., 2007). Sensory processing disorders may arise from neurological differences that impair transmission of information from unimodal to multimodal sensory integration brain regions and/or the transfer of information between the brain's hemispheres (Chang et al., 2014). Sensory processing symptoms are quite common among people with developmental disorders, especially autism (Liss, 2006; Tomchek & Dunn, 2007; Walz & Baranek, 2006). However, subgroups of otherwise typically developing children may also experience atypical responses to everyday sensations (Ahn, Miller, Milberger, & McIntosh, 2004; Ben-Sasson, Carter, & Briggs-Gowan, 2009; Van Hulle, Lemery-Chalfant, & Goldsmith, 2015).

Sensory over-responsivity (SOR) is one subtype of sensory processing disorder (Miller et al., 2007). SOR is exemplified by a tendency to perceive benign sensations as threatening, intrusive, or painful. SOR is most commonly characterized by an intolerance to noise (auditory stimuli) such as loud gymnasiums, and/or intolerance to touch (tactile stimuli) such as the feel of a hairbrush against the scalp. A small number of individuals experience adverse reactions in other sensory domains such as taste and smell (Dunn, 1994). In childhood, these extreme reactions in the face of typical sensations may impact a child's ability to master a range of essential developmental tasks and adaptive functions including social problem solving and overt signs of empathy (Ben-Sasson et al., 2009). SOR in early life may also negatively impact early relationships (Thomasgard, 2003) and positive social participation during school years (Cosbey, Johnston, & Dunn, 2010).

SOR and Internalizing Disorders

Children who score high on measures of SOR are more likely to exhibit common childhood internalizing behavior problems (Ben-Sasson et al., 2009; Carter et al., 2011; Van Hulle, Schmidt, & Goldsmith, 2012). SOR may play a unique role in the development of anxiety problems, or vice versa, starting early in development. For example, Goldsmith and colleagues (2006) found modest but significant correlations between parent ratings of SOR and general anxiety and separation distress in toddlers. Otherwise typically developing children who screen positive for SOR have a history of internalizing behavior problems as toddlers (Ben-Sasson et al., 2009) and have more concurrent symptoms of anxiety-related DSM disorders than their peers (Van Hulle et al., 2012). SOR symptoms are prevalent among children with attention deficit/hyperactivity disorder (Dunn & Bennett, 2002;

Mangeot et al., 2007), but this literature also demonstrates a strong relationship between anxiety and SOR. Among children diagnosed with ADHD, those with comorbid sensory problems had higher scores on anxiety measures than children with ADHD alone (Lane, Reynolds, & Thacker, 2010; Reynolds & Lane, 2009). In addition, children diagnosed with sensory processing disorder alone score higher on measures of anxiety than children diagnosed with ADHD alone (Miller, Nielsen, & Schoen, 2012).

Measures of anxiety are also significantly correlated with measures of SOR in adulthood. In clinical samples, adults (and children) who present with anxiety problems tend to have more SOR symptoms (Conelea, Carter, & Freeman, 2014; Hazen et al., 2008; Rieke & Anderson, 2009) than the general population. Among healthy adults, both symptoms of sensory sensitivity (similar to over-responsivity) and sensation avoiding are related to anxiety symptoms (Engel-Yeger & Dunn, 2011; Kinnealy & Fuiek, 1999).

Family Transmission of Internalizing Disorders & SOR Symptoms

Internalizing disorders co-occur among family members owing to shared genetic liability (Hettema, Neale, & Kendler, 2001; Levinson, 2006). Studies of twins, in particular, highlight genetic influences on anxiety and depression. Symptoms of SOR are likewise influenced in part by genetic factors (Keuler, Schmidt, Van Hulle, Lemery-Chalfant, & Goldsmith, 2011; Van Hulle et al., 2012). However, *parent* anxiety and depression are related to offspring behavior in part through non-genetic mechanisms (Eley et al., 2015; Lewis, Rice, Harold, Collishaw, & Thapar, 2011). For example, anxious or depressed parents may affect their offspring's internalizing behaviors through maladaptive parenting practices (Hammerton, Zammit, Thapar, & Collishaw, 2016; Leinonen, Solantaus, & Punamaki, 2003). For example, punitive, authoritarian, or absentee parenting increases risk for internalizing problems (Leinonen et al., 2003).

In contrast to research on anxiety and depression, studies of SOR rarely include parents or other first degree relatives. However, mothers of children with autism report significantly more sensory sensitivity and sensory avoidance compared to the general population (Leekam, Nieto, Libby, Wing, & Gould, 2006). Siblings of children with autism likewise reported more sensory sensitivity than children from families without a history of autism (De la Marche, Steyaert, & Noens, 2012).

Hypotheses

Our aim was to address three gaps in the literature. First, most studies of SOR symptoms rely on parent reported symptoms of both anxiety and sensory over-responsivity. Parents may under- or over-estimate offspring anxiety symptoms depending on the measure and characteristics of the parent (Briggs-Gowan, Carter, & Schwab-Stone, 1996; Cantwell, Lewinsohn, Rohde, & Seeley, 1997). Parent biases could inflate the relationship between SOR symptoms and anxiety symptoms. We asked adolescents and adults to report on their own reactions to sensory experiences and mental health symptoms. Second, few studies of SOR and anxiety include adolescent or adult participants. Thus, we extend existing literature on the relationship between anxiety and SOR to these understudied developmental periods.

Third, no studies have asked whether parent SOR is related to offspring SOR. In addition to exploring the transmission of SOR from parents to offspring, we also tested for the effect of parent SOR symptoms on offspring internalizing symptoms and the effects of parent mental health on offspring SOR symptoms.

We hypothesized that adolescents' self-reported internalizing symptoms would be significantly correlated with their self-reported SOR symptoms. Similarly, we expected parent mental health disorders, particularly anxiety disorders, would also be significantly related to parent self-reported SOR symptoms. Finally, we hypothesized that parent anxiety would directly influence both offspring internalizing and SOR symptoms, and that parent SOR symptoms would likewise directly influence both adolescent outcomes.

Method

Participants

Participants were drawn from the Wisconsin Twin Project, a statewide, birth register-based twin sample (Schmidt et al., 2012). From 1989 to 2004, families were invited to join the project 6 to 12 months after the birth of their twins. Eighty-one percent of families contacted responded favorably. At twin age seven years, families were invited to participate in a study of child temperament and behavior problems. Families who enrolled were later invited to participate in an adolescent follow-up study when twins were age 11–18 years, $M = 13.51$, $SD = 1.62$. Of the 648 families who were contacted, 118 declined to participate, 11 families enrolled in a separate study, and 18 families withdrew after initially agreeing to participate leaving a total of $N = 506$. Our work focused on data collected during this follow-up assessment. The University of Wisconsin Institutional Review Board approved this study. Parents provided consent and adolescents provided assent for their participation. All participants were paid.

