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Prevalence and patterns of sensory processing behaviors in a large clinical sample of children with prenatal alcohol exposure

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ABSTRACT

Background: Atypical behavioral responses to sensation are reported in a large proportion of children affected by prenatal alcohol exposure (PAE). Systematic examination of symptoms across the fetal alcohol spectrum in a large clinical sample is needed to inform diagnosis and intervention.

Aims: To describe the prevalence and patterns of atypical sensory processing symptoms in a clinical sample of children with PAE.

Methods: Retrospective analysis of diagnostic clinical data from the University of Washington Fetal Alcohol Syndrome Diagnostic and Prevention Network (FASDPN). Participants were ages 3 through 11 years, had a diagnosis on the fetal alcohol spectrum, and Short Sensory Profile (SSP) assessment. The proportions of children categorized with definite differences on the SSP across selected clinical and demographic features were examined with chi-square analyses.

Outcomes: The sample consisted of 325 children; 73.2 % had SSP total scores in the definite difference range. Atypical sensory processing symptoms were significantly more prevalent among children with higher reported levels of PAE. The prevalence of atypical symptoms was comparably high across age, levels of diagnostic severity, and other prenatal/postnatal risks.

Conclusions: Results lend support for altered sensory processing as another domain of brain function affected by the teratogenic impact of PAE, guiding clinical work and research.

What this paper adds

Clinically significant sensory processing differences were highly prevalent in a large clinical sample of children with prenatal alcohol exposure (PAE) and outcomes consistent with fetal alcohol spectrum disorder. Findings corroborate previous reports of atypical sensory processing in smaller samples of children with PAE. Atypical sensory processing symptoms occurred in similarly high proportions across the full fetal alcohol spectrum, and across severity of key diagnostic features [central nervous system (CNS) function, facial features and growth)]. A higher prevalence of sensory processing differences was positively and significantly associated with children who had higher reported levels PAE. Findings lend support for the neurological processing of sensation as

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another neurobehavioral domain that is vulnerable to the teratogenic impact of PAE, and inform diagnosis, intervention and areas for future research.

1. Introduction

Fetal alcohol spectrum disorders (FASD) is an umbrella term that describes the full range of physical, cognitive and behavioral impairments associated with prenatal exposure to alcohol (PAE). FASDs are estimated to occur in at least 1% of the population with prevalence estimates varying based on geography and method of diagnosis (Astley, 2011; Astley, Bledsoe, Davies, & Thorne, 2017; Roozen et al., 2016; Sampson et al., 1997). Diagnoses such as fetal alcohol syndrome (FAS), partial FAS (PFAS), static encephalopathy/alcohol exposed (SE/AE) and neurobehavioral disorder/alcohol exposed (ND/AE) fall broadly under the umbrella of FASD (Astley, 2004). The central nervous system (CNS) impairments associated with these conditions and the subsequent impact of brain-based challenge on daily activities varies among individuals and across the fetal alcohol spectrum, as do the individual strengths, supports and protective factors that foster functional performance and promote resiliency throughout the lifespan (Astley, 2010; Streissguth et al., 2004).

The teratogenic effects of PAE on the central nervous system (CNS) that have functional implications are well-documented and include, but are not limited to, cognitive, motor, memory, executive function and communication impairments (Mattson, Bernes, & Doyle, 2019). Atypical responses to sensation are reported in a large proportion of children with FASD and are widely described clinically (Astley, 2010). However, a systematic examination of the prevalence and patterns of sensory processing symptoms across the fetal alcohol spectrum is lacking. Poorly modulated responses to sensation may be an indicator of CNS dysfunction; and understanding sensory processing patterns can provide insights into maladaptive and dysregulated behaviors and inform intervention (Dunn, Little, Dean, Roberston, & Evans, 2016). The recognition of sensory processing differences in this group of vulnerable and often underserved children has implications for diagnosis and intervention that have not been fully realized due to limited empirical evidence.

Sensory processing is a term used to describe the organization of sensation for use in daily life that involves a continuum of interactions between an individual's neurological thresholds and behavioral responses to sensation (Dunn, 2001, 2007). Behavioral patterns of hyper-responsiveness (e.g., discomfort, irritability) and hypo-responsiveness to sensation (e.g., not noticing or slowed response) have been described within and across sensory domains (e.g., tactile, auditory, vestibular). These atypical or poorly modulated responses to sensation may result in sensory processing or sensory integration disorders (Miller, Anzalone, Lane, Cermak, & Osten, 2007). Sensory processing and sensory integration disorders occur in 5–16 % of children in the general population (Ahn, Miller, Milberger, & McIntosh, 2004) and at much higher proportions (up to 80 %) among children with neurodevelopmental disabilities (Cheung & Siu, 2009). Atypical responses to sensation are well documented, for example, among children with autism spectrum disorder (Ben-Sasson, Hen et al., 2009; Ben-Sasson, Carter, & Briggs-Gowan, 2009; O'Donnell, Deitz, Kartin, Nalty, & Dawson, 2012) and are part of the Diagnostic and Statistical Manual of Mental Disorders (DSM–5) diagnostic criteria for this condition (American Psychiatric Association, 2013).

Previous studies report sensory processing differences among clinical samples of children with PAE including those meeting criteria for FASD (Abele-Webster, Magill-Evans, & Pei, 2012; Carr, Agnihotri, & Keightley, 2010; Franklin, Deitz, Jirikowic, & Astley, 2008; Jirikowic, Olson, & Kartin, 2008; Wengel, Hanlon-Dearman, & Fjeldsted, 2011). Children with FASD have been described as more reactive to touch, visual, and auditory stimuli compared to peers with typical development, and patterns of sensory-seeking behaviors and sensory hypo-responsivity also have been reported (Jirikowic, Olson, & Kartin, 2008). Sensory processing problems have been associated with increased problem behaviors (Franklin, Deitz, Jirikowic, & Astley, 2008) and poorer adaptive functioning among children with FASD (Carr, Agnihotri, & Keightley, 2010; Jirikowic, Olson, & Kartin, 2008). In combination with executive function impairments (behavior dysregulation), sensory processing problems in children with FASD are associated with higher levels of parenting stress among caregivers of children with FASD (Jirikowic, Olson, & Astley, 2012). Sensory processing differences appear to be pervasive among children with FASD and these behaviors have far-reaching impacts on daily life activities and family functioning.

