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The Impact of Social Disadvantage on Autonomic Physiology of Latinx Adolescents: The Role of Environmental Risks

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Abstract

The experience of poverty embodies complex, multidimensional stressors that may adversely affect physiological and psychological domains of functioning. Compounded by racial/ethnic discrimination, the financial aspect of family poverty typically coincides with additional social and physical environmental risks such as pollution exposure, housing burden, elevated neighborhood unemployment, and lower neighborhood education levels. In this study, we investigated the associations of multidimensional social disadvantage throughout adolescence with autonomic nervous system (ANS) functioning at 17 years. 229 low-income Mexican-American adolescents (48.6% female) and their parents were assessed annually between the ages of 10 and 16. Participants' census tracts were matched with corresponding annual administrative data of neighborhood housing burden, education, unemployment, drinking water quality, and fine particulate matter. We combined measures of adolescents' electrodermal response and respiratory sinuses arrhythmia at rest and during a social exclusion challenge (*Cyberball*) to use as ANS indices of sympathetic and parasympathetic activity, respectively. Controlling for family income-to-needs, youth exposed to greater cumulative water and air pollution from ages 10–16 displayed altered patterns of autonomic functioning at rest and during the social challenge. Conversely, youth living in areas with higher housing burden displayed healthy patterns of autonomic functioning. Altogether, results suggest that toxin exposure in youths' physical environments disrupts the ANS, representing a plausible mechanism by which pollutants and social disadvantage influence later physical and mental health.

Keywords

Environmental toxins; Riskscapes Autonomic nervous system; Adolescence; Latinx; Poverty

Social disadvantage, such as socioeconomic inequality and residential segregation, often co-occurs with increased pollution exposure (Morello-Frosch & Shenassa, 2006; Morello-

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Frosch et al., 2011), and both social disadvantage and pollution exposure are systematically associated with adverse health outcomes across the lifespan (McEwen, 2000; Olvera Alvarez et al., 2018). Research has shown that disruptions to autonomic stress physiology are key mechanisms conveying the effects of chronic stress on health (Del Giudice et al., 2011; Miller et al., 2011; Kraft & Kraft, 2021). Despite mounting evidence for this biological embedding of social disadvantage, there are notable limitations to this literature (Trentacosta & Mullingan, 2020; Trentacosta et al., 2016). Most prior studies in environmental and developmental psychology have measured only one aspect of the social disadvantage experience (e.g., either poverty, neighborhood crime, or air pollution). Given the co-occurrence of these features, a narrow focus on one aspect may result in misattributing the effects of an associated stressor to the measured stressor. Additionally, most studies have used cross-sectional designs, which provide limited insight into developmental processes. Particularly absent are studies of the consequences of stressor exposure throughout the pubertal transition, a time of heightened neurobiological sensitivity to context (Berenbaum et al., 2015; Eiland & Romeo, 2013). Moreover, little research has documented the neurobiological risks of social disadvantage coupled with pollution exposure within minoritized populations (e.g., U.S. Mexican-origin youth). Pervasive structural racism and discriminatory socio-political policies have resulted in economically disadvantaged communities of color being more likely to reside in regions with greater pollution and with lower neighborhood opportunities (Acevedo-Garcia et al., 2016; Clougherty & Kubzansky, 2009; Gwynn & Thurston, 2001; Morello-Frosch & Shenassa, 2006; Morello-Frosch et al., 2011). Thus, the current study used a multidimensional approach to measure environmental toxin exposure alongside family and neighborhood socioeconomic disadvantage and tested the independent and additive effects of these poverty-related risks experienced throughout the pubertal transition in the prediction of Mexican-origin adolescents' autonomic stress regulation.

The Riskscape of Economic Inequality

Given robust evidence from research on childhood adversity demonstrating that repeated exposure to environmental and social threats contribute to deleterious health outcomes across the life span (Evans et al., 2012; Hostinar et al., 2021), scientists have investigated pathophysiological mechanisms of morbidity and mortality resulting from cumulative stress (Blair, 2010; Evans & Kim, 2012; Fagundes et al., 2013; Shields & Slavich, 2017). This body of work has focused on contextualizing the socioeconomic status (SES) gradient of health issues that emerges in adulthood by studying individual differences in "biological embedding," or the processes by which the body adapts to repeated environmental inputs over time (Boyce & Ellis, 2005; Ellis & Boyce, 2008; Hertzman, 2012). Biological embedding reflects a process of continuous, long-term physiological calibration of the brain and body and coalesces in physiological functioning that determines behavioral and health trajectories over time (Del Giudice et al., 2011; Ellis & Del Giudice, 2014). Although such neurobiological changes may be adaptations to the experienced environment, they also may convey maladaptive consequences for future health and well-being (Blair & Raver, 2012). From a biological embedding perspective, biosocial investigations of chronic stress exposure situate physiological functioning as a mediator of later health

effects, conceptualizing disrupted neurobiological functioning as both a result of prior stress exposure and a premeditating factor for later health issues (Shonkoff, Boyce, & McEwen, 2009). Further, biological embedding may be most likely to occur, and with more pronounced effects, during sensitive periods of development, which reflect maturational times when individuals are especially susceptible to influences from the environment (Miller et al., 2011). Adolescence represents one window of heightened susceptibility as it is a dynamic period of neurobiological reorganization and refinement when increasing gonadal hormones trigger maturation of multiple systems, including stimulating the brain to actively generate new, and strengthen existing, neuronal connections (Berenbaum et al., 2015; Eiland & Romeo, 2013).

For historically minoritized communities of color in the United States, experiences of chronic stress, such as living with generational poverty, and their associated impacts on health are conflated by many other sources of physical and social stress (i.e., increased pollution exposure, neighborhood disadvantage, racial/ethnic discrimination, systematic racism and oppression; Roy & Raver, 2014; Morello-Frosch & Shenassa, 2006; Morello-Frosch et al., 2011). For example, discriminatory socio-political policies and practices, including industrialization and segregation, have resulted in economically disadvantaged communities of color being more likely to reside in regions with greater pollution (Clougherty & Kubzansky, 2009; Gwynn & Thurston, 2001; Morello-Frosch & Shenassa, 2006; Morello-Frosch et al., 2011). In addition to pollution, neighborhood disadvantage and lack of equitable opportunities are also over-concentrated in communities of color (Morello-Frosch & Lopez, 2006; Myers, 2009; Osypuk & Acevedo-Garcia, 2010; Subramanian et al., 2005). These factors may individually and cumulatively increase the risk of chronic health problems (Adler & Rehkopf, 2008; Brody et al., 2018; Merikangas et al., 2010), configuring a *riskscape* of demographic and environmental disparities that characterize variations in exposure to community stressors and vulnerability to disease (Olvera Alvarez et al., 2018; Clougherty & Kubzansky, 2009; Morello-Frosch et al., 2006).

Given that social, economic and environmental inequalities are over-concentrated in communities of color, further stratifying health disparities by race and SES (Clougherty & Kubzansky, 2009; Gwynn & Thurston, 2001; Morello-Frosch & Shenassa, 2006; Morello-Frosch et al., 2011), there is a growing need for research to simultaneously account for multiple sources of chronic stress to determine the extent to which they have unique and additive effects on physiology and health across development (Hastings et al., 2022). While the past few decades of biological embedding research has highlighted the body's stress response systems and chronic inflammation as possible mechanisms by which chronic stress exposure perpetuates disease outcomes (Del Giudice et al., 2011; Fagundez et al., 2013; McEwen, 2000; Snow et al., 2018), few studies have used a developmental approach to explore how this plethora of social, economic and environmental disparities – that is, these *riskscapes* – may influence youths' autonomic stress physiology. In addition, even less is known about these processes in U.S. Latinx children and youth, which constitute the largest ethnic community in California (48%, State of California, Department of Finance, 2021) and are systematically marginalized and over-represented in poverty contexts (Bohn et al., 2020).

Environmental Risks and the Autonomic Nervous System

The autonomic nervous system (ANS) is one of the primary brain-body communication systems that serve to connect the central nervous system and peripheral organs (Porges, 2007). The ANS responds flexibly, bidirectionally and primarily in an unconscious manner to convey information between the viscera and the brain, coordinating both basic metabolic functions and responses to environmental stimuli (Hastings et al., 2014; Zisner & Beauchaine, 2016). The ANS consists of two branches, the sympathetic and parasympathetic nervous systems (SNS and PNS, respectively), that are anatomically and functionally distinct. The PNS has been characterized as the "rest-and-digest" system, serving to calm states of arousal, support restorative functions and facilitate positive social interactions. Activity of the PNS can be assessed through measures of heart rate variability, such as respiratory sinus arrhythmia (RSA), which reflect parasympathetically mediated variations in heart rate that occur in synchrony with the respiration cycle. Higher baseline RSA and mild to moderate RSA withdrawal (i.e., decreases in RSA) to challenging stimuli are hypothesized to support attention, orienting, and the flexible regulation of emotional arousal (Hastings & Kahle, 2019; Porges, 2007). In contrast, the SNS has been characterized as coordinating the "fight-or-flight" response of aggressive and defensive reactions to threats by increasing heart rate, blood pressure, and oxygen availability to the large muscle groups. It also stimulates the eccrine sweat glands to increase perspiration, allowing for cholinergic-driven SNS activity to be assessed with measures of electrodermal activity (EDA) including skin conductance responses (SCR), or acute changes in electrical conductance across the surface of the skin, with greater SCR reflecting increased SNS activity (Zisner & Beauchaine, 2016).

Allostatic load theory suggests that continued exposure to physical and psychosocial stressors disrupt the body's stress response systems with lasting costs for neural and cardiometabolic health (Boyce et al., 2021), including the ANS (McEwen, 2007). Low household income and family socioeconomic resources contribute to altered patterns of autonomic functioning throughout development, as greater duration of childhood poverty predicts heightened SNS activity at rest (Evans & Kim, 2013; Propper & Holochwost, 2013, Johnson et al., 2017). Yet, as lower-income families predominantly live in neighborhoods with fewer resources, compromised housing conditions, and more pollutants, the associations between family economic resources and ANS stress physiology may be attributable to the broader contexts within which families live. Exposure to air pollution (e.g., particulate matter 2.5 [$PM_{2.5}$]) and water contaminants (e.g., arsenic, lead, nitrates, etc.) heighten inflammatory processes and oxidative stress (Hahad et al., 2020), which may contribute to disruptions of ANS flexibility and regulation (Miller et al., 2019; Calderón-Garciduenas et al., 2015). Specifically, $PM_{2.5}$ has been linked to increased heart rate, lower heart rate variability (Cowell et al., 2019; Kim et al., 2020; Pope et al., 1999), elevated blood pressure (Ibald-Mulli et al., 2001), pulmonary problems (Gauderman et al., 2004), and increases in sympathetic dominance in response to acute stress in adolescents, even after accounting for household income (Miller et al., 2019).