At age seven years, twins were screened for behavioral problems via telephone interview with the primary caregiver with the goal of “enriching” a sub-sample with behavior problem symptoms for in-depth follow-up studies. Of the 1,012 twin participants in the current study, 305 children scored at least 1.5 standard deviations above the mean on at least one of eight parent-reported symptom scales of the Health and Behavior Questionnaire (Essex et al., 2002); 75 of those 305 were elevated on internalizing problems only. Of the remaining participants, 174 were identified as below the mean on all symptom scales, 283 are unselected cotwins, and 250 did not participate in the screening at age seven years. Six months after screening, parents and offspring participated in multi-rater, multi-method assessment of offspring behavior problems, parent mental health and family functioning. Families were invited to participate in a second follow-up study of adolescent psychopathology when offspring were ages 12–18 years old. Data for these analyses were obtained during the adolescent assessment period unless otherwise noted.

At the age seven assessment, mothers reported on family demographics including years of education, partners' years of education, and family income category (ranging from <\$10,000 to >\$200,000). A college degree was the most frequently reported highest level of education (45.0% of mothers 40.4% of fathers) and median family income was in the \$70,000 to

\$80,000 range. The majority of parents, 95% of mothers and 92.5% of fathers, self-reported their race as Caucasian. Most children (95%) lived with both biological parents at age seven years and 90% lived with both biological parents at the follow-up assessment.

Measures

Sensory Over-responsivity—During the adolescent follow-up study, parents ($n = 475$ mothers and 302 fathers) and their offspring ($n = 1010$) completed a subset of items from the Adolescent/Adult Sensory Profile (AASP; Dunn, 1999). All participants reported on their own behavior. The full AASP taps movement/vestibular processing, taste/smell processing, weak energy and under-responsiveness, in addition to touch and auditory processing. Our research focuses on tactile and auditory responsivity (Van Hulle et al., 2012); therefore, we selected the 12 tactile over-responsivity items and 10 auditory over-responsivity from the full measure. Prior to analyses, we omitted three tactile items and one auditory item that were uncorrelated with the full tactile or auditory scale. Participants were asked to rate items such as “I dislike having my back rubbed” or “I use strategies to drown out sound” on a scale from one (*almost never*) to five (*almost always*); high scores indicate more frequent over-responsivity.

Across all informants, Cronbach’s alpha was acceptable for the auditory scale, ranging from .71 to .78, but low for the tactile scale, ranging from .54 to .57. This is not surprising because auditory items tend to focus on reactions to noisy environments whereas tactile items probe reactions to a broader range of stimuli. The reliability for the full scale, however, was between .75 and .79 across respondents. Moreover, correlations between self-reported reactions to tactile and auditory sensations were remarkably consistent across respondents: $r = .48$ for mothers, $r = .43$ for fathers, and $r = .47$ for offspring, $ps < .001$. To simplify analyses, we created an overall sensory over-responsivity score for each reporter by averaging standardized tactile and auditory over-responsivity subscales.

Parent psychopathology—Trained interviewers administered the Composite International Diagnostic Index (CIDI 2.1; Robins et al., 1989) during separate phone interviews with mothers ($n = 443$) and fathers ($n = 312$). Parents were administered the CIDI during the age seven year follow-up assessment and again during the adolescent follow-up assessment. Parent diagnoses were derived from the latter assessment where available, otherwise diagnoses were derived from the age seven CIDI interview ($n = 35$ mothers and 97 fathers). We administered assessments of mood disorders (including mania and bipolar disorder), anxiety disorders (including OCD), and disorders related to alcohol use. Parents received a *lifetime* diagnosis (present/absent) for each disorder congruent with DSM-IV criteria. Parents who completed the interview at both waves were considered to have a disorder if they qualified for a diagnosis at either assessment. The majority of mothers (57.8%) and fathers (59.3%) qualified for at least 1 lifetime diagnosis.

Although anxiety disorders were the main focus of our hypotheses, to explore the specificity of findings, we included two closely related disorders: depression and alcohol abuse/dependence. Major Depression was the most commonly diagnosed disorder among mothers and is closely related to anxiety (Lahey et al., 2012). Alcohol use was the most common

diagnosis among fathers and is both a consequence and a cause of anxiety symptoms (Kushner, Abrams, & Borchardt, 2000). We reduced the total number of diagnoses to three categories: a) anxiety disorders (generalized anxiety disorder, social phobia, obsessive compulsive disorder, panic without agoraphobia, panic with agoraphobia, or agoraphobia without panic), b) single or recurring depression (mild, moderate or severe), and c) alcohol abuse or alcohol/dependency. No participants qualified for a diagnosis of mania, dysthymia, bipolar I or bipolar II. Prevalence of lifetime diagnoses in the above three categories are shown in Table 1. We excluded specific phobias and Post Traumatic Stress Disorder (prevalence rates are reported in Table 1). Specific phobia was excluded due to concerns over symptom overlap with sensory over-responsivity. PTSD was excluded over concerns that trauma induced anxiety is associated with different neurological activity than other types of anxiety disorders (Etkin & Wager, 2007).

Adolescent psychopathology—Trained interviewers administered the Diagnostic Interview Schedule for Children (DISC-IV; Fisher et al., 1997) to each twin ($n = 966$) separately over the phone. Adolescents indicated whether or not symptoms were characteristic of them. Only 16% ($n = 160$) of adolescents qualified for at least one diagnosis. However, unlike the CIDI, we were able to compute symptom counts for each DISC disorder. Correlations among the symptom counts for each anxiety-related disorder and depression ranged from .26 to .59, $ps < .01$. Depression symptom count was highly correlated with average anxiety-related disorder symptom count, $r = .60$, $p < .001$. Therefore, we created an overall internalizing composite from standardized symptom counts of depression, generalized anxiety disorder, social phobia, panic disorder, agoraphobia, and obsessive compulsive disorder. Log-transforming the internalizing composite reduced a slight positive skew, 14 to -0.67 . Specific phobia included items that might be confounded with SOR symptoms (i.e. is afraid of thunder) and was thus excluded from the internalizing composite.