Atypical responses to sensation are also described in prenatally alcohol-exposed animal models (Schneider, Moore, & Adkins, 2011). Schneider et al. (2008, 2011) reported an increased magnitude of withdrawal to tactile stimuli in association with PAE and reduced habituation to sensation in association with prenatal stress in a cohort of rhesus monkeys with prenatal alcohol and stress exposure. Further, symptoms of atypical sensory processing observed in the neonatal phase of development in these animal models were correlated with adult tactile sensory functions, suggesting developmental continuity of sensory processing symptoms (Schneider et al., 2017). Heightened reactivity to more aversive, or mildly painful stimuli has also been reported in rodent models (Rogers, Barron, & Littleton, 2004). The linkage of sensory processing disorder to PAE in animal models coupled with emerging evidence of associated neurophysiological changes and genetic influences (Schneider et al., 2008,2011,2017) bolster the need to more fully understand sensory processing disorders in children affected by PAE.

This study aimed to describe the prevalence of atypical sensory processing behaviors in a large clinical population of children systematically diagnosed with FASD, to explore risk factors associated with atypical sensory processing behaviors, and to explore sensory processing patterns across this population. The following research questions were asked:

1 What is the prevalence of atypical sensory processing behaviors among children with FASD?
2 Does the prevalence of atypical sensory processing behaviors vary by child characteristics [e.g., age, gender, level of alcohol exposure, FASD diagnosis, attention deficit hyperactivity disorder (ADHD)], or other prenatal or postnatal risk factors?

3 What are the patterns of sensory processing behaviors across sensory domains (e.g., tactile, auditory, vestibular) in children with FASD?

2. Materials and methods

2.1. Procedures

A retrospective analysis of clinical data from the Fetal Alcohol Syndrome Diagnostic and Prevention Network (FAS DPN) at the University of Washington was completed. The clinic has provided diagnostic evaluations for FASD, including FAS, since 1993. Patients of all ages (newborn to adult)newborn to adult are evaluated. The FAS DPN database currently contains over 2,000 fields of data (exposures and outcomes) on approximately 3000 patients with prenatal alcohol exposure. Data used for this study were collected with University of Washington Human Subjects approval and patient consent at the time of diagnosis.

All patients in the FAS DPN database were evaluated for FASD using the *4-Digit Diagnostic Code* (updated and coded according to criteria from the most current edition (Astley, 2004), an interdisciplinary approach to diagnosis guided by empirically validated criteria (Astley, 2004, 2013). The four digits of the FASD *4-Digit Diagnostic Code* reflect the magnitude of expression of the four key diagnostic features of FASD, in the following order: 1) growth deficiency, 2) FAS facial phenotype, 3) structural/functional CNS abnormalities, and 4) prenatal alcohol exposure. The magnitude of expression of each feature is ranked independently on a four-point Likert scale, with '1′ reflecting complete absence of the FASD feature and '4' reflecting a strong and classic presentation of the feature. Each Likert rank is specifically case defined. There are 102 4-Digit codes that fall broadly under the umbrella of FASD. These codes cluster into four clinically meaningful FASD diagnostic subcategories (Astley, 2004): fetal alcohol syndrome (FAS); partial FAS (PFAS); static encephalopathy/alcohol exposed (SE/AE); and neurobehavioral disorder/alcohol exposed (ND/AE).

2.2. Participants

Selected data from children who met the following study inclusion criteria were used: a) between 3 and 11 years old at time of diagnostic clinic visit, b) FASD diagnosis (including FAS or PFAS, SE/AE, or ND/AE), and c) completed Short Sensory Profile (SSP; McIntosh, Miller, Shyu, & Dunn, 1999). The FAS DPN began administering the SSP in the year 2000. Subjects with missing data on more than one-third of the items in any SSP domain were excluded.

2.3. Measures

2.3.1. Sensory processing behaviors

Short Sensory Profile (*SSP*: McIntosh et al., 1999) The SSP is a 38-item caregiver questionnaire that measures children's behavioral responses to sensation in daily life. The SSP is the short version of the longer 125-item Sensory Profile that is used for screening and research purposes (Dunn, 1999). The SSP examines behaviors in the sensory domains of Tactile Sensitivity, Taste/Smell Sensitivity, Movement Sensitivity, Under-responsive/Seeks Sensation, Auditory Filtering, Low Energy/ Weak, and Visual/Auditory Sensitivity. Caregivers report how frequently children respond in the way described by each item using a 5-point Likert scale (1 = almost always; 2 = frequently; 3 = occasionally; 4 = seldom; 5 = almost never). The SSP has high internal consistency for the total score (Cronbach's alpha = 0.96) and domain scores (Cronbach's alpha = 0.82 to 0.89). Lower raw scores indicate more atypical sensory processing behaviors. Raw scores are categorized as typical performance (scores at or above -1.0 standard deviation (SD) from the mean), probable differences (scores at or above -2.0 SD but lower than -1.0 from the mean) and 3) definite difference (scores below -2.0 SD from the mean).

2.3.2. FASD diagnosis and features

4-Digit Code FASD Diagnosis (FAS; PFAS; SE/AE; ND/AE). See full description above (Astley, 2004) and rankings for the following diagnostic features.

Growth Deficiency ('Growth Rank': 1 = none; 2 = mild; 3 = moderate; 4 = severe). This variable yields the first digit in the 4-Digit FASD Diagnostic Code and documents the magnitude of prenatal and/or postnatal growth deficiency (Astley, 2004).

