Early Adversity and Latent Trait Cortisol: Ethnic Racial Identity as a Potential Source of Cultural Resilience

by

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ABSTRACT

The hypothalamic pituitary adrenal (HPA) axis is a primary neuroendocrine system posited to mediate the associations between early life stress and long-term deleterious psychological and physical health outcomes. The effects of early life adversity on HPA axis functioning have been well-documented in primarily White samples, with statistical advances allowing researchers to isolate latent trait cortisol as a stable indicator of HPA axis functioning to account for day-to-day influences on diurnal cortisol patterns. However, directional associations have been mixed depending on developmental stage, demographic composition, and methodological differences across studies. The few studies of early adversity and HPA axis functioning in Hispanic/Latino/a/x samples demonstrate complex interactions between cultural processes and adversity in predicting HPA axis output. Further, nascent literature has isolated the cognitive, meaning-making, and prosocial skills involved in ethnic racial identity (ERI) and its subconstructs of exploration, resolution, and affirmation as promotive during the adolescent stage of development in Latinx youth. Such skills might better prepare youth for neurobiological stress regulation after adversity. To my knowledge, no study has examined whether ERI plays a protective role against the effects of early adversity on trait-level indicators of the HPA axis during adolescence, despite the particularly high rates of cumulative exposure to early life adversity in Latinx youth as compared to White counterparts. Guided by adaptive cultural resilience theories, this study of 197 socioeconomically diverse Latinx older-adolescents aimed to leverage recent findings of stable trait indicators of cortisol output to 1) identify consistent directional markers of the effects of early life adversity on latent trait cortisol in a Latinx

sample and 2) elucidate the degree to which ERI might act as a promotive feature for HPA axis levels and protective factor against cumulative early life adversity. Confirmatory factor analyses identified a theory-driven model as an adequate measure of latent trait cortisol. Greater exposure to early adversity predicted lower latent trait cortisol, but ERI demonstrated neither protective nor promotive effects. The present study reifies that early adversity exposure has deleterious effects on trait-level HPA axis functioning, but identifying sources of cultural resilience among Latinx youth remains critical for the future of health equity.

DEDICATION

This is dedicated to my grandparents, Saba, Dale, Safta, Mormor, and Morfar, who – through their stories and legacies – taught me to take pride in my culture, myself, and the narratives of resilience passed down through generations, l'dor v'dor.

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CHAPTER 1

INTRODUCTION

Exposure to adversity during childhood and early adolescence has the potential to lead to maladjustment during late adolescence. Early life adversity is prevalent among Hispanic/Latino[1] adolescents (Llabre et al., 2017), representing a unique distal stressor in a diverse population that is more likely to navigate culturally-specific challenges (e.g., lower socioeconomic status, acculturative stress, discrimination; Myers, 2009), in addition to normative developmental demands (e.g., pubertal and social changes). Importantly, exposure to early life adversity has been shown to be significantly associated with the functioning of neurobiological systems (e.g., the hypothalamic-pituitary-adrenal [HPA] axis) that regulate the body's stress response (Cicchetti & Rogosch, 2001), and alterations in HPA axis functioning during adolescence have been linked with increased risk for psychopathology and physical health problems in adulthood (Adam et al., 2017; Miller et al., 2018).

However, despite the fact that research suggests that approximately 25% of Latino children have experienced two or more instances of adversity (Loria & Caughy, 2018) and that Hispanic/Latino adolescents are likely to demonstrate an elevated risk for health problems like obesity and mood disorders (Haas et al., 2003; Merikangas et al., 2010), Hispanic groups (specifically Hispanic, Latino, Mexican, Puerto Rican, and Cuban)

[1] The present study includes adolescents who identify with pan-ethnic labels (e.g., Hispanic, Latino/a, and Latinx), in addition to specific nationalities as ethnicity (e.g., Mexican-American, Venezuelan). When discussing other studies, I adhere to that study's respective population term. When discussing our study, I use the term "Latinx" as a proxy for the multitude of preferred terms, to represent both Spanish-speaking individuals and individuals of Latin-American, Central-American, Spanish, and Mexican descent. I acknowledge that these groups are diverse in nature and origin, and that the conversation of academic labeling is ongoing and complex (deOnis, 2017).

broadly experience better health outcomes (Alcántara et al., 2017) and have a 17.5% lower risk of mortality compared with Whites and non-Hispanic Black populations (Ruiz et al., 2013). Thus, certain adaptive strategies embedded within Hispanic and Latino cultures during key periods of development may protect against the neurobiological consequences of early life adversity (García Coll et al., 1996). Indeed, cultural processes associated with identity development during late adolescence have been shown to be uniquely protective against the effects of concurrent life stressors on academic and emotional wellbeing (Rivas-Drake et al., 2014). The current study will help elucidate the potential differential impact of protective ethnic-racial identity processes during a critical period of socioemotional and biological development (e.g., late adolescence) on the adverse effects of early life adversity on trait-like indicators of HPA axis functioning.

Early Life Adversity and Adolescent Stress Neurobiology

Neurobiological development is particularly plastic and sensitive to the environment during early childhood and adolescence. Thus, significant challenges during these periods may lead to deleterious changes in nervous, endocrine, and immune system development (Cicchetti & Rogosch, 2001; Doom et al., 2014), resulting in impaired biological, cognitive, and affective responses later in life (Anda et al., 2006; Tarullo & Gunnar, 2006). Adverse childhood experiences (ACEs; during childhood or adolescence), including poverty, neglect, abuse (e.g., sexual, physical, emotional), or household stressors (parental mental illness, incarceration, or substance abuse), are largely identified in extant literature as significant determinants of physical health (Felitti et al., 1998). Exposure to just *one* of these categories of early life adversity has been linked with physical health risks (Baumeister et al., 2016); however, 59.3% of adolescents who report

any exposure to childhood adversity report *multiple* adversities (McLaughlin et al., 2012), and Latinx adolescents are more likely to report higher rates of exposure to early life adversity than White counterparts (Liu et al., 2018; Loria & Caughy, 2018).

In adolescent samples, more ACEs are linked with heightened sensitization to proximal stressors (Rocque et al., 2014), increased risk for psychopathology (McLaughlin et al., 2012), obesity (Davis et al., 2019), poor academic achievement (Morrow & Villodas, 2018), and global report of health outcomes (Balistreri & Alvira-Hammond, 2016). Many of these outcomes are sustained later in life – greater early adversity has been linked with a myriad of problems in adulthood, including but not limited to: sleep problems (Greenfield et al., 2011), lower perceived well-being, psychological distress, impaired daily activities (Nurius et al., 2015), hypertension (Greenfield & Marks, 2009), obesity (Ford, 2005), PTSD and other major psychopathology (Hughes et al., 2017), and inflammation (Chiang et al., 2015).