Data Analysis

We tested our hypotheses using the methodology set forth by Olsen and Kenny (2006) for structural equation modeling with interchangeable dyads. Twin pairs were considered interchangeable because they were arbitrarily assigned an order (Olsen & Kenny, 2006). Under the interchangeable dyads framework, each twin's predictor variable is estimated on both their own outcome and their cotwin's outcome with equality restrictions placed upon parameters. Regression coefficients are constrained to be equal across twin and cotwin (e.g., the effect of mother SOR on one twin's internalizing symptoms is equated to the effect on the cotwin's internalizing symptoms). Finally, equality restrictions across the twin and cotwin are also placed on the intercepts and error variances of adolescent outcomes. In the model (Figure 1) we simultaneously tested for within-trait main effects of parent mental health and SOR symptoms on adolescent internalizing symptoms and SOR symptoms (paths a and b in Figure 1) and the cross-trait effects (paths c and d). Offspring outcomes were allowed to correlate within individuals (path g) and across members of a twin pair (paths e and h) and parent predictors were allowed to correlate as well (path f). We also calculated indirect effects of parental predictors on adolescent outcomes. For example, parent SOR may influence adolescent SOR through parent and adolescent anxiety (see Figure 2):

$$\text{Mother SOR} \xrightarrow{f} \text{Mother Anxiety} \xrightarrow{b} \text{Adolescent Internalizing} \xrightarrow{g} \text{Adolescent SOR}.$$

We included offspring gender, age and family SES (average standardized mother and father years of education and family income) as predictors of adolescent outcomes. We also added parental age and family SES as predictors of parent mental health and parent SOR symptoms. We fit separate models to mother and father data. All models were fit in Mplus v. 7.3 (Muthén & Muthén, 2008), using maximum likelihood estimation with robust standard errors to maximize use of all available data.

Results

Means, standard deviations and correlations among the predictors and outcomes are shown in Table 2. For simplicity, CIDI diagnoses are collapsed into a single presence/absence score within parent. However, we tested associations between offspring outcomes and each of the three parent diagnostic categories separately. Anxiety clustered within couples, *Fisher's exact test* = .01, as did depression, *Fisher's exact test* < .001. Mother and father reported SOR symptoms were also modestly correlated, $r = .16$, $p = .005$. Adolescent females reported more internalizing symptoms than adolescent males and younger twins reported more SOR symptoms than older twins. In contrast, older parents reported more SOR symptoms than younger parents and were more likely to qualify for at least one CIDI diagnosis. Parents with lower SES were more likely to qualify for at least one CIDI diagnosis than parents with a higher SES.

Associations of internalizing symptoms/diagnoses and SOR within individuals

Among adolescents, internalizing symptoms were moderately correlated with SOR symptoms, $r = .45$ for females and $r = .41$ for males, $ps < .001$. Mothers who qualified for a diagnosis of anxiety or depression reported more frequent SOR symptoms, $t(421) = 4.65$, $p < .001$, $d = .55$ and $t(421) = 2.34$, $p = .02$, $d = .23$, respectively, than mother's without a diagnosis. Mothers' alcohol abuse/dependency was unrelated to SOR symptoms, $t(421) = 1.43$, $p = .15$, $d = .14$. Fathers who qualified for a diagnosis of anxiety reported more intense SOR symptoms, $t(228) = 2.82$, $p = .005$, $d = .61$, than father's without an anxiety diagnosis. Father diagnosis of depression, $t(228) = 0.80$, $p = .42$, $d = .13$, and alcohol abuse/dependency, $t(228) = 0.85$, $p = .40$, $d = .11$, were unrelated to self-reported SOR symptoms.

Structural equation modeling

Maternal variables—Overall model fit statistics and standardized parameter estimates are shown in Table 3. All models provided a good fit to the data. Mothers SOR symptoms significantly predicted offspring SOR symptoms (Figure 1, path a; $p = .01$ Mother diagnosis (Figure 1, path b) of anxiety, $p < .001$ and depression, $p = .002$, but not alcohol abuse/dependency ($p = .34$), predicted offspring internalizing symptoms. Finally, mother depression had a direct effect on offspring SOR symptoms (Figure 1 path d, $ps = .01$) after controlling for the effect of mother SOR symptoms on offspring SOR symptoms.

Mother SOR symptoms did *not* have a direct effect on offspring internalizing problems (Figure 1 path c, p ranged from .24 to .28). We refit the model (changing correlations to regressions where necessary) to calculate indirect effects. Figure 2 depicts the indirect paths between mother SOR and offspring internalizing symptoms (only one twin is shown for simplicity). Mother SOR symptoms had an indirect effect on adolescent internalizing symptoms via the association between Mother SOR and mother diagnosis (path $f \rightarrow b$) plus the association between adolescent SOR and adolescent internalizing symptoms (path $a \rightarrow g$). The total indirect effects equaled 0.06, $p = .004$. Likewise, mother psychopathology had an indirect effect on SOR symptoms through the association between internalizing and SOR symptoms in both the parent and offspring generations (e.g. total indirect effects = 0.05, $p = .01$ for maternal depression).

Paternal variables—Results from fitting father mental health and SOR symptoms are shown in Table 3. Father depression diagnosis predicted offspring internalizing, $p = .002$, and SOR symptoms, $p = .004$. Father alcohol abuse/dependency diagnosis likewise predicted offspring internalizing symptoms, $p = .01$, and offspring SOR symptoms, $p = .02$. However, father SOR symptoms did not predict any offspring outcomes. Unlike mothers, fathers' sensory over-responsivity had no indirect effects on adolescent internalizing symptoms or adolescent SOR symptoms because father SOR symptoms were not related to either of these diagnoses.

Post-hoc developmental considerations—The majority of parents who qualified for a CIDI diagnosis reported an age of onset that predated the birth of their twins (from 63% for maternal anxiety to 93% for paternal specific phobia), with the exception of maternal depression. A slight majority (55%) of mothers who qualified for any depression diagnosis had an age of onset that predated their twins' birth (pre-birth onset); average age of onset for this group was age 22 years. Average age of on-set for the remaining mothers who received any depression diagnosis (post-birth onset) was 35 years. Mothers with pre-birth onset reported more frequent sensory symptoms than mothers without depression, $t(420) = 2.04$, $p = .04$, $d = .19$. In contrast, SOR symptoms for mothers with post-birth onset were indistinguishable from non-depressed mothers, $t(350) = 0.68$, $p = .50$, $d = .07$. Finally, offspring of mothers with pre-birth onset reported more frequent sensory over-responsivity symptoms than children of mother's with post-birth onset, $t(263) = 1.79$, $p = .07$, $d = .22$. To explore the developmental effects further, we refit the structural equation model separately by age-of-onset group. The pattern of results were unchanged for in the pre-birth onset group. However, there was no effect of SOR or maternal depression on either of the offspring outcomes in the post-birth onset group (Table 4).