FAS Facial Phenotype ('Face Rank': 1 = none; 2 = mild; 3 = moderate; 4 = severe). This variable represents the second digit in the 4-Digit FASD Diagnostic Code and documents the magnitude of expression of FAS facial phenotype defined by short palpebral fissure lengths, a smooth philtrum, and a thin upper lip (Astley, 2004).

CNS Likelihood of Structural Abnormality ('CNS Rank': 1 = unlikely; 2 = possible; 3 = probable; 4 = definite). This variable yields the third digit in the 4-Digit FASD Diagnostic Code. These four ranks document the increasing likelihood of CNS structural abnormality. Alcohol is a teratogen that interferes with the structural development of the fetal brain. This, in turn, can lead to abnormal function. The greater the dysfunction, the higher the probability of CNS structural abnormality (Astley, Aylward et al., 2009; Astley et al., 2009; Astley, 2013). The first three CNS Ranks document the severity of CNS dysfunction (Rank 1-no dysfunction; Rank 2-mild-to-moderate dysfunction; Rank 3-severe dysfunction). CNS Ranks 1–3 are based on brain function (executive function, memory, cognition, social/adaptive skills, academic achievement, language, motor, attention, and activity level) assessed by an interdisciplinary team using standardized psychometric tools. CNS Rank 4 documents the presence of direct evidence of CNS structural and/or neurological abnormalities (e.g., microcephaly, structural brain abnormalities, a seizure disorder of prenatal origin, or other hard neurological signs).

Prenatal Alcohol Exposure ('Alcohol Rank': 1 = confirmed absence of exposure; 2 = unknown exposure; 3 = confirmed exposure; level unknown or moderate; 4 = confirmed exposure; level high). Alcohol exposure is the fourth digit in the 4-Digit FASD Diagnostic Code, which is ranked according to the quantity, timing, frequency, and certainty of exposure during pregnancy. The ranking is determined by available records, maternal report or report from others who observed exposure. A diagnosis under the umbrella of FASD requires a confirmed PAE (Rank 3 or 4) with one exception-FAS. FAS requires the Rank 4 FAS facial phenotype which is so highly specific to (caused only by) PAE that the presence of the Rank 4 FAS facial phenotype offsets the need for an independently confirmed history of alcohol exposure.

2.3.3. Other risk factors

Attention Deficit Hyperactivity Disorder (ADHD) Diagnosis This variable documents a confirmed previous ADHD diagnosis from a qualified provider or as a result of the FAS DPN clinical assessment.

Other Prenatal Risk Rank (1 = no risk; 2 = unknown risk; 3 = some risk; 4 = high risk) (Astley, 2004). Other prenatal risk factors documented in the FAS DPN clinical database include poor prenatal care, pregnancy complications, presence of other syndromes/genetic abnormalities, and prenatal exposure to other substances (e.g., medications, tobacco, illicit drugs, and/or other teratogens). The 4-Digit Code ranks the magnitude of these other prenatal risks in a single composite measure labeled "Other Prenatal Risks Rank." Rank 4 is assigned when there is exposure to another teratogen (e.g., Dilantin) or when another syndrome or genetic condition is present (e.g., Down syndrome, Fragile X, etc.). Rank 3 is assigned to all other prenatal risks. The ranking is determined by available records and caregiver or other report on intake forms and/or clinical interview (Astley, 2004)

Other Postnatal Risk Rank (1 = no risk; 2 = unknown risk; 3 = some risk; and 4 = high risk) (Astley, 2004). Postnatal risk factors documented in the FAS DPN database include perinatal complications, number of home placements, physical and/or sexual abuse, neglect, and trauma. The 4-Digit Code ranks the magnitude of these other postnatal risks in a single composite measure labeled "Other Postnatal Risks Rank". Rank 4 is used to note severe postnatal circumstances that have been shown to have a significant adverse effect on development in most instances. Examples include physical or sexual abuse, multiple home placements, and severe neglect. Rank 3 is used to note conditions akin to those in Rank 4, but the circumstances are less severe. The ranking is determined by available records and caregiver or other report on intake forms and/or clinical interview.

2.4. Data analysis

SPSS version 19.0 (IBM Corp, 2010) and MedCalc for Windows, version 18.6 (MedCalc Software) were used to conduct the analyses. Descriptive statistics (means, standard deviations, proportions) were used to profile the demographic characteristics of the study population and SSP outcomes for the total score and seven domain scores. Relationships between the SSP total score and SSP domain score categories (typical performance, probable difference, and definite difference) and selected child demographics (age, gender, ADHD; co-occurring prenatal and postnatal risk factors) and FASD diagnosis and features were examined using chi-squared (χ^2) and Fisher's exact tests.

To further describe and explore sensory processing patterns, the proportion of item-level behaviors scored by caregivers as occurring always (100 % of the time or more) or frequently (about 75 % of the time) was analyzed. In addition, SSP outcomes were dichotomized into 2 categories: 1) typical performance and probable differences (scores at or above -2.0 SD from the mean) and 2) definite difference (below -2.0 SD from the mean) to explore domain scores and sensory over-responsiveness (SOR) characteristics. A "Sensory Over-Responsiveness (SOR)" score was calculated by combining SSP Tactile Sensitivity items (1–7), Taste/Smell Sensitivity items (8–11), Movement Sensitivity items (12–14), and Visual Auditory Sensitivity items (34–38 (Mazurek et al., 2013). Lower SOR scores indicate more sensory over-responsiveness. Finally, SSP domain scores and prevalence for children with FASD with and without ADHD were contrasted. All results were considered significant at 2-sided p-values of $< .05$. P-values for post-hoc analyses were exploratory and were not corrected for multiple comparisons and should be interpreted with appropriate caution.

