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Neurodevelopmental Influences of the Early Caregiving Environment on Sensory Processing: Implications for Mental Health

A dissertation submitted in partial satisfaction of the requirements for the degree Doctor of

Philosophy in Psychology

by

Adriana Sofía Méndez Leal

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ABSTRACT OF THE DISSERTATION

Neurodevelopmental Influences of the Early Caregiving Environment on Sensory Processing: Implications for Mental Health

by

Adriana Sofía Méndez Leal Doctor of Philosophy in Psychology University of California, Los Angeles, 2024 Professor Jennifer A. Silvers, Chair

Early life adversity impacts more than half of youth worldwide and is thought to contribute to a third of adult mental illness. In particular, early caregiving adversity (ECA) – including exposure to abuse, neglect, or circumstances surrounding placement in institutional or foster care – has profound consequences for socioemotional, cognitive, and behavioral development, and is a potent contributor to later psychopathology. While ECA research has focused on high-level cognitive and socioemotional capabilities crucial to preventing psychopathology, preliminary work in children suggests that ECA may also elevate risk for lower-level sensory processing challenges. Across three multi-method studies, this dissertation introduces consistent evidence that diverse forms of severe early caregiving adversity increase risk for sensory processing challenges that persist beyond early childhood into adolescence and young adulthood, and furthermore suggests that these sensory symptoms may contribute to the

development of mental health challenges across populations. Study 1 found that two rare but profound categories of ECA are associated with elevated parent-reported sensory processing challenges in children and adolescents, and that these in turn are linked to elevated internalizing and externalizing symptoms. Study 2 showed that self-reported adversity-linked sensory challenges (and the same accompanying associations with internalizing symptoms) are present following experiences of more prevalent forms of ECA (e.g., neglect, abuse), persisting into adulthood. In addition, this study evaluated three candidate neurodevelopmental mechanisms that may contribute to the emergence of these sensory processing challenges, indicating affective and regulatory processes may be particularly important to consider in the context of enduring sensory symptoms. Lastly, Study 3 extended these findings, reporting elevated recruitment of prefrontal regulatory regions during aversive sensory stimulation in youth with histories of caregiving institutionalization, and providing novel evidence for the importance of regulatory experiences in the development and persistence of sensory processing challenges following ECA. Taken together, these studies advance developmental models of ECA by pointing to the importance of sensory processing as an underexplored and potentially tractable facet of development that may be critical to long-term mental health.

The dissertation of Adriana Sofía Méndez Leal is approved.

Andrew J. Fuligni

Adriana Galván

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Lucina Q. Uddin

Jennifer A. Silvers, Committee Chair

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Dedication

For Vivian M. Leal and Daniel J. Méndez, who taught me daily the power that stems from the synergy of logic and humanity, people and data

For Vivian G. Leal, whose joie de vivre and fearless adventuring so inspire me

Y para Julio y Carmen Méndez, quienes cultivaron una curiosidad inagotable importada de Meneses y transmitida a través de generaciones, sin la cual nada de esto sería posible.

List of Tables

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Study 1 is a version of the following journal article:

Méndez Leal, AS.*, Alba, LA.*, Cummings, KK., Jung, J., Waizman, YH., Guassi Moreira, JF., Saragosa-Harris, NM., Ninova E.,Waterman, J., Langley, AK., Tottenham, N., Silvers, JA.⁺, Green, SA⁺. Sensory processing differences as a novel link between early caregiving experiences and mental health. *Development and Psychopathology* (2022). DOI: 10.1017/S0954579422000633

ASML and LAA led pre-registration of methodology, formal analysis, and drafting of the

manuscript. ASML, LAA, SAG, and JAS contributed to conceptualization, methodology, and

revision of the manuscript. KKC, JJ, YHW, JFGM, NMSH, EN, JW, AKL, and NT contributed

to investigation and resources and provided comments on the manuscript. SAG and JAS were the

principal investigators for this study.

Figure I - 2 is an adaptation of Figure 1 in the following journal article:

Méndez Leal, AS., Silvers, JA. Neurobiological Markers of Adolescent Resilience to Early Life Adversity. Biological Psychiatry: Cognitive Neuroscience and Neuroimaging. (2021). DOI: 10.1016/j.bpsc.2020.08.004

Select Vita

Publications

- Waller, C.R., **Méndez Leal, A.S.,** & Silvers J.A. Disparities in depression and anxiety that impact self-identified sexual minorities also affect a broader group of same-gender attracted young adults. *Journal of Adolescent Health* (2023).
- Guassi Moreira, J.F., **Méndez Leal, A.S.,** Waizman, Y.H., Tashjian, S.M., Galván, A., & Silvers, J.A. Value-based neural representations predict social decision preferences. *Cerebral Cortex* (2023).

Publications (continued)

- **Méndez Leal, AS.,** Silvers, JA., Carroll, JE., Cole, S., Mahrer, N., Ross, K., Dunkel Schetter, C. Maternal Early Life Adversity Predicts Increased Pro-inflammatory Gene Transcription in the Third Trimester*. Brain, Behavior, and Immunity* (2022).
- **Méndez Leal, AS.*,** Alba, LA.*, Cummings, KK., Jung, J., Waizman, YH., Guassi Moreira, JF., Saragosa-Harris, NM., Ninova E.,Waterman, J., Langley, AK., Tottenham, N., Silvers, JA.⁺, Green, SA⁺. Sensory processing differences as a novel link between early caregiving experiences and mental health. *Development and Psychopathology* (2022).
- Guassi Moreira, JF., **Méndez Leal, AS.,**Waizman, YH., Saragosa-Harris, NH., Ninova, E., Silvers, JA. Early Caregiving Adversity Differentially Shapes Behavioral Sensitivity to Reward and Risk during Decision-Making. *Journal of Experimental Psychology: General* (2022).
- **Méndez Leal, AS.,** Silvers, JA. Neuroscientific Approaches to the Study of Self and Social Emotion Regulation During Development. *Oxford Handbook of Emotional Development*. Ed. Dukes, D., Samson, A., Walle, E. (2022).
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General Introduction

Early life adversity is both prevalent and impactful. Worldwide, more than half of youth experience at least one form of childhood adversity, and these exposures are in turn associated with elevated risk for varied physical and mental health challenges (D. W. Brown et al., 2009; Friedman et al., 2015; Kessler et al., 2010; McLaughlin et al., 2012; Murphy et al., 2017; Shonkoff et al., 2012). In particular, early caregiving adversity (ECA) – including exposure to abuse, neglect, caregiver mental illness, or circumstances surrounding placement in institutional or foster care – has profound consequences for socioemotional, cognitive, and behavioral development, and is a potent contributor to later psychopathology (Callaghan & Tottenham, 2016a, 2016b; Kessler et al., 2010; McLaughlin, DeCross, et al., 2019; Shaw & Jong, 2012; Shonkoff et al., 2012; Witt et al., 2016; Zeanah & Humphreys, 2018). Although ECA research has focused on high-level cognitive and socioemotional capabilities critical to preventing psychopathology, growing evidence suggests that ECA may confer risk for lower-level sensory processing challenges, with lasting implications for mental health (Armstrong-Heimsoth et al., 2021; Howard et al., 2020; Joseph et al., 2021; Lin et al., 2005; Schneider et al., 2007, 2008, 2008, 2017, 2017; J. Wilbarger et al., 2010). Together, the studies presented in this dissertation document sensory processing challenges experienced by children, adolescents, and adults with varied histories of ECA and provide evidence that these sensory symptoms may contribute to the development of mental health challenges in multiple populations. In addition, this research probes candidate neurodevelopmental mechanisms that may contribute to the emergence of these sensory challenges, with possible implications for the eventual development of candidate interventions.

Early Caregiving Adversity May Alter Sensory Development

Emerging work suggests that ECA greatly elevates risk for sensory processing challenges, which may in turn contribute to the elevated psychological symptomatology often observed in individuals with histories of ECA. As with investigation of sensory symptoms in other populations, empirical research on the development of sensory processing challenges following ECA has relied primarily on three foundational theories introduced by occupational therapists (Ayres, 1972; Bundy & Lane, 2020; Dunn, 1997; Miller et al., 2007), and especially on the Sensory Profile family of assessments based on one of these models (C. Brown & Dunn, 2002; Dunn & Daniels, 2002; McIntosh et al., 1999). While they differ in their details, all three frameworks attribute a prevalent subset of sensory symptoms to disordered sensory modulation, characterized by intensified or diminished responses to everyday sensory stimuli (Ayres, 1972; Dunn, 1997; Miller et al., 2007). These challenges manifest as three categories of symptoms that frequently co-occur within individuals: *Sensory over-responsivity* (SOR) is marked by intensified or prolonged reactivity to everyday sensory stimuli (e.g., bright lights, loud sounds, being touched; Ben-Sasson et al., 2009; Miller et al., 2007; Reynolds & Lane, 2008; Tomchek & Dunn, 2007). *Sensory under-responsivity* is characterized by unawareness of or delayed response to salient sensory stimulation (e.g. not reacting to novel sounds; Miller et al., 2007; Tomchek & Dunn, 2007). Lastly, *sensation seeking* involves searching for additional sensory input (e.g. seeking deep pressure; mouthing non-food items; Miller et al., 2007; Tomchek & Dunn, 2007). Many of these symptoms are common in otherwise typically developing young children (Román– Oyola & Reynolds, 2013; C. Van Hulle et al., 2015). However, when they persist beyond early childhood, they can limit an individual's ability to adapt to changing developmental contexts, and thereby contribute to family impairment, academic problems, and socialization challenges

(Ben-Sasson, Carter, et al., 2009; Carpenter et al., 2019a; Carter et al., 2011; Dellapiazza et al., 2018, 2020). This dissertation focuses on sensory modulation symptoms, and particularly on SOR, given that most experimental (and especially neuroimaging) work across populations has focused on these symptoms due to their links to symptomatology and adaptive functioning.

Empirical investigations (summarized in [Table I -](#page-22-0) 1) report sensory processing challenges at several developmental stages following varied forms of ECA. Studies have most consistently demonstrated that youth adopted from institutional (e.g. orphanage) care – an increasingly rare form of ECA characterized by reduced caregiving and a unique social and sensory deprivation – display elevated sensory processing challenges (Armstrong-Heimsoth et al., 2021; Cermak & Daunhauer, 1997; Haradon et al., 1994; Lin et al., 2005; Purvis et al., 2013; J. Wilbarger et al., 2010). These emerge in infancy and possibly continue into adolescence (Armstrong-Heimsoth et al., 2021; Cermak & Daunhauer, 1997; Haradon et al., 1994; Lin et al., 2005; Purvis et al., 2013; J. Wilbarger et al., 2010). Similar findings have been reported in adolescents and adults with varied ECA experiences that extend beyond previous institutionalization (PI) to include abuse, neglect, and other exposures (Howard et al., 2020; Jeon & Bae, 2022; Karaca Dinç et al., 2021; Pierce et al., 2021; Serafini et al., 2016). A subset of these studies have also reported differences in specific sensory symptoms experienced by individuals exposed to abuse (particularly violent abuse) as compared to neglect (Howard et al., 2020; Pierce et al., 2021). These correlational links between ECA and sensory symptoms in humans have been supported by causal experimental work in non-human primates (Schneider et al., 2007, 2008, 2017). Importantly, it is unclear whether these findings generalize to other forms of early adversity – while exposure to noncaregiving related violence (missile attacks) increases risk for sensory processing challenges

(Yochman & Pat-Horenczyk, 2020), this pattern is not observed in the context of poverty (Román-Oyola & Reynolds, 2013). Notably, small clinical studies of pilot interventions in youth with varied histories of ECA report that sensory-based treatments may improve not only sensory symptoms, but also psychological symptomatology (e.g. internalizing and externalizing behaviors), motivating further focus on sensory development following ECA (Dowdy et al., 2020; Haradon et al., 1994; Lynch et al., 2021; Purvis et al., 2013; Warner et al., 2014).

Although most research on sensory processing challenges has focused on early and middle childhood, emerging evidence indicates that it may also be important to examine sensory symptoms in other periods of development. A growing body of theoretical and empirical work suggests that specific biological periods (e.g., puberty, pregnancy) may allow for stress recalibration to the environment following experiences of early life adversity, including ECA (Davis & Narayan, 2020; Gunnar et al., 2019; Méndez Leal & Silvers, 2021). These periods may therefore present key opportunities for intervention work. While untested to date, it is possible that a similar recalibration of sensory processing correlates of ECA may occur, particularly given marked changes to sensory processing during these biological periods (Cameron, 2014; Choo & Dando, 2017; Faas et al., 2010; Steward et al., 2018; Weenen et al., 2019). It is also possible that psychosocial stress experienced at later stages of development could elicit or exacerbate sensory processing challenges for individuals previously exposed to ECA. For example, sensory symptoms are a core feature of PTSD for many individuals, and recent theoretical contributions have suggested that trauma-induced changes to sensory processing may precede and contribute to the emergence of higher-order PTSD symptoms (Engel-Yeger et al., 2013; Harricharan et al., 2021). Just as with ECA, preliminary sensory-oriented treatments for PTSD have produced

promising findings (Stoller et al., 2012). Further work is needed to understand how trajectories of sensory processing following ECA may be impacted by both biological transitions and by later experiences of stress.

Neurodevelopmental Mechanisms Underlying ECA-Driven Changes to Sensory Processing

Caregivers scaffold the development of varied functions, ranging from early attention and language acquisition to affective processes including self-regulation (Amso & Scerif, 2015; Callaghan & Tottenham, 2016a; Gee, 2016; Kuhl, 2007; Méndez Leal & Silvers, 2022; Tamis-LeMonda et al., 2014). While the mechanisms are not well characterized, theoretical and empirical evidence suggests caregiver input may similarly shape sensory development, particularly very early in life (Amso & Scerif, 2015). Disruptions to expected caregiving inputs may therefore alter the development of sensory processing, just as they impact neurobehavioral development of scaffolded higher-order processes (language, regulation, etc.; Callaghan & Tottenham, 2016b; Chen & Baram, 2016; Heleniak et al., 2016; McLaughlin, DeCross, et al., 2019; McLaughlin et al., 2020; McLaughlin, Weissman, et al., 2019). Emerging neurodevelopmental theories posit that SOR may be the result of bottom-up differences in encoding of sensory stimuli – through either altered sensory perception or initial affective appraisals of sensory input – or alternatively, may reflect altered development of top-down regulation of sensory responses (S. A. Green & Wood, 2019). Theoretically, ECA-linked sensory processing challenges, and particularly sensory modulation symptoms, may likewise reflect the impact of absent or unstable caregiver scaffolding on initial sensory responses, affective and non-affective regulation of reactions to sensory stimuli, or some combination of these.

As with other developmental processes, input from primary caregivers provides an expected contextual foundation for the salience (the modulation) of environmental cues, constantly scaffolding the interpretation of sensory signals through guided cognitive stimulation (Rosen et al., 2019). In this way, the early environment tunes neurobehavioral development, facilitated by attentional biases towards socially relevant stimuli (Johnson et al., 1991; Simion et al., 2008; Vouloumanos et al., 2010) and stimuli jointly viewed with others (a caregiver, for example; Hoehl et al., 2014; Lloyd-Fox et al., 2015; Parise et al., 2008; Suarez-Rivera et al., 2019). Through these processes, early development fundamentally reflects the needs of an individual in the context of their experienced environment (e.g. is experience-dependent), leading to increased perceptual and appraisal-based specialization to that context (e.g. perceptual narrowing, Scott et al., 2007). Navigating unpredictable or stressful environments without developmentally-expected caregiver guidance may introduce novel demands for initial modulation of responses to sensory cues – sometimes through increased sensitivity (which may become SOR), and sometimes through reduced attention to stimuli (which may eventually manifest as sensory under-responsivity). This view is consistent with empirical evidence: children exposed to chaotic caregiving environments, often characterized by reduced and inconsistent caregiving, learn to "tune out" overstimulating sensory cues (Evans, 2006; Evans & Wachs, 2010; Vernon-Feagans et al., 2013). Similarly, youth who experienced ECA display magnified behavioral and neural vigilance and threat sensitivity, possibly indicating increased attunement to relevant environmental cues required by the absence of a caregiver (Machlin et al., 2019; McLaughlin et al., 2016; Muhammad et al., 2012; Silvers et al., 2016, 2017). Notably, both SOR symptoms and these ECA-linked vigilance phenotypes are associated with altered development of the amygdala, a brain region that supports detection and appraisal of affective stimuli (Gee, 2016; Green & Wood, 2019; Silvers et al., 2017).

Figure I - 2. Neural systems linked to early caregiving adversity that may contribute to the development of SOR symptoms

Table I - 1. Comprehensive Summary of Empirical Evaluation of Links Between Early Adversity and Sensory Processing Challenges

Similarly, the limited or unstable caregiving characteristic of ECA is thought to disrupt the development of affective and non-affective regulation systems (Callaghan & Tottenham, 2016a, 2016b; Gee, 2016; Méndez Leal & Silvers, 2022), which may produce altered modulation of perceptual and affective responses to sensory stimuli, and consequently SOR or sensory under-responsivity. Empirically, ECA has been shown to alter prefrontal regulation of amygdala responses to both non-affective stimuli (in regions including the dorsolateral, ventrolateral, and dorsomedial prefrontal cortex) and affective stimuli (in regions including the ventromedial prefrontal cortex during implicit regulation, and the dorsolateral, ventrolateral, and dorsomedial cortex during explicit regulation). This altered prefrontal regulation has been linked to poor behavioral self-regulation and in principle contributes to high rates of psychopathology in youth with these experiences (Callaghan & Tottenham, 2016b; Y. Chen & Baram, 2016; Cohodes et al., 2020; Gee et al., 2013; Heleniak et al., 2016; Jenness et al., 2020; Silvers et al., 2017; Tottenham et al., 2010; VanTieghem & Tottenham, 2018; Weissman et al., 2019). Furthermore, caregiver-guided shaping of sensory processing circuits may induce altered prefrontal regulatory system development in early life, and in turn caregiver scaffolding of prefrontal regulatory development through cognitive stimulation may contribute to attentional and affective environmental tuning of perceptual and affective appraisals described above (Rosen et al., 2019). In essence, these processes may shape a feedforward loop of development that is reliant on consistent caregiver guidance. This may partially explain emerging evidence suggesting predictable caregiving is important for positive outcomes in youth (Davis et al., 2017; Smith & Pollak, 2021).

Within this framework, altered ECA-induced neurodevelopment may first emerge as sensory processing challenges, before eventually evolving into the broader psychological symptomatology experienced by individuals with histories of ECA. This is consistent with current understanding of hierarchical neurodevelopment: ECA may act directly upon sensory processing (particularly in the first years of life, when sensory cortices are rapidly developing), and these effects may shape downstream development of higher-order cognitive, socioemotional, and regulatory circuits (e.g. Rosen et al., 2019). Over time, circuits associated with both sensory and higher-level processes may continue to engage in patterns that tune towards adaptation to the absence of caregiver input, potentially inducing both sensory processing challenges and psychopathology. While it is theoretically possible that ECA independently causes sensory processing challenges in childhood and later psychological symptomatology beginning in adolescence, this is less plausible given that intervention studies report that treating sensory processing challenges in children of varied ages attenuates psychopathology in youth exposed to ECA (Dowdy et al., 2020; Fraser et al., 2017a; Haradon et al., 1994; Lynch et al., 2021; Purvis et al., 2013; Warner et al., 2014).

Sensory Processing Challenges May Impact Mental Health Outcomes

Regardless of developmental mechanisms, sensory processing challenges appear to have implications for mental health and may therefore be relevant to targeted intervention and treatment following ECA. Preliminary findings suggest sensory processing challenges prospectively predict psychological symptomatology, including the internalizing symptoms and externalizing behaviors commonly reported following ECA (Carpenter et al., 2019; Gunn et al., 2009). Similarly, sensory processing challenges are markedly over-represented in varied

developmental disorders and forms of psychopathology, and individuals with increased sensory processing challenges often experience more severe symptoms from their primary clinical diagnoses (Ben-Sasson, Hen, et al., 2009, 2009; Ben-Sasson et al., 2017; Carpenter et al., 2019a; Conelea et al., 2014; Engel-Yeger, Gonda, et al., 2016; Gunn et al., 2009; Hannant et al., 2016, 2016; Kern et al., 2006; McMahon et al., 2019; Parham et al., 2019, 2019; Serafini et al., 2017). Taken together, these findings suggest sensory processing challenges may exacerbate or contribute to other clinical outcomes, and have led some researchers to propose incorporating a sensation and perception domain to the Research Domain Criteria (Harrison et al., 2019). However, links between sensory processing challenges and psychopathology have not been fully characterized in the context of ECA, despite both theoretical and clinical relevance for sensorybased assessment and treatment.

Current Research

This dissertation examines how ECA may shape long-term sensory development, first by establishing what kinds of experiences contribute to lasting sensory processing challenges, and then by probing hypothesized mechanistic pathways for these connections using both self-report and neuroimaging techniques. In addition, this work aims to characterize possible links between sensory processing challenges and psychological symptomatology.