Although less research has centered on the impact of water contaminants on autonomic indices in children and adolescents, intermittent and pervasive lead and arsenic exposure

throughout childhood and adolescence has been linked to increases in blood pressure and hypertension in rats (Chandravanshi et al., 2019; Shvachiy et al., 2018, 2020) and co-inhibition of sympathetic and parasympathetic activation during acute stress in young adolescents (Gump et al., 2011). Likewise, different aspects of neighborhood disadvantage have also been associated with alterations in autonomic functioning. Specifically, research has found evidence linking objective measures of neighborhood SES with low heart rate reactivity in children (Portnoy et al., 2020), sympathetic dominance during sleep (Mellman et al., 2018), worsening pulmonary functioning (Lawrence et al., 2021), and increases in arterial inflammation suggesting chronic autonomic arousal (Osborne et al., 2020; Tawakol et al., 2019). In turn, heightened sympathetic and dampened parasympathetic activity may contribute to chronic inflammation, further increasing vulnerability to disease (Fagundes et al., 2013, Kraft & Kraft, 2021). Altogether, these results suggest that both environmental toxins and neighborhood disadvantage can disrupt both branches of the ANS. However, there is a dearth of studies examining the impact of environmental toxins and neighborhood disadvantage on the *coordination* of PNS and SNS activity, an arguably more compelling and informative indicator of physical and mental health problems, in comparison to studying either system in isolation (Berntson et al., 2008; Rudd & Yates, 2018; Thayer et al., 2010).

While the two ANS branches have been historically depicted as working in opposition, psychophysiological and developmental research has increasingly examined multiple forms of coordination between both branches (Berntson et al., 1994). The autonomic space model (Berntson et al., 1994, 2008) proposes autonomic balance and autonomic regulation as indices that jointly capture the relative dominance of each branch and the overall level of somatic activation, respectively. Whereas lower autonomic balance indicates sympathetic dominance and lower autonomic regulation indicates co-inhibition of PNS and SNS activity, higher autonomic balance indicates parasympathetic dominance and higher autonomic regulation indicates co-activation of PNS and SNS activity (Berntson et al., 2008, Quigley & Moore, 2018). Studies using the autonomic space model (Berntson et al., 1994, 2008) have found that sympathetic dominance and co-inhibition is associated with worse physiological and psychological health in adults and adolescents. Specifically, sympathetic dominance and co-inhibition have been linked to cardiovascular disease (Berntson et al., 2008; Thayer et al., 2010), metabolic syndrome (Licht et al., 2010, 2013; Rodríguez-Colón et al., 2015; Supriya et al., 2021), anxiety (Friedman, 2007), and depression (Brush et al., 2019; Bylsma et al., 2015; Choi et al., 2021; Stone et al., 2020). Altogether, examining the ANS across the full autonomic space can provide unique insights about biases in the ANS (branch dominance) and autonomic flexibility (branch coordination; Bylsma et al., 2015; Cohen et al., 2020).

Chronic environmental and social stressors have been linked to sympathetic dominance at rest (Berntson, 2019) and co-inhibition of both ANS branches in response to a stressor or challenge, indicating allostatic load of the ANS via blunted activity (Alkon et al., 2012; Berntson, 2019; Brook & Rajagopalan, 2021; McEwen, 2007). Altered autonomic functioning taxes the body's stress response capacity, increasing systemic inflammation and disrupting regenerative processes needed to maintain health (Berntson, 2019; Shields & Slavich, 2017; (Singh et al., 2009). Since chronic exposure to toxins occurs in the context of other social stressors such as neighborhood crime, crowding, and other hazards (Trentacosta & Mullingan, 2020; Olvera Alvarez et al., 2018), examination of independent and additive

influences of environmental toxins and neighborhood SES on altered autonomic functioning based on the coordination of both branches is needed.

Adolescence is a critical developmental window to investigate the influence of environmental riskscapes on autonomic functioning. In comparison to infancy and childhood, the *structural* maturation of peripheral systems reaches an adult-like profile by early adolescence (Porges & Furman, 2011; Quigley & Moore, 2018). However, the pubertal transition initiates increased sex hormone production that orchestrate numerous neurobiological changes, and there is continuing development of neurocircuitry and myelinogenesis of the CNS throughout adolescence, which modifies *functional* aspects of ANS activity and ANS-CNS coordination (Arain et al., 2013; Moodithaya & Avadhany, 2012; Silvetti et al., 2001; Weissman et al., 2018). Further, normative decreases in sympathetic activity (Harteveld et al., 2021) and increases in vagal activity during adolescence are critical for the maturation of prefrontal brain regions and cortical thinning (Koenig, 2020; Koenig et al., 2018). Thus, adolescence represents a sensitive period of development when environmental influences may have a heightened effect on autonomic functioning, significantly altering trajectories of normative brain development, health, and disease (Koenig, 2020). Hence, the deleterious effects of exposure to chronic stressors during adolescence may further increase vulnerability to psychopathology and cardiovascular risk, setting up the foundations for future health issues that emerge in adulthood (Rodriguez-Colon et al., 2015; Spezia Faulkner et al., 2003; Supriya et al., 2021). However, there is a relative dearth of studies examining developmental links between physical and social aspects of environmental adversity and youths' ANS functioning. Pursuing this line of inquiry would advance the field towards a better understanding of the role of altered patterns of autonomic functioning in the long-term implications of adversity on physical and mental health.

Hypotheses

In this study, we examined the overlapping nature of environmental toxins and neighborhood socioeconomic disadvantage and their effects on adolescent ANS functioning in a sample of Mexican-origin adolescents living in Northern California, over and above family income. Specifically, we focused on two sources of toxin exposure: air pollution and water contamination. Neighborhood disadvantage was measured with neighborhood levels of education, unemployment, and housing burden. Latinx children and youth are a rapidly growing population across the U.S. but are largely under-represented in neurobiological studies (Myers, 2009; Parra & Hastings, 2018). Compared to other sociodemographic groups, racial/ethnic minority families are often exposed to riskscapes, experiencing greater neighborhood poverty, unemployment, and exposure to pollutants (Morello-Frosch & Shenassa, 2006). This complex, multifaceted risk profile may uniquely contribute to altered patterns of autonomic functioning in Latinx youth. Therefore, we investigated the unique and additive effects of social and physical adversities across adolescence on the ANS functioning of Mexican-origin youth living in Northern California.

In accord with allostatic load theory and bioecological frameworks of riskscapes (Morello-Frosch & Shenassa, 2006; McEwen, 2000; Myers, 2009), we expected that (1) toxins would

be over-concentrated in areas with fewer economic resources. Additionally, we expected that (2) greater exposure to environmental toxins, neighborhood disadvantage, and lower family income would individually (e.g., the effect of one risk while holding other risks constant) and cumulatively (e.g., interpreting effects of risks altogether) predict atypical or maladaptive ANS stress physiology. In alignment with allostatic load theory and evidence linking specific profiles of autonomic function and health problems (Berntson, 2019), we hypothesized that greater stress exposure or riskscapes (e.g., greater environmental toxins and neighborhood social disadvantage) would predict higher sympathetic dominance at baseline, and co-inhibition of both branches in response to a social exclusion challenge.

Method

Participants

229 Mexican-origin adolescents (48.7% female; M_{age} = 10.85 years, SD = 0.60 at initial study recruitment; M_{age} = 17.15 years, SD = 0.42 at neurobiological assessment) and their parents participated in a neurobiological sub-study of the California Families Project (CFP), an ongoing longitudinal study examining individual, family, and community-level developmental risk and resilience factors. Participants of the main study included 674 Mexican-origin families living in Northern California with a child in the fifth grade drawn at random from school rosters from the 2006–2007 and 2007–2008 school years (Wave 1), assessed annually hereafter.

Out of the 229 Mexican-origin adolescents participating in the neurobiological study, 26.4% of participants were first generation, 65.2% were second generation, and 8.4% were third generation immigrants to the U.S. The education level of the parents of this sample ranged from none to completing a 4-year college degree (median = 9th grade for both mothers and fathers). Household income was reported to the nearest increment of $5,000 (M = $30,001–35,000, SD = 4.366, range <$5000/year to >$95,000/year), with 52% of participants' reporting <$30,001, 37.5% reporting between $30,001 and $60,000%, and 10.5% reporting $60,001 to above $95,001.

Procedure

Toxin and neighborhood data matching—Starting in fifth grade (Wave 1), CFP assessments were completed annually using questionnaires, interviews, and observational data collection in each family's home. Participants' mailing addresses were collected yearly at each visit and spatially matched to corresponding county, census tract, census block, and geolocation (latitude and longitude). Across all waves, participants lived in 98 unique census tracts within two adjacent counties, with an average of three participants in each tract (min = 1, max = 14). From age 10 to 16, 69% of participants remained in the same census tract, 19% moved once, 9% moved twice, and 3% moved three times or more. Using publicly available data, we mapped participants' geolocation and census tract to information on annual air pollution and neighborhood education levels, respectively. Unemployment, housing burden, and drinking water quality at the census-tract level were not available annually (see Measures section for details). However, these were matched by participants' census tract at each wave to account for those who relocated to a different census tract

within any given year. Figure 1 shows a visualization of the complete study timeline from Wave 1 (age 10) to Wave 7 (age 16).

Neurobiological sub-study assessment—At Wave 7, adolescents from the main CFP were invited to participate in a sub-study designed to examine neurobiological correlates underlying the etiology of depression. Given that the neurobiological sub-study was designed to examine underlying neurobiological antecedents of depression, youth were oversampled for depressive symptoms based on self-reported symptoms at 14–15 years assessed with the Computerized Diagnostic Interview Schedule for Children-IV (C-DISC; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000), and General Distress and Anhedonic Depression items of the Mood and Anxiety Symptom Questionnaire (MASQ; Watson et al., 1995). Adolescents with scores above the sample median on any of these three measures were designated as at risk for depression and this risk index was used as an inclusion criterion in the neurobiological sub-study. Despite oversampling for depressive symptomatology, none of the current participants met diagnostic criteria for major depressive disorder at 14–15 years.