3. Results

Records from 325 participants met study inclusion criteria; 43 were missing some SSP data. When item-level data were missing the average of the subject's remaining scores in the incomplete sensory domain was calculated and rounded to the closest whole number. This value replaced the missing score(s) in that domain. The adjusted item scores were rounded to the closest whole number. Clinical and sociodemographic characteristics for the total sample (n = 325) are presented in Table 1. Children had a range of diagnoses on the fetal alcohol spectrum, with the largest proportion diagnosed with ND/AE, followed by SE/AE and then FAS or PFAS (Table 1. The sociodemographic and clinical profile of this study sample is highly representative of the larger FAS DPN population of 3000 patients Astley, 2010).

3.1. Prevalence of atypical sensory processing behaviors

The proportion of children classified in each SSP category (typical performance, probable difference, definite difference) for each of the seven sensory domains and total score is shown in Fig. 1. Results indicated that 73.2 % of children in this sample were categorized with definite differences on the SSP total score. Differences were noted across all sensory domains with the highest proportions of definite differences in the domains of Auditory Filtering (81.8 %) and Under-responsive/Sensation Seeking (80.0 %). Definite differences in other domains associated with sensory over-responsiveness (SOR) were noted, but to a lesser extent (Fig. 1).

Table 1
Demographic and clinical profiles of 325 children with prenatal alcohol exposure.

Characteristic	N (valid %)
Gender	
Female	124 (38.2)
Male	201 (61.8)
Age at FASD Diagnosis (years)	
3-5.9	117 (36.0)
6-10.9	208 (64.0)
Mean (SD) Range	6.9 (2.1) 3.0–10.9
Race/Ethnicity	
Caucasian	157 (48.3)
African American	33 (10.2)
Native American/Canadian	23 (7.1)
Hispanic	14 (4.3)
Other (Including mixed race)	98 (30.2)
FASD Diagnosis[1]	
FAS	13 (4.0)
PFAS	19 (5.8)
SE/AE	96 (29.5)
ND/AE	197 (60.6)
Prenatal Alcohol Exposure: Alcohol Rank	
Rank 1: Confirmed absent	0 (0.0)
Rank 2: Unknown*	2 (0.6)
Rank 3: Confirmed/Amount moderate or unknown	160 (49.2)
Rank 4: Confirmed/Amount high	163 (50.2)
Other Prenatal Risks: Rank	
1: No risk	3 (0.9)
2: Unknown risk	12 (3.7)
3: Some risk	302 (92.9)
4: High risk	8 (2.5)
Postnatal Risks: Rank	
1: No risk	20 (6.1)
2: Unknown risk	3 (0.9)
3: Some risk	138 (42.5)
4: High risk	164 (50.5)
ADHD Diagnosis	
Yes	145 (46.9)
Caregiver at time of Diagnosis	
Biological parent	95 (30.2)
Other biological family member	44 (14.0)
Foster parent	65 (20.6)
Adoptive parent	126 (40.1)
Other	16 (5.1)

Notes. * 2 subjects with FAS had unknown prenatal alcohol exposures.
fetal alcohol spectrum disorder (FASD); fetal alcohol syndrome (FAS), partial FAS (PFAS), static encephalopathy/alcohol exposed (SE/AE); neurobehavioral disorder/alcohol exposed (ND/AE); attention deficit hyperactivity disorder (ADHD).

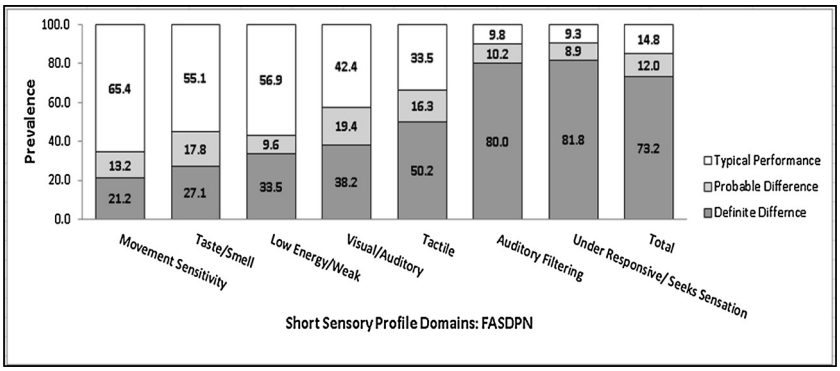

Fig. 1. Short Sensory Profile outcomes among the 325 children with prenatal alcohol exposure. Prevalence of typical performance, probable difference and definite difference across the seven SSP domains and total score.

Table 2
Associations between atypical sensory processing and clinical characteristics.

Characteristic	SSP Total Score		
	Typical/Probable Difference N (valid %) 87 (26.8)	Definite Difference N (valid %) 238 (73.2)	χ^2 (p-value)
Prenatal Alcohol Exposure			
Rank 3	54 (33.8)	106 (66.3)	8.2 (.004)
Rank 4	32 (19.6)	131 (80.4)	
FASD Diagnosis			
ND/AE	56 (28.4)	141 (71.6)	1.0 (.60)
SE/AE	22 (22.9)	74 (77.1)	
FAS/PFAS	9 (28.1)	23 (71.9)	

Notes. fetal alcohol spectrum disorder (FASD); fetal alcohol syndrome (FAS), partial FAS (PFAS), static encephalopathy/alcohol exposed (SE/AE); neurobehavioral disorder/alcohol exposed (ND/AE).

3.1.1. Sensory processing differences: FASD diagnostic characteristics

Atypical sensory processing symptoms (SSP total scores in the definite difference category) were significantly more prevalent among children with confirmed PAE at reportedly high levels (Alcohol Rank 4) than among those with confirmed PAE at unknown or reportedly lower levels (Alcohol Rank 3; Table 2). The prevalence of atypical sensory processing (SSP total scores in the definite difference range) was comparably high (72%–77%) across all FASD diagnoses (Table 2). The same pattern of distribution was observed across the four Growth Ranks, Face Ranks and CNS Ranks (data not shown). The prevalence of atypical sensory processing (SSP total scores in the definite difference range) was comparably high (67%–81.7%) across all ranks for growth, face and CNS (data not shown).