Depression age-of-onset was closely related to chronicity (recurring vs single episode). In fact, 65% of mothers who qualified for a diagnosis of recurring depression had an age-of-onset that predated their twin offspring, compared to just 37% of mothers who qualified for a diagnosis of single episode depression. When we ran analyses separately for mothers with recurring depression and single episode depression, recurring depression was related to offspring internalizing symptoms, $estimate = .23$, $p = .01$ sensory over-responsivity symptoms, $estimate = .37$, $p < .001$, but single episode depression was not related to either.

Discussion

As many as 5–13% of typically developing children experience sensory related problems (Ahn et al., 2004). That number increases considerably among children with anxiety disorders (Carter et al., 2011). Thus an understanding the pathways to sensory dysfunction could aid our understanding of the development of anxiety disorders. Our hypothesis that the positive relationship between internalizing problems and sensory over-responsivity seen in younger children would generalize to later stages of development was supported. Sensory symptoms were related to internalizing behavior problems in adolescents and mental health diagnoses in adults. Older parents and parents with lower SES reported more SOR symptoms. Most research on adult sensory dysfunction center on middle class samples, but studies of children from lower SES families report higher rates of sensory over-responsivity (Reynolds, Shepherd, & Lane, 2008; Román-Oyola & Reynolds, 2013). It is unclear why parent age is related to sensory over-responsivity.

Our hypothesis that parent SOR symptoms would predict both offspring internalizing behaviors and offspring SOR symptoms was not supported. We found small correlations between parent self-reported sensory over-responsivity and offspring self-reported sensory over-responsivity, but the SEM results suggested that parent influences on offspring sensory over-responsivity operated largely indirectly through parental transmission of anxiety/depression.

Given the relationship between anxiety and SOR symptoms, we expected that maternal anxiety in particular would be related to offspring SOR symptoms, and maternal SOR would be directly related to offspring internalizing symptoms. Neither of these hypotheses was supported. Rather, maternal *depression* had a strong impact on offspring SOR. Given the substantial overlap between qualifying for a depression diagnosis and an anxiety diagnosis (*Fishers exact test* <.001), it is unclear why maternal depression appears to uniquely relate to offspring SOR symptoms. The answer, if environmental in nature, may lie in the length of exposure to maternal psychopathology. Post-hoc analyses suggest that depression that began before the twins were born was related to offspring internalizing and SOR symptoms. Depression that began after the twins were born was not related to either outcome.

The earlier the onset of depression, the more severe and chronic it is likely to be over the course of a person's lifetime (Zisook et al., 2007). Although we did not have any information on depression timing beyond age-of-onset, the CIDI divides depression diagnoses into recurring and single episode type. Only recurring depression was related to offspring outcomes. It is possible that children exposed to severe parental depression or chronic bouts of depression develop more severe symptoms of sensory over-responsivity, although this is purely speculative. History of depression is also a strong predictor of depression during pregnancy (Rich-Edwards et al., 2006); animal studies have shown that stress during pregnancy influences the development of offspring sensory sensitivity (Schneider et al., 2007) and anxious behaviors (Vallee, Mayo, Dellu, Le Moal, & Maccari, 1997).

Likewise, father's depression but not anxiety was related to both offspring internalizing and SOR symptoms. This is not the first study to note an absence of any direct relationship between paternal anxiety and offspring anxiety. (McClure, Brennan, Hammen, & Le Brocque, 2001) also reported that paternal anxiety was unrelated to offspring anxiety in a large, high-risk sample. In contrast, a meta-analysis by (Kane & Garber, 2004) confirmed that paternal depression increases their children's risk for internalizing problems. Other studies suggest that the effect of paternal depression may depend on maternal behavior and level of father involvement (Meadows, McLanahan, & Brooks-Gunn, 2007). Father alcohol abuse/dependence was also related to internalizing and sensory over-responsivity in offspring, but that is likely due to the high comorbidity between depression and alcohol related disorders among fathers (*Fishers exact test* = .001). Nearly three quarters of fathers with a diagnosis of depression also qualified for a diagnosis of alcohol abuse/dependence.

Green and Ben-Sasson (2010) examined possible reasons that anxiety might be related to SOR. The first reason, SOR is caused by anxiety, is supported by evidence focused on hypervigilance and attention to threat that characterize anxiety. Even slightly unpleasant stimuli may take on greater salience for a child who is vigilant for external threats. A second reason, that anxiety is caused by SOR, focuses on "fear" conditioning. Because children with SOR are often confronted with environmental stimuli that they cannot control, anxious feelings may become generalized. The contemporaneous nature of most studies of SOR and psychopathology prevent researchers from establishing which of these two possibilities is most likely. However, examining the relationship between parent and offspring behaviors offers some clues. Mothers' psychopathology predicted offspring SOR but mothers' SOR symptoms did not. Similarly, father psychopathology predicted offspring SOR symptoms while fathers' own SOR symptoms did not. In fact, the indirect effects of parent mental health on offspring SOR symptoms via internalizing behaviors were equal or larger in value than the direct effects.

These results appear to support the first explanation. For example, anxious parents may be insensitive to their children's cues (Nicol-Harper, Harvey, & Stein, 2007), which leads to heightened offspring anxiety that in turn leads to hypersensitivity to the environment (Green & Ben-Sasson, 2010). However, research on autistic children indicates that sensory over-responsivity contributes to later anxious behaviors but early anxious behaviors do not lead to later sensory problems (Green, Ben-Sasson, Soto, & Carter, 2012). More longitudinal studies that include both anxiety and sensory measures are needed to establish the nature of any causal relationship. Our results cannot rule out a third explanation that — mental health problems and SOR are both caused by a common third factor. Indeed, our previous work indicates that SOR symptoms and internalizing symptoms share underlying genetic influences (Van Hulle et al., 2012). Thus, parents may pass on both genes related to sensory processing difficulties and internalizing symptoms, as well as engage in parenting practices or otherwise create environments that contribute to the manifestation of internalizing problems and maladaptive sensory integration.