These risk patterns collectively reflect allostatic load, the process by which cumulative and prolonged stress leads to physiological and psychological wear-and-tear over time (Danese & McEwen, 2012; McEwen, 1998; McEwen, 2004). Specifically, researchers of allostasis hypothesize that neurons in learning centers of the brain (e.g., hippocampus, prefrontal cortex) atrophy and neurons associated with fear response in the amygdala grow over time in response to chronic activation of stress hormones (e.g., cortisol), thus impairing physical resilience processes such as immune functioning, metabolism, and bone demineralization (McEwen, 2004). Indeed, cumulative risk models reveal that high levels of ACEs fit allostatic theory (Evans et al., 2013; Mersky et al.,

2013), and individuals who report six or more ACEs die nearly 20 years earlier, on average, than those reporting no ACEs (Brown et al., 2009).

However, despite the established prevalence of ACEs in Hispanic/Latino samples, Hispanic adults exhibit better health outcomes (Alcántara et al., 2017; Gallo et al., 2009) and lower mortality rates (Ruiz et al., 2013). This trend is similar in Latinx adolescents: despite reporting higher counts of ACEs compared to White peers, Latinx youth report the lowest mean count of health problems compared to both White and Black adolescents (Liu et al., 2018). As experiences during older adolescence and the transition to adulthood may contribute to significant alterations in mental and physical health outcomes later in life (Schulenberg et al., 2004), an examination of neurobiological mediators of allostasis in Latinx older adolescents could uncover how unique cultural processes may buffer against the deleterious health consequences of stress exposure.

Adolescent Neuroendocrinology as an Indicator of Allostasis

Cortisol is an accessible end-product of the hypothalamic-pituitary-adrenal (HPA) axis, one of the primary stress response systems in the body (Kirschbaum & Hellhammer, 1994) and a primary mediator of allostasis (McEwen, 2004). The HPA axis encompasses a cascade of adaptive neurobiological events that serve to regulate an individual's response to stressful stimuli (Sapolsky, 2004). Activation of the HPA axis triggers the hypothalamus to release corticotropin releasing hormone (CRH), leading to the release of adrenocorticotropin hormone (ACTH) from the pituitary gland and ultimately the activation of glucocorticoids (including cortisol) from the adrenal gland. Approximately 70% of the variation in cortisol output can be attributed to the time of day (Adam & Gunnar, 2001), the cycle of which is known as the diurnal cortisol rhythm, in which

cortisol levels respond to the suprachiasmatic nucleus's pulsatile regulation of circadian patterns to jointly regulate basic functioning (e.g., metabolic; Clow et al., 2010; Dallman et al., 2004) and represent within-day changes of the HPA axis (Adam, 2012).

Whereas responses to an acute stressor (e.g., reactivity) can activate the HPA axis and increase short-term cortisol output (Dickerson & Kemeny, 2004), diurnal cortisol rhythms illustrate a person's typical HPA axis functioning in naturalistic conditions. The diurnal cortisol rhythm in normative populations typically begins at high concentrations upon awakening (Pruessner et al., 1997), increases by 50-60% 30-40 minutes after waking (cortisol awakening response; CAR), and declines throughout the day (the rate of which is often referred to as the diurnal cortisol slope) until reaching a low point around midnight, then increasing again until waking (Kirschbaum & Hellhammer, 1989). Researchers may also measure a parameter called area under the curve with respect to ground (AUCg) to examine total daily cortisol output (Pruessner et al., 2003).

Normative diurnal cortisol rhythms naturally change over the course of human development, with patterns in basal cortisol maturing over the first few years of life (Gunnar & Quevedo, 2008) and cortisol levels increasing significantly during adolescence (Gunnar et al., 2009). However, the HPA axis is particularly susceptible to external influences during both early and late childhood (a critical period of neural plasticity; Heim et al., 2003). Consistent with animal literature (McCormack et al., 2003), young children exposed to neglectful, abusive, or unsupportive caregiving environments or stressful experiences often display significantly elevated diurnal cortisol patterns (i.e., hypercortisolism) early in life as compared with their peers (Tarullo & Gunnar, 2006), a

response posited to reflect an increased need for physiological resources in order to respond to the higher demands of stressful environments (Del Giudice et al., 2011).

It is important to note that predominant research on these associations uses largely White and socioeconomically middle/upper class samples; studies of cortisol output in Latinx children are sparse and demonstrate complex interactions with early life adversity and cultural processes. For example, one study of Latino children found lower mean cortisol levels only in children with high exposure to economic hardship but low levels acculturation, and high mean cortisol levels in children with low economic hardship but higher acculturation (Mendoza et al., 2017). The interplay between culture and neurobiological processes is apparent and thus crucial to consider when discussing HPA axis functioning across childhood through adolescence (Causadias et al., 2017).

Research on the directional influences of early adversity on diurnal cortisol parameters in (primarily White) *adolescents* is conflicting. This is likely a result of varying definitions of early life adversity, which often differ in terms of severity (Kuhlman et al., 2015). For example, young adolescents previously adopted from Romanian orphanages after 16 months of age evince a blunted wake-30 min cortisol slope relative to non-adopted (i.e., normative) or earlier-adopted adolescents (Leneman et al., 2018). Using a different definition of early life adversity, Meinlschmidt and Heim (2005) similarly report smaller CARs in older adolescents with exposure to early loss as compared to adolescents without early loss experience. Poor parental caregiving during childhood, in contrast, was associated with an increased CAR and increased afternoon/evening cortisol output in adolescents (Engert et al., 2011).

Other studies have found no independent main effect of general reports of childhood adversity on CAR in adolescents (e.g., Starr et al., 2017). Indeed, meta-analyses reveal little consistency in associations between maltreatment and diurnal cortisol, but suggest that overall early adversity is likely associated with a trend of hypocortisolism later in life (Bernard et al., 2017). Similarly, meta-analytic evidence conducted by Boggero and colleagues (2017) found that total cortisol output of the waking period is lower in those with post-traumatic stress symptoms.