Study 1 examines whether two broad but profound categories of ECA (experiences surrounding previous caregiving institutionalization or placement in domestic foster care) predict elevated parent-reported sensory processing challenges in children and adolescents, with a focus on SOR given its potential relevance to clinical outcomes As an initial examination of possible

implications of ECA-associated sensory processing challenges on mental health, this study explores links between sensory processing challenges and the internalizing and externalizing symptoms that are common in youth with histories of ECA.

Study 2 applies self-reported questionnaire measures in two population samples of young adults to further evaluate connections between ECA, sensory processing challenges, and mental health. Primary analyses assess whether the sensory processing challenges documented following rare, severe forms of ECA (e.g. PI caregiving) are also observed in individuals with experiences of more prevalent forms of severe early adversity like neglect and abuse. Furthermore, this study will assess whether adversity-linked sensory challenges (and possible associations with internalizing symptoms) endure into young adulthood. As a secondary analysis, Study 2 employs self-report measures to begin to probe mechanism, by examining whether these sensory processing challenges may be attributable to differences in sensory perception, affective sensory reactivity, or regulation of these responses.

Study 3 probes neural correlates sensory processing in adolescents with histories of PI caregiving, with the goal of providing further insights into the mechanistic pathways by which early adversity may contribute to sensory symptoms across development. I apply multivariate pattern expression analyses to assess overlap between individual participant's whole-brain neural responses to aversive auditory stimulation and publicly accessible meta-analytic neural "signatures" of auditory perception, affective sensory reactivity, and affective and non-affective regulation. I also examine aversive auditory-stimulation related changes in functional connectivity. Specifically, I apply psychophysiological interaction analyses using seed regions

intended to probe neural responses related to sensory perception (primary auditory cortex), affective sensory reactivity (the amygdala), and both implicit (automatic) and explicit (intentional) affective and non-affective regulation (ventromedial prefrontal cortex, ventrolateral prefrontal cortex, and dorsolateral prefrontal cortex). Insights from this mechanistic exploration may inform models of the development of sensory processing challenges following ECA, and may also be relevant to creation of further targeted treatments.

Together, these studies aim to establish the relationship between early caregiving adversity, sensory symptoms, and mental health outcomes in varied populations and contexts, and provide an initial exploration of neurodevelopmental pathways contributing to the development of these symptoms after ECA.

Study 1

Sensory processing differences as a novel link between early caregiving experiences and mental health

Development and Psychopathology, 2022

Abstract

Early caregiving adversity (ECA) is associated with elevated psychological symptomatology. While neurobehavioral ECA research has focused on socioemotional and cognitive development, ECA may also increase risk for "low-level" sensory processing challenges. However, no prior work has compared how diverse ECA exposures differentially relate to sensory processing, or, critically, how this might influence psychological outcomes. We examined sensory processing challenges in 183 8-17 year-old youth with and without histories of institutional (orphanage) or foster caregiving, with a particular focus on sensory overresponsivity (SOR), a pattern of intensified responses to sensory stimuli that may negatively impact mental health. We further tested whether sensory processing challenges are linked to elevated internalizing and externalizing symptoms common in ECA-exposed youth. Relative to non-adopted comparison youth, both groups of ECA-exposed youth had elevated sensory processing challenges, including SOR, and also had heightened internalizing and externalizing symptoms. Additionally, we found significant indirect effects of ECA on internalizing and externalizing symptoms through both general sensory processing challenges and SOR, covarying for age and sex assigned at birth. These findings suggest multiple forms of ECA confer risk for sensory processing challenges that may contribute to mental health outcomes, and motivate continuing examination of these symptoms, with possible long-term implications for screening and treatment following ECA.

Introduction

Early caregiving adversity (ECA) is characterized by environmental features that directly disrupt the caregiver–child relationship – for example, exposure to abuse, neglect, parent mental illness, parent substance abuse, or institutional (e.g., orphanage) care (Tottenham, 2020). Exposure to ECA has profound implications for socioemotional, cognitive, and behavioral development, and is a significant risk factor for the development of adolescent mental health disorders (Callaghan & Tottenham, 2016a, 2016b; Kessler et al., 2010; McLaughlin, DeCross, et al., 2019; Shaw & Jong, 2012; Shonkoff et al., 2012; Witt et al., 2016; Zeanah & Humphreys, 2018). Though ECA exposures can be quite heterogeneous, youth with histories of ECA share an increased risk for stress-related symptoms in both the internalizing (anxiety, depression, and somatic) and externalizing (rule-breaking, aggression) domains (Blake et al., 2021; Busso et al., 2017; Heleniak et al., 2016; Humphreys et al., 2015; McLaughlin et al., 2012, 2015, 2020; Witt et al., 2016). Much of the neurobehavioral research on ECA has thus focused on how exposures may impact the development of high-level cognitive and socioemotional capabilities that, if disrupted, increase risk for psychopathology (Callaghan & Tottenham, 2016b; Chen & Baram, 2016; Heleniak et al., 2016; McLaughlin, DeCross, et al., 2019; McLaughlin et al., 2020; McLaughlin, Weissman, et al., 2019). However, emerging evidence – including causal connections in primates (Schneider et al., 2008, 2017) – suggests that ECA also confers increased risk for lower-level sensory processing challenges that may also contribute to mental health outcomes (Armstrong-Heimsoth et al., 2021; Howard et al., 2020; Joseph et al., 2021; Lin et al., 2005; Schneider et al., 2008, 2017; J. Wilbarger et al., 2010).

Sensory processing challenges like those observed in youth with histories of ECA profoundly disrupt daily functioning and are linked to psychological symptomatology in both typically developing and clinical populations. These challenges often manifest in the way individuals modulate (experience and then respond to) sensory input. For example, sensory overresponsivity (SOR) is a prevalent and disruptive sensory processing challenge characterized by heightened or prolonged reactivity to sensory stimuli (e.g., bright lights, loud sounds, being touched; Ben-Sasson, Carter, & Briggs-Gowan, 2009; Miller, Anzalone, Lane, Cermak, & Osten, 2007; Reynolds & Lane, 2008; Tomchek & Dunn, 2007). Other common examples of atypical sensory processing and reactivity include sensory under-responsivity, an unawareness of or delayed response to salient sensory stimuli (e.g.. reduced pain responses, not reacting to novel sounds), and sensation seeking, which typically involves searching for sensory input (e.g. seeking out deep pressure; mouthing non-food items; Miller et al., 2007; Tomchek & Dunn, 2007). In addition to contributing to family impairment and socialization challenges (Ben-Sasson et al., 2009; Carpenter et al., 2019; Carter, Ben-Sasson, & Briggs-Gowan, 2011; Dellapiazza et al., 2020, 2018), these sensory symptoms have implications for mental health. Though the directionality of the relationship between sensory processing challenges and developmental psychopathology warrants further investigation, sensory processing challenges in general, and SOR in particular, prospectively predict later internalizing symptoms (Carpenter et al., 2019), and (to a lesser degree) are linked to externalizing behaviors (Gunn et al., 2009). While sensory processing challenges occur in otherwise typically developing youth, they are over-represented in individuals with neurodevelopmental disorders or psychopathology (Ben-Sasson et al., 2009; Ben-Sasson, Soto, Heberle, Carter, & Briggs-Gowan, 2017; Ben-Sasson & Podoly, 2017; Gunn et al., 2009; McMahon, Anand, Morris-Jones, & Rosenthal, 2019; Parham, Roush, Downing,

Michael, & McFarlane, 2019). Furthermore, within clinical populations, higher levels of sensory processing challenges are associated with greater levels of symptoms from the primary diagnosis, suggesting that sensory processing challenges may exacerbate other clinical outcomes (Ben-Sasson & Podoly, 2017; Conelea, Carter, & Freeman, 2014; Engel-Yeger, Muzio, Rinosi, Solano, & Serafini, 2016; Hannant, Cassidy, Tavassoli, & Mann, 2016; Kern et al., 2006).

Theoretical Connections Between ECA and Sensory Processing Challenges

There is both theoretical and empirical evidence to suggest that ECA can produce sensory processing challenges, which in turn may contribute to the later development of psychopathology.

Caregivers guide numerous features of development, ranging from early attention and language acquisition to affective processes including self-regulation, and may similarly shape sensory development (Amso & Scerif, 2015; Callaghan & Tottenham, 2016a; Gee, 2016; Hoff, 2006; Kuhl, 2007; Méndez Leal & Silvers, 2022; Tamis-LeMonda et al., 2014). Theoretically, the absence of stable caregiving early in life may alter sensory processing development through reduced caregiver scaffolding of initial sensory responses, regulation of attentional or affective reactions to sensory stimuli, or both. This is consistent with emerging neurodevelopmental theories of sensory over-responsivity that argue that SOR symptoms may reflect bottom-up differences in encoding of sensory stimuli – through either altered sensory perception or initial affective responses to sensory input – or alternatively, may be the result of disrupted top-down regulation of sensory responses (Amso & Scerif, 2015; Green & Wood, 2019).

In early life, the environment tunes experience-dependent neural and behavioral development (e.g. perceptual narrowing; Scott et al., 2007). Neural and behavioral evidence suggests that this tuning process is guided by attentional biases towards socially relevant stimuli (Johnson et al., 1991; Simion et al., 2008; Vouloumanos et al., 2010), and towards stimuli that are jointly viewed with others (a caregiver, for example; Hoehl, Michel, Reid, Parise, & Striano, 2014; Lloyd-Fox, Széplaki-Köllőd, Yin, & Csibra, 2015; Parise, Reid, Stets, & Striano, 2008; Suarez-Rivera, Smith, & Yu, 2019). In typical development, primary caregivers scaffold the salience of environmental cues, guiding the interpretation of sensory signals through cognitive stimulation and providing context for what is otherwise a jumble of co-occurring sights and sounds (Rosen et al., 2019). It follows that navigating unpredictable or stressful environments without a stable primary caregiver may require heightened sensitivity, which may eventually manifest as SOR. Empirically, youth with histories of ECA have heightened behavioral and neural vigilance and threat sensitivity, perhaps reflecting increased attunement to salient environmental cues (Machlin et al., 2019; McLaughlin et al., 2016; Muhammad et al., 2012; Silvers et al., 2016, 2017). Notably, both these ECA-linked phenotypes and SOR are thought to be induced by altered development of the amygdala, the brain region most commonly implicated in the detection and appraisal of emotional stimuli (Gee, 2016; Green & Wood, 2019; Silvers et al., 2017).

Another way that the absence of a stable caregiver may evoke SOR is by altering regulation of sensory systems (Amso & Scerif, 2015; Green & Wood, 2019). Given the crucial role that caregivers play in the development of attentional and affective regulation systems, and the well-documented impact of ECA on these processes (Callaghan & Tottenham, 2016a, 2016b; Gee, 2016; Méndez Leal & Silvers, 2022; Rosen et al., 2019), it is possible that the absence of stable caregiving disrupts regulation of affective responses to sensory stimuli to produce sensory processing challenges, including SOR (Amso & Scerif, 2015; Green & Wood, 2019; Rosen et al., 2019). In line with this possibility, ECA alters the development of prefrontal regulation of amygdala responses to affective and non-affective stimuli, producing poor behavioral selfregulation (Callaghan & Tottenham, 2016b; Chen & Baram, 2016; Cohodes et al., 2020; Heleniak et al., 2016; Jenness et al., 2020; Tottenham et al., 2010). The effects of ECA on these prefrontal-amygdala circuits and associated attentional and affective self-regulatory processes are theorized to underlie the high prevalence of psychopathology (particularly internalizing disorders) in youth exposed to ECA (Amso & Scerif, 2015; Callaghan & Tottenham, 2016b; Gee et al., 2013; D. Johnson et al., 2021; Rosen et al., 2019; Silvers et al., 2017; VanTieghem & Tottenham, 2018; Weissman et al., 2019). Additionally, changes to sensory processing circuits induced by altered cognitive stimulation in the context of ECA may themselves produce changes to the development of prefrontal affective and attentional regulatory systems, and vice versa (see Rosen et al., 2019 for a relevant review).

Given this evidence and that development is hierarchical, it may be that changes to neural circuitry induced by a lack of stable caregiving first manifest as sensory processing challenges in childhood, before evolving into the broader psychological symptom profiles observed in youth with these experiences. Theoretically, ECA may act directly upon sensory processing first, given that the sensory cortices are developing rapidly in the first few years of life, and this in turn could have ripple effects on other aspects of development down the road (e.g. Rosen et al., 2019). In line with this, empirical evidence in other populations suggests that sensory processing
challenges emerge prior to and prospectively predict internalizing and externalizing symptoms (Carpenter et al., 2019; Green et al., 2012; McMahon et al., 2019). For example, cross-lag analyses in youth with autism suggest that SOR emerges early and predicts later increases in anxiety, while anxiety does not predict later SOR (Green et al., 2012). While it is possible that ECA independently causes sensory processing challenges, and later in development, internalizing and externalizing problems, this seems unlikely given that several small case studies suggest treating sensory processing challenges attenuates the development of other psychopathology in individuals with histories of ECA (Dowdy et al., 2020; Fraser et al., 2017; Haradon et al., 1994; Lynch et al., 2021; Purvis et al., 2013; Warner et al., 2014).

Support for the theoretical model that ECA causes sensory processing challenges that in turn confer elevated risk for psychopathology ought to meet two criteria: first, sensory processing challenges ought to be prevalent in groups exposed to varied forms of ECA, and second, sensory symptoms ought to predict psychopathology in ECA-exposed youth. Several studies have reported that institutional (e.g. orphanage) caregiving elevates risk for sensory processing challenges (Armstrong-Heimsoth et al., 2021; Cermak & Daunhauer, 1997; Lin et al., 2005; J. Wilbarger et al., 2010). However, institutional care is an increasingly rare form of ECA characterized both by reduced caregiving and a unique social and sensory deprivation driven by a reduction in novelty. Establishing that ECA in general contributes to the development of sensory processing challenges therefore requires comparison with other forms of ECA beyond institutionalization. Wilbarger et al. (2010) found that internationally adopted youth with histories of prolonged previous institutional caregiving experienced elevated sensory processing challenges relative to non-adopted youth and *internationally* adopted youth with short-term

experiences of foster care, implying that institutional caregiving may confer a unique risk for sensory processing challenges. However, it is unclear from Wilbarger et al. whether the group differences in sensory processing challenges are related to *type* of ECA or simply to *severity.* Therefore, comparing sensory processing challenges in youth internationally adopted from institutional care to other groups with comparably severe ECA experiences – for example, youth in the United States adopted from *domestic* foster care (who have varied and often, more prolonged ECA experiences) may further clarify this finding. Although experiences surrounding placement into institutional and foster care have commonalities (e.g. separation from primary caregivers, lack of stable caregiving, and uncertainty about the future), these distinct types of caregiving adversity also typically differ on several important dimensions, including family circumstances leading to placement, the large-scale political or economic systems that determine the types of caregiving available, and qualitative features of the caregiving itself (Berens $\&$ Nelson, 2015; van IJzendoorn et al., 2020). Given that varied ECA exposures have been implicated in alterations of prefrontal-amygdala circuitry thought to underlie SOR (Callaghan & Tottenham, 2016b; Green et al., 2018, 2019; Green & Wood, 2019; Silvers et al., 2016, 2017), we would expect that diverse forms of ECA likely increase the risk of SOR. The present study allows us to test this possibility. Lastly, explicitly probing SOR and examining ties between sensory processing and mental health in middle childhood and adolescence (when most psychopathology begins to emerge; Solmi et al., 2021) may clarify the importance of sensory processing in long-term outcomes in youth with histories of ECA.

Current Study

The current cross-sectional study examined whether two broad categories of ECA (experiences surrounding previous institutionalization or placement in domestic foster care) are

associated with elevated sensory processing challenges in children and adolescents. Specifically, we explored links between ECA and sensory processing challenges in general and SOR in particular, given the latter's relationship with clinical outcomes in other populations (Carpenter et al., 2019; Green et al., 2012). We also examined whether sensory processing challenges are related to internalizing and externalizing symptoms, which are common in youth with ECA exposures. Given that varied forms of ECA exert similar deleterious effects on development in other domains, we hypothesized that both youth adopted from foster care (AFC) and previously institutionalized (PI) youth would have greater sensory processing challenges (including SOR) relative to non-adopted comparison youth, and did not have specific between-group hypotheses regarding sensory processing challenges. Additionally, we hypothesized that we would find significant indirect effects for the positive relationship between ECA and internalizing and externalizing symptoms through both general sensory processing challenges and SOR specifically. Lastly, we predicted that sensory processing challenges would be higher in participants who were placed into adoptive homes later in life (due to prolonged ECA exposure), consistent with a dose-response relationship between ECA and both sensory and psychopathology symptoms in some samples (Julian, 2013; Lin et al., 2005; Pitula et al., 2014; J. Wilbarger et al., 2010). Our a priori hypotheses and data analytic plan were pre-registered on the Open Science Framework [\(osf.io/r9e8q\)](http://www.osf.io/r9e8q).

Methods

Participants

Data were drawn from two projects examining the neurobehavioral sequelae of ECA in AFC, PI, and non-adopted comparison children and adolescents. Informed consent and assent were obtained from legal guardians and study participants, and study procedures were approved

by the Institutional Review Board. During study visits, parents/guardians were asked to complete assessments of sensory processing challenges and psychological symptomatology for their child.

As outlined in our pre-registration, child and adolescent participants were excluded from the study if they had a diagnosis of bipolar disorder, schizophrenia, autism spectrum disorder, or any known genetic conditions. While most parents completed all measures during one session, after pre-registration we discovered that psychological symptomatology measures were collected during a separate clinical intake for 7 AFC youth. Although most of these participants completed both assessments within a two-year period, one child with a larger gap between sensory and symptomatology assessments was excluded. Lastly, 6 youth in the pre-registered PI sample were later discovered to have been adopted internationally from foster (and not institutional) care and were thus excluded from the final analyses.

34 PI, 37 AFC, and 112 comparison youth aged 8-17 years had usable data and were included in analyses. Additional details about recruitment and exclusion are reported in the supplement.

Demographic Information

Chi-square analyses were performed to explore group differences in sex assigned at birth, race, and ethnicity. ANOVAs were used to assess group differences in child age, age at placement into adoptive home, and child IQ (measured using the *Wechsler Abbreviated Intelligence Scale, Second Edition;* WASI-II). Group differences in demographic information are presented in Table 1.

Variable	Comparison $(N = 112)$	PI $(N = 34)$	AFC $(N = 37)$	
	Mean (Median; SD)	Mean (Median; SD)	Mean (Median; SD)	\pmb{p}
Age	13.37 years (13.17; 2.48)	14.94 years $(15.17; 1.78)^a$	11.96 years $(10.74; 2.81)^{ac}$	< .001
Age at Placement into Adoptive Home		19.46 mths (12.75; 16.03)	37.59 mths (30.0; 33.29)	< .001
IQ	115.64 (118.0; 14.15)	104.65 (105.0; 13.31) ^a	97.61 (99.0;11.35)bc	< .001
	Count $(\%)$	Count (%)	Count (%)	\pmb{p}
Assigned Sex at Birth	Female: 50 (45%) Male: 62 (55%)	Female: 24 (71%) Male: 10 (29%)	Female: 19 (51%) Male: 18 (49%)	.03
Race				< .001
Black	9(8%)	$0(0\%)$	11 (3%)	
Asian	15 (13%)	16(47%)	$0(0\%)$	
white	64 (57%)	13 (38%) ^a	$18(49%)^b$	
Native Hawaiian or Pacific Islander	2(2%)	$0(0\%)$	$0(0\%)$	
Multiracial	19 (17%)	$1(3%)^a$	$3(8%)^b$	
Other	3(3%)	4(12%)	$0(0\%)$	
Ethnicity				< .001
Latinx/e	26 (23%)	$0 (0\%)^a$	13 (41%)	

Table 1 - 1. Sample demographic information

Note: AFC = adopted from foster care; PI = previously institutionalized. IQ was not collected in 14 AFC participants, and race/ethnicity is unknown for 5 AFC youth. Chi-square analyses were performed to explore group differences in sex assigned at birth, race, and ethnicity. ANOVA was used to explore group differences in IQ, child

age, and age at placement into adoptive home. IQ was measured using the *Wechsler Abbreviated Intelligence Scale, Second Edition* (WASI-II; Wechsler, 2011). *p* values reflect the results of each chi-square or ANOVA.

^a Denotes higher rates/scores in the Comparison group than the PI group.

b Denotes higher rates/scores in the Comparison group than the AFC group.

^cDenotes higher rates/scores in the PI group than the AFC group.

Abbreviations: Previously Institutionalized (PI); Adopted from Foster Care (AFC)

Measures

To characterize sensory experiences following ECA, we used a general measure of sensory processing challenges focused on sensory modulation (Short Sensory Profile) and a targeted assessment of SOR symptoms (SP3D Inventory), given reported links between SOR and clinical outcomes (McIntosh et al., 1999; Schoen et al., 2008). Additional measure details, discussion of the advantages of using both scales, and correlations between similar subscales across measures are reported in the supplement.