Given the proposed relation between bodily states and affect regulation (Craig, 2008), offspring of parents with affective disorders may be at risk for sensory dysregulation partly via neurological differences associated with internalizing problems. Recent studies have

identified neurological regions associated with both anxiety and SOR symptoms. For example, the insula is associated with emotional processing and anxiety (Stein, 2007), and activation in this region correlates with SOR symptoms in youths diagnosed with autism spectrum disorders (Green et al., 2015). Anxiety measures were used to validate identification of a salience-driven default mode network (Seeley et al., 2007). Connectivity between this salience network and the somatosensory cortex was positively correlated with SOR symptoms and somatosensory activation in response to mildly aversive tactile and auditory sensations (Green, Hernandez, Bookheimer, & Dapretto, 2016).

The fact that parent psychopathology is related to dysfunction in offspring is not a novel finding. But current interventions may be missing a key component of offspring dysfunction. At the moment, therapeutic interventions directly tied to sensory dysfunction are designed primarily for children with autism (Case-Smith, Weaver, & Fristad, 2015), where there is some tentative evidence that reducing sensory dysfunction reduces internalizing problems as well (Miller, Coll, & Schoen, 2007).

Limitations

Compared to a nationally representative sample (Kessler et al., 2005), prevalence of alcohol abuse/dependence was twice as high for mothers and four times as high for fathers. Fathers appeared to under-report all other mental health disorders, calling into question the generalizability of the relationship between SOR and father mental health. Lifetime prevalence rates for maternal depression and anxiety disorders were similar to those reported by Kessler et al. (2005) for all adults. However, our sample is largely Caucasian, with limited representation of families living in poverty, which limits the generalizability of our results to dissimilar populations.

Our analyses did not control for co-morbidity within parents or associations of different disorders across the two parents. Partner mental health can have an impact on parenting behaviors, which in turn influences offspring mental health (Leinonen et al., 2003). A second factor to consider is that we collapsed across multiple anxiety disorders, to obtain an adequate sample of anxious parents. Some evidence indicates that internalizing disorders are better categorized in terms of “fear” disorders such as panic disorder and specific phobia and “distress or worry” disorders such as generalized anxiety (Lahey et al., 2004). Thus, our strategy may have obscured potential disorder-specific relationships with offspring outcomes. Direction of effects is a third issue that should be examined in future research. Offspring sensory symptoms correlate with parenting stress (Gourley, Wind, Henninger, & Chinitz, 2013). However, parents generally reported an age of onset that predated the birth of their offspring, and thus children’s sensory issues are more likely to aggravate an existing disorder than lead to the onset of a disorder.

Conclusion

Despite evidence that unusual reactions to everyday sensations co-occur with heightened levels of anxiety, sensory reactions are an overlooked correlate of psychopathology. As the literature we have reviewed indicates, anxiety and symptoms of sensory over-responsivity are correlated in early and middle childhood. We found that sensory over-responsivity

continues to be associated with anxiety and depression in adolescence and adulthood. Moreover, parental anxiety has an indirect impact on offspring sensory over-responsivity through offspring's own internalizing symptoms while parental depression has a direct impact on offspring maladaptive sensory reactions.

References

- Ahn R, Miller LJ, Milberger S, McIntosh DN. Prevalence of parents' perceptions of sensory processing disorders among kindergarten children. *American Journal of Occupational Therapy*. 2004; 58:287–293. [PubMed: 15202626]
- Ben-Sasson A, Carter AS, Briggs-Gowan MJ. Sensory Over-Responsivity in elementary school: Prevalence and social-emotional correlates. *Journal of Abnormal Child Psychology*. 2009; 37(5): 705–716. <https://doi.org/10.1007/s10802-008-9295-8>. [PubMed: 19153827]
- 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; 24:749–765. [PubMed: 8970908]
- Cantwell DP, Lewinsohn PM, Rohde P, Seeley MS. Correspondence between adolescent report and parent report of psychiatric diagnostic data. *Journal of the American Academy of Child & Adolescent Psychiatry*. 1997; 36:610–619. [PubMed: 9136495]
- Carter AS, Ben-Sasson A, Briggs-Gowan MJ. Sensory over-responsivity, psychopathology, and family impairment in school-aged children. *Journal of the American Academy of Child & Adolescent Psychiatry*. 2011; 50:1210. [PubMed: 22115142]
- Case-Smith J, Weaver LL, Fristad MA. A systematic review of sensory interventions for children with autism spectrum disorders. *Autism*. 2015; 19:133–148. [PubMed: 24477447]
- Chang Y-S, Owen JP, Desai SS, Hill SS, Arnett AB, Harris J, ... Mukherjee P. Autism and Sensory Processing Disorders: Shared white matter disruption in sensory pathways but divergent connectivity in social-emotional pathways. *PLoS ONE*. 2014; 9(7):e103038. <https://doi.org/10.1371/journal.pone.0103038>. [PubMed: 25075609]
- Conelea CA, Carter AC, Freeman JB. Sensory over-responsivity in a sample of children seeking treatment for anxiety. *Journal of Developmental & Behavioral Pediatrics*. 2014; 35:510–521. [PubMed: 25186122]
- Cosbey J, Johnston SS, Dunn ML. Sensory Processing Disorders and social participation. *American Journal of Occupational Therapy*. 2010; 64:462–473. <https://doi.org/10.5014/ajot.2010.09076>. [PubMed: 20608277]
- Craig AD. Interoception and emotion: a neuroanatomical perspective. *Handbook of Emotions*. 2008; 3:272–288.
- De la Marche W, Steyaert J, Noens I. Atypical sensory processing in adolescents with an autism spectrum disorder and their non-affected siblings. *Research in Autism Spectrum Disorders*. 2012; 6(2):639–645. <https://doi.org/10.1016/j.rasd.2011.09.014>.
- Dunn W. Performance of typical children on the Sensory Profile: An item analysis. *American Journal of Occupational Therapy*. 1994; 48:967–974. [PubMed: 7530904]
- Dunn W. The Sensory Profile manual. San Antonio TX: The Psychological Corporation; 1999.
- Dunn W, Bennett D. Patterns of sensory processing in children with Attention Deficit Hyperactivity Disorder. *The Occupational Therapy Journal of Research*. 2002; 22:4–15.
- Eley TC, McAdams TA, Rijdsdijk FV, Lichtenstein P, Narusyte J, Reiss D, ... Neiderhiser JM. The intergenerational transmission of anxiety: A children-of-twins study. *American Journal of Psychiatry*. 2015; 172:630–637. <https://doi.org/10.1176/appi.ajp.2015.14070818>. [PubMed: 25906669]
- Engel-Yeger B, Dunn W. The relationship between sensory processing difficulties and anxiety level of healthy adults. *The British Journal of Occupational Therapy*. 2011; 74:210–216. <https://doi.org/10.4276/030802211X13046730116407>.