Additional meta-analyses suggest that HPA axis activity is negatively correlated with time since onset of stressor (Miller et al., 2007), aligning with allostatic theory of depletion of resources over time (McEwen, 2004). Likewise, some researchers have tested HPA axis functioning during adolescence as a biological intermediary in the development of long-term physical and mental health. For example, smaller CARs in adolescence mediated the association between greater early adversity and higher young adult body-mass index (Miller et al., 2018), and higher CAR and steeper diurnal slopes in adolescents were associated with engaging in more non-suicidal self-injury after childhood adversity (Reichl et al., 2016). Functional alterations in typical diurnal cortisol parameters in adolescence may be indicative of either hyperactive or depleting resources for biological and psychological stress-responses, but interpreting the biological significance of differences in cortisol after adversity exposure is highly complex, partially due to cortisol's lack of temporal stability (Shirtcliff et al., 2011; for discussion, see Ehrlich et al., 2016). Identifying a consistent marker of cortisol levels reflective of cumulative exposure to early adversity will serve to better illuminate the long-term effects of stress on neuroendocrine functioning and allostasis.

Latent Trait Cortisol: A Novel Indicator of Trait-Level HPA Axis Functioning

Recent meta-analyses suggest that associations between early life adversity and long-term cortisol output might be confounded, in part, by how cortisol is measured (Fogelman & Canli, 2018). Beyond methodological variables related to collection (e.g., cotton swabs versus passive drool), the majority of extant research using diurnal salivary cortisol to isolate HPA axis levels is limited to individual constructs of CAR, diurnal cortisol slope, and area under the curve (for review, see Granger et al., 2012). The HPA axis, however, constantly recalibrates in response to the changing environment (Dickerson & Kemeny, 2004), and longitudinal studies have recently determined that several commonly used indicators of diurnal cortisol patterns demonstrate limited long term stability. In fact, estimates of the percentage of variance in cortisol indices attributable to day-to-day fluctuations can range from approximately 50% (Ross et al., 2014) to 82.3-81.25% (in CAR and diurnal slope; Doane et al., 2015), with each cortisol feature changing significantly over the course of six years (Wang et al., 2014). Cortisol levels later in life are also susceptible to time and development: Wang and colleagues (2014) found that Hispanic adults' waking cortisol significantly and stably increased in a six-year follow-up as compared with Non-Hispanic Whites and Black adults. In addition, Hispanic and Black adults experienced less flattening of the diurnal slope over time.

Emerging research identifies a latent trait factor of cortisol derived from waking cortisol and 30-minute post-waking samples to reflect both within- and across-wave trait components of the variance in cortisol (Doane et al., 2015). Different researchers have utilized varying statistical methods to establish latent trait cortisol. For example, latent state-trait modeling (Steyer et al., 2015) can be applied to correlations among different

cortisol samples to isolate a latent trait cortisol (LTC) factor and latent state cortisol factor, separating individual and state-specific influences and random error variances in cortisol output. Confirmatory factor analysis (CFA; Brown, 2015) is more widely used to establish an LTC factor (Chen et al., 2017; Doane et al., 2015; Giesbrecht et al., 2015; Stroud et al., 2016, 2018); given shared communalities and intercorrelations among cortisol samples, at least 3 samples of cortisol can be used, and each sample is partitioned into common variance (i.e., the portion that is shared among indicators) and unique variance (i.e., variance unique to each sample and excluding random error; Brown, 2015; Giesbrecht et al., 2015).

LTC thus reflects the shared variance of each sample in the model. Another statistical technique that may be used to account for unique patterns of difference is a confirmatory bi-factor model (Holzinger & Swineford, 1937), in which a separate and uncorrelated "nuisance" factor is added to the model, in addition to the "general" (in this case, LTC) factor. The nuisance (or "group") factor includes specific indicators from the general factor to account for expected patterns of variance that would not be considered "common" among all samples or "unique" to individual samples. For example, only negatively worded items from a depression screening might be included in the group factor (Rodriguez et al., 2016). To my knowledge, this method has not yet been applied to latent trait cortisol, but it may be useful for accounting for expected patterns in diurnal cortisol levels (e.g., differences between the first and second cortisol samples of the day; Ross et al., 2014).

As opposed to simply measuring the average area under the curve with respect to increase (AUC_i) to obtain CAR measurements, for example, the LTC factor typically

elicits common variance of multiple morning sample cortisol levels – as the morning samples have been identified as most stable per individual across days and waves – thus distinguishing it from the CAR and diurnal slope and capturing a longitudinally trait-like indicator of allostatic load (Stroud et al., 2016). The evident stability of waking samples identified through the LTC construct is consistent with findings of the genetic heritability of cortisol, such that morning cortisol is more heritable than cortisol levels during the rest of the day (Bartels et al., 2003; Van Hulle et al., 2012).

Indeed, an examination of within- and across-wave LTC found that over 70% of the variance of within-wave (i.e., three consecutive days during one cross-sectional timepoint) LTC was attributable to across-wave (i.e., across multiple longitudinal waves of data collection periods) LTC, as compared with about 14% of CAR within-wave variance and about 18% of diurnal slope within-wave variance attributed to individual differences (Doane et al., 2015). These findings suggest the superior reliability of just one within-wave LTC indicator in capturing consistent trait-like cortisol levels (Doane et al., 2015). Given that some guidelines suggest 10 days of data collection to reliably capture "trait" indicators of diurnal *slope*, for example (Segerstrom et al., 2014), and the fact that participant non-compliance can greatly influence estimates of both CAR and diurnal slope in adolescents (Rotenberg & Mcgrath, 2014), the ability to acquire stable trait cortisol information from just 6 total samples across 3 days significantly reduces participant and researcher burden and increases reliability of cortisol indicators.

Studies of primarily White early-adolescent females from rural areas have found that greater cumulative early adversity predicted *lower* LTC (Chen et al., 2017; Stroud et al., 2016, 2018). Recent work establishing internal consistency of LTC via longitudinal

design (i.e., two different time points of LTC, one year apart) in a more ethno-racially diverse (i.e., 57% non-Hispanic white, 18% Latina/o/x or Hispanic, 12% non-Hispanic Black) sample of adolescent girls also found that greater early life stress exposure was associated with lower latent trait cortisol (Vergara-Lopez et al., 2021). In contrast, a study of ethno-racially diverse (i.e., 54% European American, 23% Latino/Hispanic descent, 13% multiracial) older adolescents assessing early adversity up until the age of 16 found that childhood and adolescent experiences of adversity were associated with *higher* LTC (Doane et al., 2015). Essex and colleagues (2011) found evidence of both hypo- and hyperarousal in trait-like cortisol in a sample of young adolescents depending on the type of (less extreme) early life adversity measured in infancy and preschool-age years. Moreover, differences in LTC levels mediated associations between early adversity and internalizing symptoms in young adolescent girls (Stroud et al., 2018), and lower LTC levels have been associated with elevated cardiovascular risk (in middle childhood; Yeung et al., 2016), suggesting LTC as a potential pathway for later vulnerability to psychopathology and allostasis.