General sensory processing challenges. The *Short Sensory Profile* (SSP; McIntosh et al., 1999) assesses a child's struggles with sensory processing. For example, parents indicate to what extent their child reacts emotionally to or avoids intense sensory stimuli (e.g., touch, sound, light, tastes), seeks out touch/movement to a disruptive degree, or is affected by sensory distractors. SSP total scores are derived from parent ratings of their child's sensory processing on all 38 items, each scored from 1 (*Always*) to 5 (*Never*). The SSP items are divided into seven subscales: Tactile Sensitivity, Taste/Smell Sensitivity, Movement Sensitivity, Visual/Auditory Sensitivity, Underresponsive/Seeks Sensation, Auditory Filtering, and Low Energy/Weak. Previous research suggests that the SSP subscales have reliability estimates in the moderate to excellent range (McIntosh et al., 1999). Lower SSP scores reflect less typical processing, with clinical categories characterized as typical sensory processing (190 to 155), or probable (154 to 142) or definite (141 to 31) sensory processing challenges.

Sensory over-responsivity. The Sensory Processing 3-Dimensions Scale Sensory Inventory (SP3D) assesses a child's responses to common, potentially aversive sensory stimuli (Schoen et al., 2008). Parents reported how bothered their child is by individual stimuli on a Likert scale ranging from 1 (*Not bothered/never avoids*) to 5 (*Extremely bothered/always avoids*)

on 42 questions. For example, parents report to what extent the sound of fluorescent lights, clothes swishing, toilets flushing, and sirens bother their child. Tactile, visual, and auditory subscales were used and combined to create a total SOR score. Previous findings have shown that the SP3D total score has high internal consistency (α = .89; Schoen et al., 2017[\).](https://www.zotero.org/google-docs/?broken=IoZRb5) SP3D scores range from 42 to 210, with higher scores corresponding to higher levels of SOR (greater impairment).

Clinical symptomatology. Internalizing symptoms and externalizing problems were measured using the Child Behavior Checklist, a parent-reported measure of mental health and behavioral symptoms for youth between the ages of 6-18 years (CBCL; Achenbach & Rescorla, 2001). On the CBCL, parents report their child's clinical symptoms on 118 questions (rated $0 =$ *Not True*, 1 = *Somewhat or Sometimes True*, or 2 = *Very True or Often True*). The internalizing subscale combines anxious/depressed, withdrawn/depressed, and somatic complaint scores. The externalizing problems subscale sums rule-breaking and aggressive behavior items. These subscales have strong evidence for reliability and both discriminant and convergent validity: there is excellent test-retest reliability for the internalizing symptoms (*r =* .91) and externalizing s[y](https://www.zotero.org/google-docs/?broken=3ULAR9)mptoms $(r = .92)$, as well as good criterion-related validity and construct validity (Achenbach & Rescorla, 2001). Due to IRB constraints, the CBCL suicidality questions were not collected, and thus were omitted from score calculations. As a result, CBCL Internalizing subscale scores were calculated without question 91, while all other subscale scores of interest were calculated as usual. To prevent truncation (Achenbach & Rescorla, 2001), all analyses used raw subscale scores rather than t-scores.

Data Analytic Plan

Statistical analyses were conducted using SPSS Version 27.0 (SPSS Inc., USA). Path analyses were conducted using the PROCESS macro (Hayes, 2017), using 95% percentile bootstrap confidence intervals (5,000 bootstraps). In line with recommendations (Lemmer & Gollwitzer, 2017; Thoemmes, 2015), we only ran statistical tests for the pre-registered crosssectional path analyses that aligned with our theoretical model (which posits that ECA causes sensory processing challenges that in turn confer elevated risk for psychopathology), and did not test alternative path models by flipping the M (sensory) and Y (psychological symptomatology) variables.

We conducted two ANCOVAs to probe differences in sensory processing between the PI and AFC groups, and to determine whether they should be examined separately or as one ECA group. We set group (AFC or PI) as the independent variable and SSP total score (general sensory processing challenges) and SP3D total score (SOR) as the respective dependent variables, with age and sex assigned at birth as covariates.

Given demonstrated relationships between ECA and both SOR and internalizing symptoms, we used two primary path analysis models to examine the impact of ECA, a multicategorical predictor (two ECA groups relative to the comparison group), on internalizing symptoms (CBCL) through sensory processing challenges, while covarying for age and sex assigned at birth. The two models respectively tested the indirect effects of our two sensory measures: SOR (SP3D score) and general sensory processing challenges (SSP score). In both models, we first examined group differences in SOR and sensory processing challenges using the path between ECA and the sensory measure of interest. We then probed indirect effects of ECA on internalizing symptoms through the two sensory measures, respectively.

Since links between sensory processing challenges and externalizing symptoms are less well-documented, we conducted two exploratory path analyses examining indirect effects of ECA on externalizing symptoms through the sensory measures, covarying for sex and age.

Our pre-registered analyses aimed to examine relative total effects (the sum of direct and indirect effects) of ECA group on psychological symptoms using these path analyses. However, because some participants had asynchronous sensory and psychological assessments, we covaried for different ages on different paths of our models. This required four multiple regressions to evaluate the total effects of ECA group (AFC or PI relative to non-adopted comparison) on internalizing and externalizing symptoms, respectively (covarying for age and sex). We also conducted a multiple regression within the combined ECA group (PI and AFC) to examine the effect of age at placement into a final adoptive home (predictors) on SOR, while covarying for sex.

To provide additional confidence in the reported findings, multiple post-hoc analyses focused on age and sex are reported in the supplement, including reanalysis of a smaller sample with age-matched groups. These results do not differ in any meaningful way from the original analyses, aside from observed differences in SOR between smaller age-matched AFC and comparison samples, which were marginally significant, presumably due to reduced statistical power.

Given the exploratory nature of our questions and that the populations in this study are very challenging to recruit (limiting statistical power), we did not correct for multiple comparisons. For this reason, we distinguished between our primary and exploratory questions of interest in both our pre-registration and below, to strike a balance between limiting multiple comparisons within the primary questions of interest while also providing as much useful

descriptive data as possible on the sensory measures collected. In addition, given our use of bootstrapping, we did not exclude outliers in our pre-registered analyses in order to preserve statistical power in a small, hard to recruit sample from a population with high inter-individual variability (Tottenham, 2012). All findings reported below therefore include all eligible participants. Post-hoc analyses excluding participants with SP3D or SSP scores more than three standard deviations from the overall sample mean (excluding 4 AFC and 2 PI participants for the SP3D and 3 AFC participants for the SSP) found nearly identical patterns of effects as those reported below. These analyses are reported in the supplement.

Results

Descriptive Results

Sample demographic information is reported in Table 1, and descriptive statistics for all measures are presented in Table 2. While all subjects completed all primary measures, IQ was not collected in 14 AFC participants, and 5 AFC youth did not provide race/ethnicity information. Both the SP3D and the SSP measures had high internal consistency reliability in this sample ($\alpha_{SP3D} = .91$, $\alpha_{SSP} = .94$). Parent-reported partial information on ECA experienced by the PI and AFC groups is reported in the supplement.

Scales	Comparison $(N = 112)$	PI $(N = 34)$	AFC $(N = 37)$
	Mean (Median; SD)	Mean (Median; SD)	Mean (Median; SD)
General Sensory Processing Challenges SSP Total Measure Range: 190-38	178.99 (183.00;11.79) Range: 190-132	169.76 (174.50; 14.10) ^a Range: 189-131	147.54 (150.00; 23.71) ^{bc} Range: 190-103
SOR SP3D Total Measure Range: 42-210	48.22 (46.00; 7.97) Range: 42-86	58.34 (52.50; 15.3) ^a Range: 42-98	58.24 (51.00; 19.26) ^b Range: 42-112
Internalizing Symptoms CBCL Internalizing Measure Range: 0-62	4.56(3.00; 4.9) Range: 0-25	11.62 $(9.5; 8.42)^a$ Range: 0-32	$12.49(11.0; 9.67)^{b}$ Range: 0-41
Externalizing Symptoms CBCL Externalizing Measure Range: 0-70	2.98(1.00; 3.7) Range: 0 -15	$7.00 (6.00; 5.82)^{a}$ Range: 0-20	15.96 (12.00; 12.44) ^{bc} Range: 0-50

Table 1 - 2. Descriptive statistics for sensory over-responsivity, general sensory processing challenges, and clinical symptomatology

Note: Reported CBCL scores are raw subscale scores. T-scores and clinical cutoffs for the CBCL are reported in the supplement.

^a Denotes elevated symptoms in the PI group relative to the Comparison group.

b Denotes elevated symptoms in the AFC group relative to the Comparison group.

^cDenotes elevated symptoms in the AFC group relative to the PI group.

Abbreviations: Previously Institutionalized (PI); Adopted from Foster Care (AFC); Sensory Processing 3-

Dimensions Scale Sensory Inventory (SP3D); Short Sensory Profile (SSP); Child Behavior Checklist (CBCL)

Differences in Sensory Processing Challenges Between ECA Groups

We found no differences between ECA groups on SP3D scores $(F(3,71) = 0.76, p = .39)$.

However, the AFC group had significantly more sensory processing challenges on the SSP than

the PI group $(F(3,71) = 10.00, p = .002)$. The AFC and PI groups were therefore examined

separately in all analyses, with ECA dummy coded and non-adopted comparison youth as the

reference group.

Sensory Processing Challenges Following ECA

As expected, youth in both ECA groups had significantly elevated sensory processing challenges (Figure 1; Table 2). Youth in the PI $(a_{PI, SP3D} = 10.72, SE = 2.57, t = 4.18, 95\% \text{ CI}$ [5.65, 15.78], $p < .001$) and AFC ($a_{AFC\ SP3D} = 9.82$, $SE = 2.45$, $t = 4.02$, 95% CI [5.14, 0.65], *p* <.001) groups had higher SP3D scores (higher SOR) than the non-adopted comparison group, covarying for age and sex. Consistent with this finding, youth in both the PI ($a_{PI,SSP} = -11.09$, $SE = 3.10$, $t = -3.56$, 95% CI [-17.22, -4.97], $p < .001$) and AFC ($a_{AFC, SSP} = -31.21$, $SE = 2.97$, *t* = -10.56 , 95% CI [-37.05, -25.38], *p* < .001) groups had significantly heightened general sensory processing challenges on the SSP (lower scores), relative to non-adopted comparison youth. This suggests that youth with histories of ECA experience elevated general sensory processing challenges and increased SOR, relative to comparison youth.

Sensory Processing Challenges by ECA Group

*Figure 1 - 1. Left: PI and AFC participants show elevated levels of sensory over-responsivity (higher SP3D scores), relative to non-adopted, comparison youth. Right: PI and AFC participants show increased levels of general sensory processing challenges (lower SSP scores) relative to non-adopted, comparison youth. **p <.001, *p <.05.*

A post-hoc chi-square analysis showed a moderate association ($\varphi = .57$, $p < .001$) between group membership (PI, AFC, and comparison) and the distribution of participants in SSP clinical categories ($\chi^2(4) = 60.19$, *p* <.001). Of the non-adopted comparison youth, 5.36% were classified as having probable and 1.7% as having definite sensory processing challenges, consistent with previous findings in younger children (Tomchek & Dunn, 2007). PI youth displayed more evidence of sensory processing challenges, with approximately 15% classified as having probable and 3% as having definite sensory processing challenges. Notably, 19% of AFC youth were considered to have probable, and an additional 40% to have definite sensory processing challenges. Group differences on the SSP and SP3D subscales are reported in the supplement for reference.

Figure 1 - 2. a) 95% percentile bootstrapped regression coefficients for a path analysis model examining the association between ECA (predictor) and internalizing problems (outcome) through SP3D total score, while controlling for age and sex assigned at birth. b) 95% percentile bootstrapped regression coefficients for a path analysis model examining the association between ECA (predictor) and externalizing problems (outcome) through SP3D total score, while controlling for age and sex assigned at birth. As in OLS regression, R2 for each component of the path analysis can be interpreted as the proportion of the variance in the outcome explained by that model (e.g. proportion of SP3D variance explained by OLS with ECA group, sex, and age predictors) **p<.001, *p<.05

Covarying for sex assigned at birth and age

Abbreviations: Previously Institutionalized (PI); Adopted from Foster Care (AFC); Sensory Processing 3- Dimensions Scale Sensory Inventory (SP3D); Short Sensory Profile (SSP); Child Behavior Checklist (CBCL) *Figure 1 - 3. a)* 95% percentile bootstrapped regression coefficients for a path analysis model examining the association between ECA (predictor) and internalizing problems (outcome) through SSP total score, while controlling for age and sex assigned at birth. b) 95% percentile bootstrapped regression coefficients for a path analysis model examining the association between ECA (predictor) and externalizing problems (outcome) through SSP total score, while controlling for age and sex assigned at birth. As in OLS regression, \mathbb{R}^2 for each component of the path analysis can be interpreted as the proportion of the variance in the outcome explained by that model (e.g. proportion of SSP variance explained by OLS with ECA group, sex, and age predictors) **p<.001, *p<.05

Covarying for sex assigned at birth and age

Abbreviations: Previously Institutionalized (PI); Adopted from Foster Care (AFC); Sensory Processing 3- Dimensions Scale Sensory Inventory (SP3D); Short Sensory Profile (SSP); Child Behavior Checklist (CBCL)

Psychological Symptomatology following ECA

There were significant total effects of ECA on both internalizing and externalizing symptoms. Both PI ($C_{PI\;INT}$ = 6.26, *SE* = 1.21, t = 5.17, 95% CI [3.87, 8.67], p < .001) and AFC ($C_{AFC INT}$ = 8.32, $SE = 1.27$, $t = 6.54$, 95% CI [5.81, 10.83], $p < .001$) youth had higher internalizing symptom scores than comparison youth, covarying for age and sex. Similarly, both PI ($C_{PI\,EXT} = 4.16$, $SE = 0.89$, $t = 4.70$, 95% CI [2.41, 6.91], $p < .001$) and AFC ($C_{AFC\,EXT} =$ 12.51, *SE =* 1.36, *t =* 9.17, 95% CI [9.81, 15.21], *p* < .001) youth had higher externalizing symptoms than comparison youth, covarying for age and sex. These results are consistent with those reported in other PI and AFC samples (e.g. Humphreys et al., 2015).

Sensory Processing Challenges and Links to Psychological Symptomatology

Findings from the path analyses were consistent with our theoretical framework, which posits that ECA inflates risk for psychological symptomatology in part through increased sensory processing challenges. First, we explored how SOR might contribute to links between ECA and internalizing symptoms. Covarying for age and sex assigned at birth, we found significant indirect effects of ECA on elevated internalizing symptoms through SOR, for both PI $(ab_{PI_SP3D_INT} = 1.37, 95\% \text{ CI} [0.36, 2.63])$ and AFC $(ab_{AFC_SP3D_INT} = 1.26, 95\% \text{ CI} [0.29,$ 2.44]) youth (Figure 1 - 2A). In a second model that examined general sensory processing challenges as a link between ECA and internalizing symptoms, we again found significant indirect effects through sensory processing challenges for both PI ($ab_{PI, SSP, INT}$ = 1.65, 95% CI [0.67, 3.04]) and AFC participants $(ab_{AFC\ SSP\ INT} = 4.64, 95\% \ CI$ [2.66, 6.95]), relative to comparison youth (Figure 1 - 3A).

We also conducted two exploratory path analyses to examine how sensory processing challenges might explain the relationship between ECA and externalizing symptoms. The first examined SOR as a link between ECA and externalizing symptoms (Figure 1 - 2B). We found significant indirect effects of PI and AFC status on externalizing symptoms through SOR (PI: $ab_{PI_SP3D_EXT}$ = 1.28, 95% CI [0.10, 2.75]; AFC: $ab_{AFC_SP3D_EXT}$ = 1.17, 95% CI [0.06, 2.6]). Similarly, we found a significant indirect effect of ECA on externalizing symptoms through sensory processing challenges (Figure 1 - 3B; PI: $ab_{PI, SSP, EXT}$ = 1.98, 95% CI [0.73, 3.76]; AFC: $ab_{AFC_SSP_EXT}$ = 5.57, 95% CI [2.78, 9.08]).

These findings support our hypothesis that sensory processing challenges and SOR symptoms may contribute to ECA-associated internalizing and externalizing symptoms.

SOR and Age at Placement into Final Adoptive Home

Our results were not consistent with a dose-response relationship between pre-adoption ECA duration and SOR ($B_{Placement} = -0.11$, $t(70) = -1.47$, 95% CI [-0.26 , 0.04], $p = .15$). Post-hoc exploratory analyses showed age at placement was not associated with SOR within the PI $(B$ Placement_PI = -0.13 , $t(33) = -0.77$, 95% CI [-0.48 , 0.22] $p = .45$) or AFC groups (*B*Placement_AFC = -0.13, $t(36) = -1.27, 95\% \text{ CI}$ [$-0.33, 0.08$], $p = .21$). Additional analyses found no associations between age and SOR symptoms across both ECA groups, as reported in the supplement.

Discussion

This study examined the impact of ECA on sensory processing challenges in youth adopted from institutional (e.g., orphanage) or foster care. We found that relative to non-adopted comparison youth, children and adolescents adopted from institutional or foster care display

elevated sensory processing challenges, including SOR. This suggests that ECA-linked sensory processing challenges persist into adolescence, in contrast with age-related reductions in sensory symptoms reported in typically developing and clinical samples of youth without known ECA (Kern et al., 2006; Little et al., 2018; Van Hulle et al., 2015). Our results also suggest that sensory processing challenges, including SOR, may contribute in part to elevated internalizing and externalizing symptoms observed in youth with histories of ECA. Taken together, our findings point to a commonality of sensory processing challenges among youth exposed to severe forms of ECA, with possible implications for mental health. Further work should examine whether similar effects are observed following more common, less severe forms of ECA.

That we observed sensory processing challenges in both PI and AFC youth both replicates and contradicts findings from a previous study, which reported sensory processing challenges (assessed using the SSP) in PI, but not AFC youth (J. Wilbarger et al., 2010). These discrepant findings in AFC youth could be explained in part by differences in time prior to placement in a final adoptive home between the current and prior studies, given that youth in the prior AFC sample were very young at adoption ($M_{\text{Age}} = 4.5$ months, range = 1-8 months) relative to our AFC sample ($M_{\text{Age}} = 37.59$ months, range = 0-108 months). However, as our current results do not suggest a dose-response relationship between duration of pre-adoption ECA and sensory processing difficulties, these differences merit further exploration of how ECA severity impacts outcomes in future work employing more targeted metrics.

Developmental heterogeneity after ECA exposure

Though the effects of ECA have primarily been documented in cognitive and affective domains (Callaghan & Tottenham, 2016a, 2016b; Chen & Baram, 2016; McLaughlin, DeCross, et al., 2019; Pechtel & Pizzagalli, 2011), our results indicate that ECA also alters "lower-level" sensory processing. Although our participant samples are not necessarily representative of all youth with similar paths to adoption, these findings suggest that across two distinct forms of ECA, each with considerable experiential heterogeneity, there is a shared elevated risk for sensory processing challenges. Though circumstances surrounding placement in institutional and foster caregiving differ on several features, they often share core adversities, including separation from primary caregivers, frequent transitions, and a lack of stable caregiving. Notably, while we observed a shared risk for sensory processing challenges in both the PI and AFC groups, there was substantial variability in sensory processing within each of these cohorts. Relative to comparison youth, the range of SOR scores was 27% wider for the PI group and 59% wider for the AFC group. This variability is consistent with a broader literature suggesting that while ECA exposure probabilistically increases the risk for psychopathology, this link is not deterministic (Kessler et al., 2010; McLaughlin et al., 2012; Tottenham, 2012).

These observations speak to the diversity of exposures that youth with histories of ECA encounter. For example, for internationally adopted PI youth, institutional placements are often the result of political, societal or economic pressures (e.g., poverty, national policies, natural disasters), and not necessarily abuse or neglect (Gunnar et al., 2007; van IJzendoorn et al., 2020). As such, the initial family separation and qualitative features of the institutional rearing environment itself (including high child to caregiver ratios, rotating staff, and resultant lower

quality caregiving) are often principal sources of ECA for these youth (Berens & Nelson, 2015; van IJzendoorn et al., 2020). By contrast, domestically adopted AFC youth have heterogeneous and varied experiences that, in addition to removal(s) from their home of origin themselves, may at times include exposure to violence, neglect (AFCARS, 2020), in addition to other systemic or family-level factors contributing to interaction with the welfare system and placement in foster care (e.g. systemic racism, poverty). The heterogeneity of exposure AFC youth experience is consistent with the present AFC sample showing more variable sensory processing challenges than PI youth. Future work should examine whether specific features of ECA (e.g., trauma, unpredictability, degree of deprivation exposure, perceptions of experiences of ECA) contribute to variability in sensory development and specific sensory symptom profiles (Cohodes et al., 2020; McLaughlin & Sheridan, 2016; Smith & Pollak, 2021). Descriptive analyses in our sample (described in the supplement) are consistent with clearer links between ECA and SOR than other sensory processing challenges, but these tentative findings merit additional exploration in future work.