- Etkin A, Wager TD. Functional neuroimaging of anxiety: A meta-analysis of emotional processing in PTSD, Social Anxiety Disorder, and Specific Phobia. *American Journal of Psychiatry*. 2007; 164:1476–1488. <https://doi.org/10.1176/appi.ajp.2007.07030504>. [PubMed: 17898336]
- Goldsmith HH, Van Hulle CA, Arneson CL, Schreiber JE, Gernsbacher MA. A population-based twin study of parentally reported tactile and auditory defensiveness in young children. *Journal of Abnormal Child Psychology*. 2006; 34:378–392. <https://doi.org/10.1007/s10802-006-9024-0>.
- Gourley L, Wind C, Henninger EM, Chinitz S. Sensory processing difficulties, behavioral problems, and parental stress in a clinical population of young children. *Journal of Child and Family Studies*. 2013; 22:912–921. <https://doi.org/10.1007/s10826-012-9650-9>. [PubMed: 24443636]
- 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; 40:1495–1504. <https://doi.org/10.1007/s10803-010-1007-x>. [PubMed: 20383658]
- Green SA, Ben-Sasson A, Soto TW, Carter AS. Anxiety and Sensory Over-Responsivity in toddlers with Autism Spectrum Disorders: Bidirectional effects across time. *Journal of Autism and Developmental Disorders*. 2012; 42:1112–1119. <https://doi.org/10.1007/s10803-011-1361-3>. [PubMed: 21935727]
- Green SA, Hernandez L, Bookheimer SY, Dapretto M. Salience network connectivity in Autism is related to brain and behavioral markers of sensory overresponsivity. *Journal of the American Academy of Child & Adolescent Psychiatry*. 2016; 55(7):618–626. [PubMed: 27343889]
- Hammerton G, Zammit S, Thapar A, Collishaw S. Explaining risk for suicidal ideation in adolescent offspring of mothers with depression. *Psychological Medicine*. 2016; 46:265–275. <https://doi.org/10.1017/S0033291715001671>. [PubMed: 26303275]
- Hazen EP, Reichert E, Piacentini J, Miguel E, Rosario M, Pauls D, Geller D. Case series: Sensory intolerance as a primary symptoms of pediatric OCD. *Annals of Clinical Psychiatry: Journal of the American Academy of Clinical Psychiatrists*. 2008; 20:199–203.
- Hettema JM, Neale MC, Kendler KS. A review and meta-analysis of the genetic epidemiology of anxiety disorders. *American Journal of Psychiatry*. 2001; 158:1568–1578. [PubMed: 11578982]
- Johnson-Ecker CL, Parham LD. The evaluation of sensory processing: A validity study using contrasting groups. *American Journal of Occupational Therapy*. 2000; 54:494–503. [PubMed: 11006809]
- Kane P, Garber J. The relations among depression in fathers, children's psychopathology, and father-child conflict: A meta-analysis. *Clinical Psychology Review*. 2004; 24:339–360. <https://doi.org/10.1016/j.cpr.2004.03.004>. [PubMed: 15245835]
- Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*. 2005; 62(6):593–602. [PubMed: 15939837]
- Keuler MM, Schmidt NL, Van Hulle C, Lemery-Chalfant K, Goldsmith HH. Sensory over-responsivity: prenatal risk factors and temperamental contributions. *Journal of Developmental & Behavioral Pediatrics*. 2011; 32:533–541. [PubMed: 21743351]
- Kinnealy M, Fuiiek M. The relationship between sensory defensiveness, anxiety, depression, and perception of pain in adults. *Occupational Therapy International*. 1999; 6:195–206.
- Koenig KP, Rudney SG. Performance challenges for children and adolescents with difficulty processing and integrating sensory information: A systematic review. *American Journal of Occupational Therapy*. 2010; 64:430–442. <https://doi.org/10.5014/ajot.2010.09073>. [PubMed: 20608274]
- Kushner M, Abrams K, Borchardt C. The relationship between anxiety disorders and alcohol use disorders: A review of major perspectives and findings. *Clinical Psychology Review*. 2000; 20(2): 149–171. [https://doi.org/10.1016/S0272-7358\(99\)00027-6](https://doi.org/10.1016/S0272-7358(99)00027-6). [PubMed: 10721495]
- Lahey BB, Applegate B, Hakes JK, Zald DH, Hariri AR, Rathouz PJ. Is there a general factor of prevalent psychopathology during adulthood? *Journal of Abnormal Psychology*. 2012; 121:971–977. <https://doi.org/10.1037/a0028355>. [PubMed: 22845652]
- Lahey BB, Applegate B, Waldman ID, Loft JD, Hankin BL, Rick J. The structure of child and adolescent psychopathology: Generating new hypotheses. *Journal of Abnormal Psychology*. 2004; 113:358–385. <https://doi.org/10.1037/0021-843X.113.3.358>. [PubMed: 15311983]