Although the few studies linking early adversity and LTC in adolescents have been directionally inconsistent (similar to findings on individual cortisol parameters like diurnal slope or CAR; Bernard et al., 2017), these discrepancies are likely a function of developmental stage (Shirtcliff et al., 2011), differential measurement and timing of adversity (Miller et al., 2007), and varying participant demographics. More research in older adolescent samples is needed in order to verify LTC directional trends and account for developmental stage after early life adversity. However, *lower* LTC and lower morning cortisol in general have been associated with deleterious outcomes, align with

allostatic theory of depletion of resources of over time, and might thus be expected in response to greater cumulative adversity in adolescents. Still, this construct has only been tested in small samples of adolescents; larger and more diverse adolescent samples have yet to be examined.

Integration of Cultural and Resilience Theory: An Adaptive Process Perspective

Associations between the particularly high rates of early life adversity in Hispanic/Latino populations (Llabre et al., 2017) and increases in stress- and adversity-related health problems in adulthood (Barrera et al., 2019) are posited to be mediated, in part, by physiological stress processes. For example, compared to their White counterparts, Hispanic adolescents exhibit significantly flatter diurnal cortisol slopes (Desantis et al., 2007). While cross-cultural variability in stress physiology is evident, systemic disparities and culturally-unique stressors (e.g., racism, discrimination) that arise as a result of social stratification systems may account, at least in part, for these differences (García Coll et al., 1996). Recent calls for increased research using a cultural neurobiology framework (Doane, Sladek, & Adam, 2018) necessitate an exploration of within-group variability in cultural factors and neuroendocrine functioning to better explain disparities in long-term health outcomes across and within racial/ethnic groups.

Risk and resilience frameworks provide a foundation from which to understand these disparities. Risk factors function to increase the likelihood of detrimental outcomes; conversely, *promotive* factors contribute to positive outcomes across all levels of risk (Masten et al., 2009). In a *protective* factor model, promotive features increase the likelihood of better outcomes specifically in the context of risk, thereby decreasing the deleterious effects of risk factors (Zimmerman et al., 2013). *Cultural protective* factors

extend this framework by acknowledging cultural processes that may ameliorate the effects of adversity and risk (Causadias, 2013). Examining cultural coping strategies, such as pride, community support, and spirituality, for example, challenge deficit models of minorities in which these practices are portrayed as sources of risk rather than resilience (Causadias & Cicchetti, 2018).

Culturally-informed theories of resilience expand on ecological frameworks (e.g., Bronfenbrenner, 1977) to include sociocultural processes and environment as important promotive and protective factors for adolescent functioning (Fuller et al., 2010; García Coll et al., 2000; Neblett et al., 2012). García Coll et al. (1996) proposed an integrative, theory-driven examination of adaptive cultures embedded *within* minority groups that uniquely shape stress regulation across developmental trajectories. Indeed, studies linking environmental stressors and diurnal cortisol parameters within Latinx adolescent samples reveal complex interactions with acculturative processes. For example, past research with the current study's sample has shown that Latino older adolescents who adopted higher mainstream U.S. cultural values exhibited higher waking cortisol levels and steeper diurnal slopes than those reporting lower levels of these values, but perceived stress in the same sample was associated with lower cortisol levels only for adolescents endorsing greater alignment with Latino ethnic heritage values (Sladek et al., 2019). In terms of familial cultural factors, Latino adolescents' perceptions of greater parental support in the same study sample has been associated with greater cortisol awakening responses, but assisting family during the day contributed to lower waking cortisol and flatter slopes the next day (Doane, Sladek, Breitenstein, et al., 2018).

While studies have found significant effects of culturally-unique *proximal* stressors on diurnal cortisol parameters (e.g., discrimination; Korous et al., 2017; Zeiders et al., 2012), including discrimination during adolescence prospectively predicting lower waking and average cortisol in Black adults (Adam et al., 2015), sparse literature has examined the interaction of cultural processes and distal risk factors of Latinx adolescent HPA axis dysfunction (e.g., exposure to cumulative early adversity). In a study of Mexican-American adults, greater Anglo-orientation acculturation (the process of adapting to an Anglo-oriented host culture, such as the United States) and early traumatic exposure were both predictive of attenuated CAR, but exposure to just one risk factor was only incrementally better than greater exposure to both risk factors (Mangold et al., 2010). Similarly, greater Anglo-oriented acculturation has been linked with blunted cortisol reactivity in the context of an acute stressor among Mexican American adolescents, whereas more bicultural youth (i.e., high on both Anglo and Mexican orientations) exhibited greater cortisol reactivity (Gonzales et al., 2018), suggesting the potential for cultural adaptations (e.g., biculturalism) to promote more adaptive HPA axis functioning. However, prior work has seldom delineated processes inherent in social stratification (e.g., cumulative early life adversity) – while accounting for within-group developmental heterogeneity of both promotive and protective factors in adolescence (García Coll et al., 2000; see Adam et al., 2015 for exception).

Ethnic Racial Identity as a Cultural Resource

Across ethnic and racial groups, developmental literature posits that the exploration and development of social and personal identity is a particularly salient feature during adolescence (Erikson, 1968; Tajfel & Turner, 1986). Ethnic-racial

minority youth, in particular, might benefit from positive experiences related to developing ethnic-racial identity (Phinney, 1990; Umaña-Taylor et al., 2004).

Ethnic-racial identity (ERI) refers to a multidimensional psychosocial meta-construct encompassing the process and content of an individual's sense of self in relation to their racial background and ethnic heritage (Umaña-Taylor et al., 2004). Acknowledging that youth's development of both racial identity and ethnic identity follow similar trajectories (Quintana, 1998) with considerable empirical and conceptual overlap between the two constructs (Casey-Cannon et al., 2011), ERI reflects a more global and intersectional definition of cultural identity (Umaña-Taylor et al., 2014). ERI includes the process through which youth seek out or are exposed to information about their ethnic-racial group (*exploration*), the extent to which youth have resolved what that group membership means to them (*resolution*), and youth's own evaluations of and affect toward that group (*affirmation*) (Sellers et al., 1998; Umaña-Taylor et al., 2014; Rivas-Drake et al., 2014). The development of ERI exploration, resolution, and affirmation in adolescence is perceived as an important developmental competency (Neblett et al., 2012; Williams et al., 2012) that emerges as a result of social factors and cognitive maturation (Pahl & Way, 2006; Schwartz et al. 2014) and contributes to attenuating the long-term maladaptive psychosocial (Rivas-Drake et al., 2014) and affective (Kiang et al., 2006) impact of current life stressors (e.g., discrimination) in ethnic-racial minority youth in the United States (Umaña-Taylor, 2016).