Potential mechanisms for development of sensory processing challenges after ECA exposure

Mechanistic pathways for the development of sensory processing challenges following ECA are not well characterized. However, key neural circuits thought to be impacted by ECA have also been implicated in the development of SOR. For example, preliminary neuroimaging evidence suggests that sensory symptoms may be driven by enhanced affective reactivity, altered top-down regulation of limbic circuitry, or both (Green et al., 2013, 2018), mirroring altered prefrontal-amygdala circuit activity observed following ECA. The present results imply that ECA-associated threat vigilance (linked to amygdala hyper-reactivity in ECA-exposed youth;

Silvers et al., 2017) may extend to the sensory domain and contribute to symptoms of both SOR and anxiety (Green & Ben-Sasson, 2010). Likewise, diminished regulation of affective responses to sensory stimuli may contribute to sensory processing challenges. Lower emotion regulation capacity is linked to SOR symptoms (McMahon et al., 2019), and SOR is associated with both reduced amygdala habituation and prefrontal down-regulation of the amygdala during aversive sensory stimulation (Green et al., 2015, 2018, 2019; Green & Wood, 2019). These findings mirror observations of altered prefrontal regulation of limbic circuitry in youth with histories of ECA during both affective and non-affective self-regulation (Callaghan & Tottenham, 2016b; Chen & Baram, 2016; Cohodes et al., 2020; Heleniak et al., 2016; Jenness et al., 2020; Tottenham et al., 2010). While altered neurobehavioral vigilance and self-regulation profiles are likely adaptations to unpredictable or threatening environments, both phenotypes convey increased risk for internalizing symptoms among youth with histories of ECA (Callaghan & Tottenham, 2016b; Gee et al., 2013; Silvers et al., 2017; VanTieghem & Tottenham, 2018; Weissman et al., 2019). Testing mechanistic pathways could further clarify the connections between sensory processing challenges and internalizing (and externalizing) symptoms observed in the present study.

Clinical Implications

Regardless of developmental mechanisms, our results are consistent with findings in other clinical populations that indicate that sensory processing challenges increase risk for a broad range of psychological and behavioral symptoms (Carpenter et al., 2019; Gourley et al., 2013; Green et al., 2012; McMahon et al., 2019). This fact has led some researchers to advocate for the addition of a sensation and perception domain to future versions of the Research Domain

Criteria (Harrison et al., 2019). These findings motivate further longitudinal exploration of sensory development in the context of ECA exposure to characterize developmental trajectories.

If replicated, the present findings motivate further work evaluating the impact of screening for sensory processing difficulties in clinical assessment and treatment in youth with histories of ECA. If additional longitudinal work establishes a directional relationship between sensory processing challenges and later psychopathology following ECA, it will be important to investigate whether monitoring or treating such challenges can support improved clinical outcomes. The present findings together with future work stand to have two implications. First, screening for sensory processing challenges could prove to be useful for early intervention in youth with histories of ECA. In some individuals, ECA-induced changes to psychosocial functioning (and underlying neural circuitry) may first manifest as sensory processing challenges -- which emerge in early childhood -- before evolving into broader psychological symptom profiles during adolescence, when psychopathology most commonly emerges (Ben-Sasson et al., 2009; Carpenter et al., 2019; Carter et al., 2011; Green et al., 2012; McLaughlin et al., 2012; Román‐Oyola & Reynolds, 2013; Solmi et al., 2021). In line with this reasoning, our findings suggest sensory processing challenges in ECA-exposed youth remain elevated in adolescence, and do not disappear following early childhood. Second, sensory processing-focused assessments and targeted treatments may improve clinical care for youth with histories of ECA. Sensory processing symptoms in populations exposed to ECA may lead to misinterpretation of behavioral and mental health symptoms by parents and clinicians alike (Conelea et al., 2014; Fernández-Andrés et al., 2015; Harrison et al., 2019; Howe & Stagg, 2016). For instance, sensory processing challenges often manifest as tantrums, aggression, and both avoidance of and

difficulty disengaging with stimulation. In addition to being psychologically taxing for youth, such responses cause distress, family impairment, and socialization challenges (Ben-Sasson et al., 2009; Carpenter et al., 2019; Carter et al., 2011; Dellapiazza et al., 2020, 2018)*.* As a result, sensory-informed assessments may lead to more accurate, targeted, and effective treatments of both sensory symptoms and psychological symptomatology.

Limitations

These findings suggest ECA is associated with altered sensory processing, and that sensory processing challenges may contribute to internalizing and externalizing symptoms. However, the present study has several limitations that should be addressed by future work. First, we have limited information about pre-adoption experiences for PI and AFC participants, including exposure to other adversities common in these populations (e.g., abuse, prenatal substance exposure). Though this precludes conclusions about the effects of specific exposures on sensory processing, that both ECA groups demonstrated elevated risk for sensory processing challenges despite heterogeneous experiences suggests that ECA generally confers risk for sensory challenges. Second, while previous findings in typically developing and clinical samples suggest SOR symptoms predict later development of psychological symptoms (Green et al., 2012; McMahon et al., 2019), our analyses used cross-sectional, observational data. Although our path analyses indicate covariation between sensory processing challenges and psychological symptomatology, we cannot draw definitive conclusions about causality or temporal ordering effects. In the present study, we tested the most theoretically plausible model but acknowledge that the directional relationships between our variables ought to be probed by future longitudinal developmental work, ideally from very early in life, including sensitive periods of sensory

development, and extending through adolescence (given that most psychopathology emerges during this life stage). Lastly, this study exclusively used parent-reported measures of sensory processing challenges and psychological symptomatology. Future studies should build upon present methods to include self-reported and behavioral measures of sensory processing and psychological symptomatology. In addition, ongoing work should probe directionality using longitudinal or experimental (e.g. animal model) designs, and evaluate whether the observed pattern of findings extends to more common and/or less severe forms of ECA than circumstances leading to adoption, potentially by characterizing early experiences using dimensional approaches (e.g. threat vs. deprivation), rather than categorical descriptors.

Conclusion

We report increased sensory processing challenges in children and adolescents exposed to heterogenous ECA (PI and AFC), and associations between ECA-linked sensory processing challenges and internalizing and externalizing symptoms. These findings motivate future work assessing whether inclusion of sensory processing challenges during screening and treatment for youth with histories of ECA may support improved clinical outcomes.

Study 2

Introduction

Growing evidence suggests that rare, severe experiences of early caregiving adversity (ECA) increase risk for sensory processing challenges across childhood and adolescence (Armstrong-Heimsoth et al., 2021; Howard et al., 2020; Joseph et al., 2021; Lin et al., 2005; Schneider et al., 2007, 2008, 2008, 2017, 2017; J. Wilbarger et al., 2010). These symptoms, characterized by altered responses to and regulation of responses to everyday sensory stimuli, can be fundamentally disruptive to daily functioning (Ayres, 1972; Dunn, 1997; Miller et al., 2007). In addition to causing difficulties on their own, sensory processing challenges appear to elevate risk for varied forms of psychopathology, including internalizing symptoms, which are common after ECA (Carpenter et al., 2019b; Gunn et al., 2009; Kessler et al., 2010; McLaughlin et al., 2012). However, the majority of existing research examining the connection between ECA and sensory processing challenges has been conducted in young children who have experienced profound and rare forms of early adversity, including circumstances surrounding placement in and adoption from institutional or foster care settings (Armstrong-Heimsoth et al., 2021; Cermak & Daunhauer, 1997; Lin et al., 2005; Méndez Leal et al., 2022; J. Wilbarger et al., 2010). The current study assesses sensory processing challenges following more prevalent forms of ECA in adults, in the hopes of characterizing the generalizability of previously reported links between ECA and sensory symptoms.

Common forms of abuse and neglect are both prevalent and impactful: one in seven children living in the United States has experienced abuse or neglect within the last year, and 41% of the international adult population reports similar histories during childhood, with even higher rates within clinical populations (*CDC Fast Facts: Preventing Child Abuse & Neglect*, 2022; MacDonald et al., 2016). However, despite the impact of these experiences on mental health in the population at large, the relationship between these more prevalent adversities and increased risk for sensory processing challenges is not well-established. Additionally, most prior research on sensory symptoms following ECA has been focused on youth (Armstrong-Heimsoth et al., 2021; Howard et al., 2020; Joseph et al., 2021; Lin et al., 2005; Schneider et al., 2007, 2008, 2008, 2017, 2017; J. Wilbarger et al., 2010). Notably, epidemiological work has shown ECA elevates risk for varied forms of psychopathology (many of which are in turn associated with elevated prevalence of sensory processing challenges) across the lifespan, and that many of these symptoms may only emerge beginning in adolescence or young adulthood (Ben-Sasson et al., 2017, 2019; Ben-Sasson & Podoly, 2017; Gunn et al., 2009; Kessler et al., 2010; McLaughlin et al., 2012; McMahon et al., 2019; Parham et al., 2019; Solmi et al., 2021). As such, evaluating prevalence and patterns in ECA-linked sensory processing challenges across the lifespan may provide important information about developmental trajectories and inform clinical understanding.

While some evidence in some clinical populations has suggested sensory processing challenges may reduce with age, findings are inconsistent and have not focused on ECA. Building on prior work in younger children, in Study 1 (Méndez Leal et al., 2022) I found that parent-reported sensory processing challenges (including SOR) experienced by youth with institutional and foster caregiving histories extend into adolescence, and are linked to internalizing symptoms in youth. These findings have been recently supported by work in one population sample of adolescents (Jeon $\&$ Bae, 2022) and one population sample of adults

(Karaca Dinç et al., 2021), both of which identified links between childhood abuse and neglect and sensory processing challenges. Thus, in the present study, I sought to further this work and test whether less rare but still severe forms of ECA (e.g., abuse, neglect) also increase risk for sensory processing challenges in multiple samples of young adults, leveraging self-reported measures.

A final goal of this study was to explore potential mechanistic pathways by which ECA may induce sensory processing challenges, as this may inform both developmental models and clinical practice. As noted by a recent taxonomy of sensory processing challenges (He et al., 2023), the current literature on sensory processing challenges in youth across populations often uses similar terminology to assess discrete and different elements of sensory processing challenges, typically assessed using distinct categories of measurement approaches (Ben-Sasson et al., 2019; Glod et al., 2015; Gunderson et al., 2023; He et al., 2023; Lane, 2020; Passarello et al., 2022). These categories of symptoms can be divided into categories including perceptual differences (in discrimination, localization, or detection, for example) and affective sensory responses (e.g. having aversions to certain sensory stimuli), among others (He et al., 2023). These sensory processing challenges are often assessed using independent measurements: for example, affective sensory responses are typically examined using questionnaires, while perceptual responses like localization or detection are often indexed using clinical observational measures or behavioral paradigms. While common terms are often used interchangeably to describe sensory processing challenges in each of these domains, and at times symptoms at both levels are treated as though they are reflective of the same phenotype, this may mask underlying variability of experiences. For example, while many individuals with autism spectrum disorders

appear to have both altered experiences of sensory perception and elevated affective sensory symptoms, but these are at most weakly associated within individuals, indicating they may reflect unique underlying phenotypes (He et al., 2021; Schulz & Stevenson, 2022; Williams et al., 2019).

With this in mind, in this study, I attempt to identify the broad categories of sensory symptoms experienced by youth with histories of ECA, using the same modality (self-reported questionnaire measures, including a recently developed assessment of low-level sensory perception) to assess sensory processing challenges at multiple levels within individuals. Here, I probe both low-level perceptual and affective sensory experiences to attempt to characterize ECA-linked sensory experiences. In addition, I also incorporate assessments of both affective and non-affective self-regulation, given both well-established evidence that ECA induces alterations to self-regulation that have important consequences for mental health, and prevalent theoretical models that sensory processing challenges reflect disrupted top-down modulation of responses to sensory experiences (Y. Chen & Baram, 2016; S. A. Green & Wood, 2019; Heleniak et al., 2016; McLaughlin et al., 2020; Weissman et al., 2019). Given neurodevelopmental evidence for hierarchical development, where low-level sensory circuits are thought to develop in infancy and early childhood, followed by affective development across childhood and protracted development of independent self-regulation in adolescence and beyond, these symptom patterns may also speak to mechanistic developmental pathways by which ECA induces changes to sensory processing.

Current Study

The current study seeks to characterize the relationship between more prevalent, severe forms of early life adversity (e.g. abuse, neglect) and sensory over- and under- responsivity

symptoms in adults, with a focus on links to mental health outcomes. I examined the relationship between highly adverse, more prevalent forms of ECA and self-reported questionnaire measures of sensory processing challenges in adults. While I focused on SOR given prior work, I also examined how ECA related to sensory under-responsivity symptoms in adults, and how SOR and SUR related to each other within individuals. In addition, I explored possible developmental timing effects in this sample, in the hopes of informing future longitudinal work. A final, purely exploratory goal of this study was to examine three non-exclusive mechanisms (sensory perception, affective sensory responses, and self-regulation) that might contribute to links between ECA and SOR. Specifically, I assessed whether concurrent measures of sensory perception, affective sensory responses, and of affective and attentional self-regulation were associated with ECA.

Our a priori hypotheses and analysis plan were pre-registered on the Open Science Framework [\(https://osf.io/wa6sc;](https://osf.io/wa6sc) [https://osf.io/mu3eb\)](https://osf.io/mu3eb). In line with previous research, I hypothesized that increased overall ECA exposure would be associated with reduced sensory under-responsivity and elevated SOR. Furthermore, I predicted that more overall ECA exposure would be associated with higher levels of internalizing symptoms through elevated SOR, covarying for age and assigned sex at birth. As part of a series of exploratory analyses examining developmental timing effects, I also predicted that ECA experiences earlier in development (indexed through earliest and mean age of onset) would be associated with both elevated SOR and internalizing symptoms. Based on previous research, I predicted that ECA would be associated with increased affective sensory responses and decreased self-regulation (Callaghan &

Tottenham, 2016b; Wilbarger & Cook, 2011) in our exploratory analyses. I did not anticipate a relationship between ECA and sensory perception differences.

Methods

Participants

Study 2A

We collected questionnaire data from participants aged 18-30 living in the United States, using Amazon's Mechanical Turk platform in February 2021. Responders that failed 1 or more attention checks or provided different answers to repeated simple questions (e.g. "How old are you") were excluded. The final eligible sample included 227 participants.

Study 2B

In order to further probe the effects reported in Study 1, I collected the same measures in April and May 2023 from young adults aged 18-25 enrolled as undergraduates at the University of California, Los Angeles. These participants were also asked to complete additional measures related to candidate mechanistic processes of interest. Participants received course credit for their participation in this research, although participants that failed 2 or more attention checks or provided different answers to repeated simple questions were excluded from analyses. The final eligible sample included 263 participants.

Exclusionary criteria

Given our focus on sensory processing, participants across both studies who identified as deaf, hard of hearing, blind, visually impaired, or as having limited or low vision were excluded

from the primary analyses, but fully compensated for their time through payment or course credit, as appropriate. To ensure findings were not secondary to comorbid autism, all analyses were re-run post-hoc excluding participants with ASD diagnoses, with no substantive changes to my findings.

Measures

Early life adversity: The Childhood Trauma Questionnaire Short Form (CTQ) is a 28-item screening measure that assesses a broad range of experiences of maltreatment in childhood and has been validated in both clinical and population samples with varied experiences of ECA (Bernstein et al., 2003). For each item, participants were asked to report how often given examples of ECA occurred during their childhood prior to age 14, with responses ranging from Never True (1) to Very Often True (5). In addition, participants were asked to report at what age (3-14) they first noticed that example occurring. Total scores on this measure range from 28 to 140 and are derived from the sum of responses to three validity items and responses from five, five-item subscales that capture physical abuse, sexual abuse, emotional abuse, physical neglect, and emotional neglect.

Sensory Over-Responsivity/Sensory Under-Responsivity: To assess self-reported SOR and SUR in adults, I administered the Glasgow Sensory Questionnaire (GSQ; Robertson & Simmons, 2019). While this measure was developed to study sensory symptoms in the context of autism and autistic traits in the general population, it has since been validated in varied clinical and population samples, across cultural contexts (Kuiper et al., 2019; Panagiotidi et al., 2018; Sapey-Triomphe et al., 2018; Takayama et al., 2014; Ward et al., 2017, 2021). This 42-item measure

assesses SOR (referred to on the GSQ as "hypersensitivity") and sensory under-responsivity (GSQ "hyposensitivity") across seven sensory modalities: visual, auditory, tactile, gustatory, olfactory, and proprioception. The six questions associated with each modality are in turn divided into three SOR/hypersensitivity items and three sensory underresponsivity/hyposensitivity items. Participants are asked to report how often they experience specific sensory events, with responses ranging from Never (0) to Always (4). I used the summed GSQ hypersensitivity (SOR) and hyposensitivity (sensory under-responsivity) scores separately throughout. Each of these indices ranges from 0 to 84.

Sensory perception: In order to evaluate sensory perception independent of affective sensory reactivity, I administered the Sensory Perception Quotient (Tavassoli et al., 2014), which was expressly designed for this purpose. This 92-item measure assesses detection and discrimination abilities for vision, hearing, touch, smell and taste from Strongly Agree to Strongly Disagree, using questions that are targeted at specific biological processes (e.g. specific receptors associated with detection of vibrations). I used the summed SPQ perceptual hypersensitivity (range 0-68) and perceptual hyposensitivity (range 0 to 90) scores produced by the revised scoring method (Taylor et al., 2020) separately throughout, with higher scores reflecting more affected sensory perception.

Self-Regulation: I used the Brief Self-Control Scale and the Extended-Emotion Regulation Questionnaire (Gross & John, 2003; Guassi Moreira et al., 2021; Tangney et al., 2004) to probe affective and non-affective self-regulation. The Brief Self-Control Scale is a 13-item measure of applied self-control that asks participants to rate how often they engage in specific behaviors,

from Not at All (1) to Very Much (5). Scores range from 13-65. The Extended-Emotion Regulation Questionnaire asks participants to report how much they engage in five emotion strategies (situation selection, selective attention, distraction, reappraisal, suppression) on 23 items (Likert 1: strongly disagree -7: strongly agree). Subscales are scored separately and used to characterize overall patterns of regulation.

Internalizing Symptoms: Internalizing symptoms were indexed using a common, wellvalidated measure, the Mini Mood and Anxiety Symptoms Questionnaire (Mini MASQ; Clark and Watson, 1995). This short questionnaire captures general distress symptoms common to both anxiety and depression, as well as indices of anxious arousal and anhedonic depression. Participants are presented with 26 feelings, sensations, or problems, and asked how often they experienced them in the past week, ranging from Not At All (1) to Extremely Often (5). Given our interest in internalizing symptoms generally, rather than specifically anxiety or depression, I used the General Distress score as a primary outcome measure in this analysis. Scores on this 8 item subscale range from 8 to 40.

Analysis

The general goals of this study were to report potential effects of more prevalent severe forms of ECA on SOR measures (e.g. GSQ hypersensitivity) and to evaluate possible mediation of relationships between ECA and internalizing symptomatology by SOR. Additionally, I sought to evaluate whether ECA is associated with sensory under-responsivity symptoms and conducted initial exploration of candidate mechanistic pathways that may contribute to sensory symptoms following ECA.

Primary Analyses

Statistical analyses were conducted using SPSS Version 28 (SPSS Inc., USA). Path analyses were performed using the PROCESS SPSS macro version 3.4. These analyses used 95% percentile bootstrap confidence intervals, with 5,000 bootstraps (Hayes, 2017). In line with recommendations (Lemmer & Gollwitzer, 2017; Thoemmes, 2015) for the primary analyses I only performed statistical tests for the pre-registered path analyses that aligned with our theoretical model.

To establish that the expected relationship between early adversity and internalizing symptoms is present in our sample, I first performed a linear regression, with CTQ total, age, and sex as predictors, and general distress on the Mini MASQ as an outcome measure. After establishing that there was a significant relationship between CTQ total and general distress on the Mini MASQ in both sample 2A and sample 2B, I then used one primary path analysis model to examine the impact of ECA (assessed using the CTQ Total Score) on internalizing symptoms (Mini MASQ General Distress score) through SOR (GSQ Hypersensitivity Total Score) in each sample, while covarying for age and sex assigned at birth. Specifically, I examined the relationship between ECA (CTQ) and SOR (GSQ), and then probed indirect effects of ECA on internalizing symptoms through SOR.

To examine the relationship between ECA and sensory under-responsivity symptoms, I performed a standard linear regression, with the CTQ total, age, and sex as predictors, and sensory under-responsivity (hypo-sensitivity) on the GSQ as an outcome. In addition, I conducted four multiple regressions to explore the impact of timing of caregiving adversity on

sensory and psychological symptomatology, with earliest or mean age of onset of ECA, age, and sex assigned at birth as predictors, and SOR (GSQ) or internalizing symptoms (mini MASQ) as outcomes. Lastly, I examined the co-occurrence of SOR and sensory under-responsivity symptoms within individuals using a linear regression.