- Lane SJ, Reynolds S, Thacker L. Sensory over-responsivity and ADHD: differentiating using electrodermal responses, cortisol, and anxiety. *Frontiers in Integrative Neuroscience*. 2010; 4 <https://doi.org/10.3389/fnint.2010.00008>.
- Leekam SR, Nieto C, Libby SJ, Wing L, Gould J. Describing the sensory abnormalities of children and adults with Autism. *Journal of Autism and Developmental Disorders*. 2006; 37:894–910. <https://doi.org/10.1007/s10803-006-0218-7>.
- Leinonen JA, Solantaus TS, Punamaki RL. Parental mental health and children's adjustment: the quality of marital interaction and parenting as mediating factors. *Journal of Child Psychology and Psychiatry*. 2003; 44:227–241. <https://doi.org/10.1111/1469-7610.t01-1-00116>. [PubMed: 12587859]
- Levinson DF. The genetics of Depression: A review. *Biological Psychiatry*. 2006; 60:84–92. <https://doi.org/10.1016/j.biopsych.2005.08.024>. [PubMed: 16300747]
- Lewis G, Rice F, Harold GT, Collishaw S, Thapar A. Investigating environmental links between parent Depression and child depressive/anxiety symptoms using an Assisted Conception Design. *Journal of the American Academy of Child & Adolescent Psychiatry*. 2011; 50:451–459e1. <https://doi.org/10.1016/j.jaac.2011.01.015>. [PubMed: 21515194]
- Liss M. Sensory and attention abnormalities in autistic spectrum disorders. *Autism*. 2006; 10(2):155–172. <https://doi.org/10.1177/1362361306062021>. [PubMed: 16613865]
- Mangeot SD, Miller LJ, McIntosh DN, McGrath-Clarke J, Simon J, Hagerman RJ, Goldson E. Sensory modulation dysfunction in children with attention-deficit-hyperactivity disorder. *Developmental Medicine & Child Neurology*. 2007; 43:399–406. <https://doi.org/10.1111/j.1469-8749.2001.tb00228.x>.
- McClure EB, Brennan PA, Hammen CL, Le Brocque RM. Parental anxiety disorders, child anxiety disorder, and the perceived parent-child relationship in an Australian high-risk sample. *Journal of Abnormal Child Psychology*. 2001; 29:1–10. [PubMed: 11316331]
- Meadows SO, McLanahan SS, Brooks-Gunn J. Parental Depression and Anxiety and early childhood behavior problems across family types. *Journal of Marriage and Family*. 2007; 69:1162–1177. <https://doi.org/10.1111/j.1741-3737.2007.00439.x>.
- Miller LJ, Coll JR, Schoen SA. A randomized controlled pilot study of the effectiveness of occupational therapy for children with Sensory Modulation Disorder. *American Journal of Occupational Therapy*. 2007; 61:228–238. <https://doi.org/10.5014/ajot.61.2.228>. [PubMed: 17436845]
- Miller LJ, Anzalone M, Lane SJ, Cermak SA, Osten E. Concept evolution in sensory integration: A proposed nosology for diagnosis. *American Journal of Occupational Therapy*. 2007; 61:135–140. [PubMed: 17436834]
- Miller LJ, Nielsen DM, Schoen SA. Attention deficit hyperactivity disorder and sensory modulation disorder: A comparison of behavior and physiology. *Research in Developmental Disabilities*. 2012; 33:804–818. <https://doi.org/10.1016/j.ridd.2011.12.005>. [PubMed: 22236629]
- Nicol-Harper R, Harvey AG, Stein A. Interactions between mothers and infants: Impact of maternal anxiety. *Infant Behavior and Development*. 2007; 30:161–167. <https://doi.org/10.1016/j.infbeh.2006.08.005>. [PubMed: 17292789]
- Olsen JA, Kenny DA. Structural equation modeling with interchangeable dyads. *Psychological Methods*. 2006; 11:127–141. <https://doi.org/10.1037/1082-989X.11.2.127>. [PubMed: 16784334]
- Reynolds S, Lane SJ. Sensory over-responsivity and anxiety in children with ADHD. *American Journal of Occupational Therapy*. 2009; 63:433–440. [PubMed: 19708472]
- Reynolds S, Shepherd J, Lane S. Sensory Modulation Disorders in a Minority Head Start Population: Preliminary Prevalence and Characterization. *Journal of Occupational Therapy, Schools, & Early Intervention*. 2008; 1:186–198. <https://doi.org/10.1080/19411240802589031>.
- Rich-Edwards JW, Kleinman K, Abrams A, Harlow B, McLaughlin TJ, Joffe H, Gillman M. Sociodemographic predictors of antenatal and postpartum depressive symptoms among women in a medical group practice. *Journal of Epidemiology & Community Health*. 2006; 60:221–227. <https://doi.org/10.1136/jech.2005.039370>. [PubMed: 16476752]
- Rieke EF, Anderson D. Adolescent/Adult Sensory Profile and obsessive-compulsive disorder. *American Journal of Occupational Therapy*. 2009; 63:138–145. [PubMed: 19432051]

- Robins LN, Wing J, Wittchen HU, Helzer JE, Babor TF, Burke J, ... Towle LH. The Composite International Diagnostic Interview: An epidemiologic instrument suitable for use in conjunction with different diagnostic systems and in different cultures. *Archives of General Psychiatry*. 1989; 45:1069–1077.
- Román-Oyola R, Reynolds S. Prevalence of Sensory Modulation Disorder among Puerto Rican preschoolers: An analysis focused on socioeconomic status variables: Sensory Modulation, PR Preschoolers and SES. *Occupational Therapy International*. 2013; 20:144–154. <https://doi.org/10.1002/oti.1353>. [PubMed: 23696328]
- Schmidt NL, Van Hulle CA, Brooker RJ, Meyer LR, Lemery-Chalfant K, Goldsmith HH. Wisconsin Twin Research: early development, childhood psychopathology, Autism, and Sensory Over-responsivity. *Twin Research and Human Genetics*. 2012; 16:376–384. <https://doi.org/10.1017/thg.2012.105>. [PubMed: 23200241]
- Schneider ML, Moore CF, Gajewski LL, Laughlin NK, Larson JA, Gay CL, ... DeJesus OT. Sensory processing disorders in a nonhuman primate model: evidence for occupational therapy practice. *The American Journal of Occupational Therapy*. 2007; 61:247–253. [PubMed: 17436847]
- Seeley WW, Menon V, Schatzberg AF, Keller J, Glover GH, Kenna H, ... Greicius MD. Dissociable Intrinsic Connectivity Networks for Salience Processing and Executive Control. *Journal of Neuroscience*. 2007; 27:2349–2356. <https://doi.org/10.1523/JNEUROSCI.5587-06.2007>. [PubMed: 17329432]
- Stein M. Increased amygdala and insula activation during emotion processing in anxiety-prone subjects. *American Journal of Psychiatry*. 2007; 164:318. <https://doi.org/10.1176/appi.ajp.164.2.318>. [PubMed: 17267796]
- Thomasgard M. Working with challenging young children: Relations between child temperament and response to novelty and sensory processing. *Clinical Pediatrics*. 2003; 42:197–204. [PubMed: 12739917]
- Tomchek SD, Dunn W. Sensory processing in children with and without autism: a comparative study using the short sensory profile. *The American Journal of Occupational Therapy*. 2007; 61:190–200. [PubMed: 17436841]
- Vallee M, Mayo W, Dellu F, Le Moal M, Maccari S. Prenatal stress induces high anxiety and postnatal handling induces low anxiety in adult offspring: Correlation with stress induced corticosterone secretion. *Journal of Neuroscience*. 1997; 17:2626–2636. [PubMed: 9065522]
- Van Hulle CA, Lemery-Chalfant K, Goldsmith HH. Trajectories of Sensory Over-Responsivity from early to middle childhood: Birth and temperament risk factors. *PloS One*. 2015; 10:e0129968. <https://doi.org/10.1371/journal.pone.0129968>. [PubMed: 26107259]
- Van Hulle CA, Schmidt NL, Goldsmith HH. Is sensory over-responsivity distinguishable from childhood behavior problems? A phenotypic and genetic analysis. *Journal of Child Psychology and Psychiatry*. 2012; 53:64–72. <https://doi.org/10.1111/j.1469-7610.2011.02432.x>. [PubMed: 21797864]
- Walz NC, Baranek GT. Sensory processing patterns in persons with Angelman syndrome. *The American Journal of Occupational Therapy*. 2006; 60:472–479. [PubMed: 16915878]
- Zisook S, Lesser I, Stewart JW, Wisniewski SR, Balasubramani GK, Fava M, ... Rush AJ. Effect of age at onset on the course of Major Depressive Disorder. *American Journal of Psychiatry*. 2007; 164:1539–1546. <https://doi.org/10.1176/appi.ajp.2007.06101757>. [PubMed: 17898345]

Figure 1.