Neblett and colleagues (2012) hypothesized that ERI functions as promotive for racial/ethnic minority youth by way of boosting self-esteem (Brody et al., 2006), encouraging healthy cognitive appraisal strategies (e.g., attributional bias in the context

of discrimination; Seaton et al., 2010; Sellers et al., 2006), augmenting sophisticated and diverse coping skills (i.e., youth with higher ERI might spend more time conceptualizing and thinking about their identity and thus might find alternative coping mechanisms through that development; Neblett et al., 2004; Umaña-Taylor et al., 2008), and meaning-making (Kiang & Fuligni, 2010). ERI affirmation, in particular, is identified as a particularly salient promotive factor in fostering positive psychosocial outcomes in Latino youth (Rivas-Drake et al., 2014), perhaps due to the affective components of evaluating and developing one's emotional appraisal of their identity. ERI affirmation also tends to evince a steeper increase during school transitions (e.g., the transition from junior high to high school; French et al., 2006). In relation to neurobiological indicators, preliminary evidence has linked higher ERI affirmation with steeper diurnal cortisol slopes in Mexican-American adolescents (Zeiders et al., 2018), demonstrating the potential promotive effects of constructs of ERI on HPA axis functioning.

Meta-analyses reveal that ERI exploration and resolution confer protection against experiences of discrimination in Latinx individuals (Yip et al., 2019). ERI affirmation may also act as a protective agent: Mexican-American middle-schoolers who experienced high levels of discrimination but high ERI affirmation maintained high self-esteem as compared with peers with lower ERI affirmation (Romero & Roberts, 2003). However, Neblett and colleagues (2012) note that ERI is largely studied as a promotive factor, and only a few studies have investigated the construct as *protective*, particularly one that interacts with distal stressors to buffer youth against maladaptive outcomes.

Despite demonstrations that ERI is both promotive of positive physical and mental health outcomes in Latinx youth and protective against perceived discrimination,

there remains a dearth of literature examining how identity, and ERI specifically, interacts with globally maladaptive developmental contexts, such as exposure to abuse, neglect, or poverty (Berman et al., 2020; Tyrell et al., 2019). Direct effects between adversity and ERI have been examined: studies of refugee samples reveal that certain experiences of adversity (e.g., forced relocation) may increase an individual's awareness and commitment to their overall ERI (Bilge, 2018), and greater severity of childhood maltreatment in foster contexts has been associated with lower ERI private regard (i.e., affirmation) across ethnic/racial minority youth (including Latinx youth; Tyrell et al., 2019), but these studies exclude the potential for ERI to serve as a source of resilience in the context of adversity among youth with greater ERI. Only one study to date (to my knowledge), comparing Black and non-Hispanic White adolescents, has examined ERI as a protective factor in the context of early life adversity, finding that higher levels of ERI affirmation and belonging mitigated the association between cumulative ACEs and lower occupational expectancies (Moses et al., 2019). No study, to my knowledge, has explicated whether ERI both promotes better HPA axis functioning and serves as a buffer in the context of cumulative early life adversity.

The Present Study and Hypotheses

Despite emerging evidence that certain cultural processes (e.g., acculturation) can have deleterious effects on cortisol levels in trauma-exposed Mexican-Americans (Mangold et al., 2010), no study has examined cultural constructs as both promotive and protective factors against the effects of early life adversity on HPA axis activity, particularly with stable trait-level indicators of cortisol levels in adolescence as an indicator of allostasis. Stable trait indicators of cortisol - such as LTC - have yet to be

explored in Latinx samples, and research has yet to adequately elucidate if and how ERI in adolescence may buffer against the negative effects of early life adversity on trait-level HPA axis functioning. As such, this study will leverage existing cultural and developmental theory (Doane, Sladek, & Adam, 2018; García Coll et al., 1996) to understand adaptive cultural processes within a Latinx sample (e.g., ERI) that may both promote stable indicators of HPA axis functioning (e.g. latent trait cortisol) and buffer the negative effects of cumulative early life stressors on these cortisol indicators during a pivotal period of development (i.e., late adolescence). The study aims and hypotheses were as follows:

The first aim was to derive a latent factor from morning cortisol levels (6 total samples of waking and waking +30 minutes across 3 days) to identify a common trait-level cortisol factor, and to determine whether early life adversity (ACEs) was associated with the latent trait cortisol (LTC) factor (similar to the results obtained by Stroud, Chen, Doane, & Granger, 2016 and Doane et al., 2015) (Figure 2). **(Hypothesis 1)** I predicted that the data-driven model of LTC would fit best (Chen et al., 2017; Doane & Thurston, 2014; Giesbrecht et al., 2015; Stroud et al., 2016), and there would be a significant negative association between early life adversity and latent trait cortisol levels. Previous literature is mixed regarding the direction of the association between early life adversity and LTC, with Stroud et al. (2015) reporting a negative association in a sample of young adolescents and Doane et al. (2015) reporting a positive association in older adolescents. Although this sample is developmentally similar to Doane et al. (2015), meta-analyses of diurnal cortisol parameters after certain types of adversity (e.g., chronic stress, childhood maltreatment, sexual abuse, racial discrimination) indicate that wake-up and morning

cortisol levels tend to be lower after early adversity, albeit with small-to-medium but significant effect sizes (Bernard et al., 2017; Korous et al., 2017; Miller et al., 2007). Therefore, a small-to-medium negative association was posited for the association between early life adversity and LTC.

The second aim was to examine whether ethnic-racial identity (ERI) was associated with LTC, and to test whether subscales of ethnic racial identity (exploration, resolution, and affirmation) differentially predicted the latent trait cortisol factor. **(Hypothesis 2)** I hypothesized that ERI would be moderately (i.e., small effect size) positively associated with LTC. **(Hypothesis 3)** Based on preliminary findings from Zeiders, Causadias, and White (2018) and links with positive psychosocial development (Rivas-Drake et al., 2014), I predicted that the affirmation subscale would have the strongest positive association with LTC, and exploration and resolution would not be significantly associated with LTC.