Secondary Analyses

In Study 2B, I also conducted a purely exploratory analysis examining correspondence (correlations) between profiles of sensory perception (SPQ), affective sensory responses (GSQ total), and self-regulation (BSCS, E-ERQ subscales) within individuals, and examine how these profiles relate to experiences of ECA (CTQ). I used separate linear regressions to examine the relationship between ECA (CTQ) and each outcome of interest, controlling for age and assigned sex at birth. To inform a developing field, I also report zero-order correlations between all perception, affective sensory response, and self-regulation measures below.

Lastly, I applied two exploratory models to examine the relative impacts of ECA (CTQ Total Score) on internalizing symptoms (Mini MASQ General Distress) through measures of candidate mechanistic processes (visualized in Figure 2-1). In the first parallel multiple path analysis model, I examined specific indirect effects of ECA on internalizing through each of three candidate mechanistic measures: perceptual sensitivity (SPQ Hypersensitivity Total Score), SOR (GSQ Hypersensitivity Total Score), and positive self-regulation (indexed by the Brief Self Control Scale Total Score). To assess the contribution of affective regulation, in the second parallel multiple path analysis model, I examined the specific indirect effects of ECA on internalizing through perceptual sensitivity (SPQ Hypersensitivity Total Score), SOR (GSQ

Hypersensitivity Total Score), and reappraisal, a form of affective self-regulation associated with positive outcomes (indexed by the Extended Emotion Regulation Questionnaire Reappraisal Score). Both models included age and sex assigned at birth as covariates.

Results

Descriptive Results

Sample demographic information and descriptive statistics for the primary measures of interest are reported in Table 1 and Table 2. 1 participant who reported they were losing their hearing in one ear was excluded from Study 2A, but fully compensated for their time.

ECA is Associated with Elevated SOR and SUR in Adulthood

As expected, higher reported ECA was associated with increased SOR symptoms in adulthood in both Sample 2A ($a_{SOR_2A} = 0.38$, $SE = 0.05$, $t = 7.59$, 95% CI [0.28, 0.48], $p <$.001), and Sample 2B $(a_{SOR_2B} = 0.42, SE = 0.07, t = 6.32, 95\% \text{ CI} [0.29, 0.55], p < .001)$, covarying for age and sex. In contrast with our predictions, higher reported ECA was also associated with elevated SUR symptoms in adulthood in both samples ($a_{\text{SUR 2A}} = 0.35$, $SE =$ 0.04 , $t = 8.55$, 95% CI $[0.27, 0.43]$, $p < .001$; $a_{\text{SUR}_2B} = 0.39$, $SE = 0.06$, $t = 6.52$, 95% CI $[0.27, 0.43]$ 0.50], $p < .001$). These findings (visualized in Figure 1) suggest that prevalent forms of ECA (e.g. abuse, neglect) increase risk for two key forms of sensory processing challenges, with symptoms persisting into adulthood.
Figure 2 - 1. Parallel path analysis models used to explore mechanisms associated with sensory processing challenges

	Study 2A (MTurk)	Study 2B (SONA)
Scales	Mean (Median; SD)	Mean (Median; SD)
ECA CTQ Total Measure Range: 28 -140	49.42 (42.00; 15.2) Range: 32-103	47.19 (44.0; 9.58) Range: 33 - 86
<i>SOR</i> GSQ Hypersensitivity Total Measure Range: 0 - 84	20.32 (19.0; 12.83) Range: 0-64	27.42 (27.0; 10.95) Range: 1 - 68
Sensory Under-Responsivity GSQ Hyposensitivity Total Measure Range: 0 - 84	17.89 (16.0; 10.82) Range: 0-54	25.53 (26.0; 9.78) Range: 2 - 60
Internalizing Symptoms Mini MASQ Measure Range: 8 - 40	14.77 (12.0; 7.58) Range: 8 - 40	19.03 (18.0; 6.98) Range: 8 - 36

Table 2 - 2. Descriptives for ECA, SOR, sensory under-responsivity, and internalizing symptoms

Effects of Timing of ECA on Links to Sensory Symptoms and Psychological Symptomatology

Median onset of ECA exposures were not associated with SOR in either sample (β_{2A} = -0.17 , SE = 0.34, *t* = -0.49, p = .63; β_{2B} = -0.263, SE = 0.312, *t* = -0.842, *p* = .401), nor were they predictive of internalizing symptoms (β_{2A} = 0.09, SE = 0.22, *t* = 0.448, p = .65; β_{2B} = -0.02, SE = 0.20, $t = -0.08$, $p = .93$). However, there was a significant relationship between earlier first age of onset of ECA and elevated SOR in both samples.

ECA, Sensory Processing Challenges, and Links to Psychological Symptomatology

Findings from the primary path analyses were consistent with our theoretical framework, which posits that ECA inflates risk for psychological symptomatology in part through SOR. As reported in numerous previous studies, there was a significant total effect of ECA on internalizing symptoms. Covarying for age and sex assigned at birth, participants with higher

ECA reported elevated internalizing symptoms on the Mini MASQ ($c_{2A} = .15$, $SE = .03$, $t = 4.7$, 95% CI $[0.08, 0.21]$, $p < .001$; $c_{2B} = 0.13$, $SE = 0.04$, $t = 2.93$, 95% CI $[0.042, 0.215]$, $p = .003$). Additionally, I found significant indirect effects of ECA on elevated internalizing symptoms through SOR ($ab_{2A} = .08$, $SE = .02$, 95% CI [0.05, 0.13]; $ab_{2B} = .09$, $SE = .02$, 95% CI [0.05, 0.15].

Figure 2 - 2. A) ECA is associated with elevated SOR in adulthood across multiple samples. B) ECA is associated with elevated SUR in adulthood across multiple samples

	CTQ	SPQ Hyper	GSQ Hyper	GSQ Hypo	SPQ Hypo	BSCS	EERQ Reappraisal
SPQ Hyper	$.226***$	\overline{a}					
\boldsymbol{p}	< .001						
	$.372**$	$.520**$	\overline{a}				
GSQ Hyper \boldsymbol{p}	< .001	< .001					
GSQ Hypo	$.369**$	$.477**$	$.802**$	\overline{a}			
\boldsymbol{p}	< .001	< .001	< .001				
SPQ Hypo	$.131*$.017	$.122*$	$.142*$	\overline{a}		
\boldsymbol{p}	.035	.782	.050	.022			
BSCS	$.215***$	$-.045$	$.281**$	$.303*$	$.189**$	$\mathord{\hspace{1pt}\text{--}\hspace{1pt}}$	
\boldsymbol{p}	< .001	.467	< .001	< .001	.002		
EERQ Reappraisal	$-.169**$	$-.027$	$-.093$	$-.075$	$-.084$	$.166***$	
\boldsymbol{p}	.006	.663	.135	.229	.175	.007	
MASQ General Distress	$.310**$	$.229***$	$.415***$	$.353*$.034	$-.329**$	$-.295***$
\boldsymbol{p}	< .001	< .001	< .001	< .001	.584	< .001	< .001

Table 2 - 3. Zero-order correlations between mechanistic assessments of interest

ECA, Candidate Mechanism-Linked Experiences, and Psychological Symptomatology

As shown in Chapter 2 Table 1, there were significant primary relationships between ECA all variables of interest, supporting the idea that sensory symptoms following ECA may represent disruptions at the perceptual, affective, and regulatory levels. Likewise, internalizing symptoms were significantly associated with all variables except sensory hyposensitivity (SPQ). However, when assessed in both of the parallel multiple path analysis models (visualized in Figure *2 –* 1, reported in Table *2 - 4*) the specific indirect effect of ECA on internalizing symptoms through perceptual hypersensitivity was no longer significant, while specific indirect effects of affective sensory symptoms and both measures of self-regulation were. This provides preliminary indication that affective and regulatory components may be particularly relevant in the context of sensory processing challenges and mental health following ECA.

Exploratory Model 1: Non-Affective Regulation	Effect	SE	CI 25%	CI 75%
Direct Effect of CTQ on MASQ GD	0.1034	0.0433	0.0182	0.1887
Total Indirect Effect	0.1178	0.0255	0.0735	0.1735
Specific Indirect Effects through:				
Sensory hypersensitivity (SPQ)	0.0067	0.0109	-0.0158	0.0287
Sensory over-responsivity (affective, GSQ)	0.0717	0.0239	0.0318	0.1263
Non-affective self-regulation (BSCS)	0.0395	0.0148	0.0152	0.073
Exploratory Model 2: Affective Regulation				
Direct Effect of CTQ on MASQ GD	0.1005	0.043	0.0158	0.1853
Total Indirect Effect	0.1207	0.0268	0.0748	0.1801
Specific Indirect Effects through:				
Sensory hypersensitivity (SPQ)	0.0027	0.0109	-0.0213	0.0233
Sensory over-responsivity (affective, GSQ)	0.0879	0.0252	0.0447	0.1433
Affective self-regulation (EERQ Reappraisal)	0.0301	0.0135	0.0087	0.061

Table 2 - 4. Results from exploratory parallel path analysis models

Affective, but not Perceptual Sensory Symptoms Co-Occur Within Individuals

Lastly, I assessed co-occurrence of symptoms of SOR and SUR, as well as perceptual hyper and hyposensitivity. SOR and sensory under-responsivity symptoms were very highly correlated in both samples, consistent with previous findings in younger clinical populations (*β2A* $= 0.86$, *SE* = 0.04, *t* = 26.95, *p* <.001; $\beta_{2B} = 0.80$, *SE* = 0.03, *t* = 21.61, *p* <.001). By contrast,

SPQ hyper and hyposensitivity measures appear to be uncorrelated ($\beta_{2B} = 0.02$, *SE* = 0.064, *t* = .277, *p* =.78), suggesting they may capture independent patterns of symptoms.

Discussion

The current study examined the prevalence of sensory over responsivity following severe, prevalent forms of early caregiving adversity in multiple samples of young adults. Across samples, I find evidence that early caregiving adversity (specifically exposure to prevalent forms of abuse and neglect) is associated with SOR, and that both ECA and subsequent SOR symptoms are linked to internalizing symptoms in young adulthood. Furthermore, I find that sensory under responsivity symptoms are also present into adulthood following ECA and find consistent evidence that SOR and SUR symptoms frequently co-occur within individuals.

In addition, I report preliminary cross-sectional analyses suggesting that these sensory symptoms may reflect adversity-linked changes to and regulation of affective sensory responses, more than direct changes to low level sensory perception, providing a foundation for future longitudinal and experimental investigation. While I did find evidence of a relationship between perceptual hypersensitivity and early caregiving experiences, our findings are most consistent with theories positing that sensory processing challenges reflect primarily affective and selfregulation experiences (often described as modulation), more so than low level perceptual differences (Ayres, 1972; Dunn, 1997; S. A. Green & Wood, 2019; Miller et al., 2007).

This also may inform our understanding of the enduring presence of these ECA-linked sensory symptoms into young adulthood. While raw perceptual materials may develop early in childhood and be modulated by the environment to a small degree over time, given our findings and the protracted emotion regulation development that occurs during development (Silvers et al., 2012), it may be that the window for interpreting the meaning of perceptual cues may be most meaningfully extended through young adulthood through affective experiences. Our findings dovetail with other recent work in one population sample of adolescents (Jeon & Bae, 2022) and one population sample of adults (Karaca Dinç et al., 2021), both of which report links between childhood abuse and neglect and sensory processing challenges. While the adult study primarily reported links between early caregiving adversity and general psychopathology through sensory sensitivity symptoms, the adolescent findings also indicate that early caregiving adversity and sensory symptoms in youth are linked to both general negative affect and reduced emotion regulation on self-reported questionnaires (Jeon & Bae, 2022; Karaca Dinç et al., 2021). Taken together, our findings provide consistent support for the importance of sensory and affective experiences following early caregiving adversity for mental health.

In addition, I find evidence that sensory under responsivity symptoms are also present into adulthood, and that sensory over and under responsivity symptoms frequently co-occur within adult individuals, as has been reported in clinical samples. I also report early evidence that this co-occurrence is primarily present in measures of affective sensory responses, rather than lower-level perceptual experiences where over-responsivity and under-responsivity may be more distinct. Our findings suggest the need to explore whether ECA-linked SUR and SOR as they are commonly assessed in the literature reflect distinct but overlapping symptom patterns or in fact reflect one underlying phenotype, and to examine perceptual and affective sensory symptoms concurrently whenever possible.

Of note, independent of early caregiving experiences, here I report high incidence of sensory over and under responsivity in general samples of young adults. This is striking given emerging evidence that sensory processing difficulties are a strong predictor of subsequent psychopathology in young people(Schwarzlose et al., 2023). In addition, I find increased sensory symptoms appeared to be associated with varied symptoms of distress, including internalizing symptoms, decreased positive (and increased negative) self-regulation, and even elevated sleep problems. Given the rapidly changing developmental demands introduced during adolescence and young adulthood, including elevated capacity for learning, adaptation, and exploration, rapidly expanding social emotional and relationship-building opportunities, and concurrent romantic and sexual development, understanding and characterizing underlying mechanisms for the development of sensory processing challenges may provide unique and under-explored opportunities to support and improve well-being in young people.

Limitations and Future Directions

Leveraging self-reported measures of sensory perception, affective sensory responses, and self-regulation experiences across multiple samples, my findings indicate that prevalent, severe forms of ECA are associated with elevated SOR and SUR in young adulthood. However, the present work leaves several opportunities for continued investigation. First, this analysis only includes self-reported questionnaire measures, and does not include behavioral measures of sensory perception, affective sensory experiences, or self-regulation, or objective measures of ECA. While continued research has demonstrated that self-reported adversity and emotionrelated experiences are highly correlated with outcomes, ongoing work might benefit from

observational, behavioral and experimental investigation of these relationships. Additionally, all data included in this study is cross-sectional, and therefore prevents conclusions about causality or directionality in the effects I have observed, particularly for links between sensory symptoms and concurrent mental health challenges. Future work should leverage long-term longitudinal observational studies and sensory-based intervention work to begin to explore directionality effects for the primary reported relationships between early caregiving adversity, sensory experiences, and internalizing symptoms.

Consistent with recent recommendations (van den Boogert et al., 2022) the studies above suggest this emerging field would benefit from a systematic effort to develop consistent terminology and well-validated measurements for each of the symptom profiles of interest, with a focus on behavioral observation and experiment-based assessments, particularly for adolescents and adults. Existing work on sensory processing challenges has used varied and inconsistent terminology to describe similar patterns of symptoms (Ben-Sasson et al., 2019; Glod et al., 2015; Gunderson et al., 2023; He et al., 2023; Lane, 2020) and relied primarily on a limited number of parent-reported questionnaire based assessments of sensory processing, even though parent-reported symptoms are often uncorrelated with self-reported experiences in youth. Incorporating self-reported symptoms into studies of sensory processing challenges in adolescents and young adults may therefore provide more complete understandings of symptoms in these populations. While some recent work (e.g. Jung et al., 2021) has applied observational assessments in adolescent populations, our findings suggest that the field would benefit from targeted assessments of enduring symptoms relevant to the daily experiences of adolescents and young adults, regardless of adversity history and/or comorbid clinical diagnoses.

Interim Conclusion

This study examined the prevalence of sensory processing challenges following abuse and neglect (two prevalent severe forms of ECA) in two samples of young adults. In line with Study 1, I find that ECA is associated with elevated SOR and SUR in young adults, and that ECA, SOR, and SUR all are linked to internalizing symptoms. Additionally, I report evidence that SOR and SUR symptoms frequently co-occur within individuals, alongside preliminary findings that suggests affective sensory and regulatory phenotypes might be particularly relevant to mental health following ECA.

Study 3:

Introduction

Youth who have experienced previous institutional (e.g. orphanage) caregiving display heightened levels of sensory processing challenges that can have profound impacts on daily life (Armstrong-Heimsoth et al., 2021; Howard et al., 2020; Joseph et al., 2021; Lin et al., 2005; Schneider et al., 2007, 2008, 2008, 2017, 2017; J. Wilbarger et al., 2010). In particular, previously institutionalized children and adolescents display elevated sensory over-responsivity (SOR), a pattern of prolonged or intensified responses to everyday stimuli that has been connected to clinical outcomes in varied populations (Méndez Leal et al., 2022; Reynolds & Lane, 2008; J. Wilbarger et al., 2010). While to our knowledge no published neuroimaging work to date has assessed how these early institutional caregiving experiences might alter the neural bases of sensory perception, extensive neuroimaging work has demonstrated that unstable caregiving experiences are associated with altered affective and self-regulatory neurodevelopment, both in childhood and in the adolescent period Callaghan & Tottenham, 2016b; Y. Chen & Baram, 2016; Cohodes et al., 2020; Gee et al., 2013; Heleniak et al., 2016; Jenness et al., 2020; Silvers et al., 2017; Tottenham et al., 2010; VanTieghem & Tottenham, 2018; Weissman et al., 2019). Given what is known about neurodevelopment following ECA, sensory processing challenges following early caregiving adversity may therefore reflect changes to low-level sensory perception (for example, perceiving a sound to be louder than others might), altered affective responses (e.g., finding the sound to be unpleasant), or altered regulation of initial affective or perceptual response (e.g., having difficulty controlling the intensity or duration of one's emotional response to the sound).

Previous findings from the ECA literature provide insights into how ECA may act on the affective and regulatory pathways proposed. Empirical research suggests that ECA alters neural circuitry involved in responding to affectively salient stimuli, including the amygdala (Dannlowski et al., 2013; Fareri & Tottenham, 2016; Silvers et al., 2017).Similarly, ECA appears to modulate prefrontal regulation of responses to both affective stimuli (in regions including the ventromedial prefrontal cortex, or during implicit/automatic regulation, and the dorsolateral, ventrolateral, and dorsomedial cortex during explicit/deliberate regulation) and to non-affective stimuli (in regions including the dorsolateral, ventrolateral, and dorsomedial prefrontal cortex). ECA-associated alterations in prefrontal circuitry have in turn been associated with diminished behavioral self-regulation ability, and are thought to contribute to elevated rates of psychopathology in youth with these experiences (Callaghan & Tottenham, 2016b; Y. Chen & Baram, 2016; Cohodes et al., 2020; Gee et al., 2013; Heleniak et al., 2016; Jenness et al., 2020; Silvers et al., 2017; Tottenham et al., 2010; VanTieghem & Tottenham, 2018; Weissman et al., 2019). Thus, there is evidence that ECA shapes neural circuitry relevant for both generating and regulating affective responses to a variety of affectively salient stimuli.

However, to date very little is known about the neurodevelopmental impacts of ECA on sensory perception in humans. Neuroimaging research in clinical populations (primarily conducted in individuals with autism spectrum disorders, or ASD) has connected symptoms of sensory processing challenges to neural responses thought to be associated with altered sensory perception, affective reactivity, and self-regulation. Work in youth with ASD has reported altered neural responses to sensory stimuli in low-level sensory regions, including primary sensory cortices (Edgar et al., 2015; Jassim et al., 2021; Orekhova et al., 2012; C. E. Robertson

& Baron-Cohen, 2017; Williams et al., 2020), as has research investigating SOR symptoms in particular (S. A. Green et al., 2013, 2019; Orekhova et al., 2012). In addition, SOR symptoms have been linked to both altered responses to and habituation of responses to sensory stimuli in affective regions (including the amygdala, the insula, and the salience network more broadly; Green et al., 2013, 2015, 2016, 2019). Similarly, SOR symptoms have been associated with altered responses in prefrontal regulatory regions, including reduced prefrontal down-regulation of affective regions (including the amygdala) during aversive sensory stimulation (Green et al., 2015, 2018, 2019; Green & Wood, 2019). Likewise, a recent meta-analysis reported youth with ASD displayed diminished activity in regulatory prefrontal regions that are also altered by ECA, including dorsolateral and medial prefrontal cortex (Jassim et al., 2021).

However, emerging evidence suggests that patterns of sensory processing challenges may vary depending on the clinical population studied, and therefore different mechanistic processes may contribute to sensory symptoms in youth with ECA, depending on the context (He et al., 2021; van den Boogert et al., 2022). The present study sought to fill this gap in the literature by simultaneously examining three non-exclusive candidate developmental functions (altered perception, affective sensory reactivity, or affective or non-affective self-regulation) that may be shaped by ECA and later contribute to sensory challenges.

Current Study

The current study probes candidate mechanisms underlying SOR after ECA exposure in two parts. First, I apply multivariate pattern expression analyses to fMRI data collected from a population of PI and comparison adolescents. Specifically, I assess overlap between individual

participant's whole-brain neural responses to aversive auditory stimulation and publicly accessible meta-analytic neural "signatures" of auditory perception, affective reactivity, and affective and non-affective regulation drawn from Neurosynth (Yarkoni et al., 2011). I hypothesized PI youth would have increased affective reactivity pattern expression and decreased affective and non-affective regulation pattern expression, relative to comparison youth, and that increased affective reactivity and decreased regulation pattern expression estimates would be associated with SOR.