3.1.2. Sensory processing differences: child characteristics and risk factors

The prevalence of atypical sensory processing behaviors (SSP total scores in the definite difference range) did not vary significantly by age or clinical ranking of other prenatal risk factors or postnatal risk factors. Although a higher proportion of children with ADHD (73.1 %) had definite differences in the SSP total score than those without ADHD (68.9 %), differences in the total score were not statistically significant ($\chi^2 = 3.2$, $p = .07$). Significant differences by gender were found. Males were significantly more likely (78.1 %) to present with total scores in the definite difference range than females (65.3 %; $\chi^2 = 6.4$, $p = .011$).

3.2. Sensory processing patterns: exploratory analyses across behaviors and domains

Sensory processing patterns were further explored in three ways. Fig. 2 illustrates the proportion of item-level behaviors that were ranked by caregivers as occurring always (100 % of the time) or frequently (about 75 % of the time) within each of the seven sensory processing domains. Data show the frequency of specific sensory processing behaviors that are most problematic in daily life.

Sensory processing patterns were also compared by dichotomizing SSP total score outcomes into 2 categories, those with 1) typical performance and probable differences (scores at or above -2.0 SD from the mean) or 2) definite difference (at or below -2.0 SD from the mean) (see Fig. 3). Children with definite differences on the SSP total score were about twice as likely to have definite differences in both the Under-responsive/Seeks Sensation and Auditory Filtering domains as compared to those without. However, atypical behaviors in these two domains were the predominant symptoms in both groups. In contrast, the proportion of children with definite differences in the other sensory processing domains was consistently higher for those with definite differences on the SSP total score. For example, there was approximately a10-fold increase in the prevalence of definite differences in the domain of Movement Sensitivity; an 8-fold increase in the prevalence of definite differences in the domains of Tactile Sensitivity and Visual and Auditory Sensitivity; and a 4-fold increase in Taste/Smell Sensitivity.

These four sensory processing domain scores have items that comprise a score some have coined as the SOR score (Mazurek et al., 2013). Subsequently, we explored if the distribution of SOR scores differed between those who did and did not receive a total score in the definite difference range. We found the distribution of SOR scores was significantly different between groups (Area under the ROC curve (AUC) 0.935, $p < 0.0001$) and could be used to sort the groups with significant accuracy (81.5 % accuracy at the most accurate cut-point). The distribution of SOR scores differed significantly more between the two groups than either the Auditory Filtering score (AUC 0.860, significantly different than SOR, $p = 0.0092$) or the Under-responsive/Seeks Sensation score (AUC 0.870, significantly different than SOR, $p = 0.0141$).

Finally, because of the high prevalence of co-occurring ADHD in this sample and the atypical sensory processing behaviors that have been documented in other studies of children with ADHD (Panagiotidi, Overton, & Stafford, 2018), we also explored SSP patterns for the children with FASD in this sample who had ADHD compared to those who did not have ADHD (see Fig. 4). While the overall pattern across the seven domains in both groups was similar, definite differences in the Under-responsive/Sensation Seeking domain (89.0 % vs 76.2 %, respectively; $\chi^2 = 8.9$, $p = 012$) and Auditory Filtering domain (88.3 % vs 72.6 %, respectively; $\chi^2 = 11.9$ $p = 001$) were significantly more prevalent in children with co-occurring ADHD relative to those without ADHD, and males were significantly more likely to have a co-occurring diagnosis of ADHD (53.6 %) than females (35.7 %) ($\chi^2 = 9.3$, $p = .002$).

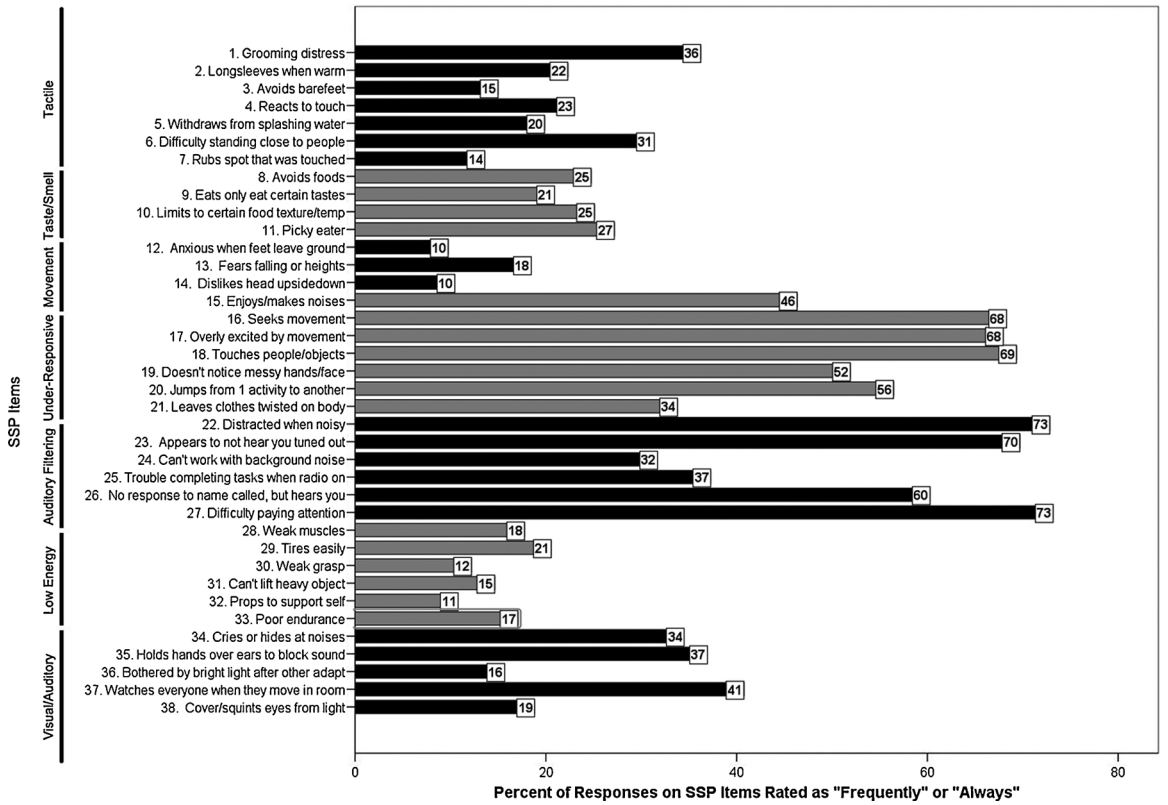

Fig. 2. Proportion of SSP item-level behaviors by domain ranked by caregivers as occuring Always (100% of the time or Frequently (about 75% of the time).