The third aim was to test whether associations between early life adversity and latent trait cortisol differed depending on level of ethnic racial identity (Figure 3). (**Hypothesis 4)** I predicted an interaction between ACEs and ERI, such that the association between ACEs and LTC would be negative and stronger in adolescents with lower levels of ERI, and weaker but still negative in adolescents with higher levels of ERI. I also conducted exploratory analyses to determine whether individual subscales of ERI interacted with early life adversity in the prediction of latent trait cortisol.

CHAPTER 2

METHOD

Participants

Participants included 209 self-identified Latino/a and Hispanic adolescents (M_{age}=18.1 [16.0-19.0], 64.4% female), recruited from December 2016 - July 2017 (notably very shortly after a U.S. election that was particularly stressful for particular subgroups of college students, yet prior to the onset of the novel coronavirus [COVID-19] pandemic, during which students of color may have exhibited greater risk for stress; Gusman et al., 2021; Hagan et al., 2018). Participants were recruited prior to enrollment at a large Southwestern U.S. university as part of an ongoing multimethod longitudinal study of Hispanic/Latino students transitioning to college (see Doane et al., 2018). Recruitment occurred during orientation sessions, through e-mail, text messages, phone calls conducted in both English and Spanish, university and community partnerships, and by word of mouth. Bilingual staff answered questions from potential participants and caregivers throughout recruitment. Participants were included if they were accepted to the university and paid an initial financial deposit or selected to defer payment, were seniors in high school, identified as Latino(a)/Hispanic, and lived within 60 miles of the university during their senior year in high school.

Two hundred thirty-nine adolescents consented to the study and 209 (87.4%) participated in study procedures during the first wave of data collection (5.9% were excluded based on criteria and 6.7% did not respond to scheduling requests after initial consent). After accounting for missing data (i.e., missing saliva samples) and strict exclusion criteria for gold-standard salivary cortisol data cleaning (Stalder et al., 2016),

the analytic sample included 197 participants total (see reasons for exclusion in Procedures and Salivary Cortisol sections below). Participants attended 92 different high schools (ranging from 6% to 96% Latinx/Hispanic enrollment; *M* = 53% Latinx/Hispanic enrollment; *SD* = 26%) from the surrounding metropolitan area and included a range of Hispanic/Latino identities with similar proportions of nationalities among the broader population of U.S. Latino youth (Patten, 2016). Specifically, the majority of the first wave of participants identified as being of Mexican (85.1%) or South or Central American (10.1%) descent, while others identified as Cuban (5.3%) or other Latino/a/x or Hispanic heritage (3.3%), and 18.2% identified as biethnic (e.g., Latino and Native American). In terms of immigrant generation, 10.6% of the sample reported being first-generation immigrants (born outside the U.S.), 62% second generation, and 27.4% third generation or greater. Reports of subjective social status (e.g., middle or working class) and socioeconomic background indicated by parental education levels varied, with over half of the sample (55.3%) reporting parental education as being high school degree or less.

Procedure

The institutional review board of the university approved all procedures. After providing written consent (from self or parent if under the age of 18), participants completed the first wave of questionnaires and salivary cortisol collection during either the spring of their senior year of high school (64.5%) or the summer prior to college entrance (34.5%). Procedures were conducted in the participants' homes or in the university laboratory, with instructions given by trained study personnel. Participants

were compensated for their participation. A one-time questionnaire included measures of adverse childhood experiences, ethnic racial identity, and demographic variables.

Participants completed the salivary cortisol collection procedures during the week following the first visit. Trained study personnel provided participants with instructions to wear a wrist-based accelerometer (i.e., actigraph; Ambulatory Monitoring, Inc, Ardsley, NY USA) to detect sleep and aid in determining wake times, complete 4-5 diary questionnaires per day for 7 days (M= 26.57, SD=4.35), and provide salivary samples via passive drool for three consecutive typical weekdays (typically Monday, Tuesday, and Wednesday) at home. Specifically, on collection days, participants provided saliva immediately upon awakening (M_{time}=7:17 am; SD=1.70 hr), 30 minutes after waking (M_{time} = 7:50 am; SD=1.70 hr), twice during the day (approx. 3 and 8 hours after the waking sample to avoid mealtimes; M_{time}=12:21 pm; SD=1.85 hr; M_{time}=5:00pm; SD=1.68 hr), and bedtime (M_{time}=11:26 pm; SD=1.45 hr). Daily text reminders were sent to participants to facilitate compliance with the protocol. Study personnel answered participant questions throughout the protocol and, at the end of the study week, collected completed materials from participant homes and compensated each participant.

Participants were asked to complete 15 saliva samples total (n = 206, M = 14.56, SD = 1.61; 3,018 samples). Fifteen of the diary reports corresponded with the 15 saliva sample collections (M = 13.97; SD = 2.24; 2,877 total corresponding diaries). Study personnel instructed participants not to eat, drink, or brush their teeth during the hour before each saliva sample. Straws for passive drool were provided from a MEMS 6TM (Aardex) track cap compliance device, an objective measurement tool that recorded the exact sample time upon opening the track cap. For each saliva sample and diary entry,

participants recorded the time of collection and diary completion and pressed a button on the actigraph watch (serving as secondary indicators of bedtimes, wake times, and diary completion). Brief diary entries assessing stressors and behaviors experienced in the last hour or across the day were completed immediately after each saliva sample on web-based smartphones (with the option to complete on paper if Internet was not available). Recent eating, exercise, caffeine use, nicotine use, medication use, sleep, and pain were all reported by participants as well, in order to account for potential covariates in cortisol analyses.

Measures

Adverse Childhood Experiences. Participants completed a 10-item baseline measure of the Adverse Childhood Experiences Scale (ACES; Felitti et al., 1998; Wingenfeld et al., 2011). The ACEs questionnaire, which was developed as part of a large-scale study by the Centers for Disease Control and Kaiser Permanente and has demonstrated adequate psychometric properties in Latinx samples (Llabre et al., 2017), assessed participant exposure to established adverse events (Felitti et al., 1998) during participants' first 16 years of life. Participants responded "yes" or "no" to questions asking whether they have been exposed to: emotional, physical, or sexual abuse; physical or emotional neglect; domestic violence; household substance abuse or mental illness; household member incarceration; or parental separation or divorce. The total ACEs score was calculated by counting the number of events endorsed, with possible scores ranging from 0 to 10. Internal consistency is not required for the ACEs count score, but these items tend to cluster, and internal consistency was $\alpha = .70$ in this sample.