Second, I conducted an exploratory psychophysiological interaction (PPI) analysis to examine sensory-related changes in functional connectivity linked to seed regions implicated in each of the three mechanistic pathways of interest. To test alterations associated with perception, I used primary auditory cortex seeds, given that auditory cortex has been associated with altered perception of simple sensory stimuli in autism (Edgar et al., 2015; H. L. Green et al., 2022; Millin et al., 2018; Orekhova et al., 2012; O'Sullivan et al., 2019; C. E. Robertson & Baron-Cohen, 2017; Williams et al., 2020, 2021). To test for differences in reactivity and regulation, I used amygdala seeds (affective reactivity), alongside various prefrontal regulatory regions including the ventromedial prefrontal cortex (vmPFC; linked to implicit affective regulation) as well as dorsolateral (dlPFC), ventrolateral (vlPFC), and dorsomedial (dmPFC) cortex regions associated with explicit affective and non-affective regulation (Buhle et al., 2014; Diekhof et al., 2011).

Method

Participants

Participants for this project were recruited from the fourth wave of an ongoing longitudinal study examining socioemotional neurodevelopment in PI and comparison youth (described in Study 1). Sample demographics are described in Chapter 3 Table 1. Parent-reported sensory symptoms were only assessed in youth who were younger than 18 at the time of data collection.

	Comparison $(N = 43)$	PI $(N = 30)$
	Mean (Median; SD)	Mean (Median; SD)
Age	15.26 (14.46; 3.71)	16.5(16.04; 2.78)
	Count $(\%)$	Count $(\%)$
Assigned Sex at Birth	Female: 21 (49%) Male: 22 (51%)	Female: 20 (67%) Male: 10 (33%)
Race		
Black	7	$\overline{0}$
Asian	12	16
white	24	13
Native Hawaiian or Pacific Islander		
Multiracial		$\mathbf{1}$
Other		
Ethnicity		
Latinx/e	$\overline{4}$	

Table 3 - 1. Demographic information for comparison and PI participants in final sample

fMRI Task

Data for this analysis was collected using an aversive auditory stimulation fMRI task described in previous research (Silvers et al., 2016). In the scanner, participants were presented with one of two shapes and asked to press a button when they see the border of the shape begin to thicken (sample trial shown in Figure 1). During three 27-second blocks of this game ("aversive auditory"), participants were exposed to a loud, metallic, aversive auditory stimulus (Neumann et al., 2008) that was co-presented with the shape and similarly terminated when the button was pressed. During three interleaved blocks, participants played the game with the same shape as in the aversive auditory blocks, but without the aversive sound ("silent"). Participants wore noise cancelling headphones tuned to the scanning sequence, and the volume of the auditory stimulus was calibrated such that each individual participant was asked to confirm that it was "loud, but not painful" during a test scan prior to completing the task. All analyses compared the game presented with the auditory stimulus ("aversive auditory stimulation") with the same game played without the noise ("silent").

Figure 3 - 1. Visualization of Aversive Sensory Stimulation Neuroimaging Task Data acquisition

Neuroimaging data for this analysis were collected using a 3T Siemens Prisma scanner with a 32-channel head coil and a parallel image acquisition system (GRAPPA). T2* echoplanar images were acquired during a single functional run (33 axial slices; TR: 2000; TE: 30 ms; flip angle: 75° ; FOV: 192 mm²; voxel resolution: $3 \times 3 \times 4$ mm³). Functional images were registered to a whole-brain high-resolution T1-weighted, MPRAGE image (TR = 2400 ms, TE = 2.22 ms, flip angle = 8° , FOV = 256 mm², 0.8 mm³ isotropic voxels, 208 slices).

Preprocessing

All preprocessing and analyses were conducted using the fMRI Expert Analysis Tool from the FMRIB Software Library package (FSL, www.fsl.fmrib.ox.ac.uk). Following visual inspection of functional and anatomical images for artifacts, non-brain tissue was removed from all images using FSL's Brain Extraction Tool. Functional images were then spatially realigned using MCFLIRT (to correct for head motion) and high-pass filtered at 100 s to address scanner drift. Volumes with head motion that was greater than a .9 mm framewise displacement were censored. Participants were excluded from final analyses if > 20% of volumes exceeded this threshold, or if they did not respond behaviorally to at least 85% of trials. In total, 8 participants were excluded due to data quality issues, resulting in the final sample reported in Chapter 2 Table 1. Functional images for each participant were then pre-whitened, registered to their high-resolution MPRAGE scan using FSL's registration tool (Greve & Fischl, 2009), and normalized to standard MNI space (MNI 152 NLIN sixth-generation T1 template, 2 mm³). Data were then smoothed at 5 mm FWHM prior to univariate analysis of task responses and PPI analysis, and at 1 mm FWHM prior to pattern expression analyses.

Univariate Analysis of Aversive Sensory Stimulation Task

Given that the experimental task employs a block design with rapid auditory stimulus presentations (approximately every second, less than the length of the TR) that would produce biased estimates if modeled individually (Mumford, 2014), I modeled block-level responses to aversive auditory stimulation. To examine average responses to our contrast of interest (aversive auditory> silent), I created a generalized linear model (GLM) with individual regressors for aversive, silent, and a third task condition, convolved with a standard hemodynamic response function. I also included the first derivatives of each of these regressors in the GLM, to account for individual variability in hemodynamic responses and slice timing effects. I statistically controlled for head motion using independent regressors for individual volumes that exceed a motion threshold cutoff of 0.9 mm framewise displacement (effectively censoring these

volumes). In addition, I used FSL's motion parameters, consisting of 6 regressors (for x, y, z, pitch, roll, yaw directions), their first derivatives, and the squares of both of these (for a total of 24 regressors). I then performed mixed effect group-level analyses on the auditory stimulation contrast using FSL's FLAME1 module, using a cluster defining threshold of $Z = 2.3$, a familywise error rate of *p*< 0.05 and Random Field Theory cluster correction to address multiple comparisons.

Block-Level Whole-Brain Activity Estimation for Pattern Expression Analysis

As in the univariate analysis, I modeled block-level responses to aversive auditory stimulation. However, to produce the most accurate final estimate of pattern expression possible, I created separate GLMs for each of the three individual block-level responses to auditory stimulation. In each GLM, the target block was modeled as its own regressor, the other two auditory stimulation blocks were modeled together in one nuisance regressor, and remaining stimulus block types (silent and the final condition included in this task but not included in this analysis) were modeled in additional nuisance regressors. As in the first analysis, I included the first derivatives of each of these regressors and FSL's motion parameters in all three GLMs, and censored volumes with excessive motion, using a threshold cutoff of 0.9 mm framewise displacement.

Extracting Individual Pattern Expression Estimates for Candidate Mechanistic Signatures

We used parameter estimates for each of the three blocks to create a linear contrast image (of auditory stimulation relative to the silent task condition), and then used the unthresholded z statistic images from this contrast to extract pattern expression estimates for each of the neural

signatures of interest (Guassi Moreira et al., 2023; Van Oudenhove et al., 2020). I applied pattern expression analyses to assess the extent to which each individual's block-level whole-brain patterns of brain activity during aversive auditory stimulation reflected meta-analytic neural signatures of auditory perception, affective reactivity, and affective regulation. These neural signature maps were drawn from Neurosynth (Yarkoni et al., 2011), a publicly accessible resource that automates meta-analyses of fMRI studies. Specifically, I used the meta-analytic uniformity maps generated by the terms "auditory stimuli" (auditory perception, 115 studies), "reactivity" (affective reactivity, 84 studies), "emotion regulation," (affective regulation, 598 studies), and "cognitive control," (non-affective regulation, 598) respectively. In order to examine the distinct contributions between affective reactivity and regulatory signatures, I subtracted the affective reactivity signature from both the emotion regulation and cognitive control maps, and removed any clusters comprised of fewer than 10 voxels to eliminate artifacts. Applying these meta-analytic maps provides a benefit over ROI selection because it allows whole-brain, empirically-founded selection of voxels of interests, reducing the chance that important neural task responses are occurring in voxels not included in my mask of interest.

I extracted multivariate patterns from the four signature maps (in standard MNI 152 space) and from each of the three unthresholded z-stat images (one per block) using NiftiMasker(). I then performed two parallel analyses, minimally smoothing multivariate patterns at 1 mm and 4mm FWHM, respectively. This is in line with recommendations for pattern expression analyses in which the granularity of the neural signatures of interest is not wellcharacterized and the goal is both to assess fine-grained individual responses and to develop a candidate marker that might be applied to future investigations (Weaverdyck et al., 2020).

Following smoothing, I took the dot product between the two types of multivariate patterns (neural signature from Neurosynth, whole brain single-block activity estimate) to produce a single pattern expression estimate value for each signature (4; perception, affective reactivity, emotion regulation, and cognitive control) and each block (3), for a total of 12 pattern expression estimates per participant, in line with prior work.

Statistical Analyses for Pattern Expression

Upon extracting parameter estimates for each of the three signature maps of interest and for each block $(3x 4 = 12$ pattern expression estimates per individual), I averaged pattern expression estimates across blocks to produce one estimate per signature per participant, and divided all pattern expression values (which are unitless) by 10^5 for visualization and analysis purposes. I then examined group differences between PI and comparison participants on each of these pattern expression estimates, using three separate linear regressions with PI status (0: comparison, 1: PI) as a categorical predictor, assigned sex at birth and age as covariates, and average pattern expression estimate (for each signature of interest) as the outcome.

Given that I have already reported PI youth have elevated sensory processing challenges (SSP) and SOR (SP3D) relative to comparison youth in Study 1, I did not repeat group comparisons of sensory symptoms. Because sensory measures for this wave of the study were only collected for participants under the age of 18, within-group sample size of usable neural and questionnaire data for participants was limited, so I only examined links between pattern expression estimates for each signature and sensory symptoms using linear regression across the whole sample, not within groups.

Psychophysiological Interaction Analysis

We conducted exploratory psychophysiological interaction analyses using seed regions associated with each proposed candidate mechanistic process.

ROI Definition for PPI Analyses

Figure 3 - 2. Left vlPFC (orange), left dlPFC (gray) and right dlPFC (green) ROIs from metaanalysis (Buhle et al., 2014). Not shown: right dmPFC and right vmPFC ROIs

Structural ROI masks (primary auditory cortex and amygdala) were created using the Harvard Oxford Atlas in FSL. Prefrontal functional ROIs were created using meta-analyses of implicit emotion regulation (right vmPFC; drawn from (Diekhof et al., 2011) and explicit emotion regulation (cognitive reappraisal; left vlPFC, right dmPFC, bilateral dlPFC; (Buhle et al., 2014). Given variability in size and function of these large, distributed functional regions, for consistency (and comparison) I created 7 mm radius spheres centered around the global maxima reported for each region in the meta-analyses. Future analyses might also include ROIs centered around local maxima reported in these meta analyses.

PPI Modeling

For each seed region, using FSL's Feat tool I created a separate GLM in which I modeled the 3 task conditions, an additional regressor for the average time course in the seed region, drawn from preprocessed data and mean centered, 3 task x seed time course interaction terms, and the motion regressors included in the previous models for each participant. In line with recommendations for block-designed data(Di & Biswal, 2017; O'Reilly et al., 2012), I did not apply deconvolution prior to analysis. A linear contrast of the seed x aversive auditory - seed x silent interaction terms was computed to compare task-related changes in functional connectivity.

Results

Manipulation Check

As a manipulation check, I computed the average whole brain response to the contrast of interest (aversive auditory isilent) and report these findings in Chapter 3 Figure 2. Analyses were conducted using FSL's FLAME1 module, using a cluster defining threshold of $Z = 2.3$, a familywise error rate of *p* < 0.05 and Random Field Theory cluster correction to address multiple comparisons. The largest peaks were present in bilateral primary auditory cortex, consistent with our expectations for the contrast of interest, and other global maxima were found in the supplementary motor area and visual cortex, consistent with task demands. I found no relationship between parent-reported symptoms of SOR on the SP3D and whole-brain responses to the contrast of interest.

Cluster peak region aversive auditory > silent *Voxels p value Max Z value R/L Peak MNI Coordinates* **X Y Z** Superior temporal gyrus (primary auditory cortex) $18001 \le 0.0001 \le 10.8 \le R \le 60 \le 18 \le 8$ *Frontal operculum* | | 10.8 | R | 46 | -16 | 0 *Heschl's gyrus* | | | 10.3 | R | 46 | -18 | 4 *Heschl's gyrus* | | 9.95 | R | 44 | -20 | 10 *Central opercular cortex* \vert \vert 9.66 \vert R \vert 52 \vert -4 \vert -2 Superior temporal gyrus (primary auditory cortex) $\begin{vmatrix} 11262 \\ \end{vmatrix} < .0001$ 10.8 L $\begin{vmatrix} -44 \\ -32 \end{vmatrix}$ -32 10 *Heschl's gyrus* | | 10.8 | L | -38 | -30 | 10 *Heschl's gyrus* | | | 10.6 | L | -46 | -14 | -2 *Planum Polare* | | | 10.5 | L | -48 | -8 | -4 *Superior temporal gyrus* 10.1 L -50 -22 8 *Superior temporal gyrus* | \vert 9.09 | L \vert -68 | -34 | 18 Supplementary motor area 2303 *< .0001* 5.17 R 12 4 64 *Supplementary motor area* | | | 5.09 R | 10 | 8 | 62 *Superior frontal gyrus* 1 4.94 R 12 12 62 *Superior frontal gyrus* | 4.73 R 8 18 56 *Superior frontal gyrus* | 4.46 R 6 | 18 | 44 *Superior frontal gyrus* | 3.71 | L | -16 | 4 | 64 *Superior frontal gyrus* | 5.17 R | 12 | 4 | 64 Calcarine sulcus (visual cortex) $\begin{vmatrix} 1971 & | \ 1971 & | \end{vmatrix}$ 6.0001 4.44 R 20 -62 8 *Lateral occipital cortex* \vert \vert \vert 4.13 \vert R \vert 10 \vert -74 \vert 14 *Intracalcarine cortex* | | 3.89 | L | -12 | -70 | 8 *Lingual gyrus* | | 3.79 | L | -20 | -60 | 0 *Intracalcarine cortex* | 3.55 R | 14 | -78 | 10 Cerebellum | 865 |< .0001 | 4.9 | L | -16 | -74 | -44 *Cerebellum* 1 4.65 L -22 -74 -46 *Cerebellum* | | 4 | L | -8 | -76 | -30 *Cerebellum* | | 3.94 | L | -24 | -68 | -28 *Cerebellum* | | 3.68 | L | -8 | -78 | -24 *Cerebellum* 1 3.56 L -18 -72 -24

Table 3 - 2. Global and local maxima from significant clusters from whole-brain analysis for aversive auditory stimulation contrast

Figure 3 - 3. Whole-brain average responses to aversive auditory stimulation contrast

ECA Group Differences in Whole-Brain Responses to Aversive Sensory Stimulation

As shown in [Figure 3 -](#page-97-0) 4, previously institutionalized youth demonstrate elevated responses to aversive sensory stimulation in the left middle and inferior frontal gyri (dlPFC and vlPFC), relative to comparison youth. Global and local maxima for the group comparison for this contrast are reported in [Table 3 -](#page-98-0) 3.

Figure 3 - 4. Relative to comparison youth, PI youth demonstrate elevated recruitment of dlPFC and vlPFC during aversive auditory stimulation

Table 3 - 3. Global and local maxima from group comparison for whole-brain analysis for aversive auditory stimulation contrast

Cluster peak region			Voxels $ p$ value $ Max Z$ value $ R/L $ Peak MNI Coordinates			
aversive auditory $>$ silent						
Middle frontal gyrus	905	< .0001	3.63	-48	18	48
Middle frontal gyrus			3.62	-46	22	50
Frontal pole			3.44	-50	42	12
Inferior frontal gyrus			3.4	-50	36	16
Middle frontal gyrus			3.33	-48	18	32
Middle frontal gyrus			3.29	-52	16	

Figure 3 - 5. A) PI youth display elevated pattern expression of neural signatures associated with cognitive control during auditory stimulation B) No relationship between pattern expression estimates for mechanistic signatures of interest and parent-reported SOR symptoms

PI Youth Display Elevated Non-Affective Self-Regulation Pattern Expression

I found no significant group differences in pattern expression for mechanistic signatures of auditory perception, affective reactivity, or emotion regulation, but did find elevated pattern expression for the signature associated with cognitive control in PI youth. While not significant, consistent with my expectations, pattern expression estimates for affective self-regulation mirrored those observed for non-affective regulation (cognitive control). These findings are shown in Chapter 3 Figure 5 and in Table 3 - 4. [Group differences in pattern expression](#page-99-0) [estimates for mechanistic signatures.](#page-99-0) After covarying for participant age and sex, I found that PI youth had elevated pattern expression for both neural signatures of auditory perception and cognitive control relative to comparison youth, while there remained no group differences for affective reactivity or emotion regulation estimates.

Without covariates:		Std error	t	p	CI 25%	CI 75%
Auditory Perception	1.3365	0.707	1.89	0.063	-0.074	2.747
Affective Reactivity	0.0239	0.217	0.11	0.913	-0.409	0.457
Emotion Regulation	0.7484	0.435	1.722	0.089	-0.118	1.615
Cognitive Control	1.2884	0.623	2.067	0.042	0.045	2.531
Covarying for age and sex:	β	Std error	t	\boldsymbol{p}	CI 25%	CI 75%
Auditory Perception	1.6056	0.734	2.188	0.032	0.141	3.07
Affective Reactivity	0.064	0.227	0.282	0.779	-0.389	0.517
Emotion Regulation	0.8782	0.453	1.938	0.057	-0.026	1.782

Table 3 - 4. Group differences in pattern expression estimates for mechanistic signatures

Elevated Aversive Auditory Stimulation-Related Functional Connectivity Between Auditory Cortex and rostrolateralPFC in PI Youth

PPI analyses conducted with a right and left auditory cortex seeds revealed elevated taskrelated functional connectivity with right rostrolateral prefrontal cortex for PI youth, relative to comparison youth (results from the analysis using a left auditory cortex seed shown in [Figure 3 -](#page-100-0) 2). This suggests greater task-related connectivity in between these two regions during the aversive auditory condition, relative to the silent condition, for the PI youth. Additionally, I report increased task-related connectivity between bilateral cerebellum and both vlPFC and right auditory cortex in PI vs comparison youth. I did not find any group differences in connectivity when using seeds from the amygdala, vmPFC or dmPFC.

Figure 3 - 6. PI youth display elevated connectivity between bilateral primary auditory cortex and rostrolateral prefrontal cortex (left primary auditory seed shown)

Discussion

The current study explored candidate neurodevelopmental mechanisms for the development of sensory over responsivity symptoms following exposure to early caregiving adversity. Across multiple measures, I report evidence of elevated recruitment of prefrontal regulatory regions in PI vs comparison youth during aversive auditory stimulation, preliminarily suggesting a potential role of self-regulation and modulation processes known to be impacted by ECA in sensory processing in this group. Specifically, I report elevated pattern expression of a meta-analytic signature for non-affective regulation in PI youth, relative to comparison. In line with these findings, I also found increased recruitment of dorsolateral and ventrolateral prefrontal cortex during auditory stimulation in PI youth, as well as increased functional connectivity between auditory cortex and rostrolateral prefrontal cortex. Notably (and contrary to our hypotheses), I did not find evidence in line with reactivity-driven SOR symptoms in this population, although auditory perception-focused pattern expression was significantly elevated in PI youth after covarying for age and sex.

The increased pattern expression associated with cognitive control and elevated recruitment of prefrontal regulatory regions in PI youth may reflect more effortful or elaborative processing of sensory stimuli, or alternatively, they may reflect compensatory engagement of regulatory regions to help manage affective responses to sensory stimuli. While the current sample of participants for whom I have SOR symptom information is too small, in the final sample it may be informative to parse these possible explanations by evaluating whether greater auditory cortex-rlPFC connectivity predicts reduced SOR. In either case, the current findings suggest that, in line with pre-existing theory (Ayres, 1972; Dunn, 1997; S. A. Green & Wood, 2019; Miller et al., 2007), SOR symptoms following ECA may at least in part reflect altered selfregulation. While the current findings provide purely correlational evidence, they suggest that it may be worthwhile to explore these effects in the context of deliberate self-regulation to sensory stimuli in both affective and non-affective contexts.

Limitations

While our findings provide preliminary evidence of elevated recruitment of prefrontal regulatory regions during aversive sensory stimulation in PI youth, this study has a few important limitations. First, this analysis would benefit from increased sample size, particularly for the PI group. Due to data collection protocols, parent-reported sensory over responsivity symptoms were only available for youth who were under the age of 18 at the time of data collection, further reducing the sample for which I could concurrently examine sensory symptoms and neuroimaging data and preventing analysis of the relationship between symptoms and neural responses within groups. Ongoing work in a larger sample my examine the relationship between SOR symptoms and the elevated auditory cortex to rfPFC symptoms we observed in this sample, for example. Additionally, the task used for this analysis uses one very aversive, repeated sound in a block design, potentially masking variability in responses to different types, valences, and intensities of sensory stimuli. Future work might benefit from event-related task designs that incorporate variability in stimuli and do not extinguish aversive stimuli with a motor response. In addition, tasks that expressly examine each mechanistic pathway of interest (e.g. discrimination tasks, tasks directly regulating responses to sensory stimulation) may also clarify the present findings. Lastly, while the NeuroSynth signature maps provide a reference point from which to examine pattern expression in this sample, future work might benefit from generating participant-specific signatures of candidate processes of interest, for more individualized, fine-grained analysis.