Approximately 15.5 weeks after their Wave 7 visit, on average, youth visited an imaging research center at the university medical center, where they participated in a simultaneous electrocardiogram (ECG) and skin conductance recording during a functional magnetic resonance image (fMRI) scan. Figure 2 shows a visualization of the neurobiological sub-study complete protocol for the MRI scan and ANS acquisition. Over an hour after participants' arrival, baseline ECG and skin conductance were recorded outside the scanner for three minutes while participants were lying on their backs in the scanning bed. Adolescents then underwent fMRI scanning procedures, completing three tasks. ANS activity during *Cyberball* (Williams et al., 2000), a social exclusion challenge task, was examined for the current analyses. After the scan, participants filled out questionnaires. The study site's Institutional Review Board, approved the main CFP study and neurobiological sub-study. Participants' parents provided informed consent and adolescents provided assent; both were compensated for their participation.

Measures

Neighborhood-level data—Particulate Matter 2.5 ($PM_{2.5}$) was estimated using air quality monitoring station data made publicly available through the California Air Resources Board online database (https://www.arb.ca.gov/aqmis2/aqdselect.php?tab=daily). $PM_{2.5}$ is a mixture of aerosolized liquid and solid particles measuring 2.5 micrometers or less in diameter and includes organic chemicals, dust, metals, and allergens. Specific compositions of $PM_{2.5}$ vary by local and regional conditions, time of year, and weather. Hourly $PM_{2.5}$ data were calculated using inverse distance weighting interpolation (Al-Hamdan et al., 2009) between participants' home address geolocation and the three nearest monitoring stations with available data (average distance between participants and three sensors = 11.30 kms, $SD = 6.57$). Values were averaged across days and months so that each participant had one value per wave for a total of 7 years (2006/2007–2012/2013, $M = 9.45$, $SD = 1.15$). Missing data for $PM_{2.5}$ ranged from 0.4% ($n = 1$; Wave 1) to 3% ($n = 7$; Wave 2). Across waves, approximately 7.58%, (*min* 0; Waves 4–7, *max* = 35%; Wave 2) of participants were above

the Environmental protection agency's (EPA) maximum contaminant level of 12 μg/m3 (see Table S1 for year-to-year details).

Drinking water quality was assessed using data compiled by the California Environmental Protection Agency (CalEnviroScreen 3.0 [CES 3.0]; OEHHA, 2017; https://data.ca.gov/dataset/calenviroscreen-3–0-results/resource/89b3f4e9–0bf8–4690-8c6f-715a717f3fae) and reflects the total level of 13 known contaminants, including Arsenic, Cadmium, Chromium Hexavalent, Dibromochloropropane, Lead, Nitrate, Perchlorate, Radium 226 and 228, Total Trihalomethanes, Tetrachloroethylene, Trichloroethylene, 1,2,3-Trichloropropane, and Uranium within the state of California over the 2005–2013 compliance cycle. The overall drinking water score from the CES 3.0 reflects the cumulative concentrations of all contaminants per census tract. Water data was matched by participants' census tract at each wave ($M = 420$, $SD = 294$). Based on missing home mailing addresses used to determine census tracts, missing data for drinking water ranged from 3% ($n = 7$; Wave 7) to 19% ($n = 43$; Wave 2). Across waves, no families were above EPA's maximum contaminant level thresholds for nine out of the 13 contaminants. Only one and two families were above EPA's cutoff of 10 μg/L for arsenic and 0.2 μg/L for Dibromochloropropane, respectively. However, approximately 39% of participants were above the cutoff of 10 mg/L of nitrate in water and 34% were above the cutoff of 10 μg/L for chromium hexavalent, on average across years (for year-to-year data, see Table S1 in the supplemental material).

Housing burden represents the percent of owned or rented low-income households (i.e., families who reported income that was less than 80% of the local area median family income) that are severely burdened by mortgage or rent costs (i.e., greater than 50% of income allocated to housing costs) within a census tract. Housing burden was downloaded from the U.S. Department of Housing and Urban Development public access website (https://www.huduser.gov/portal/datasets/cp.html#2006–2013). This data was collected once every three years (2006–2010 and 2010–2013) and matched to participants' census tract at each wave ($M = 13.68$, $SD = 6.02$). Based on missing home mailing addresses used to determine census tracts, missing data for housing burden ranged from 3% ($n = 7$; Wave 7) to 19% ($n = 43$; Wave 2). Across waves, 9.17% of participants lived in census tracts above the 70th percentile of housing burden in the state of California (CES 3.0, 2017).

Unemployment reflects the percent of the population over age 16 and eligible for the labor force that is unemployed. This measure excluded retired people, students, homemakers, those not seeking work, active-duty military personnel, and those who were institutionalized (e.g., individuals currently in prison, nursing homes, or mental health facilities). Unemployment data were extracted from the American Community Survey of the U.S. Census Bureau (https://www.census.gov/programs-surveys/acs/about.html). Only one measurement occasion was available, covering the years 2006–2010, which was matched yearly to participants' census tracts between Wave 1 and Wave 4 ($M = 8.87$, $SD = 4.64$). Missing data for unemployment ranged from 8% ($n = 18$; Wave 4) to 19% ($n = 43$; Wave 2). Across waves, 19.65% of participants lived in census tracts above the 70th percentile of unemployment in the state of California (CES 3.0, 2017).

Education was defined as the percent of the population aged 25 and over who earned a four-year college degree or higher. Education data were collected yearly by the California Department of Public Health (https://data.chhs.ca.gov/dataset/educational-attainment) and matched by participants' census tract at each wave ($M = 18.31$, $SD = 11.18$). Missing data for education ranged from 3% ($n = 7$; Wave 7) to 19% ($n = 43$; Wave 2). Across waves, 42.36 % of participants lived in census tracts below the 30th percentile of educational attainment in the state of California (CDPH Office of Health Equity, Health Research and Statistics Unit, 2018).

Individual-level data

Family income-to-needs.: Mothers reported on the family's annual total household income when adolescents were 10–16 years old (7 waves of data) to the nearest $5,000 increment (i.e., $30,001–35,000). Each increment corresponded to a number between 1 ("less than $5,000") and 20 ("$95,001 or more"). Mothers also reported the number of people living in the household. Annual income-to-needs ratios were calculated by dividing the family's reported income by the income value corresponding with the poverty line for a family of that size that year, as indicated by the U. S. Census Bureau (https://www.census.gov/data/tables/time-series/demo/income-poverty/historical-poverty-thresholds.html). Income-to-needs ratios ranged from 0 to 5 with ratios of 1 or less indicating poverty. Missing data for income-to-needs ratios ranged from 5% ($n = 12$; Wave 5) to 31% ($n = 72$; Wave 3).

MRI experience including a social exclusion challenge—At the neurobiological assessment (see Figure 2) participants underwent MRI scanning procedures during which they played *Cyberball*, beginning on average 40 minutes after entering the scanner. *Cyberball* is a virtual ball-toss game designed to elicit feelings of ostracism, social exclusion, and rejection (Williams et al., 2000). In line with prior *Cyberball* studies, this task was chosen to represent a mild social challenge that is salient during adolescence and has been shown to elicit individual differences in physiological reactivity in the ANS (Holterman et al., 2016; Kaufman et al., 2018) and the Hypothalamic-Pituitary-Adrenal (HPA) axis (Beekman et al., 2016; Johnson et al., 2021). Participants were told they would play a simulated ball-tossing game with two computerized players and were asked to imagine, as vividly as possible, that they were playing with their peers. Participants viewed a projection screen in the scanner, with three cartoon figures representing the two other players of no apparent gender or race/ethnicity and the third representing the participant. While playing the game, the ball was thrown back and forth among the players, with the participant choosing the recipient of their throws using a button and the computer selecting the throws between the two virtual players.

Cyberball includes both inclusion and exclusion trials. In the inclusion trials, participants had equal opportunities to toss the ball to the two virtual players. However, during exclusion trials, the virtual players tossed the ball to the participant once before excluding them for the rest of the trial. There were six rounds of inclusion trials and six rounds of exclusion trials, each with 10–11 ball tosses, presented in a pseudo-random order with inclusion trials over-concentrated near the beginning and exclusion trials over-concentrated toward the end of the task. Trial durations ranged from 22–32 seconds, with 7–8 second intervals between

each trial. In total, the task ran for approximately 9 minutes. Although *Cyberball* was administered during a neuroimaging scan (Schriber et al., 2018), the present report examined adolescents' autonomic stress responses, and thus, neuroimaging data were excluded.

Autonomic nervous system data acquisition and preprocessing

Respiratory sinus arrhythmia.: ECG data were obtained before and during the fMRI scan using three electrodes on the chest linked to Biopac fMRI compatible wireless signal recording (Biopac Systems, USA) via Siemens' telnet MPCU at 400 Hz. The data were transformed into an ASCII-formatted string of amplitude values, which was then input into the Mindware HRV program (Mindware Technologies, Gahanna, OH). RSA was computed using the inter-beat-interval sequence. The delay time between consecutive local maxima in the QRS complex (R-spikes) was used to calculate inter-beat intervals (Berntson et al., 1997). Trained research assistants visually reviewed ECG data for accurate identification of R-spikes and corrected the data when the automated program misinterpreted the R-spikes. The frequency band utilized to measure RSA was 0.12–0.40, reflecting the age-normative respiratory frequency band (Dollar et al., 2020). RSA is calculated by Mindware as the natural log of spectral power in this frequency range. Baseline RSA was calculated in 30-second epochs which were averaged across the three-minute baseline. RSA was calculated over the duration of each exclusion trial in *Cyberball*, then averaged across the six exclusion trials. Inclusion trials were not included in the current analyses, and as such we refer to task RSA as "RSA during a social challenge". RSA data for 10 individuals during baseline and for 27 during *Cyberball* were missing due to human error, equipment errors during sample collection, or high-frequency noise in the ECG signal caused by MRI interference. Two outliers (> +/− 3 *S.D.* from the mean) were identified for baseline RSA and winsorized prior to analysis (Wilcox, 2012). RSA activity decreased from baseline to the social challenge, $t(194) = 10.75$ $p < .001$.