Ethnic Racial Identity. Participants responded to the 9-item Brief Form of the Ethnic Identity Scale (EIS-B; Douglass & Umaña-Taylor, 2015) derived from Umaña-Taylor et al.'s (2004) conceptualization of ethnic racial identity (ERI) as three dimensional components of exploration, resolution, and affirmation. The EIS-B has demonstrated reliability and validity in Latino/Hispanic and diverse adolescent samples (Douglass & Umaña-Taylor, 2015), and ERI subscales have demonstrated associations with depressive symptoms, positive social functioning, self-esteem, well-being, internalizing, externalizing, academic achievement, academic attitudes, and health risk outcomes among ethnic and racial minority youth (Rivas-Drake et al., 2014). Each subscale includes 3 items, examples of which include "I have attended events that have helped me learn more about my ethnicity" (exploration), "I am clear about what my ethnicity means to me" (resolution), and "I wish I were of a different ethnicity" (affirmation). Likert-scale responses range from 1 (*does not describe me at all*) to 5 (*describes me very well*). Responses are reverse coded in the affirmation subscale. Means are calculated for the total 9-items and for each scale, with higher scores indicating higher total ERI ($\alpha = .76$), exploration ($\alpha = .84$), resolution ($\alpha = .91$), and affirmation ($\alpha = .79$).

Salivary Cortisol. Saliva was collected via passive drool (i.e., participants expelled saliva through a small straw into a plastic vial) in the home environment and stored in participants' refrigerators until materials were brought to the lab and stored at -80 °C. Following best practices for obtaining CAR based on sleep-cortisol associations (Stalder et al., 2016), actigraphy watches were used to capture sleep qualities and wake times to accurately record cortisol response. Once the study was completed, saliva

samples were sent on dry ice and sent via courier over 3 days to Biochemisches Labor at the University of Trier in Germany for assay. Precautions followed guidelines for handling and transporting salivary biomarkers (Granger et al., 2012). Saliva was assayed for cortisol in duplicate using a solid phase time-resolved fluorescence immunoassay with fluorometric endpoint detection (DELFIA; Dressendörfer et al., 1992). The average concentration from both assays was used to measure cortisol in nanomoles per liter, aside from 9 samples for which only one assay was available. The intraassay coefficient of variation ranged from 7.1% to 9.0%.

The present study used only waking and waking +30 minutes samples across the three days of saliva collection. Two-hundred and six participants were in the full analytic sample, as two participants did not provide saliva samples and one was using corticosteroid medication at the time of saliva sampling which led to extreme outlier cortisol values. Based on procedures outlined in Doane et al. (2018), actigraph, track cap, and self-report data were then inspected to determine compliant saliva samples (Desantis et al., 2010; Doane & Zeiders, 2014), as cortisol estimates (particularly morning samples) have been shown to be susceptible to saliva sampling noncompliance in previous studies (Stalder et al., 2016). Participants completed 1,109 total diary reports for all 6 waking and waking +30 minute samples combined (M = 5.38 diaries per participant, SD = 0.89). If track cap-detected times were within 15 minutes of participant's actigraph waketimes, waking samples were considered compliant (79.5% of waking samples). The waking + 30 min. sample was considered compliant if the track cap detected collection 23 to 37 minutes after the track cap-detected times for the waking sample (67.4% of waking + 30 min samples). These compliance rates include the more stringent criteria that actigraph or

track cap data must be available for samples to be considered compliant (i.e., noncompliance assumed if missing compliance information). Thus, cortisol values from samples that did not adhere to timing (10.9% of all samples) were treated as missing data in analyses, in line with expert consensus on methods to reduce bias (Stalder et al., 2016). Samples that were 3 standard deviations beyond the mean were excluded (1% of samples; Ghosh & Vogt, 2012). In total, 353 samples out of 1,210 received morning (i.e., waking and waking +30 minutes) samples were excluded. Table 3 lists all reasons for exclusion and number of samples excluded per exclusion criteria. Strict compliance criteria resulted in 9 participants being fully excluded (analytic n = 197; see Table 5 for sample size and means per salivary cortisol sample in both the full sample and the sample accounting for strict compliance criteria). Following suggested practice for addressing outliers in cortisol measurement (Nicolson, 2008), the natural log function was used to transform raw cortisol values to account for the positive skew of cortisol distribution (skew =2.08 before transforming, –0.70 after transforming). Following the same guidelines, individual cortisol samples were also examined in relation to body mass index, preexisting conditions, female menstrual cycle, and use of exogenous corticosteroids during the data cleaning process, and binary variables were created for each of these factors to examine bivariate correlations between individual samples (e.g., waking samples) and these potential covariates.

Covariates. Oral contraceptive and exogenous corticosteroid or psychotropic medication usage have been shown to inflate cortisol output (Kirschbaum et al., 1999; Stalder et al., 2016) and were thus included as dummy-variable covariates (1 = taking birth control, 0 = not taking birth control; 1 = relevant medications, 0 = no medications)

predicting the latent trait cortisol factor. Following established guidelines for the analysis of cortisol values (Adam et al., 2009; Granger et al., 2012), the following variables, which were assessed on each saliva collection day in the hour before each saliva sample, were considered as potential covariates affecting cortisol outcomes and were kept in the model if they were significantly correlated with a respective saliva sample (a practice in line with prior literature establishing an LTC factor; e.g., Stroud et al., 2016a, 2016b): caffeine use, nicotine, alcohol, perceptions of pain, exercise, and last meal. Immigrant generation based on family nativity information (two dummy codes based on sample proportions; first dummy variable: 1 = youth, parents, and grandparents foreign-born, 0 = youth, parents, or grandparents U.S. born; second dummy variable: 1 = youth U.S. born but at least one parent foreign-born, 0 = youth, parents, and grandparents all foreign-born or youth, parents, and grandparents all U.S. born; Suárez-Orozco et al., 2015; Umaña-Taylor et al., 2009), parent education (two dummy codes based on sample proportions; first dummy variable: 1 = Some college, 0 = high school degree or less, or college degree or more; second dummy variable: 1 = college degree or more, 0 = some college or less; Myers, 2009), high school ethnic composition (i.e., proportion of Hispanic/Latino/x students; Benner & Graham, 2009), and sex (0 = female; 1= male; Desantis et al., 2007; Desantis et al., 2011; Kirschbaum et al., 1999) were included in path models in which LTC was the outcome to account for potential socio-contextual influences on pathways influencing general outcomes and/or physiological output (e.g., cortisol) in Latinx youth.