Ongoing work might also explore network neuroscience-based assessments of sensory processing. Recent research suggests greater expansion of functional connectivity gradients (and therefore diminished integration between visual and default mode networks) is associated with sensory symptoms in the general population, and a recent study in the Adolescent Brain Cognitive Development cohort (participants aged 9-12) cohort suggests SOR is associated with decreased functional connectivity within and between sensorimotor networks, and increased connectivity between sensorimotor and salience networks (del Río et al., 2022; Schwarzlose et al., 2023). Similarly, findings from work in youth with ASD suggests here, too, SOR symptoms were associated with elevated connectivity between the salience network and primary sensory areas(S. A. Green et al., 2016). Importantly, recent evidence suggests there may be sex-related differences in SOR-related salience network functional connectivity patterns (Cummings et al., 2020). Network investigations will also allow for explorations of theories arguing that sensory symptoms in autism may reflect altered temporal processing (C. E. Robertson & Baron-Cohen, 2017), or relatedly that sensory processing challenges in ASD are indicative of differences in core-periphery brain dynamics (Roy & Uddin, 2021). Lastly, investigation of possible contributions by the thalamus to sensory symptoms may be an important contribution, particularly as an assessment of possible contributions of perceptual mechanisms to ECA-linked sensory symptoms. A growing body of literature has implicated thalamocortical connectivity in SOR in individuals with autism (beginning as early as infancy), suggesting this may be a ripe area for investigation following ECA(S. A. Green et al., 2017; Wagner et al., 2023).

Interim Conclusion

This study probed candidate neurodevelopmental mechanisms that may underlie the development of SOR following experiences of ECA by applying multivariate pattern expression and PPI analyses to fMRI data collected during aversive auditory stimulation. While I found no relationship between SOR symptoms and neural responses to aversive auditory stimulation, I

report that PI youth display elevated pattern expression of a meta-analytic signature of cognitive control, and increased recruitment of prefrontal regulatory regions relative to comparison youth. These findings provide a foundation for ongoing experimental work explicitly evaluating mechanisms underlying sensory processing challenges following exposure to ECA.

General Discussion

This dissertation sought to characterize the relationship between ECA and sensory processing challenges in adolescence and young adulthood, employing parent-reported, selfreported, and neuroimaging measures to examine varied facets of sensory development. Together, the studies above provide consistent evidence that varied forms of severe caregiving adversity elevate risk for sensory processing challenges, and that these sensory symptoms persist beyond early childhood and into adolescence and young adulthood.

In Study 1, I found that two broad but profound categories of ECA (experiences surrounding previous caregiving institutionalization or placement in domestic foster care) are associated with elevated parent-reported sensory processing challenges (and specifically SOR) in children and adolescents. In addition, I reported findings suggesting that these sensory processing challenges may contribute to elevated internalizing and externalizing symptoms in the same sample, suggesting enduring sensory symptoms in adolescence may be relevant for mental health during this period.

In Study 2, I collected self-reported questionnaire measures in multiple population samples of adults to further explore connections between ECA, sensory processing challenges, and mental health. Again, I reported that the same sensory processing challenges documented following rare severe forms of ECA (e.g., PI caregiving, removal from the home and placement in foster care) are also present in individuals with experiences of more common forms of early adversity (e.g. neglect, abuse). In addition, I confirmed that adversity-linked sensory challenges (and their accompanying associations with internalizing symptoms) persist into adulthood, report on preliminary relationships between various facets of sensory processing within individuals, and report that affective and regulatory processes may be particularly important to consider in the context of enduring sensory symptoms.

Lastly, Study 3 examined neural correlates of SOR in adolescents with histories of PI caregiving, with the goal of providing further insights into the mechanistic pathways by which early adversity may contribute to sensory symptoms across development. I applied multivariate pattern expression analyses to assess overlap between individual participant's whole-brain neural responses to aversive auditory stimulation and publicly accessible meta-analytic neural "signatures" of 1) auditory perception, 2) affective sensory responses, and 3) affective and nonaffective regulation, finding elevated expression of non-affective regulation patterns in PI youth. In addition, I report elevated recruitment of prefrontal regulatory regions in PI youth, and elevated aversive auditory stimulation-related functional connectivity between auditory cortex and rostrolateral prefrontal cortex. While correlational, these findings motivate further work investigating the relationship between self-regulation and sensory processing challenges following ECA.

This dissertation leverages both self-reported symptoms and neuroimaging techniques to probe candidate mechanisms for the development of sensory processing challenges following ECA, providing preliminary evidence for the importance of affective and regulatory experiences in the development and persistence of SOR. Strikingly, we find very similar relationships between ECA and sensory processing challenges across four samples with very different ECA histories (PI, AFC, MTurk population sample, UCLA SONA sample). In addition, this research

suggests that sensory processing challenges may be relevant for ongoing mental health following experiences of ECA. Taken together, these studies stand to inform developmental models of ECA, and to highlight potential opportunities for continuing research on clinical intervention for an underexplored facet of development that may be critical to long-term health.

Dissociating components of sensory processing challenges

My findings align with field-wide recommendations to examine various facets of sensory experiences (e.g. perceptual, affective sensory, and regulatory) in tandem, using multimodal techniques. Using questionnaires, my findings from Study 2 demonstrate unique patterns in sensory processing challenges that differ depending on the type of sensory experiences measured (sensory perception vs. affective sensory responses). Given the nascency of the field, research in clinical (e.g. ASD) and typically developing populations has often focused on single aspects relevant to sensory processing in isolation, using inconsistent terminology and distinct measurement approaches (He et al., 2023). For example, perception-focused research might employ behavioral assessments of sensory detection, while clinically-oriented occupational therapy research might investigate affective experiences of sensory stimuli in the classroom. Findings from both of these classes of studies are often assessed without characterization of the other categories of symptoms and reported as pertaining to sensory processing, as a result of field-wide discrepancies in terminology that have been cited as a major limitation in ongoing sensory research in ASD (Ben-Sasson et al., 2019; Glod et al., 2015; Gunderson et al., 2023; He et al., 2023; Lane, 2020). In conjunction with other recent field recommendations, Study 2 points to the need for parallel measurement of various aspects of sensory function across multiple levels, and for work towards field-wide standards in terminology and reporting.
Towards this goal, ongoing work may benefit from complementary and interdisciplinary behavioral, physiological, and neuroimaging paradigms to allow for parallel assessments of symptoms that can be applied in varied populations. Parent-reported questionnaires focused on affective sensory experiences (e.g. the Short Sensory Profile used in Study 1) are utilized in much research on the development of sensory challenges. While these have been well-validated in varied populations, many have significant limitations regarding construct validity, reliability, solely measuring subjective affective experience, and not relying on individual's reports of their own experiences (Gunderson et al., 2023; Ujiie & Wakabayashi, 2015). Incorporation of selfreported measures of symptoms like those I applied in Study 2 may provide additional benefit in developmental samples, particularly given recent research on the importance of self-reported, rather than externally reported, exposures to adversity on mental health (Francis et al., 2023). Characterization of sensory processing challenges would also benefit from the incorporation of combined approaches, including behavioral assessments and physiological and neuroimaging paradigms, to allow for the evaluation of mechanistic underpinnings of symptoms in tandem (see Jung et al., 2021 for an example).

Tailored measures may provide further benefit by allowing for characterization of unique patterns of sensory processing challenges in individual populations, including in adolescents and following ECA. Recent evidence suggests that despite shared prevalence of sensory processing challenges across clinical groups, different clinical populations may in fact experience distinct profiles of sensory symptoms (He et al., 2021; van den Boogert et al., 2022). Incorporating population-tailored assessments may allow for further clarification of these patterns. For example, although recent work has attempted to assess sensory processing challenges in joint

questionnaire, behavioral and observational measures (e.g. SP3-D Assessment), many of these are designed primarily for younger children, still rely heavily on parent-reported symptoms have yet to be fully validated in large scale samples or in older children or adolescents (Passarello et al., 2022). Likewise, neuroimaging research may be by nature challenging to individuals with high sensory processing challenges. fMRI or EEG measures (which can be overwhelming due to scanner noise and application-related moisture, respectively) might be adapted to utilize fNIRS paradigms, and promising virtual-reality-related sensory assessments may consider vestibular effects on sensitive populations. Together, tailored multimodal assessments may inform understanding of sensory experiences, and provide a critical foundation for ongoing mechanistic and intervention-focused work.

Longitudinal and lifespan approaches to investigating sensory processing

In Study 1 and Study 2, I documented an enduring association between ECA and sensory processing challenges into adolescence and young adulthood, and in Study 2 I reported high incidence of sensory processing challenges in two samples of young adults. These findings motivate characterizing trajectories of sensory symptoms in large scale longitudinal studies in ECA-focused, clinical, and generalized contexts, respectively. In addition to providing causal information about the development of sensory processing challenges following prevalent ECA, such work could allow for ongoing evaluation of joint trajectories of development of sensory symptoms and mental health symptoms that may support decoupling of co-morbid conditions. This work is particularly important given increasing evidence that sensory processing challenges may be a transdiagnostic phenotype associated with psychopathology (Harrison et al., 2019; van den Boogert et al., 2022). Furthermore, although my results from Study 2 provide evidence that

specific sensory processing challenges (SOR and SUR) may co-occur within a population sample of adults (mirroring similar findings in children; Y.-J. Chen et al., 2022; van den Boogert et al., 2022), this has not been fully explored in long-scale longitudinal work, preventing full characterization of profiles of sensory symptoms within individuals. This may be particularly important to consider given reported changes in profiles of sensory symptoms in clinical populations over time, and recent findings that rate of change in severity of sensory symptoms is predictive of later outcomes (Y.-J. Chen et al., 2022; van den Boogert et al., 2022).

Lastly, my findings point to the importance of characterizing sensory processing challenges in otherwise typically developing populations, and particularly in adolescents and young adults from representative samples. In Study 2 I report a large distribution of sensory processing challenges in two population samples of young adults, with potent links to various outcomes associated with well-being across domains. A limited number of studies have characterized trajectories of sensory processing challenges across early childhood, often with the primary goal of predicting (Carpenter et al., 2019a; Y.-J. Chen et al., 2022; C. Van Hulle et al., 2015). While sensory processing challenges are common in infancy (~20% prevalence), some studies have reported that most symptoms resolve before school age (Van Hulle et al., 2015). However, a recent examination in over 11,000 9-12-year-old youth in the Adolescent Brain Cognitive Development estimated SOR prevalence at 18% (Schwarzlose et al., 2023). While this study used a single-item index of sensory processing, leaving room for further confirmation, this finding points to the importance of characterizing sensory processing in typical adolescent development. However, although one study (Van Hulle et al., 2018) has tracked possible heritability of sensory processing challenges in adult parents and their adolescent children, to my

knowledge no work to date has directly reported trajectories of sensory processing challenges in adolescence in otherwise typically developing youth. Given the prevalence of sensory processing challenges reported in my Study 2 sample and the ABCD cohort, and the myriad impacts of sensory processing challenges on adaptive functioning and potential relevance to later social, psychological, academic, and other outcomes, understanding these pathways should be prioritized in future large scale neurodevelopmental studies of typical development in the general population (e.g. ongoing waves of the ABCD study and other large projects). In the meantime, publication or open access release of data from the many studies examining typically developing adolescents and young adults as comparisons for clinical samples may provide an important starting point for the characterization of variability in adolescent sensory development. With this in mind, I plan to share the data used in Study 2 on the Open Science Framework.

Future Directions

While cumulatively, my studies suggest that the early environment modulates the development of sensory processing, future work is needed to explicitly characterize these connections.

Implications for Empirical Research on Evidence-Based Interventions

This research points to the need for ongoing research developing and evaluating evidence-based interventions addressing sensory symptoms (both general and adversity-linked) in childhood, but also in adolescence and adulthood. The majority of studies exploring sensorybased treatments following ECA have been conducted in small, specialized samples and without thorough examination of efficacy or treatment outcomes (Fraser et al., 2017b; Joseph et al.,

2021; McGreevy & Boland, 2020). In addition, much of this work (including in ECA-linked contexts) has focused on addressing sensory symptoms in early and middle childhood, in the hopes of providing treatment during a tractable period and preventing ongoing symptoms across the lifespan. However, the research presented in Study 1 and Study 2 of this dissertation suggests that sensory processing challenges are both prevalent and long lasting following early caregiving adversity, extending into adolescence and young adulthood. Recent developments in experimental options for sensory-based treatments (e.g. neuromodulation, virtual reality, etc.), and promising evidence from increasingly popular sensory-related interventions for other mental health conditions like the Self-Soothing with the Five Senses skills taught in DBT, traumainformed yoga, and both in person and online EMDR for PTSD indicate the potential clinical value of further research into targeted intervention development (Becker & Zayfert, 2001; Lenferink et al., 2020; Passarello et al., 2022; Perlini et al., 2020; Stoller et al., 2012). Conversely, the relationship between affective sensory responses, self-regulation, and mental health I report in Study 2 (in tandem with the elevated recruitment of prefrontal regulatory regions I observe during sensory stimulation in PI youth in Study 3) points to the importance of integrating affective and self-regulation approaches in sensory-based therapies. Although these are already the targets of some existing sensory interventions, our findings point to possible value in promoting collaborative intervention research with joint teams of psychologists and occupational therapists. Given both the prevalence and seeming functional impact of these sensory processing challenges documented in Study 2, outcome-focused research into evidencebased sensory interventions for adolescent and adult symptoms may be a promising (and scalable) avenue for promoting well-being (Becker & Zayfert, 2001; Lenferink et al., 2020; Passarello et al., 2022; Perlini et al., 2020; Stoller et al., 2012).

98

As a result, sensory-based interventions may also present prime opportunities to support youth with histories of ECA into adolescence, young adulthood and beyond. Emerging theories based on recent empirical findings suggest that the onset of puberty may provide unique opportunities for recalibration of biological systems altered by early adversity, particularly if an individual is in an improved environment at the time of pubertal onset (Gunnar et al., 2019; Méndez Leal & Silvers, 2021). Although clinical work in youth with histories of ECA often focuses on early intervention, these findings suggest that adolescence may be an optimal time for introduction of novel therapies.

Additionally, independent of ECA, sensory processing challenges may simultaneously be particularly impactful in adolescence and young adulthood, and also particularly amenable to intervention during this period. Adolescence provides novel opportunities for exploration and learning, as well as the consistent introduction of new professional, social, and romantic contexts where sensory processing difficulties might be particularly challenging. My findings from Study 1 and Study 2 are consistent with this view – regardless of developmental ordering, individuals experiencing heightened sensory processing challenges in our samples report distress across other domains, and in our qualitative data report that their sensory symptoms are profoundly affecting their daily experiences. However, this period may also provide a unique opportunity for improvement of symptoms in otherwise typically developing youth, as this period is marked by rapid development of effective affective and non-affective self-regulation ability (shown in Study 2 to be associated with reduced sensory processing challenges; (Karbach & Unger, 2014; Luna, 2009; Silvers et al., 2012). While ongoing work should explicitly evaluate the impact of these sensory symptoms on social and learning behaviors in youth of this age, this is indicative

of the importance and possible opportunity for supporting wellbeing through sensory intervention.

Conclusion

This dissertation examines links between early caregiving adversity and sensory processing challenges in adolescence and young adulthood, employing parent-reported, selfreported, and neuroimaging measures. In addition, I examine multiple features of sensory development following adversity and provide an exploration of candidate mechanisms underlying the development of sensory symptoms. The work above demonstrates that both rare and prevalent forms of severe caregiving adversity increase risk for sensory processing challenges that endure beyond early childhood and into adolescence and young adulthood, and that these symptoms may be related to affective and self-regulation processes known to be altered by ECA. Furthermore, these studies show consistent links between ECA-associated sensory processing challenges and concurrent internalizing symptoms, suggesting sensory symptoms may be important to consider in supporting well-being following ECA. Taken together, these studies stand to inform developmental models of ECA, and to highlight potential opportunities for ongoing research on clinical intervention for an underexplored facet of development that may be critical to long-term health.

100

Study 1 Supplement

Additional Participant Information

Recruitment

Youth adopted from domestic foster care (AFC) were recruited from two adoptionrelated programs to participate in a study examining neurobiological and behavioral mechanisms underlying sensory processing challenges following ECA. Importantly, youth were not recruited based on the presence of sensory processing challenges. Study staff contacted AFC participants and their families by providing flyers to clinicians working with adopted children, presenting to adoptive families and clinicians, and posting on social media outlets. Non-adopted comparison participants in this sample were recruited through flyers posted throughout the community (schools, university campus, and around the metropolitan area), on social media, and from an active waiting list of families interested in participating in research. These comparison participants were initially recruited for a study examining sensory processing challenges in youth with autism spectrum disorders. Given that autism is most prevalent in individuals assigned male at birth, youth assigned male at birth were oversampled in this comparison group. Participants were between the ages of 8-17 years and had no known history of early caregiving adversity.

Internationally adopted previously institutionalized (PI) youth were originally recruited from adoption-related programs. The data used in this analysis was collected from PI and nonadopted PI-comparison youth as part of the fourth wave of an ongoing longitudinal study. These participants were originally recruited through a combination of flyers and word of mouth in various targeted communities, including international adoption family networks, online adoption family support groups, and adoption agencies. In addition, participants were recruited from local

101

early childhood education centers, the campus, local public posting areas in the metropolitan area, and varied community institutions, including schools, religious organizations, community centers, professional offices, after-school facilities, community gatherings, and activity fairs.

The two comparison groups (from the AFC and PI studies, respectively) were equivalent on all demographic variables except for sex assigned at birth (in part because of over-recruitment of males in the AFC comparison sample) and were therefore combined to yield one joint comparison group prior to all analyses.

Pre-Adoption Experiences

Overall, AFC youth in this sample were adopted much later than PI youth and had a larger number of placements. For example, AFC youth had an average of 7 placements prior to arrival in their final adoptive home. In contrast, to our knowledge 86% of PI participants were placed in an institution within the first 18 months of life $($ > 50% within the first month) and adopted directly from the institution. Nearly all PI participants had only 1-2 placements (including the institution) prior to final adoption.

AFC:

We do not have information about why AFC participants were removed from their initial homes. However, a subset $(N = 25)$ of AFC participants had their parents report additional detail about experiences of ECA prior to adoption, while 21 reported on the number of foster care placements. It should be noted that parents often do not have full information on their adopted

children, so the below statistics should be considered examples of the types of adversity commonly experienced by this population but the percentages are likely not representative. For example, of the 65% who did not report prenatal exposure to substances, it does not mean these children were *not* exposed, but just that the adoptive parents lack this information:

Type of ECA Experience	$N (Total = 25)$	$%$ (of subset)
Neglect	16	70
Prenatal Exposure to Substances	8	35
Physical Abuse	4	17
Witnessing Violence in the Home	6	26
Sexual Abuse	13	57
Other	Experienced homelessness $=$ 3 Malnutrition $= 1$ Failed finalized adoption $= 1$	22
	M(SD)	Range
Mean Number of ECA Experienced	2.09(1.44)	$1 - 5$
Mean Number of Foster Care Placements	1.52(1.72)	$0 - 7$

Supplemental Table 1. Parent reported pre-adoption ECA for a subset of AFC youth

Abbreviations: Early Caregiving Adversity (ECA)

PI:

The countries that the PI youth in this study were adopted from are listed in the table below for all participants. In addition, 91.2% (N = 31) of parents reported having visited the institutions their children were living in, and provided their subjective impressions of the building quality, facility cleanliness, quantity of caregiving, and quality of caregiving in the institutions also reported below. In general, most parents reported moderate to high building

quality and facility cleanliness. Average reports of quantity and quality of caregiving were

middling, with a high degree of variability. Lastly, 62% (N = 21) of PI adoptive parents said they

were told their child had a special relationship with a caregiver prior to adoption.

Supplemental Table 2. Parent reported caregiving history for PI youth

Country Adopted from: Azerbaijan China Kazakhstan Russia South Korea	1 12 7 13 1
Parental Impressions of Institution (1-10): Building Quality ($1 = poor$, $10 = nice$) <i>Facility Cleanliness</i> ($1 = poor$, $10 = excellent$) Quantity of Caregiving ($1 =$ too few caregivers, $10 =$ many caregivers) Quality of Caregiving (1 = very poor, 10 = very good)	$6.73(2.72; 1-10)$ $8.05(1.63; 4.5-10)$ $5.98(3.09;1-10)$ $6.50(3.11, 1-10)$
Parent Reported Placement History Caregiving Institution Only Placed in institution 0-1 months after birth, adopted from institution Placed in institution 2-6 months after birth, adopted from institution Placed in institution 7-18 months after birth, adopted from institution Placed in institution >18 months after birth, adopted from institution	18 $\overline{4}$ $\overline{4}$ 3
Caregiving Institution + Other Out of Home Placements Placed in institution, 6-9 months after birth, after extended hospital stay Adopted from institution Placed in institution $<$ 6 months after birth, in foster care for some period*	$\overline{2}$ 3

***** one of these children also had an extended hospital stay (age 0-3 months)

Note: While all parents reported country of origin and a brief placement history ($N = 34$), parental impressions of the institution were available for 31/34 participants (91.2%)

Additional Information Regarding Study Measures

Measure Selection

We included analysis of both the Short Sensory Profile and the SP3D checklist to provide

a more complete assessment of links between ECA and sensory development. While there are

some similarities between "sensitivity" items on the SSP and SOR items on the SP3D checklist,

they assess these symptoms using different (but complementary) approaches.