Skin conductance data were obtained before and during the fMRI scan with two electrodes on the palm of the non-dominant hand using Biopac MP150 equipment and AcqKnowledge 4.1 software, with a gain of 10 μS (Biopac Systems, USA). To account for high-frequency noise in the fMRI signal, a rolling filter was applied using Mindware EDA software (Mindware Technologies, Gahanna, OH). For each 30 second segment, the number of nonspecific skin conductance responses (SCR) with an amplitude change of 0.05 μS was counted (Braithwaite et al., 2015). Nonspecific SCR is a reliable measure of SNS activity that has been linked to subjective arousal, negative mood, and cardiac SNS activity measurements (Kelsey, 1991; Nikula, 1991). SCR were counted for each 30-second epoch during baseline acquisition, and then averaged across epochs to index baseline SCR. Similarly, SCR were counted within each exclusion trial of *Cyberball*, and then averaged across trials to index social exclusion task SCR. We refer to task SCR as "SCR during a social challenge". Data for seven individuals during baseline acquisition and for 15 during *Cyberball* were missing due to human error, device breakdown during sample collection, or high-frequency noise caused by MRI interference. Seven outliers (> +/− 3 *S.D.* from the mean) were winsorized prior to analysis. There was not a significant change in SCR, $t(205) = 0.45$, $p = .653$ between baseline and *Cyberball* exclusion trials.

Covariates.: Analyses included gender, age at scan visit, depression-risk recruitment status, and body-mass index (BMI) as covariates to account for individual differences in participant characteristics shown by prior work to impact ANS functioning (Harteveld et al., 2021; Hollenstein et al., 2012; Koenig et al., 2014; Nelson et al., 2021). Depression-risk recruitment status reflects a dichotomous recruitment variable (1 = scored above the median on any recruitment measure, N = 175, 0 = scored below the median on all measures, N = 54). Depression-risk was controlled for in analyses to account for any bias attributed to oversampling for past depressive symptoms. No participants were missing data for gender and depression risk. Nine participants were missing data for age and BMI (4% of sample), respectively. Further, our models were adjusted for time difference (in years) between participant's Wave 7 visit and scan date, which ranged from 0 (same wave 7 visit and scan year) to 1.5 years (*M* time difference = 6.2 months). All covariates were centered prior to analyses to assist interpretability.

Analytic Strategy

Missing data for census tracts ranged from 3% ($n = 7$; Wave 7) to 17% ($n = 41$; Wave 2) and 31% of participants moved at least once throughout data collection. After adjusting for multiple comparisons, participants who moved more often were more likely to have lower income-to-needs (Spearman $r = -.22$, $p = .02$), but no other differences among our predictors of interest or physiological outcomes were found.

Aligned with past studies that calculated autonomic balance and regulation using measures of EDA to assess SNS activity (Stone et al., 2020; Visnovcova et al., 2013), the current study calculated ANS coordination using standardized (Z-scored) averages of RSA and SCR at baseline and during the social exclusion challenge. Berntson and colleagues (2008) autonomic space calculation was originally computed using pre-ejection period as an index of SNS activity, wherein *lower* values indicate heightened SNS. However, when using SCR, *higher* values reflect greater SNS activity. Therefore, autonomic balance was computed as RSAz - SCRz (Stone et al., 2020), such that higher values indicate parasympathetic dominance and lower values sympathetic dominance. Autonomic regulation was computed as: RSAz + SCRz (Stone et al., 2020), such that higher values indicate co-activation of both branches and lower values indicate co-inhibition.

To test the effect of neighborhood- and individual-level risk factors on the prediction of adolescents' ANS functioning accounting for the nested structure of the data, we conducted a two-step structural equation model using Lavaan (Rosseel, 2012) and the Lavaan.survey package (Oberski, 2014) in R-studio (R version 4.0.2). A path diagram of the full model is presented in Supplemental Materials. In step 1, measures of neighborhood unemployment (Waves 1–4, ages 10 to 13) and family income-to-needs, neighborhood education, housing burden, $PM_{2.5}$, and drinking water quality (Waves 1–7, ages 10 to 16) were used to calculate latent variables for each construct (named Unemployment, Family Income-to-needs, Education, Housing Burden, PM 2.5, and Water Quality, respectively) with factor loadings set so that the intercept represented average exposure across all waves for each risk factor. Autonomic balance (branch dominance) and autonomic regulation (branch coordination) at baseline and during the social exclusion challenge were regressed on these

latent variables and covariates. Covariances between autonomic balance and regulation, significantly related covariates (i.e., gender with BMI and depression-risk), and all latent risk factors were calculated. Full information maximum likelihood (FIML) estimation accounted for missing data and model fit was assessed with the chi-square (χ^2) goodness of fit statistic, the comparative fit index (*CFI*; Bentler, 1990), and the root mean square error of approximation (*RMSEA*; Browne & Cudeck, 1992). Model fit was considered acceptable if the χ^2 *p*-value was nonsignificant, *CFI* was > .90, and *RMSEA* was < .08. In step two, the previously specified models were run through lavaan.survey (Oberski, 2014), a package that allows for complex sampling designs and accounts for clustered data. In our analysis, we specified the function to account for the clustering of the census tract that families spent the most time in across the years of the study.

Although the use of an autonomic space framework is a useful way to explore overall levels and track how PNS and SNS operate simultaneously, examining both branches as separate indicators may inform whether dominance and activation are driven by the coordination of both branches or by one specific branch, on average. Thus, we conducted post-hoc exploratory analyses using average baseline and RSA and SCR scores during the social exclusion challenge instead of autonomic balance and regulation. RSA and SCR at baseline and during exclusion were regressed on latent environmental variables and covariates. Covariances between RSA and SCR, significantly related covariates (i.e., gender with BMI and depression-risk), and all latent risk factors were calculated.

Results

Descriptive statistics and zero-order correlations corrected for multiple comparisons for all measures are presented in Table 1. On average, participants' income-to-needs ratio was 118% of the federal poverty line, with 47.16% of the sample living at or below the federal poverty line. For year-to-year descriptives for each of toxin and neighborhood disadvantage variables see Table S1 in the supplemental materials.

In testing Hypothesis 1, correlations among study variables were examined to test whether greater levels of air and water toxins were significantly over-concentrated in areas characterized by economic disadvantage. As expected, most zero-order correlations between neighborhood-level SES and exposure to toxins were significant, with lower neighborhood SES and greater toxin exposure clustering together over the years. Participants who had lower income-to-needs lived in areas with higher concentrations of $PM_{2.5}$ and in census tracts with higher unemployment, housing burden, and lower education. However, neither income-to-needs, $PM_{2.5}$, or unemployment were associated with water quality. Few correlations between autonomic functioning and environmental risks were significant; individuals with higher latent average exposure to $PM_{2.5}$ had sympathetic dominance at baseline and during the social challenge.

Autonomic balance values were positively correlated with RSA and negatively correlated with SCR both at rest and during the social challenge, consistent with its denotation as a spectrum from parasympathetic to sympathetic dominance (Alen et al., 2020). Likewise, autonomic regulation was positively associated with RSA and SCR at baseline and during

the social challenge, indicating relative co-activation of both branches. As expected in studies using the autonomic space framework (Wiley et al., 2021), autonomic balance and regulation were not associated with each other within the same task. However, participants with higher parasympathetic dominance at rest also had higher parasympathetic dominance and co-activation of both branches during the social challenge. Likewise, those with higher co-activation at rest had increased parasympathetic dominance and co-activation during the social challenge.

Regarding Hypothesis 2, we constructed a path model defining latent variables for all environmental risks across Wave 1 to 7 (see Figure S1 in supplemental materials) with the purpose of testing the unique and additive effects of each latent risk predicting adolescents' ANS physiology. Our model demonstrated close fit to the data. While the statistical index of fit was significant, X^2 (1037) = 2001.536, $p < .001$ (common in large samples), indices of practical fit were good, with *CFI* = 0.91; *TLI* = 0.905; *RMSEA* = .064, 90% CI [.060, .068]. Factor loadings and intercepts for the latent variables were all significant at $p < .001$, and standardized factor loadings tended to be moderate to large in magnitude, ranging from .69 to .98 (See Table 2). Intercept variances were significant, suggesting there were inter-individual differences in adolescents' exposure to risk factors. Significant covariances were consistent with zero-order correlations.

Key results of the structural equation model are shown in Figure 3 and full model results are shown in Table 3. As hypothesized, $PM_{2.5}$ was negatively associated with autonomic balance at rest and during the social exclusion challenge, indicating higher sympathetic dominance. No significant associations were found between $PM_{2.5}$ and autonomic regulation. Similar to the effect of $PM_{2.5}$, youth with lower family income-to-needs and poor drinking water quality exhibited higher sympathetic dominance (lower autonomic balance) at rest. Additionally, lower family income-to-needs and poor drinking water quality was related to co-inhibition of both branches (lower autonomic regulation) in response to the social challenge. Contrary to hypothesis 2, the opposite pattern was found for housing burden; youth living in census tracts where lower-income families paid more towards rent or mortgage loans exhibited a pattern of parasympathetic dominance (higher autonomic balance) at rest and co-activation of both branches (higher autonomic regulation) during the social challenge. Neither neighborhood unemployment nor education were associated with ANS functioning.

Regarding covariates, there were significant positive associations of gender, age, and depression-risk with autonomic balance, as well as a negative association with BMI. Older adolescents, females, and adolescents with prior risk for depression (indexed by subclinical depressive symptoms on the C-DISC and MASQ approximately 2.5 years before the scan) had parasympathetic dominance at rest and during the social challenge. Adolescents with elevated BMI had sympathetic dominance at rest.

Post-hoc analysis with RSA and SCR

Lastly, we examined each ANS branch as a separate indicator to explore whether the coordination of both branches or one specific branch was driving autonomic balance and regulation (see Table S2 in supplemental materials for full model results). At baseline,

participants with higher latent average exposure to $PM_{2.5}$ exhibited lower RSA ($B = -0.302$, $SE = 0.04$, $p < .001$) and higher SCR ($B = 0.283$, $SE = 0.098$, $p < .001$), indicating reciprocal coordination of both branches. Conversely, poor drinking water quality was associated with elevated baseline SCR only ($B = 0.167$, $SE = 0.045$, $p = .010$). Higher housing burden ($B = 0.231$ $SE = 0.136$, $p = .005$) and family income-to-needs ($B = 0.247$, $SE = 0.088$, $p < .001$) were associated with elevated baseline RSA only.

Considering ANS indices during the social exclusion challenge, poor drinking water quality was associated with lower RSA ($B = -0.124$, $SE = 0.024$, $p = .048$) and with lower SCR ($B = -0.205$, $SE = 0.048$, $p = .001$), and housing burden was positively associated with RSA ($B = 0.279$, $SE = 0.178$, $p = .002$) and with SCR ($B = 0.331$, $SE = 0.359$, $p < .001$), indicating co-inhibition and co-activation of both branches, respectively. $PM_{2.5}$ was negatively associated with RSA only ($B = -0.250$, $SE = 0.052$, $p = .001$), and high income-to-needs ratio ($B = 0.292$, $SE = 0.116$, $p < .001$) was associated with elevated RSA only.