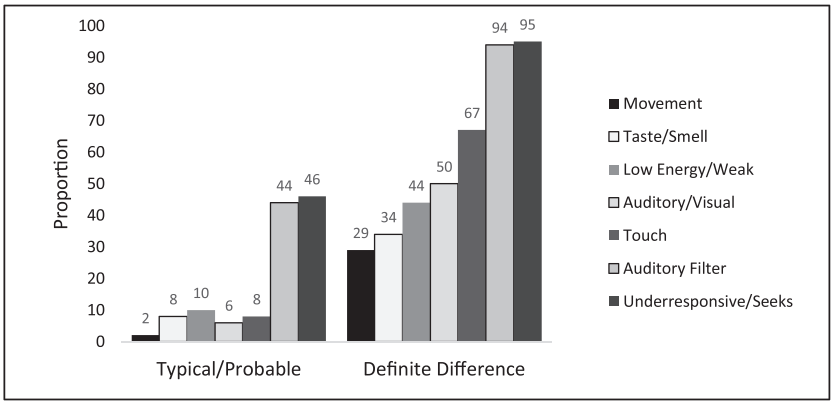

Fig. 3. Comparison of SSP domain scores by total score categories (definite difference vs. typical performance/probable difference. Bars reflect the proportion of the children with definite difference by SSP domain.

4. Discussion

This study is the first to report the prevalence of symptoms of sensory processing dysfunction in a large clinical sample of children with PAE and to demonstrate a significant positive association between prevalence of atypical sensory processing and PAE. Atypical sensory processing symptoms, as determined by total scores in the definite difference category of the SSP were prevalent in a high proportion (73.7 %) of children ages 3–11 years old, and significantly more prevalent among children with higher reported levels of confirmed PAE (Alcohol Rank 4) as compared to children with unknown or lower reported levels of confirmed PAE (Alcohol Rank 3). Males in this sample also had a significantly higher prevalence of sensory processing differences than females, but prevalence did not differ by other selected demographic or risk factors such as age, ADHD diagnosis, or level of other prenatal or postnatal risks. Results from this large clinical sample corroborate and strengthen findings that atypical sensory processing symptoms occur frequently

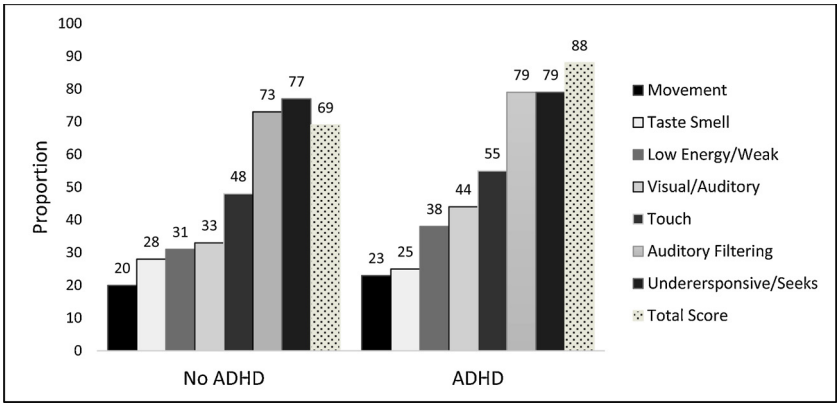

Fig. 4. Comparison of SSP domain scores for children with prenatal alcohol exposure who have co-occurring ADHD and those without ADHD.

among children with PAE, and that higher levels of PAE may induce more impairments in this domain of CNS functioning. While the latter finding needs replication and more research is needed to understand how PAE and sensory processing may be linked, direct associations between PAE and atypical sensory processing behaviors have been shown in alcohol-exposed animal models (Schneider et al., 2008, 2017).

The high prevalence of sensory processing differences across diagnoses on the fetal alcohol spectrum is consistent with prevalence estimates reported in previous studies of children with PAE that used smaller clinical samples (Abele-Webster et al., 2012; Carr, Agnihotri, & Keightley, 2010; Franklin, Deitz, Jirikowic, & Astley, 2008; Jirikowic, Olson, & Kartin, 2008). Even when using the conservative cut-off of -2.0 SD from the mean, almost three quarters (73.7 %) of the children with PAE had atypical responses to sensation. This prevalence falls at the higher end of the range of prevalence estimates (30%–80%) reported for other children with neurodevelopmental disabilities (Ben-Sasson, Hen et al., 2009; Ben-Sasson, Carter et al., 2009; Tomchek & Dunn, 2007). The item-level behaviors analyzed for this study shed light on how these behaviors affect participation in daily activities (e.g., distress during grooming; difficulty functioning with background noise, seeking all sorts of movement). Atypical sensory processing behaviors clearly occur and are a clinically significant problem among children with PAE.

As expected, atypical sensory processing behaviors were highly prevalent across the full fetal alcohol spectrum and severity of diagnostic features. Notably, impaired sensory processing occurred across all levels of CNS dysfunction that ranged from mild to moderate/severe. A striking new finding is that the prevalence of atypical sensory behaviors was significantly higher in children with higher levels of confirmed PAE. This association between PAE and atypical sensory processing behaviors in a large clinical sample lends support for sensory processing and integration as another CNS domain that is vulnerable to the teratogenic impact of alcohol. These clinical findings are substantiated by findings in animal models where less optimal sensory processing function (tactile over-responsivity and vestibular function) has been described in primates exposed to moderate levels of alcohol as compared to non-exposed controls (Schneider et al., 2008,2017, 2011).