The SSP provides a general measure of sensory issues across multiple aspects of functioning, including sensory seeking, sensory under-responsivity, and difficulty filtering sensory information, as well as SOR. In addition, the SSP has been extensively validated and is the measure most commonly used in developmental research on sensory processing challenges (including work on early adversity). This measure therefore provides a helpful point of comparison with other relevant work. Importantly, the SSP focuses primarily on affective expressions of responses to sensory stimuli, asking parents to report on patterns of behavior and including both physical and social stimuli (e.g., grooming, being touched, responding to name).

We administered the SP3D checklist as a more tailored estimate of SOR. I were most interested in SOR a priori because I felt SOR was most likely to be impacted in youth with histories of ECA given the neurodevelopmental mechanisms I believe underlie the emergence of sensory differences in this population, and because SOR symptoms have been most clearly linked to mental health outcomes. I therefore selected the SP3D because it was developed with the primary goal of providing more specific assessment of a child's response to their regular sensory environment, with an explicit focus on assessing SOR from the perspective of multiple sensory modalities. As a result, it was designed in a checklist format, with parents asked to what extent their children were bothered by commonly encountered stimuli.

Supplemental Analyses

Descriptions of supplemental analyses conducted as part of this study are included below. Unless otherwise noted, these analyses were included in the original pre-registration.

105

Correspondence Between Measures of Sensory Over-Responsivity

To examine consistency across measures, an SSP SOR composite score (intended as a parallel to the SP3D SOR measure) was calculated using the Tactile Sensitivity and Visual/Auditory Sensitivity subscales. In addition, to examine whether observed differences in general processing challenges on the SSP were solely the result of overlap between SOR items across measures, I also calculated an SSP total score that omitted items from the two SSP subscales with overlap with the SP3D (the SSP Tactile Sensitivity and Visual/Auditory Sensitivity subscales). Neither of these composite scores were used in any primary analyses.

We conducted a series of linear regressions to examine concordance between different measures of sensory over-responsivity (the SSP and SP3D) across sensory modalities. Specifically, I compared a composite measure of the SSP Tactile and Visual/Auditory sensitivity scales to the SP3D total score, a measure of tactile, visual, and auditory SOR. In addition, I compared symptoms reported on the SSP and SP3D subscales for each of these sensory modalities. As expected, I found high correspondence between all SP3D measures and analogous SSP scores, as shown in *Supplemental Table 1.*

An unregistered exploratory analysis of the SSP that excluded the two subscales with overlap with the SP3D (the SSP tactile sensitivity and visual/auditory sensitivity subscales) revealed very similar results to the SSP findings reported in the main text (although with decreased effect sizes). There were still group differences between the AFC and PI groups on total non-SOR SSP score $(F(3,71) = 9.71 p = .003)$, so I again analyzed the two ECA groups separately. Consistent with this finding, youth in both the PI ($a_{PI,SSP} = -7.57$, *SE* = 2.22, *t* =

106

 $-3.42, 95\%$ CI [$-11.95, -3.20$], $p < .001$) and AFC ($a_{AFC|SSP} = -21.29$, $SE = 2.11$, $t = -10.08$,

95% CI $[-25.45, -17.12]$, $p < .001$) groups had significantly heightened general sensory processing challenges on the SSP (lower scores), relative to non-adopted comparison youth. In a model that examined general sensory processing challenges as a link between ECA and internalizing symptoms, I again found significant indirect effects through non-SOR general sensory processing challenges for both PI ($ab_{PI,SSP,INT} = 1.51, 95\%$ CI [0.57-2.81]) and AFC participants ($ab_{AFC \; SSP \; INT}$ = 4.24, 95% CI [2.26-6.53]), relative to comparison youth. Similarly, I found a significant indirect effect of ECA on externalizing symptoms through non-SOR sensory processing challenges (PI: $ab_{PI, SSP, EXT} = 1.73, 95\%$ CI [0.62-3.31]; AFC: $ab_{AFC_SSP_EXT}$ = 4.86, 95% CI [2.48-7.78]).

Note: Concordance was assessed in the whole sample $(N = 183)$. The SSP sensitivity score was derived using the Tactile Sensitivity and Visual/Auditory Sensitivity subscales to create a comparable score to the SP3D total.

Abbreviations: Short Sensory Profile (SSP); Sensory Processing 3-Dimensions Checklist (SP3D); Sensory Over-Responsivity (SOR)

These findings suggest that the general sensory processing challenges reported in the main text are not purely driven by SOR items.

Sensory Measure Subscales by Group

Sensory measure subscale score distributions for each group are documented in

Supplemental Table 2 and *Supplemental Table 3.*

Supplemental Table 2. SP3D subscale scores for total, auditory, visual, and tactile domains in comparison, PI, and AFC participants.

ANOVA was used to explore group differences in subscale scores, and associated p values are reported in the table. Pairwise group differences were then probed using t-tests:

^a Denotes that the PI group has higher scores (higher SOR) than the Comparison group.

 b Denotes that the AFC group has higher scores (higher SOR) than the Comparison group.

Abbreviations: Sensory Processing 3-Dimensions Checklist (SP3D); Sensory Over-Responsivity (SOR); Previously Institutionalized (PI); Adopted from Foster Care (AFC)

Supplemental Table 3. Mean SSP subscale scores for total, tactile sensitivity, auditory filtering, movement sensitivity, visual/auditory sensitivity, taste sensitivity, sensory under-responsivity, and low energy/weakness domains among comparison, PI, and AFC participants.

ANOVA was used to explore group differences in subscale scores, and associated p values are reported in the table. Pairwise group differences were then probed using t-tests:

^a Denotes that the PI group has lower scores (greater general sensory processing challenges) than the Comparison group

 \overline{b} Denotes that the AFC group has lower scores (greater general sensory processing challenges) than the Comparison group, suggesting more sensory symptoms.

^c Denotes that AFC group has lower scores (greater general sensory processing challenges) than the PI group *Abbreviations:* Short Sensory Profile (SSP); Sensory Over-Responsivity (SOR); Previously Institutionalized (PI); Adopted from Foster Care (AFC)

SSP Categories by Group

Supplemental Table 4. Sample SSP Clinical Categories by Group

Note: Probable Sensory Processing Challenges and Definite Sensory Processing Challenges categories correspond to the Probable and Definite Difference categories from the SSP *Abbreviations:* Early Caregiving Adversity (ECA); Short Sensory Profile (SSP); Previously Institutionalized (PI); Adopted from Foster Care (AFC)

Descriptive Statistics for CBCL T-Scores by Group

Descriptive statistics for CBCL T-scores are provided in *[Supplemental Table 5](#page-124-0)* and visualized in

[Supplemental Figure 1](#page-125-0) and *[Supplemental Figure 2.](#page-125-1)*

Supplemental Table 5. Sample Clinical Descriptive Statistics

Note: CBCL internalizing T-scores in this sample may underestimate symptoms, because raw scores were calculated without question 91. Internalizing and externalizing T-scores above 70 are considered to be in the clinical range; scores between 65 and 70 are considered to be in the borderline clinical range. *Abbreviations:* Child Behavior Checklist (CBCL); Previously Institutionalized (PI); Adopted from Foster Care (AFC)

Supplemental Figure 1. Visual representation of CBCL internalizing and externalizing scores for comparison, PI, and AFC participants.

Supplemental Figure 2. Visual representation of CBCL internalizing and externalizing scores with clinical cutoffs for comparison, PI, and AFC participants. T-scores above 70 are considered to be in the clinical range; scores between 65 and 70 are considered to be in the borderline clinical range.

Abbreviations: Previously Institutionalized (PI); Adopted from Foster Care (AFC)

Early Caregiving Adversity, Age, and Sensory Processing Challenges

Based on previous findings, I predicted that sensory processing challenges would decrease with age. With this in mind, I pre-registered an analysis of age-SOR associations within the larger ECA group (AFC +PI). I chose not to conduct a moderation analysis given I predicted

the same (negative) relationship between age and symptoms in the two ECA groups. Instead, I performed a planned linear regression examining the relationship between age and SOR symptoms within the overall ECA group (PI and AFC). Given age differences between the AFC and PI groups in the updated sample, I performed a post-hoc linear regression within each of the individual ECA groups.

SOR symptoms in PI and AFC youth were not correlated with age, covarying for sex assigned at birth ($B_{Age} = -0.68$, $t(70) = -0.93$, 95% CI [-2.14-0.78], $p = .36$). Post-hoc exploratory follow-up analyses showed no association between age and SOR in either the PI $(B_{AgePI} = 0.23, t(33) = 0.15, 95\% \text{ CI}$ [-2.92-3.37], $p = .89$) or AFC groups $(B_{AgeAFC} = -1.54,$ *t*(36) = -1.42,95% CI [-3.74 -0.67], *p =* .17).

Post-Hoc Exclusion of Outliers and Reanalysis

We made the decision when pre-registering our exclusion criteria to not exclude outliers, in order to preserve statistical power in a relatively small sample for a hard to recruit population that has documented high inter-individual variability (Tottenham, 2012). All primary analyses were conducted using bootstrap resampling to provide greater confidence in our estimate of the examined effect sizes.

To provide additional confidence that our findings were not the result of influential outliers, all SP3D SOR analyses were re-run (post-hoc), excluding participants with SP3D SOR total scores greater than (or less than) 3 SDs from the overall sample mean of 49.23 (SD = 8.83).

The remaining sample ($N = 145$) included 33 AFC participants (4 excluded), 32 PI participants (2 excluded) and 112 comparison participants (0 excluded).

All SP3D SOR analyses remained significant in the direction of the original results. Specifically:

- As before, I found no differences between ECA groups on SP3D scores $(F(3, 64) =$ 1.95, $p = 0.168$). Again, the AFC group had significantly more sensory processing challenges on the SSP than the PI group ($F(3, 64) = 10.5$, $p = .002$).

- Youth in the PI $(a_{PI_SP3D} = 7.87, SE = 1.97, t = 4.00, p < .001, 95\% \text{ CI } [3.97-11.75])$ and AFC ($a_{AFC S P3D} = 4.82$, $SE = 1.90$, $t = 2.53$, $p = .01$, 95% CI [1.07-8.58]) groups had higher SP3D scores (higher SOR) than the non-adopted comparison group, covarying for age and sex

- Covarying for age and sex assigned at birth, I found significant indirect effects of ECA on elevated internalizing symptoms through SOR, for both PI ($ab_{PI, SP3D, INT} = 1.38$, $SE =$ 0.55, 95% CI [0.37- 2.51]) and AFC $(ab_{AFC\ SP3D\ INT} = 0.85, SE = 0.46, 95\% \ CI [0.08-1.86])$ youth.

- I found significant indirect effects of PI and AFC status on externalizing symptoms through SOR (PI: $ab_{PI \text{ } SP3D \text{ } EXT}$ = 1.16, SE = 0.54, 95% CI [0.29, 2.41]; AFC: $ab_{AFC \ SP3D \ EXT} = 0.71$, $SE = 0.43,95\% \ CI [0.07, 1.72]$.

Likewise, all SSP analyses were re-run (post-hoc), excluding participants with SSP total scores less than (or greater than) 3 SDs from the overall sample mean of 170.92 (SD = 19.58).

The remaining sample ($N = 180$) included 34 AFC participants (3 excluded), 34 PI participants (0 excluded) and 112 comparison participants (0 excluded).

Specifically:

- As before, I found no differences between ECA groups on SP3D scores (*F*(3,67) = 1.08, $p = .30$). Again, the AFC group had significantly more sensory processing challenges on the SSP than the PI group ($F(3,67) = 9.69$, $p = .003$).

- Youth in the PI $(a_{PI \ SP3D} = 10.12, SE = 2.36, t = 4.29, p < .001,95\% \text{ CI } [5.47-14.78])$ and AFC ($a_{AFC_SP3D} = 7.32$, $SE = 2.31$, $t = 3.17$, $p = .002$, 95% CI [2.77-11.87]) groups had higher SP3D scores (higher SOR) than the non-adopted comparison group, covarying for age and sex. Consistent with this finding, youth in both the PI ($a_{PI, SSP} = -10.63$, *SE* = 2.93, *t* = -3.63, 95% CI [-16.40, -4.85], $p < .001$) and AFC ($a_{AFC|SSP} = -27.94$, $SE = 2.86$, $t = -9.77$, 95% CI [-33.59, -22.3 , $p < .001$) groups had significantly heightened general sensory processing challenges on the SSP (lower scores), relative to non-adopted comparison youth.

- Covarying for age and sex assigned at birth, I found significant indirect effects of ECA on elevated internalizing and externalizing symptoms through SOR, for both PI $(ab_{PI_SP3D_INT} = 1.56$, $SE = 0.64$, 95% CI [0.38- 2.93]; $ab_{PI_SP3D_EXT} = 1.31$, $SE = 0.56$, 95% CI [0.31- 2.52) and AFC $(ab_{AFC_{SP3D_{INT}}} = 1.13, SE = 0.54, 95\% \text{ CI } [0.20-2.32];$ $ab_{AFC\ SP3D\ EXT} = 0.95, SE = 0.51, 95\% \ CI [0.14-2.13])$ youth.

- Covarying for age and sex assigned at birth, I found significant indirect effects of ECA on elevated internalizing and externalizing symptoms through general sensory processing challenges, for both PI $(ab_{PI \; SSP \; INT} = 1.80$, $SE = 0.70$, 95% CI [0.65- 3.36]; $ab_{PI_SSP_EXT}$ = 2.11, SE = 0.82, 95% CI [0.77- 3.94) and AFC $(ab_{AFC_{SSP_{INT}}}$ = 4.74, SE = 1.24, 95% CI [2.59-7.41]; $ab_{AFCSSPEXT} = 5.55$, $SE = 1.56$, 95% CI [2.95-9.00]) youth.

Post-Hoc Reanalysis in an Age-Matched Sample

To provide additional confidence that our findings were not the result of age differences between groups, all analyses were re-run (post-hoc) using only participants between ages 11 and 18. This age range ensured that the resultant sample had no differences between ages across groups, while maximizing sample size.

The remaining sample ($N = 144$) included 20 AFC participants (17 excluded), 34 PI participants (0 excluded) and 90 comparison participants (22 excluded). Our findings are summarized below:

Differences in Sensory Processing Challenges Between ECA Groups:

As before, I found no differences between ECA groups on SP3D scores $(F(3,53) = 1.93, p = .17)$. However, the AFC group had significantly more sensory processing challenges on the SSP than the PI group $(F(3,53) = 8.52, p = .005)$. The AFC and PI groups were therefore examined separately in all analyses, with ECA dummy coded and non-adopted comparison youth as the reference group.

Sensory Processing Challenges Following ECA:

- As before, age-matched PI youth had higher SOR (higher SP3D scores; $a_{PI,SP3D} =$ 10.06, *SE =* 2.27, *t* = 4.44, 95% CI [5.58 -14.54], *p* < .001) and heightened general sensory processing challenges (lower SSP scores; $a_{PI,SSP} = -10.79$, $SE = 2.99$, $t = -3.61$, 95% CI [-16.70, -4.88], $p < .001$ than the non-adopted comparison group, covarying for age and sex.

- As before, age-matched AFC youth had heightened general sensory processing

challenges (lower SSP scores; $a_{AFC|SSP} = -27.31$, $SE = 3.57$, $t = -7.65$, 95% CI [-34.37, -20.25], $p < .001$) than the non-adopted comparison group, covarying for age and sex. However, although the direction of the effect remained the same, the age-matched AFC sample of AFC youth no longer had significantly elevated SOR (higher SP3D scores; $a_{AFC SPSD} = 4.84$, *SE* = 2.71, *t* = 1.79, 95% CI $[-0.52 - 10.19]$, $p = .08$) than the non-adopted comparison group, covarying for age and sex.

Psychological Symptomatology following ECA:

As in the original analysis, there were significant total effects of ECA on both internalizing and externalizing symptoms. Both PI ($c_{PI\;INT}$ = 6.28, *SE* = 1.3, *t* = 44.84, 95% CI [3.71, 8.84], $p < .001$) and AFC ($c_{AFCINT} = 8.34$, $SE = 1.57$, $t = 5.23$, 95% CI [5.22 – 11.46], *p <* .001) youth had higher internalizing symptom scores than comparison youth, covarying for age and sex. Similarly, both PI ($c_{PL_EXT} = 4.30$, $SE = 0.91$, $t = 4.75$, 95% CI [2.51, 6.1], $p <$.001) and AFC ($c_{AFC_{EXT}}$ = 9.99, *SE* = 1.32, t = 7.55, 95% CI [7.34 – 12.62], p < .001) youth had higher externalizing symptoms than comparison youth, covarying for age and sex.

Sensory Processing Challenges and Links to Psychological Symptomatology:

- Age-matched PI youth: covarying for age and sex assigned at birth, I again found significant indirect effects of previous institutionalization on elevated internalizing and externalizing symptoms through SOR $(ab_{PI, SP3D, INT} = 1.76, 95\% \text{ CI} [0.56-3.19];$ $ab_{PI_SP3D_EXT}$ = 1.06, 95% CI [0.14 -2.09]) and through general processing challenges

 $(ab_{PI_SSP_INT} = 1.90, 95\% \text{ CI} [0.7-3.63]; ab_{PI_SSP_EXT} = 1.45, 95\% \text{ CI} [0.51-2.85]),$ relative to comparison youth.

- Age-matched AFC youth: covarying for age and sex assigned at birth, I again found significant indirect effects of AFC status on elevated internalizing and externalizing symptoms through general processing challenges (ab_{AFC} SSP INT = 4.82, 95% CI [2.45-8.01]; $ab_{AFC_SSP_EXT}$ = 3.68, 95% CI [1.62-6.37]), but not SOR $(ab_{AFC_{SP3D_{INT}}}$ = 0.85, 95% CI [-0.12-2.1]; $ab_{AFC\ SP3D\ EXT} = 0.51, 95\% \ CI$ [-0.08 -1.51]), relative to comparison youth.

Early Caregiving Adversity, Age, and Sensory Processing Challenges within the age matched sample:

SOR symptoms in PI and AFC youth were not correlated with age, covarying for sex assigned at birth ($B_{Age} = 0.62$, $t(53) = -0.60$, 95% CI [-1.44-2.67], $p = .55$). Unregistered exploratory follow-up analyses showed no association between age and SOR in either the PI $(B_{Age_PI} = 0.23, t(33) = 0.15, 95\% \text{ CI}$ [-2.92-3.37], $p = .89$) or AFC groups $(B_{Age_AFC} = 1.68,$ *t*(19) = -0.12, 95% CI [-2.72 – 3.06], *p* = .90).

Examination of Sex Differences Between Groups

Individuals assigned female at birth are often over-represented in internationally adopted previously institutionalized samples as a result of varied political and social factors that impact both circumstances leading to placement in an institution and the process of international adoption. Consistent with this, individuals assigned female at birth are disproportionately represented in our PI sample (~71%). The comparison and AFC groups have approximately even proportions of individuals assigned male and individuals assigned female at birth.

All analyses covaried for assigned sex at birth. In the primary models (which included group membership), sex was not significantly associated with SOR symptoms in $(B_{Female\;SOR} = -$ 1.09, $t = -0.58$, $p = .56$, CI = [$-4.85 - 2.65$]). Sex was significantly associated with SSP scores in the primary models (B_{Female_SSP} = 4.79, $t = -0.86$, $p = .39$, CI = [0.25 – 9.32]), indicating that individuals assigned male at birth had more elevated sensory processing challenges than individuals assigned female at birth. Given this and that limited data suggest sensory symptoms are more common in males than females in youth with and without experiences of ECA (J. Wilbarger et al., 2010), if anything the over-representation of females in the PI group may be resulting in underestimation of the impact of PI experiences on sensory symptoms.

Relationship between SSP Auditory Filtering Score and ADHD Symptoms

In addition to our focal analyses of the CBCL internalizing and externalizing subscale, I calculated ADHD subscale scores for all participants as part of our assessment of the relationship between measures. While the SSP is the most commonly used questionnaire index of sensory processing challenges in youth, critics of the measure argue that it may conflate sensory processing issues with symptoms of ADHD. In order to parse these effects in the context of ECA, I conducted an exploratory multiple regression. ADHD symptoms were significantly associated with more atypical SSP auditory filtering (β = -0.50, $t(182)$ = -8.70, $p < .001$).

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