Discussion

Exposure to air and water pollutants and greater neighborhood disadvantage from pre-to-mid-adolescence was linked to altered patterns of autonomic functioning at rest and during a social exclusion task within an MRI scanner in a sample of Mexican-origin youth from predominantly low-income backgrounds. In accord with bioecological frameworks of riskscapes (Morello-Frosch & Shenassa, 2006; Morello-Frosch et al., 2011), we found that low family income-to-needs coincided with greater exposure to air pollution, greater neighborhood unemployment and housing burden, and lower neighborhood education. Likewise, environmental toxins were over-concentrated in areas with lower education and higher housing burden. Based on allostatic load theory and prior evidence linking these risk factors to altered patterns of autonomic functioning and disease (Berntson et al., 2008; McEwen, 2007), we hypothesized that toxin exposure would be linked to sympathetic dominance at rest and co-inhibition of both branches during a social challenge. We found partial support for this hypothesis, as associations varied across specific risk factors. Overall, youth living in areas with poor drinking water quality from age 10 to 16 displayed a profile consistent with altered patterns of autonomic functioning (i.e., relative sympathetic dominance at rest and co-inhibition of both branches during acute stress). Similarly, exposure to $PM_{2.5}$ predicted relative sympathetic dominance at rest and during the social challenge. Interestingly, post-hoc analyses examining RSA and SCR as separate indicators of the two ANS branches showed that both toxins were associated with sympathetic activation at baseline and with parasympathetic inhibition during the task. Further, $PM_{2.5}$ was also related to reciprocal parasympathetic inhibition at baseline and poor drinking quality with sympathetic inhibition during the task. Regarding neighborhood disadvantage, no associations were found between baseline or task ANS indices and neighborhood education or unemployment, after controlling for exposure to environmental toxins and family income-to-needs. However, youth living in census tracts with higher housing burden, that is, in areas where low-income households were severely burdened by mortgage or rent costs, displayed a healthy pattern of parasympathetic dominance at rest and co-activation of both branches during the social challenge. Altogether, our study provides novel evidence that, over and above family income and other demographic

factors, toxin exposure in youths' physical environments disrupt the ANS, suggesting one plausible mechanism by which the context of poverty influences later physical and mental health. Since neurobiological changes triggered by puberty create more opportunities for external factors to have a lasting impact on internal processes (Eiland & Romeo, 2013; Koening, 2020), adolescence might be a particularly pernicious period for the experience of environmental riskscapes to increase the physiological potential for adverse health consequences throughout the lifespan (Evans et al., 2012).

Environmental toxins such as water quality and air pollution are critical threats to public health worldwide (Trentacosta et al., 2016). Our findings demonstrate that exposure to air and water toxins and low family income contributed to the upregulation of the SNS at rest, and, in addition, water toxins were related to reduced parasympathetic and sympathetic activity during a social exclusion challenge. Exactly how long-term toxin exposure increases SNS activity in humans remains relatively unexplored. Air pollutants and chronic exposure to water contaminants such as nitrate, lead, and arsenic, increase neural inflammation and activation which may interrupt the normal functioning of the CNS and ANS (Balasubramanian et al., 2013; Chandravanshi et al., 2019; Shvachiy et al., 2020; Ying et al., 2014). Air pollutants have also been shown to increase circulating adrenal corticosteroid levels, potentiating sympathetic neural activity (Li et al., 2017). Clearly, greater integration of research across multiple fields is needed to understand the complex mechanistic relations linking water toxin exposure to ANS functioning. The chronic autonomic consequences of day-to-day toxin exposure may play a key role in studies of poverty or socioeconomic adversity resulting in "stress getting under the skin" to affect peripheral stress physiology, yet this has received scant attention in developmental research (Miller et al., 2019; Trentacosta & Mulligan, 2020).

Sympathetic dominance typically decreases throughout adolescence (Harteveld et al., 2021; Quigley & Moore, 2018) and normative increases in vagal activity throughout development contribute to healthier brain development (Koening, 2020). Hence, the evidence for environmental adversity earlier in adolescence predicting heightened sympathetic dominance in late adolescence may reflect the consequences of prolonged wear-and-tear on the ANS (McEwen, 2007). This eroding of normative healthy ANS functioning could place excessive metabolic and energetic demands on bodily systems, increasing systemic inflammation and incurring potentially negative consequences for physical and mental health. Direct links between lower resting autonomic balance and increases in inflammation are evident as early as late childhood (Alen et al., 2020). Elevated SNS activation and reduced PNS activation promote the systemic release of epinephrine and norepinephrine, increasing the production of proinflammatory cytokines, resulting in oxidative stress and disrupting regenerative processes needed to maintain health (Berntson, 2019; Kemp & Quintana, 2013; Quigley & Moore, 2018; Shields & Slavich, 2017). Thus, increases in immune responsivity and neuroinflammation associated with childhood social adversity are candidate mechanisms for the links between adolescent's altered patterns of autonomic functioning with future cardiovascular diseases in adulthood (Boyce & Hertzman, 2018; Rajagopalan et al., 2018).

Although prior research has found evidence for the detrimental effects of low neighborhood SES - lower education and greater unemployment - on ANS functioning (Mellman et al., 2018; Portnoy et al., 2020; Tawakol et al., 2019), our models yielded no findings over and above toxin exposure and family income. Notably, as none of these prior studies controlled for environmental toxins, it is possible that low neighborhood SES serves as a proxy for toxin exposure. Alternatively, the lack of significant findings may be attributed to our study sample. Since the main CFP study and neurobiological sub-study were not designed to investigate the effects of neighborhood environmental toxin exposure on youths' ANS physiology, average census-tract levels and variability of education and unemployment might be lower than would be expected for a general population sample. In comparison to California, neighborhood education attainment was low, but so was unemployment. This particular sample configuration (Latinx, low-income, less educated, but predominately employed) might be driving null findings for neighborhood SES.

Contrary to our null findings for neighborhood education and unemployment, cumulative housing burden was associated with a profile that corresponds to a healthy ANS pattern: PNS dominance at rest and PNS-SNS co-activation during acute stress (Alkon et al., 2011; Berntson, 2019). Prior research suggests that the impact of housing burden and affordability is not straightforward, as "families' decisions about where to live are complex" (Warren & Font, 2015, p. 12). Prior studies have failed to find consistent associations between housing affordability and children's health, behavior, or academic achievement (Coley et al., 2013; Leventhal & Newman, 2010; Warren & Font, 2015). This is partly explained by the presence of financial moderators of the relationship between housing and child development, including family composition and income level, race/ethnicity, and housing tenure (ownership or rent; Acevedo-Garcia et al., 2016; Dunn, 2020; Shamsuddin & Campbell, 2021). Since there is a positive gradient between housing affordability and neighborhood opportunities for children, low-income families often face a tradeoff between affordability and neighborhood quality (Acevedo-Garcia et al., 2016). This might reflect economically-challenged families striving to invest in high-priced housing to increase their access to quality schools, lower crime rates, green/blue spaces, and other services that might counteract the effects of the family's limited financial resources, but also incur financial pressure as a result of increased housing cost burden on the family (Leventhal & Newman, 2010; Coley et al., 2013). Among low-income families, studies have found an inverted u-shape association between housing cost burden and children's cognitive achievement (S. Newman & Holupka, 2016), and between housing cost burden and child enrichment expenditures (S. J. Newman & Holupka, 2015). While low-income families living in areas with high housing burden have a higher probability of experiencing significant material hardship since they have less disposable income after accounting for housing costs, low-income families living in areas with low housing burden have a higher likelihood of living in poor quality housing units and neighborhoods with under-resourced schools (Newman & Holupka, 2015; Acevedo-Garcia et al., 2016). Therefore, moderate levels of housing burden have been linked to better cognitive development and higher parental investment in child enrichment, which is particularly relevant in the context of our sample. On average, housing burden was in the 24.06% percentile of the state of California, and only 9.17% of participants lived in census tracts above the 70th percentile. Given that our sample had a

lower to moderate prevalence of housing burden and was mostly low-income (90% lived at or below 200% of the federal poverty line), our results could reflect tradeoffs between disposable income and neighborhood quality. However, this interpretation may be subject to other moderators.

Across the United States, low-income Latinx families experience high levels of imbalance in this tradeoff, being more likely to overpay for housing relative to the opportunities provided to children (Acevedo-Garcia et al., 2016). In addition, homeowners and renters experience burden differently because of the significant tax advantages of home ownership in the United States in comparison to rent (Dunn, 2020; Department of Housing and Urban Development [US HUD], 2019), and low-quality neighborhoods are still unaffordable for low-income renters (Acevedo-Garcia et al., 2016). In our sample, neighborhood housing burden was associated with greater neighborhood air pollution and unemployment, poor drinking water quality, decreased neighborhood education, and lower family income, but also to healthier ANS functioning in adolescents. Since our measure of housing burden did not differentiate housing tenure and this is the first study to report evidence linking neighborhood housing burden to ANS functioning in youth while adjusting for important confounders, more research is needed to investigate what housing burden represents in the context of these low-income Latinx families.

Finally, youth who experienced higher family income-to-needs from age 10 to 16 displayed a pattern consistent with flexible autonomic regulation, exhibiting relative parasympathetic dominance at rest and co-activation during the social challenge, even after accounting for toxin exposure and neighborhood SES. The post-hoc analyses indicated that this profile was driven by higher parasympathetic activity since income-to-needs was not associated with sympathetic activity. As mentioned above, most of this sample experienced significant financial hardship. Thus, it seems unlikely that access to material resources or absence of financial pressure could fully account for this association. Since the experience of financial hardship embodies a wide range of potential co-occurring stressors (e.g., neglect, crime and violence exposure, family conflict), exactly what our measure of income-to-needs is capturing is unclear. One possibility is that parents' ability to provide effective and engaged caregiving may be compromised by increasing financial distress leading to a lower quality of caregiving (Conger & Conger, 2002; Martin et al., 2019). However, there is considerable heterogeneity of parents' perceptions and reactions to financial distress (Masten et al., 2021) and this explanation is not true of all family dynamics. Despite this, caregivers play a pivotal role in the development of ANS functioning, specifically for parasympathetic activation due to its link to social engagement and the regulation of social behavior (Alkon et al., 2012; Johnson et al., 2014; Katz et al., 2020; Porges & Furman, 2011; X. Zhang et al., 2020). Unpacking family-level indicators in addition to toxin exposure may further enrich the field's understanding of risk, resilience, and variation in the outcomes of childhood and adolescent adversity.

Theoretical implications

By examining the neurobiological impact of chronic poverty and toxin exposure in a sample of Mexican-origin adolescents, the current study contributes to the growing

literature re-centering diverse communities, following the overreliance of WEIRD (White, Educated, Industrialized, Rich, and Democratic) samples in research (Henrich et al., 2010). Contextualizing the experience of economic inequality within the broader environment of pollution and neighborhood disadvantage is in line with Myers' (2009) biopsychosocial perspective of cumulative vulnerability and minority health and expands the field's understanding of how social disadvantage and chronic stress may alter autonomic physiology across diverse communities. Compared to impoverished White communities, impoverished Latinx and other ethnically/racially minoritized groups are more likely to live in over-polluted, underfunded areas (Clougherty & Kubzansky, 2009; Gwynn & Thurston, 2001; Morello-Frosch & Shenassa, 2006; Morello-Frosch et al., 2011), and thus are more likely to experience neighborhood crime and crowding, all while living within a sociopolitical climate classified by systematic racism, oppression, and discrimination (Roy & Raver, 2014).

A key theoretical strength of the current study is its investigation of multiple sources of chronic stress and their independent and additive influences on adolescents' autonomic physiology. Historically, psychological research has approached the study of chronic stress from a global perspective, essentially equating the sources of stress in terms of their impact on physiology and largely ignoring arguments of specificity. For example, the cumulative risk (CR) perspective requires risk factors to be dichotomized in measurement (0 = no risk; 1 = risk) with the final CR score reflecting an overall sum of these dichotomized experiences (Evans et al., 2013). Studies of allostatic load take a similar approach to measure the cumulative physiological wear and tear resulting from chronic stress by calculating total allostatic load as a sum of all the physiological parameters measured (e.g., blood pressure, cortisol, epinephrine, etc. dichotomized based on percentiles; Seeman et al., 2001). These cumulative models have drawbacks, including the loss of information reflecting risk intensity (Evans et al., 2013). In contrast, studies investigating independent *and* additive effects of multiple risk factors contribute to the field's understanding of developmental trajectories in terms of equifinality (i.e., different developmental outcomes can be achieved through a variety of causes) and multifinality (i.e., the same developmental event can cause a variety of different outcomes dependent on individual factors). By connecting overlapping exposures to low family income and air and water pollution over the course of adolescence to subsequent patterns of autonomic functioning in Mexican-origin youths, this study contributes to the growing body of literature incorporating economic, sociological, and neurobiological lenses to understand the contexts and consequences of growing up in poverty for marginalized and minoritized communities.

Importantly, whether or not alterations to normative stress physiology are adaptive in the context in which they develop may underpin the variability found in developmental trajectories of health and well-being (Del Guidice et al., 2011; Ellis & Del Giudice, 2014; Hostinar et al., 2021). In studying developing stress physiological systems, understanding the entire context in which patterns emerge is crucial for discerning whether profiles of stress responsivity are adaptive or maladaptive (Del Guidice et al., 2011; Ellis & Del Giudice, 2014; Hostinar et al., 2021). Investigating the neurobiological impact of experiencing chronic environmental stress in both the physical (toxins) and social domains (neighborhood disadvantage) provides a unique opportunity for unpacking physical versus

psychological mechanisms driving alterations in ANS functioning. Regarding the social domain, variation in neighborhood social context predicting alterations in normative autonomic activity can be interpreted as divergent profiles of adaptation resulting from differences in the severity of the neighborhood stressor (Del Guidice et al., 2011; Hostinar et al., 2021). However, when considering the objective nature of the physical domain, it is difficult to envision toxin exposure predicting similar alterations as anything other than an adverse environmental impact on normative functioning. As such, differentiating between biological and psychological factors underlying the neurobiological consequences of chronic stress requires domain-specificity.

Evidence of alterations to autonomic functioning following poverty and toxin exposure has implications for physical and mental health. If left uninterrupted, ANS dysfunction has serious implications for normative brain development (Koenig, 2020), elevated systemic inflammation (Fagundes et al., 2013; Kraft & Kraft, 2021; Shields & Slavich, 2017), and relatedly, increases in physical and psychological health problems, including cardiovascular disease (Berntson et al., 2008; Thayer et al., 2010), metabolic syndrome (Rodriguez-Colon et al., 2015), anxiety (Friedman, 2007), and depression (Stone et al., 2020). During adolescence, neurobiological sensitivity to the environment is heightened (Berenbaum et al., 2015; Eiland and Romeo, 2013) and alterations to ANS functioning are proposed to disrupt trajectories of brain development, stress and immune system physiology (Koenig, 2020). Thus, our findings together with past literature could suggest that ANS dysfunction is one plausible mechanism by which exposure to the context of poverty and environmental toxins increases the risk of physical and mental health problems, including metabolic syndrome, diabetes, and internalizing problems (Koenig, 2020; Rodriguez-Colon et al., 2015; Berntson, 2019). This contributes to health disparities for people of color, who disproportionately experience racist medical practices including inequitable access to and quality of medical treatment (Substance Abuse and Mental Health Services Administration [SAMHSA], 2020). This inequality compounds the inordinate elevation of cardiovascular morbidity and psychopathology in marginalized racial and ethnic groups as the lack of treatment inevitably worsens health outcomes.

Limitations and future directions

The present study was not without limitations. First, since this study was not originally designed to investigate the effects of environmental toxins and neighborhood socioeconomic status on youths' stress physiology, our findings must be interpreted in light of our specific sample of Mexican-origin youth from predominantly low-income households living in Northern California. While informative and useful for strengthening the field's understanding of the social, economic, and environmental challenges this community faces, these findings are not especially generalizable to other community samples or to the general population, as levels and variability of environmental toxin exposure, neighborhood SES, and income might differ substantially.

Secondly, the administrative data publicly available to us were highly variable in their granularity. Some variables were available at a yearly level (i.e., $PM_{2.5}$ and neighborhood education), while other indicators only had fewer measurement occasions that covered the

same time period (water quality, housing burden), with the exception of neighborhood unemployment. We attempted to address this issue by merging the data yearly depending on their census tract of residence, which also allowed us account for participants' mobility between census tracts. Since our analyses explored the unique effects of average exposure to each of these risks across adolescence by modelling each predictor as a latent common factor across time, we do not believe that this difference resulted in extreme bias. It is plausible that differences in granularity might have contributed to measurement error, which can be partially addressed using structural equation models (Rhemtulla, 2016). Additionally, our analysis was unable to parse within-tract differences in water exposure rates, where contaminants (e.g., lead) also depend on household and plumbing infrastructure (Clark et al., 1985).

Information on socioeconomic disadvantage and toxin exposure prior to age 10 was not collected, thus limiting the consideration of how exposure during infancy and early childhood may have contributed to adolescents' autonomic activity. Social and economic adversity early in life has been linked with chronic inflammation and autonomic dysfunction (Garcia Duenas, Olvera Alvarez et al., 2018, Fagundes et al., 2013), which may persist into adolescence (Koenig, 2020). Likewise, it is possible that toxin exposure and neighborhood effects on the ANS are partially explained by environmental continuity, or the similarities between current and prior exposure. Further, although neurobiological changes triggered by puberty might create more opportunities for external factors to influence internal physiological processes (Berenbaum et al., 2015; Eiland & Romeo, 2013), we did not control for pubertal stage in our analyses, as this measure was not available during our early adolescent time-point. Thus, it is crucial to interpret our findings regarding prolonged sensitivity of the ANS related to prior experiences of poverty and pollution throughout early to mid-adolescence in light of these limitations. However, our results suggest that chronic socioeconomic and environmental threats may continue to have effects on neurobiological maturation beyond the childhood period, and future work should examine pubertal stage, timing, and tempo as possible moderators of this association.

Next, there is ongoing debate about the use of different baseline ANS recording conditions in developmental research (Beauchaine et al., 2019; Utendale, 2014). Traditional baseline tasks require participants to sit still and quietly for a period equal to or longer than three minutes to achieve a wakeful resting state. Other studies sometimes use "vanilla" baselines (e.g. watching a neutral, non-evocative video) to ensure participants sit still for a set period of time, or present them with comparable demands to a task without the specific challenge of the task (Zisner & Beauchaine, 2016). However, the vanilla baseline approach may itself induce ANS reactivity through attention allocation mechanisms (Shader et al., 2018). To avoid attention-induced confounds and minimize novelty effects (Zisner & Beauchaine, 2016), we assessed ANS activity during a movement- and stimulus-free quiet baseline condition outside the MRI scanner, approximately one hour after arriving at the research center. Given that we do not compare a restful condition inside the scanner to the social exclusion task, we cannot disentangle whether participants' autonomic activity during Cyberball was a response to the psychological experience of the specific task or to the more general experience of doing a task within an unfamiliar and potentially intimidating MRI scanner.

Additionally, it is important to note that the current analysis uses data from a study that was designed to test different aims and hypotheses, *Cyberball* was not selected with an a-priori knowledge that participants' physiological reactions to the social challenge would be used in future studies to examine the neurobiological impact of neighborhood socioeconomic disadvantage and environmental toxin exposure. It is possible that ANS reactivity to other tasks would show different associations with environmental toxins, and we recognize that this is an important area of future research. However, the fact that we found significant differences in ANS responses to *Cyberball* were related to toxin exposure might suggest that these associations would be even stronger if we were to choose a task that was more directly related to the nature of the provocations that we measured in the environment. Further, this study included a single time point capturing mid- to late-adolescents' autonomic physiology, limiting our ability to test changes in stress physiology resulting from social disadvantage and chronic stress exposure. Longitudinal studies that are already collecting physiological data could be leveraged to explore whether neighborhood disadvantage and environmental toxin exposure predicts alterations to developing stress physiology, especially in ethnically and economically diverse adolescents (Trentacosta & Mullingan, 2020).

Despite strong evidence for the negative effects of chronic stress on adolescent psychophysiology, many studies in environmental and developmental psychology take a unidimensional approach by measuring one source of disadvantage (e.g., low family income, water contamination, $PM_{2.5}$) as it relates to one physiological system (e.g., ANS) using cross-sectional designs. In studying the health consequences of chronic stress, an important consideration is whether effects are driven by mechanisms of psychology, biology, or a mix of both. The current findings echo prior work (Olvera-Alvarez et al., 2018; Trentacosta et al., 2016) providing evidence that environmental toxin exposure catalyzes a chain of biological alterations across many bodily systems, which may ultimately increase risk of serious physical and psychological health problems later in life. Designing and conducting multidimensional explorations of chronic stress as it relates to multisystem stress physiology (e.g., concurrent HPA functioning and inflammation) would further inform the field's understanding of how stress "gets under the skin" to disrupt health over time.

Contrary to previous studies demonstrating the negative contributions of low neighborhood SES on ANS functioning, we found no evidence for this over and above toxin exposure and family income. This could potentially be explained by the fact that neighborhood SES is typically measured as a proxy for a host of other co-occurring social threats that have been linked to neurobiological, physical, and mental health problems in youth via upregulation of stress response systems (Kim et al., 2013). Among these, community crime and violence (Mellman et al., 2018; Tawakol et al., 2019), lack of social cohesion and sense of safety (Hill et al., 2005), access to illicit substances (Leventhal & Newman, 2010), or abandoned dwellings and noise (Coley et al., 2013; Hahad et al., 2019). It is plausible these null findings reflect an intertwining of both biological and psychological mechanisms we could not account for in the current study design. Additionally, our measure of neighborhood unemployment only covered the ages of 10 to 13, and no data at the census tract level was found for subsequent years. Given that impoverished Latinx youth experience higher rates of community crime, neighborhood violence, and systemic discrimination (DeNavas-Walt, 2010), future studies should combine objective and subjective measures of neighborhood

quality to capture participants' exposure to threatening environments that could potentially disrupt the ANS.

We did not explore individual-level and demographic variables as they interact with risk factors to impact developing neurobiology. Current integrated socio-environmental models of health and well-being (Olvera Alvarez et al., 2018, Fagundes et al., 2013) and novel empirical findings exploring *interactions* between these distal environmental factors, proximal psychosocial stressors (e.g., life stressors, parenting; Manczack et al., 2020), and individual-level risk factors (psychological and physiological characteristics, Miller et al., 2019) suggest that the nature of the relations between environment and health may be multifaceted and interdependent. In the current study, depression-risk as indexed by depressive symptoms two and a half years earlier was included as a covariate due to an oversampling for adolescents experiencing depressive symptoms. Our analyses revealed an unexpected effect linking greater depression-risk to parasympathetic dominance at rest and during acute stress above and beyond that of poverty and toxin exposure, indicating that depressive symptomatology plays a unique role in parasympathetic activity. Gender, age, and BMI were also included as covariates, each with unique effects in autonomic balance and regulation. Future studies should explore possible moderation effects of history of depressive symptoms, biological sex, age, and BMI suggested by the findings herein.

Lastly, future research taking a strengths-based approach by incorporating indices of natural environmental supports (e.g., green and blue spaces) and community resilience (e.g., neighborhood cohesiveness, women's health organizations, and family support networks) would appropriately balance the primarily risk-focused line of inquiry we have discussed. Given past work showing the many physical and psychological health benefits associated with green/blue spaces (Crouse et al., 2018; Mygind et al., 2021; Thompson et al., 2012, 2014; Y. Zhang et al., 2020) and community social support (Anthony & O'Brien, 2002; Farrell et al., 2015; Wight et al., 2006), this is an important direction for future studies.

Conclusions

In sum, our results compliment the field's understanding of environmental inequality by demonstrating evidence of environmental toxins and poverty "getting under the skin", the co-occurrence and overlapping presence of environmental toxins and social stressors, and their unique and additive impacts on developing autonomic stress physiology. Building on research demonstrating the health benefits of air and water pollution reduction programs, community revitalization initiatives, and access to green spaces early in development (Pew Research Center, 2018; Robert Wood Johnson Foundation, 2011; Schraufnagel et al., 2019; Wood & Smyth, 2020), this study underscores the need to increase funding for programs targeting communities historically and currently disadvantaged and disenfranchised in the existing sociopolitical structure. Moreover, these findings contribute to the growing literature exploring how racial, social and environmental inequalities jointly impact health and well-being across the lifespan (Hastings et al., 2022; Morello-Frosch et al., 2006; 2011; Olvera Alvarez et al., 2018). This line of inquiry has the potential to inform policy and public health initiatives designed to dismantle institutional and environmental racism and contribute to the

remediation of the systemic inequality experienced by racial/ethnic and economic minorities in the United States.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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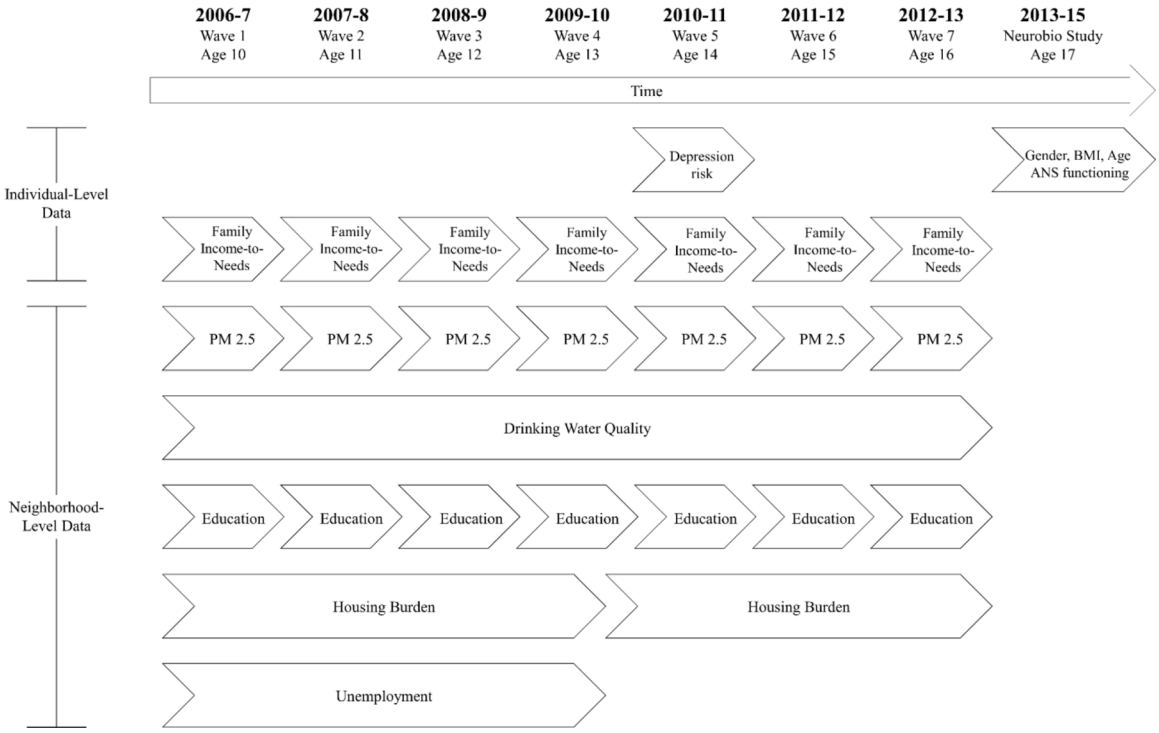

Figure 1. Visualization of complete study timeline matched with public toxin and neighborhood data

Note. Family Income-to-Needs, $PM_{2.5}$, and neighborhood educational attainment were available yearly. Drinking water quality reflects one cumulative score spanning 2005–2013. Neighborhood housing burden reflects two cumulative scores spanning 2006–2010 and 2010–2013. Neighborhood unemployment reflects one cumulative score spanning 2006–2010.

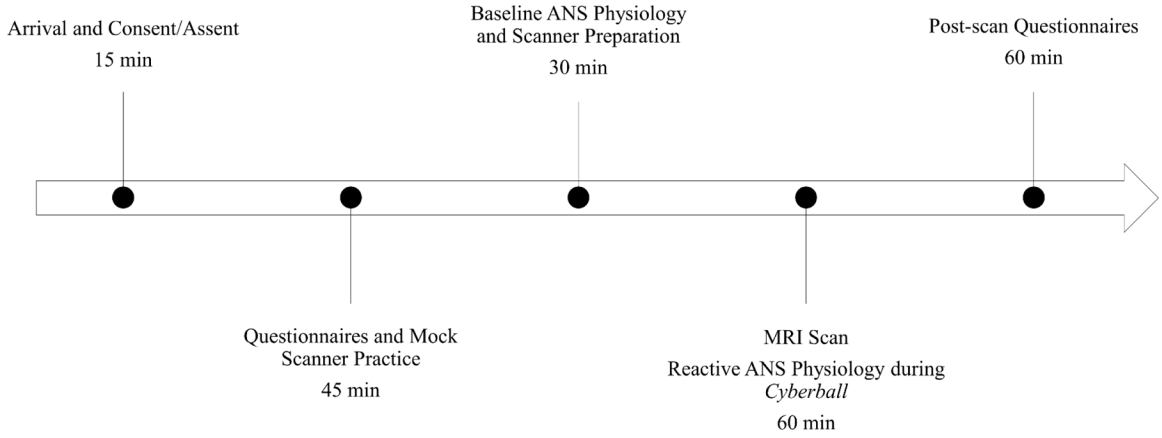

Figure 2.
Visualization of neurobiological sub-study timeline

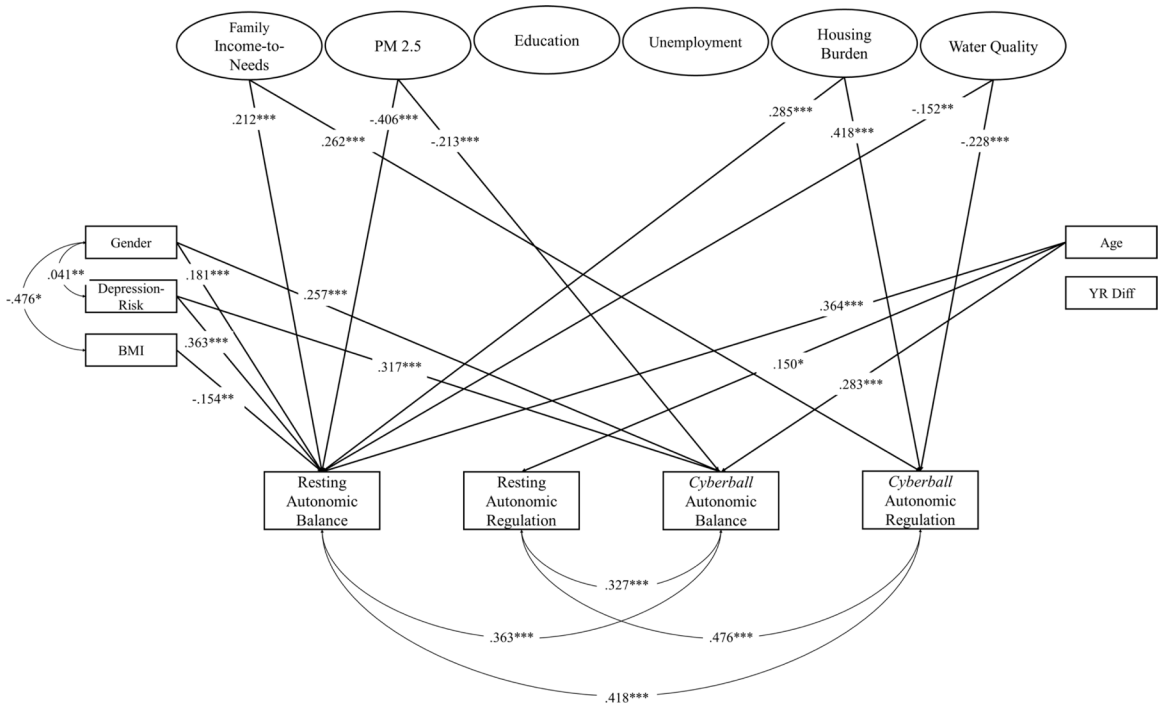

Figure 3. Full Model Results Path Diagram

Note. Only significant paths displayed. *Cyberball* refers to the social exclusion task. Covariances between environmental risks are not displayed.

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Table 1

Descriptives and Zero-order Correlations of Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1 Gender	--																	
2 Depression risk	**0.17***	--																
3 Age	−0.01	−0.11	--															
4 BMI	**0.15****	−0.01	0.05	--														
5 Family Income-to-Needs	0.01	−0.10	0.04	*0.12* [t]	--													
6 Neighborhood Unemployment	0.00	−0.02	−0.04	−0.10	**−0.25*****	--												
7 Neighborhood Education	0.00	−0.07	0.05	0.09	**0.46*****	**−0.37*****	--											
8 Neighborhood Housing Burden	−0.06	0.03	−0.11	0.02	**−0.41*****	**0.24*****	**−0.52*****	--										
9 PM 2.5	−0.02	−0.04	0.07	−0.09	**−0.19*****	**0.23*****	**−0.30*****	**0.39*****	--									
10 Water quality	0.07	0.06	−0.02	−0.01	0.01	0.09	**−0.32*****	**0.40***	0.08	--								
11 Baseline RSA	**0.19** [*]	0.01	0.04	**−0.23*****	0.03	−0.05	0.11	−0.11	−0.05	−0.10	--							
12 Baseline SCR	**−0.16**[*]	*−0.13* [t]	0.01	**0.14** [*]	−−0.01	0.05	−0.03	0.02	*0.12* [t]	0.03	*0.13* [t]	--						
13 *Cyberball* RSA	**0.15** [*]	0.04	0.04	**−0.26*****	−0.01	0.01	0.04	−0.09	−0.03	0.00	**0.66*****	0.04	--					
14 *Cyberball* SCR	**−0.16**[*]	−0.05	0.02	**0.14** [*]	−0.06	0.07	−0.02	0.08	0.03	−0.04	−0.08	**0.20*****	0.03	--				
15 Resting Autonomic Balance	**0.24*****	0.10	0.04	**−0.27*****	0.02	−0.07	0.12	−0.10	**−0.14**[*]	−0.1	**0.66*****	**−0.66*****	**0.47*****	**−0.21*****	--			
16 Resting Autonomic Regulation	0.02	−0.10	0.04	−0.050	0.01	−0.01	0.05	−0.06	0.07	−0.05	**0.75*****	**0.75*****	**0.46*****	0.07	−0.01	--		

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
17 *Cyberball* Autonomic Balance	**0.21*** **	0.09	−0.01	**−0.29*** **	0.03	-0.03	0.06	−0.10	**−0.16***	0.00	**0.55*** **	−0.10	**0.71*** **	**−0.68*** **	**0.49*** **	**0.30*** **	--	
18 *Cyberball* Autonomic Regulation	0.00	−0.02	0.05	−0.10	−0.06	0.05	−0.01	0.00	0.03	−0.04	**0.41*** **	**0.17** *	**0.73*** **	**0.70*** **	**0.19** *	**0.37*** **	0.04	--
Mean	48%	0.76	17.15	25.35	1.18	8.48	16.93	12.16	10.89	405.84	6.82	2.45	6.17	2.44	−0.03	−0.02	0.00	0.00
SD	--	0.43	0.42	5.65	0.66	4.20	10.76	5.63	1.39	288.19	0.98	2.25	1.11	1.91	1.32	1.50	1.37	1.41
Min	1	0	15.72	15.75	0.22	0.23	2.28	0.14	8.36	66.63	4.04	0.00	3.28	0.00	−3.77	−3.39	−4.08	−3.05
Max	2	1	18	44.87	3.73	20.26	32.7	32.7	13.81	973.88	9.09	9.09	9.10	7.87	2.63	4.9	2.61	3.73

Note. Environmental risks and Family Income-to-Needs are latent means derived from an SEM model including Waves 1 to 7 of these variables only. For year-to-year descriptives see Table S1. Neighborhood Unemployment, Education, and Housing Burden are measured as percentages. *Cyberball* refers to the social exclusion task.

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Table 2

Factor loadings and intercepts for Toxin and SES latent variables

	Factor Loading Est	(SE)	95% CI	
Income-to-Needs				
Wave 1	1	0	1	1
Wave 2	0.915	0.021	0.875	0.955
Wave 3	0.966	0.024	0.918	1.014
Wave 4	0.939	0.024	0.891	0.987
Wave 5	0.963	0.023	0.917	1.009
Wave 6	1.048	0.025	0.998	1.097
Wave 7	1.078	0.030	1.020	1.137
PM 2.5				
Wave 1	1	0	1	1
Wave 2	0.687	0.028	0.633	0.742
Wave 3	0.834	0.008	0.818	0.851
Wave 4	0.806	0.006	0.794	0.818
Wave 5	0.802	0.006	0.790	0.814
Wave 6	0.796	0.005	0.785	0.806
Wave 7	0.814	0.006	0.803	0.825
Education				
Wave 1	1	0	1	1
Wave 2	1.016	0.026	0.966	1.067
Wave 3	1.048	0.026	0.998	1.098
Wave 4	1.070	0.028	1.016	1.124
Wave 5	1.022	0.030	0.964	1.080
Wave 6	1.024	0.030	0.966	1.082
Wave 7	1.071	0.037	0.998	1.144
Unemployment				
Wave 1	1	0	1	1
Wave 2	1.121	0.022	1.078	1.164
Wave 3	1.155	0.021	1.114	1.196
Wave 4	1.175	0.027	1.123	1.228
Housing Burden				
Wave 1	1	0	1	1
Wave 2	0.967	0.007	0.953	0.981
Wave 3	1.017	0.016	0.985	1.050
Wave 4	1.184	0.029	1.127	1.240
Wave 5	1.139	0.025	1.091	1.187
Wave 6	1.149	0.026	1.099	1.200
Wave 7	1.142	0.027	1.090	1.194

	Factor Loading Est	(SE)	95% CI	
Water Quality				
Wave 1	1	0	1	1
Wave 2	1.026	0.013	1.000	1.052
Wave 3	1.053	0.026	1.001	1.104
Wave 4	0.958	0.028	0.904	1.012
Wave 5	1.059	0.026	1.008	1.111
Wave 6	1.037	0.026	0.986	1.088
Wave 7	0.967	0.026	0.917	1.018

Table 3.

Autonomic Balance and Regulation Full Model Parameters

	Resting Autonomic Balance			**Resting Autonomic Regulation**			***Cyberball* Autonomic Balance**			***Cyberball* Autonomic Regulation**		
Predictor	***B***	**(SE)**	***p***	***B***	**(SE)**	***p***	***B***	**(SE)**	***p***	***B***	**(SE)**	***p***
Gender	**0.181**	**0.127**	**0.000**	0.069	0.167	0.310	**0.257**	**0.160**	**0.000**	−0.052	0.201	0.424
Depression Risk	**0.363**	**0.152**	**0.000**	0.044	0.200	0.520	**0.317**	**0.191**	**0.000**	−0.034	0.240	0.607
Age	**0.364**	**0.157**	**0.000**	**0.15**	**0.206**	**0.035**	**0.283**	**0.197**	**0.000**	0.052	0.247	0.441
BMI	**−0.154**	**0.011**	**0.003**	0.058	0.014	0.410	−0.068	0.014	0.241	−0.056	0.017	0.410
YR diff	0.061	0.137	0.240	−0.046	0.180	0.516	−0.075	0.173	0.196	***0.131***	***0.217***	***0.054***
Family ITN	**0.212**	**0.112**	**0.000**	***0.149***	***0.148***	***0.064***	***0.112***	***0.141***	***0.091***	**0.262**	**0.177**	**0.001**
PM 2.5	**−0.406**	**0.051**	**0.000**	−0.030	0.067	0.715	**−0.213**	**0.064**	**0.001**	−0.115	0.080	0.141
Water Quality	**−0.152**	**0.024**	**0.003**	0.073	0.031	0.288	0.078	0.030	0.169	**−0.228**	**0.037**	**0.001**
Education	−0.057	0.075	0.376	0.087	0.099	0.322	***−0.138***	***0.095***	***0.058***	0.010	0.119	0.904
Unemployment	−0.082	0.163	0.129	−0.065	0.214	0.381	−0.063	0.204	0.303	−0.068	0.256	0.336
Housing Burden	**0.285**	**0.174**	**0.000**	0.049	0.227	0.628	−0.073	0.218	0.383	**0.418**	**0.247**	**0.000**
Fit Statistics	*CFI*	*TLI*	*SRMR*	*RMSEA*								
	0.912	0.905	0.077	0.064								

Note. B coefficients are standardized. ITN = Income-to-Needs. *Cyberball* refers to the social exclusion task. YR difference denotes the time difference (in years) between participant's Wave 7 visit and scan date. Higher values in autonomic balance and regulation indicate parasympathetic dominance and co-activation of both branches, respectively.

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