Interestingly, the prevalence of atypical sensory processing behaviors in this clinical sample of children did not differ based on reported levels of other prenatal risks (e.g., pregnancy complications, other prenatal exposures, other syndromes/genetic abnormalities) or postnatal risks (e.g., physical/sexual abuse, neglect, trauma). The high prevalence of adverse childhood experiences reported in this clinical sample, coupled with findings by Schneider et al. (2017) that revealed a main effect of prenatal stress exposure in relation to atypical sensory processing in alcohol and stress exposed primate models prompted a post hoc analyses. The analyses explored whether more specific prenatal (i.e., exposure to tobacco or other illicit drugs, pregnancy complications, family history of developmental disorders, etc.) and postnatal (i.e., physical abuse, sexual abuse, neglect, multiple home placements) risks affected sensory processing outcomes. These analyses (data not shown) also did not reveal any significant relationships between SSP outcomes and specific risk factors. It is possible that in both analyses the presence of other prenatal and postnatal risk factors were not reported clinically with sufficient accuracy to detect true relationships with sensory processing outcomes.

Adversity and complex trauma have been implicated as risk factors that alter neurobehavioral development in areas that include self-regulation and sensory processing (van der Kolk, 2003). However, comparable systematic or descriptive clinical studies of sensory processing behaviors in children with complex trauma are limited for comparison. Purvis, Brooks, Cross, and Becker Razuri (2013) reported sensory processing deficits occurred in a small sample (n = 19) of adopted children 3–14 years old with histories of early deprivation or abuse. However, similar to other studies that describe the neurobehavioral implications of adverse childhood experiences and sensory processing in children, the presence (or absence of) PAE is not clearly accounted for (Fraser, MacKenzie, & Versnel, 2017; Rinne-Albers, van der Wee, Lamers-Winkelman, & Vermeiren, 2013). Future research that examines sensory processing in children with PAE who are also at high risk for other adverse childhood experiences such as trauma, abuse and neglect (Price, Cook, Norgate, & Mukherjee, 2017) must account for the potential interaction of both PAE as a teratogen and cumulative environmental risk factors as they collectively may alter developmental outcomes and trajectories.

Males in this sample had a significantly higher prevalence of sensory processing differences than females, but prevalence estimates across the SSP total score did not differ by other selected demographic factors (age or ADHD diagnosis). Gender differences,

developmental implications and the overlap of sensory processing symptoms with other neurobehavioral characteristics (e.g., inattention) are noteworthy because they have been examined in other groups of children with sensory processing problems and neurodevelopmental disabilities, but findings are mixed and inconclusive (Ghanizadeh, 2011; Lane, Reynolds, & Thacker, 2010; Miller, Nielsen, & Schoen, 2012; Panagiotidi et al., 2018). Specific to children with FASD, Abele-Webster et al. (2012) also reported differences in the domains of Under-responsive/Sensation Seeking and Auditory Filtering to be the most prevalent among a small clinical sample (n = 26) of children with FASD, but found only weak correlations between sensory processing and attention problems as measured by the Connors Parent Rating Scales-Revised (1997). More recently, SSP Auditory Filtering behaviors were extensively examined by McLaughlin et al. (2019) as a proxy for listening behaviors among children with FASD. Listening problems, attributed to suprathreshold auditory processing deficits, were highly prevalent across all diagnosis on the fetal alcohol spectrum and occurred in the absence of hearing loss. Auditory Filtering differences were also significantly correlated with age and ADHD.

These relationships and potential shared mechanisms underlying sensory processing impairments are being investigated through biobehavioral studies of children with and without clinical sensory processing deficits. For example, Davies, Chang, and Gavin (2009) measured auditory event-related potential (ERP) responses using electroencephalography (EEG) in children with sensory processing disorders, children with typical development and adults. They reported differences in the maturational trajectories of sensory gating, the brain's capacity to regulate sensitivity to sensory stimuli, between children with and without clinical sensory processing disorders (SPD). The children with SPD also showed a diminished ability to filter repeated auditory input and did not selectively regulate sensitivity to sensory stimuli. Owen et al. (2013) reported disrupted white matter microstructure integrity examined through diffusion tensor imaging (DTI) in a sample of 8–11 year-old males with and without SPD. The differences noted in posterior cerebral tracts were strongly correlated with behavioral measures of atypical sensory processing and integration. The authors concluded that abnormal white matter may be a biomarker for children with SPD with potential to distinguish this disorder from other clinical conditions such as autism and ADHD. Neuroimaging and neurophysiological studies of sensory processing need replication in children with FASD to corroborate behavioral findings, disentangle co-morbid symptoms, and examine these brain-behavior relationships in the presence of PAE as a teratogen.

The predominant patterns of differences in Under-responsive/Sensation Seeking and Auditory Filtering domains coupled with differences, but to a lesser degree, in domains that represent sensory over-responsiveness are congruent with profiles reported in previous studies of children with FASD (Abele-Webster et al., 2012; Carr, Agnihotri, & Keightley, 2010; Franklin, Deitz, Jirikowic, & Astley, 2008). The distinction of subtypes for children with PAE is beyond the scope of the present study and limited by the use of the SSP, however, the robust patterns of under-responsive/sensation seeking tendencies are notable, but wider ranging differences (21%–50%) in sensory domains that represent sensory-over-responsiveness, suggest more analyses of subtypes is an important area of future study. Our exploratory analysis also suggests that the presence of more frequent sensory-over responsive behaviors may potentially differentiate atypical sensory processing from other overlapping symptoms in this population, such as inattention. Clinical sensory processing subtypes and their relation to behavior patterns as well as neurobehavioral and diagnostic characteristics within and between different populations of children are important areas of focus of current research (Little, Dean, Tomchek, & Dunn, 2017; Little, Dean, Tomchek, & Dunn, 2018; Miller, Schoen, Mulligan, & Sullivan, 2017).