Parent-offspring transmission model.

Note: SOR = mean sensory over-responsivity symptoms; Dx = diagnosis of anxiety, depression, or alcohol related disorder, in the 3 SEMs fit for each parent; Int = mean adolescent internalizing symptoms; TA = twin A and TB = twin B (randomly ordered). Paths with the identical letters are constrained to have equal value; parent effects are shown in black. Not shown: parent age and family SES were included as predictors of parent diagnosis and SOR symptoms; offspring age, gender, and family SES were included as predictors of offspring internalizing and SOR symptoms.

Figure 2.

Model of indirect effects of maternal SOR symptoms on offspring internalizing problems

Note: SOR = sensory over-responsivity symptoms, Dx = maternal anxiety, depression, or alcohol related disorder, Int = adolescent internalizing symptoms; TA = twin A and TB = twin B (randomly ordered). Paths with the identical letters have equal value; bold black lines indicate indirect paths from maternal SOR to offspring internalizing symptoms. Not shown: parent age and family SES were included as predictors of parent diagnosis and SOR symptoms; offspring age, gender, and family SES were included as predictors of offspring internalizing and SOR symptoms.

Table 1

Prevalence of lifetime diagnoses among parents using the Composite International Diagnostic Index

	Mother (<i>n</i> = 443)	Father (<i>n</i> = 312)
Alcohol Abuse/Dependence	129 (29%)	167 (53%)
Major Depression ^a	133 (30%)	46 (14%)
Anxiety ^b	96 (22%)	39 (12%)
Specific Phobia ^c	77 (18%)	26 (8%)
PTSD ^c	37 (8%)	9 (3%)
Multiple Diagnoses	162 (37%)	74 (23.7%)

^aSingle episode (mild, moderate, severe) or recurring (mild, moderate, severe)^bAt least one diagnosis of Generalized Anxiety Disorder, Social phobia, Obsessive Compulsive Disorder, Panic Disorder with Agoraphobia, Panic Disorder without Agoraphobia, or Agoraphobia without Panic Disorder.^cExcluded in study analyses.

Table 2

Means (SD) and inter-correlations by informant.

	M (SD)	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
Offspring											
1. Family SES	0.0 (0.8)										
2. SOR	2.5 (0.5)	-.02									
3. Internalizing (INT)	-0.3 (0.8)	-.05	.44**								
4. Age (years)	13.5 (1.7)	-.06	-.10**	-.05							
5. Gender (% female)	50.1	.02	-.01	-.15**	.06						
6. SOR	2.2 (0.5)	-.06	.12**	.05	.04	-.03					
7. Any CIDI Dx (%)	57.9	-.17**	.15**	.15**	.00	-.07*	.21**				
8. Age	40.8 (5.8)	.26*	.03	.08*	.00	.01	.13**	-.10			
9. SOR	2.2 (0.4)	.00	.08*	.03	.08*	.04	.16**	.01	-.02		
10. Any CIDI Dx (%)	59.3	-.14**	.12**	.15**	.06	-.02	.04	.09	.01	.13	
11. Age (years)	42.8 (6.5)	.06*	.04	.02	.05	-.04	.10*	.01	.62**	-.02	.04

*
p<.05;**
p<.01

Note: SES = socio-economic status, SOR = mean sensory over-responsivity score, INT = mean adolescent internalizing symptom score, CIDI Dx = any anxiety, depression, or alcohol related diagnosis on the Composite International Diagnostic Index.

Table 3

Standardized parameter estimates from fitting parent-offspring structural equation models.

	Mothers						Fathers					
	Adolescent INT			Adolescent SOR			Adolescent INT			Adolescent SOR		
	β	p		β	p	RMSEA	β	p		β	p	RMSEA
Parent Anxiety ^a	0.13	.003		0.05	.19	0.028	-.0005	.93		0.05	.33	.031 (.012–.046)
Parent SOR	0.05	.27		0.12	.002	(.003–.044)	0.04	.41		0.08	.09	
SES	-.02	.61		-.01	.74		-.04	.40		-.02	.43	
Gender (Offspring)	-.11	.002		0.02	.49		-.12	.001		0.02	.54	
Age (Offspring)	-.02	.66		-.04	.005		-.01	.83		-.02	.15	
Parent Depression ^b	0.14	.001		0.14	<.001	.000	0.13	.003		0.13	.002	.017 (.005–.026)
Parent SOR	0.06	.19		0.11	.003	(.000–.029)	0.02	.65		0.08	.09	
SES	-.02	.60		-.003	.95		-.04	.42		-.02	.68	
Gender (Offspring)	-.12	.001		0.02	.52		-.12	<.001		.02	.60	
Age (Offspring)	-.02	.64		-.04	.002		-.03	.46		-.02	.17	
Parent Alcohol ^c	-.04	.34		0.00	.99	.027	0.13	.01		0.13	.005	.030
Parent SOR	0.07	.09		0.13	.001	(.000–.044)	0.03	.47		0.08	.08	(.011–.046)
SES	-.04	.33		-.02	.65		-.01	.81		0.01	.90	
Gender (Offspring)	-.12	.001		0.02	.53		-.12	<.001		0.02	.59	
Age (Offspring)	-.02	.58		-.03	.008		-.02	.68		-.03	.03	

Note: RMSEA = root mean square error of approximation, CFI = Comparative Fit Index, SES = socio-economic status, SOR = mean sensory over-responsivity symptoms, INT = mean adolescent internalizing symptoms

^aSingle episode (mild, moderate, severe) or recurring (mild, moderate, severe)

^bAt least one diagnosis of Generalized Anxiety Disorder, Social phobia, Obsessive Compulsive Disorder, Panic Disorder with Agoraphobia, Panic Disorder without Agoraphobia, or Agoraphobia without Panic Disorder.

^cDiagnosis of alcohol abuse or alcohol dependency

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript