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Patterns of poverty across adolescence predict salivary cortisol stress responses in Mexican-origin youths

Lisa E. Johnson [a,b,*], Luis A. Parra [b,c,d], Elisa Ugarte [b,c], David G. Weissman [e], Sasha G. Han [f], Richard W. Robins [a], Amanda E. Guyer [b,c], Paul D. Hastings [a,b]

[a] Department of Psychology, University of California, Davis, USA
[b] Center for Mind and Brain, University of California, Davis, USA
[c] Department of Human Ecology, University of California, Davis, USA
[d] Suzanne Dworak-Peck School of Social Work, University of Southern California, USA
[e] Department of Psychology, Harvard University, USA
[f] Department of Psychology, St. Bonaventure University, USA

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ABSTRACT

Poverty is a chronic stressor associated with disruptions in psychophysiological development during adolescence. This study examined associations of chronic poverty and income changes experienced from pre- to mid-adolescence with hypothalamic-pituitary-adrenal (HPA) axis stress responses in late adolescence. Participants ($N = 229$) were adolescents of Mexican-origin (48.7% female). Household income (converted to income-to-needs ratios) was assessed annually when children were 10–16 years old. At 17 years, adolescents completed *Cyberball*, a social exclusion simulation task while undergoing a functional magnetic resonance imaging scan. Saliva samples were collected prior to and five times over a 50-minute period following the scan, from which salivary cortisol was assayed. Results showed that differential trajectories of poverty from ages 10–16 predicted HPA axis activity at age 17. Relative to others, distinct HPA suppression (hyporeactivity) was demonstrated by youth who started adolescence in deep poverty and were still living in poverty at age 16 despite experiencing some income gains. Youth from more economically secure families evinced typical cortisol increases following the lab stressor. These results suggest that subsequent HPA functioning varies as a function of economic status throughout adolescence, and that efforts to increase family income may promote healthy HPA functioning for youths in the most impoverished circumstances.

1. Introduction

Poverty, a state in which essential financial needs for a minimum standard of living are unmet, is linked with disruptions to psychological and neurobiological functioning in children and adolescents (Page et al., 2016). The chronicity of poverty exposure in particular may shape its impact on development (Leung and Shek, 2011). Persistent poverty can jeopardize adolescent well-being to a greater extent than temporary poverty (Evans et al., 2012), with fluctuations in family income also affecting development, particularly in low-income environments (Duncan et al., 2014).

Chronic exposure to stressors like poverty is linked with children's adrenocortical functioning (Evans et al., 2012). Adolescence is a developmentally-sensitive period when chronic poverty may have heightened effects (Mazza et al., 2017), as the neurobiological maturation triggered by puberty creates more opportunities for external factors to influence internal processes (Eiland and Romeo, 2013), with potential long-lasting adverse health consequences (Evans et al., 2012). Over 50% of children under 18 in California are Latinx, and as evident across the U. S., Latinx youth and families are structurally and systematically marginalized and over-represented in poverty contexts (Annie E. Casey Foundation, 2017; Myers, 2009) that can disrupt HPA activity at rest and in response to stressors. Yet, this same community has been under-represented in neurobiological studies, creating a gap in research on the links among poverty dynamics during adolescence and marginalized adolescents' hypothalamic-pituitary-adrenal (HPA) functioning.

According to the biopsychosocial model of cumulative vulnerability and minority health (Myers, 2009), people at the intersections of

* Correspondence to: Center for Mind and Brain, University of California, Davis, 202 Cousteau Pl., CA 95618, USA.
E-mail address: lisjohnson@ucdavis.edu (L.E. Johnson).

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marginalized social group memberships are often disadvantaged and may experience disruptions to their HPA functioning (Parra and Hastings, 2018). Relative to other sociodemographic groups, racial/ethnic minority families experience greater exposure to community crime, crowding, and systematic discrimination (Roy and Raver, 2014), all of which are associated with poverty. This complex, multi-faceted risk profile may contribute to disruptions in physiological functioning for Latinx youth that differ from those observed in other, previously studied populations. Thus, we examined effects of the timing and chronicity of poverty across adolescence on Mexican-origin adolescents' basal and acute HPA activity.

1.1. Poverty and the HPA axis

About 20% of American children live in poverty, and another 20% live in low-income households (Koball and Jiang, 2018). Poverty has been linked with disruptions in every level of neurobiological functioning (Page et al., 2016), including hypoactivation of the HPA axis (Evans et al., 2012; Lupien et al., 2000), with reduced basal and diurnal cortisol levels found in children (Zalewski et al., 2012) and adolescents (Marsman et al., 2012) living in poverty. Childhood poverty predicts disrupted HPA functioning in early and middle adolescence (Evans and Kim, 2012), with longer duration of exposure predicting reduced acute cortisol reactivity to stressors (Evans et al., 2012). Whether chronicity of poverty across adolescence matters for youths' subsequent HPA functioning has received less attention.

Poverty is a dynamic context that fluctuates with familial and societal factors (Leung and Shek, 2011). For children in lower-income homes, income gains are positively associated with better health outcomes (Duncan et al., 2014). Income losses are posited to increase psychological distress, possibly due to both diminished resources and increased relative deprivation (Runciman and Runciman, 1966). Poverty is nonbinary, as economic adversity is experienced both below and above the poverty line (Koball and Jiang, 2018). Experiencing income losses when in a precarious economic state (e.g., hovering around the poverty line) may be particularly disruptive to physiological regulation since relatively small changes in income may incur greater financial and emotional distress as basic material needs can no longer be met (Runciman and Runciman, 1966).

Poverty experienced earlier, compared to later, in adolescence may have a pronounced impact on later stress regulation due to the rapid neurobiological maturation and increased adrenocortical sensitivity associated with puberty (Dahl and Gunnar, 2009). Weissman et al. (2018) found that for Mexican-origin adolescents living in poverty, increasing family income from 10 to 16 years predicted greater functional connectivity of the brain's default mode network (DMN); income changes were unrelated to functional connectivity in adolescents living in more affluent families. Because the DMN supports social cognitive functions, increased DMN connectivity may reflect an adaptation of improved capacity for social information processing and navigation of complex social environments (Weissman et al., 2018). We are unaware of a comparable prospective longitudinal study linking poverty dynamics across adolescence with adrenocortical functioning.

1.2. Adolescent adrenocortical functioning

Basal and acute adrenocortical activity is greater in adolescence than in childhood and adulthood (Dahl and Gunnar, 2009). Adolescents mount an acute HPA response to being in a magnetic resonance imaging (MRI) scanner (Eatough et al., 2009), which was the context of the current study. Additionally, the HPA axis is particularly sensitive to unpredictable, uncontrollable, and personally relevant social stressors (Gunnar and Quevedo, 2007). Adolescence is characterized by increased peer orientation and sensitivity to social acceptance or rejection (Schriber and Guyer, 2016). In the present study, *Cyberball* (Williams et al., 2000) was used to elicit an acute stress response to social exclusion. Although *Cyberball* has not consistently produced HPA reactivity in adults (Radke et al., 2018), elevated cortisol following *Cyberball* has been observed in adults with stronger need-to-belong (Beekman et al., 2016), and in African-American adults who experience the exclusion as racially-motivated (Peterson et al., 2020). Need-to-belong reflects the motivation for social acceptance that normatively peaks in adolescence (Schriber and Guyer, 2016). Latinx adolescents are members of a marginalized community that experiences systemic social exclusion (Parra and Hastings, 2018). Collectively, it is plausible that family poverty in early adolescence may influence Mexican-origin adolescents' acute HPA responses to social exclusion via *Cyberball* experienced within an MRI scanner.

Theories differ regarding the likely nature of that predictive association. Allostatic load theory posits that chronic stress presents a constant demand on bodily resources and can incite architectural and molecular changes to neural systems (McEwen, 2000). The resultant breakdown of many bodily systems can produce either exaggerated stress responses, the *hyperactivity hypothesis* (Gold et al., 1988), or underactive stress responses, the *hypoactivity hypothesis* (Kudielka et al., 2009). Neither adaptation is beneficial, as both have been associated with psychopathology in adolescence (Chrousos, 2009). Both hyperactivity and hypoactivity may be evident, but at different points of exposure to chronic stressors, such as poverty (Miller et al., 2007). Months or years of chronic stressor exposure may exhaust the capacity of the HPA axis to mount acute responses, evidenced as hyporeactivity (Ulrich-Lai and Herman, 2009). We expected the duration and stability of family poverty across adolescence to predict adolescents' subsequent HPA activity in accord with the hypoactivity hypothesis.

1.3. Hypotheses

In a large sample of Mexican-origin adolescents, we examined how experiences of chronic poverty and changes in family income during pre- to mid-adolescence predicted basal cortisol and acute HPA responses in late adolescence to a social exclusion task administered during an MRI. In accord with allostatic load theory, we expected that (1) experiences of poverty over the course of adolescence would predict HPA hypoactivity, and (2) deeper and more persistent poverty would exaggerate this association, such that youths who experienced deep poverty for a prolonged period of time would show the most evidence of cortisol hypoactivity (i.e., low basal levels and no acute response). Given the neurobiological significance of the early adolescent period (Dahl and Gunnar, 2009), among youths who experienced changes in poverty across adolescence, we expected to find (3) a developmental timing effect with poverty exposure in early adolescence predicting stronger physiological effects than exposure in later adolescence.

2. Method

2.1. Participants

Participants included 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 HPA assessment) and their parents enrolled in a neurobiological sub-study of the California Families Project (CFP), a prospective, longitudinal study designed to examine developmental risk and resilience factors. Participants of the main CFP 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.

2.2. Procedure

The main CFP study assessments were completed annually starting in fifth grade using questionnaire, interview, and observational data collection methods administered in each family's home. The sub-study

of the CFP that included the current study participants was conducted at a university imaging research center, and all visits were scheduled to start in the afternoon to early evening. The sub-study was designed to examine neurobiological mechanisms underlying depression and thus oversampled for adolescents from the main CFP based on adolescent reports of depressive symptoms on the Computerized Diagnostic Interview Schedule for Children-IV (C-DISC; Shaffer et al., 2000), and General Distress and Anhedonic Depression items of the Mood and Anxiety Symptom Questionnaire (MASQ; Watson et al., 1995) at 14 years. Scores above the sample median on any of these three measures indicated presence of risk for depression. A dichotomous recruitment variable referring to depression-risk (1 = *scored above the median on any recruitment measure; n* = 175, 0 = *scored below the median on all measures; n* = 54) was controlled for in analyses. All participants' parents provided informed consent and adolescents provided assent. This study was approved by the university's Institutional Review Board. All participants were compensated for their time and efforts.

2.3. Measures

Mothers reported the family's annual total household income when adolescents were 10–16 years old (7 waves of data), to the nearest $5000 increment (i.e., $30,001–35,000). Each increment corresponded to a number between 1 ("less than $5000″) and 20 ("$95,001 or more"). Mothers also reported the number of people living in the household. Following previous work with this sample (Weissman et al., 2018), 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, 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% (Wave 4) to 31% (Wave 2).

2.4. MRI experience including a social exclusion stressor

Fig. 1 shows a visualization of the present study's complete protocol for the MRI scan and neuroendocrine collection components; only the components pertinent to the current analyses are reported here. At 17 years, adolescents completed functional MRI (fMRI) scans. During the scan, participants completed three tasks. *Cyberball*, the social stressor task, was the second task administered, beginning on average 40 min after entering the scanner and ending approximately 10 min before the end of the scanning protocol. *Cyberball* (Williams et al., 2000) is a widely used, virtual ball-toss game designed to elicit feelings of ostracism, social exclusion, and rejection. 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 sex 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 an equal number of opportunities to toss the ball to the virtual players. During exclusion trials, the two virtual players included the participant by tossing the ball to them once, before excluding them entirely. There were 6 rounds of inclusion trials and 6 rounds of exclusion trials, each with 10–11 ball tosses of game play, presented in pseudo-random order with inclusion trials over-concentrated near the beginning, and exclusion trials over-concentrated at the end of the task. The task lasted approximately 9 min. Although *Cyberball* was administered during a neuroimaging scan, the current study examined adolescents' HPA responses, and thus, only cortisol data were included. Ten adolescents were ineligible for scanning; eight completed the task on a laptop computer outside of the scanner, for whom cortisol samples were used. Two participants experienced computer malfunctions with the task, therefore their cortisol samples were not used in analyses.

2.5. HPA axis assessment

Research assistants collected seven saliva samples using absorbent salivettes (Salivettes™, Salimetrics Inc., State College, PA) and two passive drool samples, before and after participants completed *Cyberball* and the MRI scan to assess HPA responses via salivary cortisol output. The basal sample (Sample 1) was collected approximately 1 hour after arriving at the research center, immediately prior to beginning the MRI scan preparation (M = 4:44 PM, SD = 2:20). During that first hour, adolescents acclimated to the scanning environment, trained on scanner tasks, and completed non-evocative questionnaires. Samples 2, 3, 4, 5, and 6 were collected 10, 20, 30, 40, and 50 min post-*Cyberball* (after the last *Cyberball* trial, corresponding to 1–40 min post-MRI scan, with sample 2 collected within a few minutes of having exited the scanner). Adolescents left the scanner room after providing Sample 2 and returned to a comfortable waiting room where they completed questionnaires while providing the remaining saliva samples. The first 25 min of questionnaires included measures of their experiences of the tasks administered in the scanner, and non-evocative questionnaires selected to minimize potential influence on cortisol output. The final 15 min of questionnaires included mental health assessments, administered last to avoid potential spillover of reactivity to the measures in salivary cortisol. Additional measures and tasks that were potentially more evocative were administered after the final saliva sample was collected. The current study used Samples 1–6 as they reflect the time course of HPA response to the *Cyberball* exclusion trials. One other Salivette sample and the two passive drool samples were excluded from analyses as they were not pertinent to the current research questions.

Saliva samples were assayed for cortisol at the Proteomics Core Facility at the UC Davis Genome Institute, using the standardized salivary protocol from Salimetrics Saliva Lab. All samples were assayed in duplicate using a high sensitivity enzyme immunoassay kit (Salimetrics

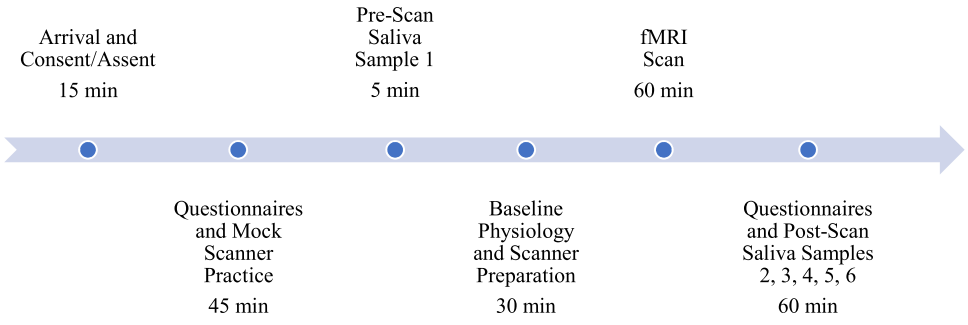

Fig. 1. Study visit timeline. Participants arrived and completed consent and assent procedures. This was followed by 45 min of questionnaires and practice in a mock scanner. After this, a pre-scanner saliva sample was taken. Participants then completed a 60-minute fMRI scan (during which *Cyberball* was administered between minutes 40 and 50 of the scan). Immediately following the scan, another saliva sample was taken. Following this, 4 more samples were taken every 10 min post-Cyberball. During this period of post-Cyberball sample collection, the participants were completing more questionnaires.

Inc., State College, PA) that had a minimum detection ranging from 0.007 to 1.8 μg/dL; intra- and inter-assay variabilities were 1.37% and 8.45%, respectively. Fourteen participants were missing all salivary cortisol values due to experimenter error in conducting the assays; those plates were discarded. There was blood contamination in all 6 samples for 1 participant, and too little saliva in 1 sample from another participant for assaying; these 7 samples were discarded. Any samples ($n = 17$) for which the raw data value was more than 3 *SD*s above or below the mean were considered outliers and removed from the data set prior to log transformation. Raw cortisol values (μg/dL) were $\log_{10}$ transformed to correct for leptokurtic and positive skews. Percent of valid, usable samples for adolescents ranged from 88% (Sample 1) to 90% (Sample 3).

2.6. Analytic strategy

Three sets of analyses were conducted to test study aims. First, a latent growth curve analysis in a structural equation model framework (McArdle and Epstein, 1987) was used to assess overall (linear) change in income-to-needs. This analysis was conducted in R-studio (R version 3.4.4) using the lavaan package (Rosseel, 2012) and full-information maximum likelihood (FIML) estimation of missing data. Repeated measures of income-to-needs ratios were used to calculate the latent intercept and slope of each participant's family income-to-needs across the 7 waves of data, centered at Wave 7 (16 years). Factor loadings of the income-to-needs intercept and slope were set so the intercept represented average income-to-needs at age 16 and the slope represented linear change in income-to-needs from age 10 to 16 years. Therefore, positive values of income-to-needs slope reflect increases in family income-to-needs ratios from 10 to 16 years, values of income-to-needs slope near zero reflect little or no change in income-to-needs ratios across the years of study, and negative values of income-to-needs slope reflect decreases in income-to-needs from 10 to 16 years.

As the majority of studies have focused on contemporaneous associations between income and physiology (Leung and Shek, 2011), we set the income-to-needs intercept at the wave most proximal in time to our HPA assessment. In doing so, our analyses could address the developmental timing of poverty exposure. If adolescents' current economic state drove the association between income and HPA functioning, we would expect only main effects of income-to-needs intercept. If poverty in early adolescence drove the effect, we would expect a main effect of income-to-needs slope or a significant income-to-needs intercept-by-slope interaction. Model fit was assessed with the chi-square (χ^2) goodness of fit statistic, comparative fit index (*CFI*; Bentler, 1990) and root mean square error of approximation (*RMSEA*; Browne and Cudeck, 1992). Model fit was considered excellent if the χ^2 *p*-value was non-significant, *CFI* was >0.95, *RMSEA* was <0.05, with the upper-bound of its confidence interval <0.08. The chi-square test is extremely sensitive to sample size and commonly results in a significant *p*-value when the sample size is large.

Second, growth trajectories of salivary cortisol in response to the fMRI-administered social exclusion task were examined using a two-level growth model with random intercepts and slopes (Goldstein, 2003) in Mplus 8.3 (Muthén and Muthén, 1998-2017) using maximum likelihood estimation. This approach was chosen based on prior use of multilevel, mixed, or hierarchical linear modeling to analyze both diurnal rhythms and acute responses in cortisol data (Shirtcliff et al., 2005). Interclass correlations were calculated from a no growth model to analyze between- and within-person variance in cortisol. We tested models with fixed and random effects of linear and quadratic time on cortisol, controlling for sample collection times as a time-varying covariate. Model fit comparisons were assessed using Akaike Information Criterions (AIC) and Bayesian Information Criterions (BIC) with lower values indicating better fit (Kuha, 2004).

Third, to address Hypothesis 1, a main effects model was run by combining analyses one and two, incorporating extracted growth factors for income-to-needs intercept and slope in predicting cortisol trajectories at the between-person level (fixed effects) with a random effect for quadratic time. Hypothesis 1 would be supported if we found significant main effects of income-to-needs intercept or slope, such that lower income at age 16 (low income intercept) or income losses over the adolescent period (negative income slope) predicted lower basal cortisol or decreased cortisol reactivity. To address Hypotheses 2 and 3, an interaction model including the income-to-needs intercept-by-slope interaction was conducted to test the effect of income change on cortisol as a function of average income-to-needs at age 16 (income intercept-by-slope interaction). Hypothesis 2 would be supported if we found a significant intercept-by-slope interaction, such that youths who remained in deep poverty throughout the adolescent period (e.g., youth in poverty at age 16 with a history of poverty across adolescence; low intercept and flat slope) evinced HPA hypoactivity as demonstrated by blunted basal or reactive cortisol. Hypothesis 3 pertains to dynamic changes in poverty across adolescence, and would be supported if we found a significant intercept-by-slope interaction, such that youths who started adolescence in deeper poverty than they were experiencing later in adolescence (e.g., a positive income slope combined with low income intercept) would manifest greater HPA hypoactivity than youths in families that were less impoverished in early adolescence than in mid-adolescence.

All independent and control variables were grand-mean centered prior to running the prediction models. Significant interaction effects ($p < 0.05$) were probed using the Johnson-Neyman regions of significance (RoS) technique and an online calculator (quantpsy.org/interact; Preacher et al., 2006). Simple slopes of the relation between income-to-needs intercept (average income-to-needs at age 16) and cortisol value was examined at −1 *SD* income slope (reflecting income-to-needs decreasing across ages 10–16), average income slope (reflecting small increases but relative stability in income-to-needs across adolescence), and +1 *SD* income slope (reflecting greater increases in income-to-needs). An alternative analytic model with the income-to-needs intercept centered at age 10 is provided in Supplemental Materials.

2.7. Covariates

The cortisol-only growth model and both of the prediction models (main effects and interaction) included depression-risk, sex, and medication use as covariates at the between-person level, and sample collection times at the within-person level. Sex and sample collection times were included to account for salivary cortisol's diurnal rhythm and potential sex differences in HPA responses (Kudielka et al., 2009). Depression-risk and medication use (e.g., corticosteroids, birth control) were included to ensure recruitment criteria did not impact HPA activity. Possibly due to the low variability in age at testing, age did not significantly correlate with any study measure and thus was not controlled for.

3. Results

3.1. Descriptive analyses

Table 1 displays descriptive statistics of all study variables, and partial correlations of the relation between income-to-needs from ages 10 to 16 years and salivary cortisol samples 1, 2, 3, 4, 5, and 6, controlling for depression-risk, sex, medication use, and sample collection times. Average total household income increments at age 10 was $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. Average household roster was 5.47 people (*SD* = 1.548). Using household roster to convert total household income into income-to-needs ratio, the mean income-to-needs at age 10 was 120% of the federal poverty line, with 47.8% of the sample living at or below the federal poverty line.

Table 1
Descriptive statistics and partial correlation coefficients.

Measure	*M*	*SD*	Cortisol 1	Cortisol 2	Cortisol 3	Cortisol 4	Cortisol 5	Cortisol 6
ItN 10 y	1.20	0.82	0.055	0.177*	0.175*	0.178*	0.196**	0.164*
ItN 11 y	1.14	0.72	0.271***	0.199**	0.169*	0.250***	0.238***	0.205**
ItN 12 y	1.22	0.86	0.082	0.161^{t}	0.165*	0.149^{t}	0.110	0.066
ItN 13 y	1.11	0.72	0.043	0.138^{t}	0.122^{t}	0.188**	0.192**	0.156*
ItN 14 y	1.14	0.76	-0.033	0.146*	0.167*	0.178*	0.170*	0.101
ItN 15 y	1.19	0.78	-0.017	0.135^{t}	0.109	0.149^{t}	0.098	0.054
ItN 16 y	1.28	0.81	-0.017	0.079	0.080	0.103	0.117^{t}	0.066
Sample 1 Collection Time (hh.mm)	16.56	2.36	–	–	–	–	–	–
Sex	1.49	0.50	–	–	–	–	–	–
Depression-risk	0.76	0.43	–	–	–	–	–	–
Medication Usage	0.12	0.33	–	–	–	–	–	–
M	–	–	–0.976	–1.048	–1.067	–1.053	–1.034	–1.033
SD	–	–	0.240	0.268	0.267	0.249	0.273	0.287

Notes: hh.mm = military time, hours and minutes since midnight; Sex = dummy coded where 1 = Male, 2 = Female; Depression-risk dummy coded where 0 = scored below the median on any depression symptom measure, 1 = scored above the median; ItN = Income-to-needs ratios; Cortisol # = Cortisol Sample # (measured in μg/dL). Partial correlations control for sample time, sex, medications, and depression-risk. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$, $^{t}p < 0.10$.

At the mean level, participants did not show evidence of acute HPA reactivity to the MRI scanner and *Cyberball* activities. Rather, cortisol decreased from sample 1 to 3 (20 min post-*Cyberball* stress task, 10 min post-MRI scan completion), paired $t(216) = -5.007$, 95% CI [−0.1217, − 0.0539], $p < 0.001$, and then increased from sample 3–6 (50 min post-*Cyberball* stressor task, 40 min post-MRI scan completion), paired $t(218) = 2.315$, 95% CI [0.0046, 0.0575], $p = 0.002$. By the final sample (6), on average, cortisol had not returned to pre-task levels, paired $t(212) = -2.721$, 95% CI [−0.0925, −0.0148], $p = 0.007$, consistent with a negative diurnal slope. Partial correlations revealed a fairly consistent pattern of higher income-to-needs across the first 5 waves (10–14 years) being associated with higher cortisol levels in most samples. This pattern was particularly pronounced for income-to-needs at ages 10 and 11.

3.2. Income dynamics

Income-to-needs was modeled using a latent linear growth model which showed acceptable fit to the data (χ^2 (23) = 70.956, $p < 0.001$, *CFI* = 0.961, *RMSEA* = 0.097). The mean parameter estimate for the intercept ($\mu_i = 1.208$, $p < 0.001$) was the average income-to-needs ratio at age 16 across all adolescents, and the estimate for the slope ($\mu_s = 0.016$, $p = 0.025$) was the average value of the change in income-to-needs across all time points. Across the sample, adolescents experienced increasing income-to-needs ratios at a rate of 1.6% each year such that at age 16 the average income-to-needs ratio was 1.208 (i.e., marginally above the poverty line). The intercept variance was significant ($\sigma_i = 0.519$, $p < 0.0001$), as was the slope variance ($\sigma_s = 0.005$, $p < 0.0001$), indicating significant inter-individual differences in adolescents' income-to-needs trajectories. Income-to-needs intercept was significantly correlated with slope ($r = 0.423$, $p = 0.001$), such that adolescents who experienced increases in income-to-needs ratio across ages 10–16 were likely to have higher average income-to-needs at age 16.

3.3. Salivary cortisol responses

Salivary cortisol was modeled using two-level modeling. The interclass correlation from the no growth model calculated between-person variance to be 69.59%, suggesting substantial inter-individual variation in adolescents' salivary cortisol responses to the lab stressor. After testing growth models controlling for sample times, the best fitting model was a quadratic growth curve model with random effects for intercept, linear slope, and quadratic slope (AIC = −542, BIC = −428). The mean parameter estimate for the intercept (i.e., basal cortisol; $\mu_i = -1.024$, $p < 0.001$) indicated the average basal cortisol value across adolescents before entering the scanner. The cortisol intercept had significant variance at the within ($\sigma_i = 0.012$, $p < 0.0001$) and between level ($\sigma_i = 0.044$, $p < 0.0001$). The mean linear slope ($\mu_i = -0.048$, $p = 0.038$) indicated the average linear change in cortisol across all samples. This average linear slope value represented the average decrease in cortisol from baseline to 50 min post-*Cyberball*. The linear slope had significant variance at the between level ($\sigma_i = 0.058$, $p < 0.001$). The mean quadratic slope ($\mu_{s1} = 0.022$, $p = 0.004$) is the average quadratic change in cortisol across all samples. This average quadratic slope value represents the average change in linear slope of cortisol across samples, from baseline to 50 min post-*Cyberball*, with more positive values indicating smaller decreases, or increases, in cortisol across progressive samples (a U-shaped quadratic slope). The variance of the quadratic slope was significant at the between level ($\sigma_{s1} = 0.006$, $p < 0.0001$). Cortisol intercept was significantly positively correlated with linear slope ($r = 0.503$, $p = 0.012$), indicating greater cortisol at baseline was associated with greater decreases across the following samples, but was not significantly correlated with the quadratic slope ($r = 0.390$, $p = ns$). Linear slope was significantly correlated with quadratic slope ($r = 0.948$, $p < 0.001$), such that adolescents who had smaller decreases, or even increases, in cortisol post-*Cyberball* were likely to exhibit slower quadratic change across the samples (i.e., a shallower or flatter U-shaped curve).

3.4. Main effect and interaction models

The final prediction model incorporated the linear growth model of income and the quadratic growth model of cortisol responsivity (Fig. 2). The best fitting model (i.e., having the lowest AIC and BIC values) included regression paths for the income variables and between-person covariates on the random intercept, linear and quadratic slope. Table 2 displays the model fits, unstandardized regression weights, and confidence intervals for the main effects and interaction models.

3.4.1. Main effects model: effect of income-to-needs intercept and slope on basal and acute cortisol

In testing Hypothesis 1, results revealed no significant main effects of income-to-needs intercept and slope predicting cortisol intercept (Table 2). There were significant negative associations of both saliva sample collection time and sex with cortisol intercept. Adolescents tested later in the afternoon or early evening, and female adolescents, had lower basal cortisol levels prior to the scan than adolescents tested earlier, and males. In testing Hypothesis 2, income-to-needs intercept significantly predicted linear and quadratic cortisol, such that lower average income-to-needs ratios at age 16 predicted greater initial decreases (linear slope) followed by greater increases across the later samples (quadratic slope), or stronger U-shaped curves. Conversely, higher average income-to-needs at age 16 predicted shallower, or flatter,

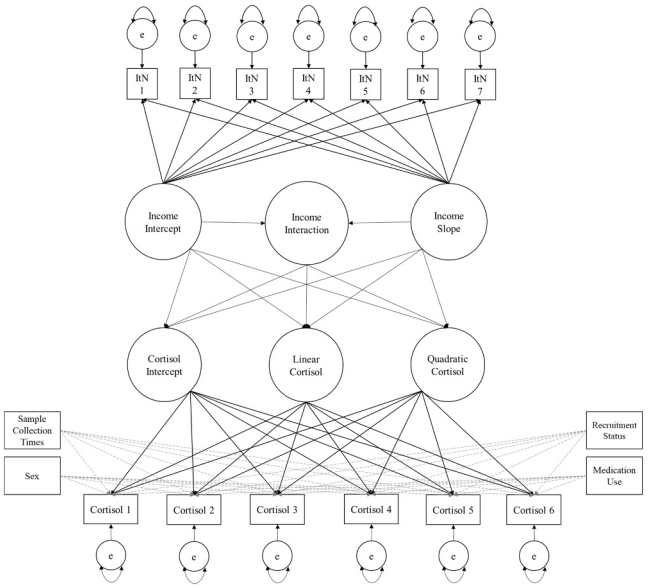

Fig. 2. Path diagram of average income-to-needs (intercept), income-to-needs change (slope), and income-to-needs intercept X slope interaction predicting acute HPA responses. *Note*: ItN = Income-to-needs ratios; Cortisol # = Cortisol Sample #; Income = Income-to-needs; Factor loadings for Income-to-needs intercept = 1, 1, 1, 1, 1, 1; Income-to-needs slope = −6, −5, −4, −3, −2, −1, 0; Cortisol intercept = 1, 1, 1, 1, 1, 1; Linear Cortisol = 0, 0.5, 1, 1.5, 2, 2.5; Quadratic Cortisol = 0, 0.25, 1, 2.25, 4, 6.25.

U-shaped curves of cortisol reactivity. Income-to-needs slope was not a significant predictor of linear or quadratic cortisol slopes. Lastly, adolescents' risk for depression (indexed by sub-clinical depressive symptoms on the C-DISC and MASQ) positively predicted linear cortisol slope, such that youths who reported greater depressive symptoms evinced flatter linear slopes, indicating less cortisol decline post-stressor.

3.4.2. Interaction model: effect of average income-to-needs by change in income-to-needs on acute cortisol response

In testing Hypothesis 3, the significant effects of average income-to-needs at age 16 predicting linear and quadratic cortisol were qualified by significant income-to-needs intercept by income-to-needs slope interactions. To interpret the interactions predicting linear and quadratic cortisol further, simple slopes were probed at ±1 *SD* of income-to-needs slope (Fig. 3a and b). There was a significant negative association between income-to-needs intercept and linear cortisol slope for higher (+1 *SD*; increasing income-to-needs; $\beta = -0.140$, $p = 0.0001$) and average income-to-needs slope (M_{slope}; stable income-to-needs; $\beta = -0.082$, $p = 0.011$) but not for lower income-to-needs slope (−1 *SD*; decreasing income-to-needs; $\beta = -0.023$, $p = 0.586$). Likewise, there was a significant negative association between income-to-needs intercept and quadratic cortisol slope for higher (+1 *SD*; $\beta = -0.046$, $p = 0.0003$) and average income-to-needs slope (M_{slope}; $\beta = -0.024$, $p = 0.034$) but not for lower income-to-needs slope (−1 *SD*; $\beta = -0.002$, $p = 0.901$). Thus, the most distinct HPA response profiles were exhibited by youths who entered adolescence living in deep poverty and, despite experiencing some income gains over the years, were still living below the poverty line at age 16. These youths evidenced a pattern of sharp cortisol decline across the initial samples, followed by slight increases in the final two samples (40–50 min post-*Cyberball*).

To clearly illustrate the nature of the two interaction effects, trajectories of cortisol were plotted at ± 1 *SD* of income-to-needs slope and intercept (Fig. 3c). Adolescents from families with lower income-to-needs at age 16 (−1 *SD* intercept) with a history of annual increases in income-to-needs from age 10 to age 16 (+1 *SD* slope) (hence, deeper poverty in early adolescence, but still impoverished at age 16) produced a cortisol profile characterized by a pronounced decrease in circulating levels across the first 4 samples, followed by a clear increase in the 5th and 6th samples. In contrast, adolescents from families whose income-to-needs remained stable across the years (M_{slope}) and reported average income-to-needs at age 16 ($M_{intercept}$) displayed a cortisol profile characterized by slow and small decreases in cortisol across the first 4 samples, followed by a slight increase in the 5th and 6th samples. Finally, adolescents from families with higher income-to-needs at age 16 (i.e., +1 *SD* intercept) with histories of increasing or decreasing income-

Table 2
Parameter estimates and model fit indices for the prediction of adolescents' HPA response profiles in relation to income dynamics across adolescence, with income-to-needs intercept centered at age 16.

	Main Effects Model											
	Basal cortisol				Linear cortisol				Quadratic cortisol			
Predictor	b	(SE)	95% CI		b	(SE)	95% CI		b	(SE)	95% CI	
Fixed Effects												
Time	**–0.032**	**0.005**	**–0.042**	**–0.021**								
ItN Intercept (16 y)	0.008	0.025	–0.041	0.049	**0.098**	**0.032**	**0.035**	**0.160**	**–0.030**	**0.011**	**–0.052**	**–0.008**
ItN Slope	–0.344	0.380	–1.089	0.282	–0.481	0.482	–1.425	0.463	0.095	0.169	–0.236	0.426
Sex	**–0.112**	**0.031**	**-0.173**	**-0.060**	0.056	0.040	-0.022	0.134	-0.015	0.014	-0.042	0.013
Medication Usage	0.066	0.048	–0.027	0.145	0.058	0.060	–0.060	0.176	–0.020	0.021	–0.062	0.022
Depression-risk	–0.040	0.037	–0.111	0.020	**0.098**	**0.047**	**0.007**	**0.190**	–0.029	0.016	–0.061	0.003
Fit Statistics	–2 Log Likelihood		AIC	BIC								
	–1250		2564	2730								
	Interaction Model											
	Basal Cortisol				Linear Cortisol				Quadratic Cortisol			
Predictor	b	(SE)	95% CI		b	(SE)	95% CI		b	(SE)	95% CI	
Fixed Effects												
Time	**–0.032**	**0.005**	**–0.042**	**–0.021**								
ItN Intercept (16 y)	0.011	0.025	–0.039	0.061	**0.082**	**0.032**	**0.019**	**0.145**	**–0.024**	**0.011**	**–0.046**	**–0.002**
ItN Slope	–0.219	0.428	–1.058	0.620	**–1.078**	**0.535**	**–2.128**	**–0.029**	0.320	0.187	–0.047	0.686
ItN I X S Interaction	–0.260	0.419	–1.082	0.562	**1.302**	**0.529**	**0.266**	**2.238**	**–0.494**	**0.185**	**–0.856**	**–0.131**
Sex	**–0.114**	**0.031**	**–0.175**	**–0.052**	0.065	0.040	–0.013	0.143	–0.018	0.014	–0.045	0.009
Medication Usage	0.067	0.048	–0.026	0.161	0.056	0.060	–0.061	0.173	–0.019	0.021	–0.060	0.022
Depression-risk	–0.039	0.037	–0.111	0.032	**0.095**	**0.046**	**0.004**	**0.185**	–0.028	0.016	–0.059	0.004
Fit Statistics	–2 Log Likelihood		AIC	BIC								
	–842		1761	1958								

Note: Time = Sample Collection Time; ItN = Income-to-needs ratios; *AIC* = Akaike Information Criterion; BIC = Bayesian Information Criterion; Significant 95% CIs are presented in bold.

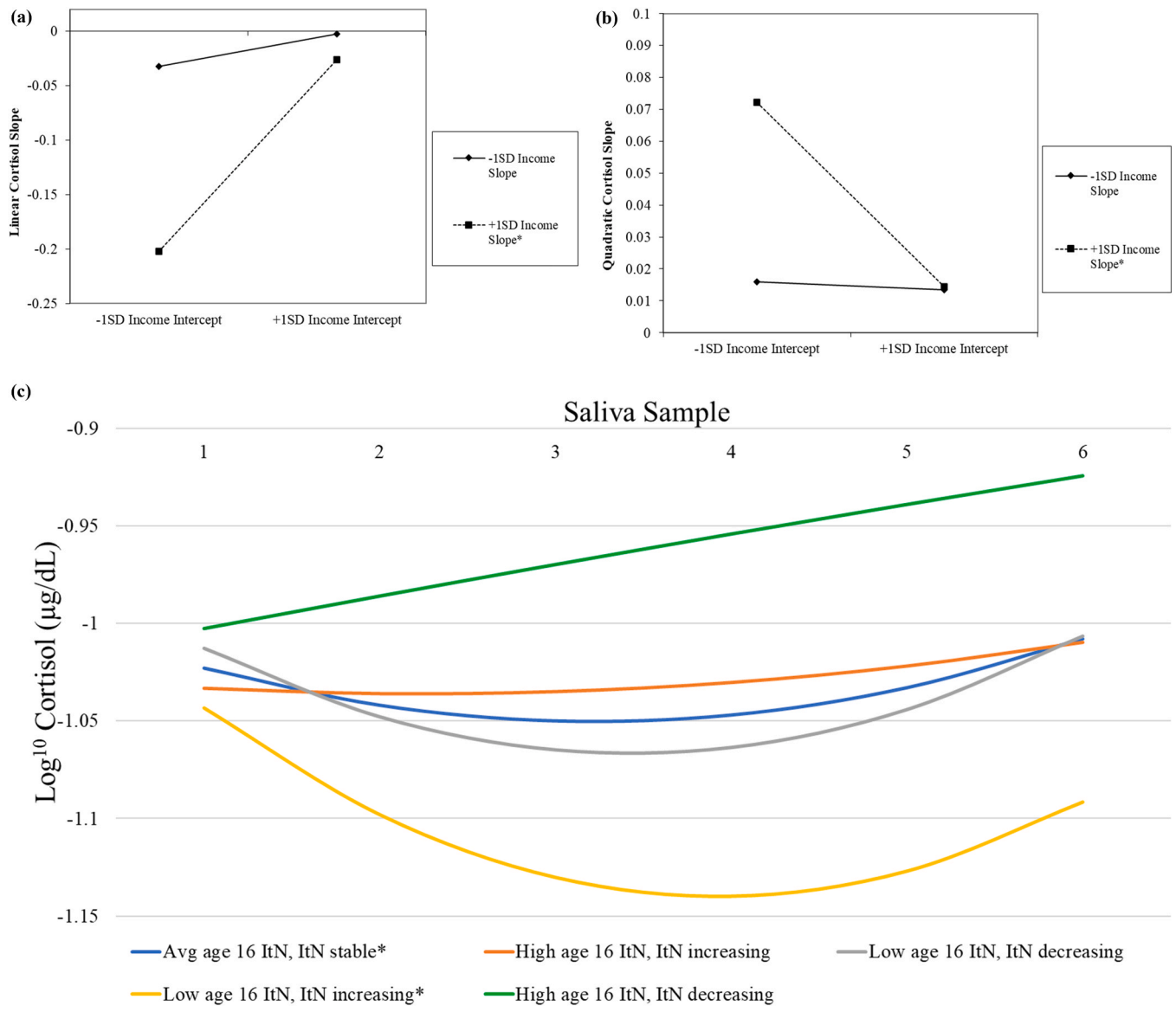

Fig. 3. (a) Prediction of linear slope of cortisol from family income-to-needs at age 16 for Mexican-origin adolescents who experienced increasing income-to-needs (+1 SD slope) versus decreasing income-to-needs (–1 SD slope) across 10–16 years. (a) displays simple slopes of the income-to-needs intercept-by-slope interaction predicting linear cortisol. Income-to-needs intercept (average income-to-needs at age 16) predicted the linear slope of cortisol when income-to-needs increased (+1 SD slope; $\beta = -0.140$, $p = 0.011$) or remained stable across ages 10–16 (M_{slope}; $\beta = -0.082$, $p = 0.011$), but not when income-to-needs decreased across adolescence (−1 SD slope; $\beta = -0.023$, $p = 0.586$). (b) Prediction of quadratic slope of cortisol from family income-to-needs at age 16 for Mexican-origin adolescents who experienced increasing income-to-needs (+1 SD slope) versus decreasing income-to-needs (–1 SD slope) across 10–16 years. (b) displays simple slopes of the income-to-needs intercept-by-slope interaction predicting quadratic cortisol. Income-to-needs intercept (average income-to-needs at age 16) predicted the quadratic slope of cortisol when income-to-needs increased (+1 SD slope; $\beta = -0.046$, $p = 0.003$) or remained stable across ages 10–16 (M_{slope}; $\beta = -0.024$, $p = 0.034$), but not when income-to-needs decreased (−1 SD; $\beta = -0.002$, $p = 0.901$). (c) Projected quadratic slopes of cortisol at the Regions of Significance (ROS) boundaries for the interaction between income-to-needs intercept and slope. (c) displays the projected quadratic slopes of cortisol for all participants, and for lower (−1 SD) or higher (+1 SD) income-to-needs intercept with trajectories of decreasing (−1 SD) or increasing (+1 SD) income-to-needs from ages 10–16. Adolescents from families with lower income-to-needs at age 16, with a history of increasing income-to-needs across ages 10–16, evinced sharp decreases in cortisol with a nadir at 30 min after *Cyberball* (sample 4), followed by cortisol increases across the final 2 samples. Adolescents from families with average income-to-needs at age 16 who experienced stable income-to-needs across adolescence evinced small cortisol decreases across the first 3 samples, followed by slight increases in cortisol from 30 to 50 min post-*Cyberball*. The quadratic slopes of lower versus higher income-to-needs slopes were not significantly different from each other for high income-to-needs intercept. * slopes are significantly different at $p < 0.05$; ItN = Income-to-needs ratios.

to-needs had relatively unchanging or increasing cortisol levels, respectively, across the samples.

Lastly, we analyzed RoS with respect to X (i.e., income-to-needs intercept) to determine the specific levels at which the income-to-needs intercept-by-slope interactions become significant in predicting cortisol. The upper and lower bounds for the RoS on linear cortisol slope with respect to income-to-needs intercept were 2.938 and 0.026, respectively, where values *outside* this region are significant. The upper bound reflects a value of income-to-needs intercept which is beyond 2 *SD*s above the mean (1.251), the commonly-used cut-off for meaningful interpretation (Roisman et al., 2012). Thus, history of income-to-needs change (i.e., negative versus positive income-to-needs slope) did not predict significantly different linear cortisol for youths with higher income-to-needs at age 16. The lower bound of the ROS

corresponds with average or lower values of income-to-needs intercept, such that when income-to-needs intercept was average or low, the linear slope of cortisol for youths who experienced increasing income-to-needs from 10 to 16 years was significantly different from the linear slope of cortisol for youths who experienced decreasing income-to-needs from 10 to 16 years. The former characterizes youths who started adolescence in deeper poverty that lessened somewhat over time (i.e., positive income-to-needs slope), but for whom income-to-needs was still low at age 16. These youth evinced strong, negative linear cortisol slopes, or HPA hyporeactivity. Conversely, youth who did not start adolescence as deeply in poverty, but for whom income-to-needs decreased to a comparably low level at age 16 (i.e., average or lower income-to-needs intercept paired with negative income-to-needs slope), displayed weak, positive linear cortisol slopes (see Fig. 3c).

The upper and lower bounds for the RoS on quadratic cortisol slope with respect to income-to-needs intercept were 1.909 and −0.156, respectively, where values *outside* this region are significant. Similar to the RoS on linear cortisol slope, the upper bound is uninterpretable as it reflects a value of income-to-needs intercept beyond 2 *SD*s above the mean. The lower bound corresponds with a value of income-to-needs intercept approximately one-fourth *SD* below the mean, indicating that for youths with lower income-to-needs at age 16, quadratic slopes of cortisol significantly differed according to their history of income-to-needs change. Specifically, deep poverty at the onset of adolescence (i.e., experienced increasing income-to-needs over ages 10–16, yet remaining in poverty at age 16), predicted a faster return toward baseline levels post-stressor (e.g., sharper U-shaped curve across the samples). Contrastingly, youths who did not start adolescence in poverty, yet experienced decreasing income-to-needs across the years, displayed the weakest quadratic slope (i.e., cortisol values did not decrease in the last samples following initial spike).

4. Discussion

Chronic poverty and changing economic resources from pre- to mid-adolescence predicted HPA responses to a social exclusion task administered within a MRI scanner in Mexican-origin youths from predominantly low-income families. On average, participants evinced a pattern of slight initial decreases in HPA activity from baseline following the MRI, then slight increases as further time passed. Yet, there was considerable heterogeneity of HPA response to the MRI, with some youths showing increased activity relative to baseline, consistent with HPA arousal or reactivity, and others showing strongly decreased activity, consistent with HPA suppression. Although income-to-needs was not related to baseline cortisol levels prior to the MRI scan, youths' experiences of family poverty across adolescence distinguished patterns of HPA responses to the protocol. Youths who started adolescence in a state of deep poverty and, despite some income gains over time, remained in poverty from 10–16 displayed dampened activity. Conversely, youths from middle-income homes, living above the poverty line across adolescence, displayed stronger (albeit prolonged) activity. Increased cortisol following acute stress is considered normative (Gunnar and Quevedo, 2007), suggesting that severe and prolonged poverty across adolescence is associated with atypically low HPA reactivity, whereas Mexican-origin youths not experiencing poverty evinced HPA reactivity. The current findings extend prior evidence for concurrent associations between environmental and familial stress and hypocortisolism (Joos et al., 2019; Kwak et al., 2017) in low-income young adolescents. Although our study design precludes direct examination of the influences of possible co-occurring stressors that low-income Mexican-American families may be facing, these findings suggest living in poverty during adolescence, particularly early adolescence, may potentially lead to hypocortisolism in HPA responses (Evans et al., 2012), consistent with the hypoactivity hypothesis of the allostatic load framework (McEwen, 2000).

By probing family income as a dynamic condition that can fluctuate across time, the present study identified variations in post-stressor cortisol responses as a function of timing and duration of previous poverty exposure. Past studies indicate that income gains may positively impact children living in poverty (Duncan et al., 2014; Fernald and Gunnar, 2009), and we have observed that for Mexican-origin adolescents living in impoverished families, increasing income-to-needs across adolescence predicted greater functional connectivity of the DMN (Weissman et al., 2018). Consistent with this, the slight yet distinct cortisol increases of youths from higher income families at age 16 with a history of income gains across 10–16 (Fig. 3c, Orange line) was arguably the response pattern, among those patterns demonstrated by the current sample, that was most similar to what has been termed typical in prior studies (Gunnar and Quevedo, 2007). Conversely, and more clearly, income losses appeared to impact adrenocortical responsiveness for youths from economically-secure families. Youths who had higher income-to-needs at age 10 that decreased across adolescence (Fig. 3c, Green line) displayed significant and prolonged cortisol increases, without recovery after 50 min. As changing income is appraised or weighed against one's recent economic context (Runciman and Runciman, 1966), income losses may be perceived as especially threatening for those adolescents who had not previously experienced economic distress or deprivation. For these adolescents, their family's financial security may suddenly seem perilous, leading to increased psychological distress which may exaggerate physiological stress reactivity.

For youths from families reporting low income-to-needs at age 16, income losses versus gains across 10–16 differentiated youths according to their family's economic state in early adolescence. Youths from families who were more financially secure at the beginning of adolescence but subsequently experienced income losses resulting in low income at age 16 (Fig. 3c, Gray line) evinced relatively reduced HPA activity with cortisol initially decreasing, followed by a return toward pre-stressor (baseline) levels. Youths from families in deep poverty at the beginning of adolescence and, despite some income gains, remaining impoverished across the adolescent years (Fig. 3c, Gold line) displayed sharp and steady cortisol decreases across the saliva samples, indicative of greater HPA suppression relative to the other youths in this sample.

Strikingly, our interaction analyses revealed that reduced HPA activity following social stress was demonstrated by adolescents who were living in the most impoverished conditions at age 10 (Gold line). In comparison to the adolescents who were living at a *comparably low socioeconomic status (SES) level at age 16* but whose families *were more economically-secure at age 10* (Gray line), these youths experienced severe economic hardship during a period of significant neurobiological sensitivity to their environmental context (Eiland and Romeo, 2013; Dahl and Gunnar, 2009). Thus, the differences in HPA responses displayed between these two groups point to the early adolescent period as especially salient for understanding the subsequent impact of family income on developing neurobiology. Observing physiological differences as a function of our earliest available measure of family income-to-needs contributes to the extensive literature detailing the effects of childhood poverty on health broadly (Page et al., 2016), and HPA functioning specifically (Evans et al., 2012; Lupien et al., 2000). It is plausible that the effects demonstrated herein reflect a downstream impact of cumulative poverty exposure during childhood. Future analyses incorporating measures of income across childhood and adolescence, as well as in tandem with cortisol responses, will be crucial for clarifying the effects of environmental stress across multiple sensitive periods of development.

4.1. Theoretical implications

By examining the neurobiological effects of poverty dynamics in a sample of Mexican-origin adolescents across a range of economic conditions, the current study addressed concerns about the over-reliance on WEIRD (White, Educated, Industrialized, Rich, Democratic) samples in psychological and neurobiological research (Henrich et al., 2010).

Characterizing the experience of poverty in our sample of Mexican-origin families corroborates Myers' (2009) biopsychosocial model of cumulative vulnerability and minority health and expands the field's understanding of how chronic stressful conditions may alter stress physiology across diverse communities. As well as reflecting material scarcity, poverty is often seen as a proxy measure for co-occurring risk factors (Page et al., 2016) that differ in intensity based on racial and ethnic group membership. Compared to impoverished White communities, impoverished Latinx and other ethnically/racially marginalized communities experience higher rates of community crime and crowding, as well as increased exposure to systematic oppression and discrimination (Roy and Raver, 2014).

Investigations of between- and within-culture variation across diverse communities in the field of developmental neurobiology are rare (Parra and Hastings, 2018), yet the existing studies highlight key areas warranting further elaboration. DeSantis et al. (2007) demonstrated that, compared to their White peers, Latinx youths have flatter diurnal cortisol slopes, driven by lower waking and higher evening levels. Latinx adolescents with more acculturated values consistent with the majority U.S. culture evidence steeper diurnal cortisol slopes than do their peers with values closer to their ethnic cultural traditions (Sladek et al., 2019). Further, Tackett et al. (2017) found that low family SES accounted for significant variance in the effect of ethnicity predicting decreased cortisol reactivity for Hispanic early adolescents. By connecting severe poverty in early adolescence and prolonged poverty exposure over the course of adolescence to subsequent pattern of attenuated cortisol reactivity in Mexican-origin youths, our study contributes to the growing body of literature incorporating economic, sociological, and neurobiological approaches to understand the contexts and consequences of growing up in poverty for diverse communities.

Although the current study was primarily informed by McEwen's (2000) allostatic load theory, elements of the adaptive calibration model also pertain (ACM; Del Giudice et al., 2011). For Mexican-origin and other minority communities in the U.S., deep poverty reflects scarcity, threat, and unpredictability, which if severe and traumatic, may induce "unemotional" profiles of multiple stress systems (Del Giudice et al., 2011). With respect to the HPA axis, the "unemotional" profile is characterized by low basal and acute activity; the latter parallels the pattern evinced by Mexican-origin adolescents from chronically-impoverished families in the current study. However, the ACM posits that the "unemotional" profile is male-biased, and no sex differences emerged in our analyses of HPA arousal as a function of economic distress. Whether the reduced post-stressor activity of Mexican-origin adolescents raised in poverty reflects the "unemotional" profile detailed by the ACM is an open question requiring further investigation.

Our observation of hyporeactivity has implications for the field of psychopathology. If left unregulated, HPA suppression may have serious implications for mental health (Chrousos, 2009), particularly for adolescents at risk for depression, a group represented in the current sample. With increasing recognition of adolescence as a period of heightened neurobiological sensitivity to environmental context (Eiland and Romeo, 2013), our findings together with past literature could suggest that reactive hypocortisolism is a plausible mechanism by which growing up in poverty undermines mental health at various stages across the lifespan. Thus, support for the hypoactivity hypothesis suggests that chronic family poverty leading to alterations of normal HPA functioning during adolescence may potentially increase risk for the development of psychopathology, particularly depression (Chrousos, 2009). This is distressing given that risk for psychopathology is disproportionately elevated for racial and ethnic marginalized adolescents (McLaughlin et al., 2007).

Building on research demonstrating the long-lasting effects of governmental assistance programs on children's and adolescents' development, including HPA functioning (Fernald and Gunnar, 2009), this study highlights early adolescence as a period when interventions may confer additional benefits. Income augmentation services targeting parents of early adolescents may disrupt the pathophysiological processes possibly extending from prolonged poverty exposure. This work underscores the need to increase funding for programs providing economic support to communities historically and currently disadvantaged and disenfranchised in the existing sociopolitical structure.

4.2. Limitations and future directions

Several limitations of the current study should be recognized. The study protocol did not allow us to fully distinguish participants' adrenocortical responses to being in an MRI scanner from responses specific to the social exclusion task. The saliva sampling protocol was timed to capture HPA responses to *Cyberball*, and although reactivity post-task was not evident in the average cortisol values across adolescents, analyses indicated that a subset of the adolescents did react to *Cyberball* in the expected manner of a social stressor (Gunnar and Quevedo, 2007). Finding differential activity following *Cyberball* aligns with past studies indicating inconsistencies regarding the capacity of *Cyberball* to elicit acute HPA responses (Beekman et al., 2016). However, Eatough et al. (2009) demonstrated that adolescents mount acute HPA responses to the MRI scan itself. Thus, our findings must be interpreted as differences in non-specific, post-stressor HPA responses as a function of socioeconomic distress.

The fact that adolescents from more economically secure families did not show recovery from HPA activation could suggest that some of the participants found the post-scan experience of completing additional questionnaires for 40 min to be evocative or unpleasant. Administering relatively innocuous questionnaires following an acute stressor task is fairly common, but it may have been more advantageous to follow the scan with 20–30 min of restful activities. Additionally, youths may have perceived the assessment environment as uncontrollable, uncertain, or alienating. It is possible that the novelty of the overall research experience contributed to prolonged cortisol release in some youths. Further study will be needed to determine whether the association between modestly higher income-to-needs in adolescence is associated with prolonged HPA reactivity or reduced recovery.

Poverty can entail a range of co-occurring stressors (e.g., conflict, neglect, scarcity, crime), all of which may impact adrenocortical functioning. Designing careful studies unpacking poverty profiles and examining their intersection with other marginalized social statuses would deepen the field's understanding of the equifinality and multifinality of downstream sequelae of childhood adversity. Similarly, information on family economic circumstances prior to age 10 was not collected; thus, the present study was unable to consider how income dynamics in childhood may have contributed to adolescents' HPA activity. Poverty in early childhood has been linked with elevated HPA activity (Fernald and Gunnar, 2009; Lupien et al., 2000), which may persist into adolescence (Evans et al., 2012). The current study provided evidence for prolonged sensitivity of the HPA axis at a later time point to relative fluctuations in economic context throughout early to mid-adolescence, suggesting that poverty may continue to have effects on neurobiological maturation beyond the childhood period.

The current analyses did not link HPA activity to indicators of adolescent adjustment. Depression-risk, indexed by depressive symptoms, was included as a covariate because the study oversampled for adolescents experiencing depressive symptoms, and analyses revealed an effect of depression-risk on linear cortisol slope above and beyond that of economic distress, indicating that depressive symptomatology plays a significant role in post-stressor HPA responses. Future studies should explore whether the transition from pre-clinical symptoms to clinical depression alters cortisol response profiles in ethnically and economically diverse adolescents. We are currently exploring this line of inquiry in further analyses. Additionally, we did not measure and could not account for time from awakening and menstrual cycle phase, both of which are known to influence the stress response (Kudielka et al., 2009).

Lastly, we were unable to measure families' public safety net usage. This is an important direction for future studies, to better understand and improve financial assistance programs and other economic interventions that may protect the developing stress neurobiology of diverse adolescents living in poverty.

4.3. Conclusions

The current study uniquely captures the intersection at which two marginalized social statuses (living in low SES contexts and being Latinx) interact and impact adrenocortical functioning, thus contributing to a more nuanced perspective on poverty, ethnicity, and the development of adrenocortical responses. Differential HPA profiles emerged when parsing the duration and timing of poverty exposure. Mexican-origin adolescents who were living in deep poverty at age 10 and, despite experiencing some income gains, were still living below the poverty line at age 16 evinced reduced HPA activity following acute social stress. Conversely, adolescents from more economically secure families evinced increases in cortisol considered more typical, based on studies of predominantly economically secure and White samples. While further examination and replication of these findings is warranted, these results add to literature building a contextually-sensitive theory of stress regulation in Latinx adolescents. Critically, the poverty dynamics that continue to unfold across adolescence, a time when young people experience significant neurobiological changes and increased risk for psychopathology, have a marked effect on subsequent stress physiological sensitivity (Dahl and Gunnar, 2009; McLaughlin et al., 2007).

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Conflict of interest

This manuscript has not been published and is not under consideration for publication elsewhere. All authors agree to the authorship order, content of this manuscript, and have no conflicts of interest to disclose.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.psyneuen.2021.105340.

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