Because of the overarching interest in sensory behaviors and sensory processing patterns between different diagnostic groups and the lack of previous studies that include a comparison group of children with FASD, exploratory post hoc contrasts were made using data from a previous study that reported SSP outcomes for children of similar age on the autism spectrum. Fig. 5 shows contrasts between outcomes across the three SSP classification categories of typical, probable and definite differences using outcomes from 42 children with ASD (O'Donnell et al., 2012) in comparison to the current sample of children with FASD.

Notably, the proportion of sensory processing definite differences for the total score and five out of seven domain scores were higher for children with FASD compared to children with ASD. Children with FASD showed more atypical behaviors in the definite difference category across all domains except for the Taste/Smell domain where the children with ASD had a greater proportion of definite differences; and the Movement Sensitivity domain, where the proportion of definite differences was comparable. Results reveal specific sensory domains (e.g., oral sensory processing) that may be different, as well as sensory behaviors that may overlap between children with FASD as compared to children with ASD-a group with well-documented sensory processing differences (Ben-Sasson, Hen et al., 2009; Ben-Sasson, Carter et al., 2009; Caminha & Lampreia, 2012) and diagnostic criteria that includes atypical sensory symptoms. While these exploratory analyses must be interpreted with caution, findings further demonstrate the need to include children with FASD in systematic comparisons of sensory processing with other clinical groups. Systematic comparisons can enrich larger scale research efforts that aim to improve the early identification of sensory processing impairments in children at-risk; inform individualized, targeted interventions; and examine shared neurological mechanisms underlying sensory processing disorders (Little et al., 2018).

4.1. Limitations

One study limitation is the use of caregiver reported outcome to measure sensory processing differences. Caregivers may be biased in their reporting of symptoms. However, caregiver report is an accepted clinical means of measuring ecologically valid behaviors that reflect daily function and responses in different environments. Likewise, the SSP is a short version of a more comprehensive questionnaire. However, the SSP has adequate to good psychometric reliability and validity and has been used extensively in research (Tomchek & Dunn, 2007). Because this was a retrospective study, our data do not reflect the use of the newer and perhaps more refined tools that can measure more and different dimensions of sensory processing (Jorquera-Cabrera, Romero-Ayuso, Rodriguez-Gil, & Triviño-Juárez, 2017). The precise measurement of sensory processing for assessment and intervention purposes remains a

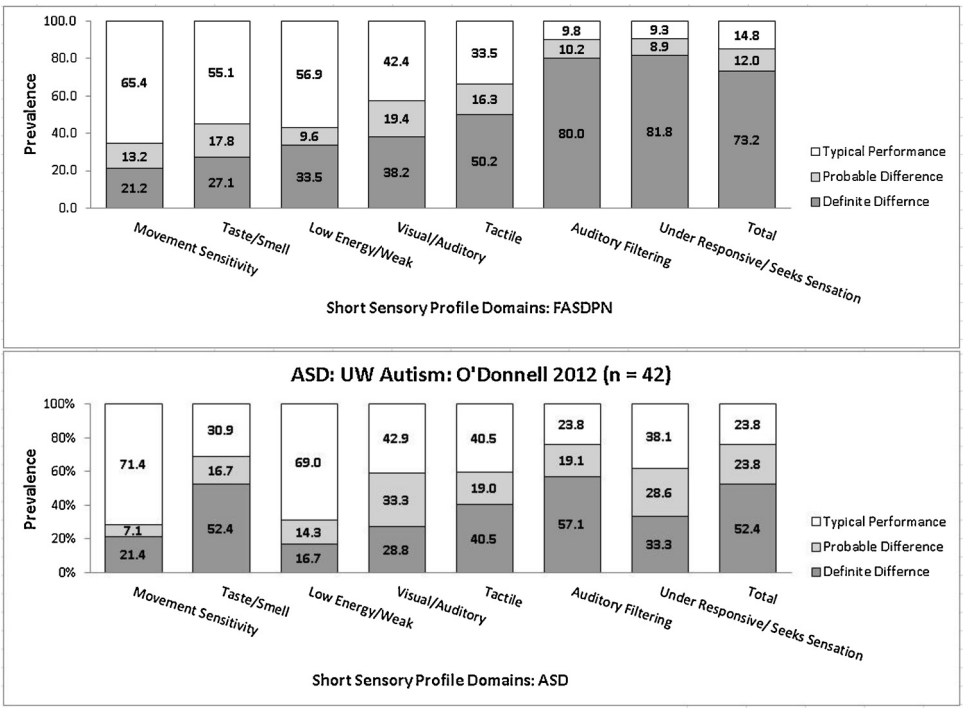

Fig. 5. Comparison of SSP outcomes among A) the 325 children with FASD in the current study and B) 42 children with autism spectrum disorder in a previous published study (O'Donnell et al., 2012). Proportion of of typical performance, probable difference and definite difference across the seven SSP domains and total score.

research and clinical priority, and combined approaches that use questionnaires, direct clinical observation and performance-based assessments are recommended Tvassoli et al. (2019).

5. Conclusion

Results demonstrate that clinically significant sensory processing differences are highly prevalent in a large clinical sample of children with PAE and diagnostic outcomes on the fetal alcohol spectrum. Higher prevalence of sensory processing differences among children with higher levels of PAE suggests that neurological processing of sensation may be vulnerable to the teratogenic impact of PAE. From a clinical standpoint, this reinforces that sensory processing behaviors warrant attention in diagnostic assessments since they occur across the full spectrum of diagnosis. The recognition of sensory processing differences is also an important source of data to inform interventions that can help reframe challenging behaviors, provide positive behavioral supports and accommodations and help tailor environments that enable successful participation in home, school and community.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.ridd.2020.103617.

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