Data Analytic Plan

Analyses were conducted in Mplus 8.3 (Muthén & & Muthén, 1998-2012). Full information maximum likelihood (FIML) was used to handle missing data and is robust

to non-normal data distributions (Savalei & Rhemtulla, 2012). Meta-analyses indicate that studies examining effects of chronic exposure to stress on cortisol indicators require a sample size of at least 160 individuals to obtain adequate power (Miller et al., 2007), and power analyses recommended a sample size of 200 to obtain statistical power of .8 in a single factor model (Soper, 2021). Thus, our initial sample of 206 participants was considered acceptable for obtaining adequate effect sizes. Of note, however, the analytic sample size of 197 participants was just below the threshold for obtaining an adequate effect size, but above meta-analytic recommendations.

Preliminary analyses were conducted to obtain descriptive statistics and assess skewness and kurtosis of sample distribution. Model fit was determined with a χ^2 test (a *p*-value > .05 indicates good fit), a comparative fit index (CFI; >.90 suggests good fit) and the root mean square error of approximation (RMSEA; <.05 reflects good fit; Hu & Bentler, 1998). The fit of individual model parameters was established by examining the magnitude and statistical significance of standardized (STDYX in MPlus) factor loadings, with standardized factor loadings greater than or equal to |.50| considered adequate (Hair et al., 2010). Cortisol specific covariates (i.e., sample-level and person-level) were included when modeling LTC, and person-level covariates were included in analyses when LTC served as the outcome.

In **Aim 1**, confirmatory factor analysis was used to model LTC using the waking and 30-minute post-waking samples from the 3 days of collection (6 samples per person), to test the structure and dimensionality of latent trait cortisol indicators from the 3 days of collection. All factor loadings and unique variances were estimated. The latent cortisol

factor variance was constrained to 1.0 in order to set the scale of measurement for the latent variable.

Three different approaches were used to determine optimal patterns and methods of correlating errors between samples (see Figure 1). First, samples errors in the confirmatory factor analysis were correlated using methods established in prior literature (here referred to as the *data model*; Chen et al., 2017; Doane et al., 2015; Stroud et al., 2016), such that modification indices were utilized to determine which sample errors to correlate in order to obtain adequate model fit. Additional exploratory analyses tested alternative ways of modeling LTC, including a theoretical single-factor model of LTC (*theoretical model*) with sample error correlations established based on theoretical relations between cortisol samples (i.e., covarying sample errors within the same days to account for day-level fluctuations and similarities; Ross et al., 2014); and a confirmatory *bi-factor model* (Holzinger & Swineford, 1937), which included the single-factor LTC model (general factor) with no correlations between cortisol sample errors, and a separate factor (group factor) including only waking +30 samples from each day to account for inherent differences between waking and waking +30 samples within days. The correlation between the general and group factor was constrained to 0. The measurement model with both adequate fit and sound theoretical foundations was used in the remaining path models.

To test associations between LTC and early adversity, the same confirmatory analysis process was used with covariations based on the model with the best fit and theoretical justification, in addition to estimating the sum score of endorsed ACEs, and relevant person-level covariates (i.e., immigrant generation, parent education, sex), as

predictors of LTC. Unique variances of the predictor variables and covariates were also estimated. **Aim 2** included the same confirmatory analysis procedure, but with the sum score of ERI as the predictor of LTC. Additional analyses included exploration, resolution, and affirmation subscales of ERI as predictors of LTC in the separate models (due to concerns about multicollinearity among subscales) to test the unique effects of each subscale on LTC. Unique variances were estimated for each predictor and covariate. In **Aim 3**, early adversity and ERI were simultaneously included as predictors of LTC, with an interaction term for ACEs and ERI as an additional predictor. Predictor variables were centered at their mean. Exploratory analyses included the same models but with an interaction term for ACEs and each ERI subscale (in separate models) instead of total ERI.

Three sets of planned sensitivity analyses were conducted. Due to the significant proportion of cortisol samples excluded from analyses based on suggested compliance criteria, the first set of sensitivity analyses ran the same analyses as above but used all available (n = 1,210) cortisol samples, rather than excluding samples (n = 353 excluded samples; 28.6%) that were non-compliant. The second set of sensitivity analyses were conducted to determine effects of nativity (e.g., Mexican-origin), given that paradoxical findings of health outcomes in Latinx samples are posited to potentially reflect the overrepresentation or lack of adequate representation of various Hispanic subgroups (Myers, 2009). The same primary analyses were conducted in a sub-sample comprised of Mexican-origin only participants, to determine whether findings differed by this group which makes up a substantial proportion of the study's larger sample. Finally, path models were conducted using the bi-factor method of estimating LTC.

CHAPTER 3

RESULTS

Descriptive Statistics and Preliminary Analyses

Table 1 includes the descriptive statistics of all study variables and sample demographics. Preliminary descriptive analyses revealed the distributions of primary study variables were acceptable (i.e., skewness < | 1 |, kurtosis < | 7 |; West et al., 1995), with the exception of ERI affirmation (skewness: -3.50, kurtosis: 13.67). Descriptive statistics for the distribution of endorsed types of early adversity are included in Table 2. A descriptive scatterplot of the associations between early life adversity and both waking and waking +30 minute salivary cortisol samples are included in Figure 4.

Table 4 shows correlations between all study variables. ERI resolution significantly correlated with ERI exploration ($r = .51$, $p < .001$) and affirmation ($r = .19$, $p < .01$), but ERI exploration and affirmation were not significantly correlated ($r = .10$, $p = .138$). Total ERI was significantly correlated with parent's obtaining an advanced degree ($r = .16$, $p = .026$) and being a second-generation immigrant ($r = .15$, $p = .031$). ERI exploration was significantly correlated with oral contraceptive use ($r = .17$, $p = .012$) and being a second-generation immigrant ($r = .17$, $p = .019$). Second generation immigrant status was also correlated with ERI resolution ($r = .15$, $p = .03$). Early adversity (i.e., ACEs sum score) did not significantly correlate with total ERI or ERI subscales, but it was significantly correlated with average waking +30 min samples ($r = -.20$, $p < .01$), having parents who completed some college ($r = .14$, $p = .045$), parents who obtained an advanced degree ($r = .17$, $p = .015$), and high school ethnic composition ($r = .14$, $p = .047$).

Aim 1: Latent Trait Cortisol and Early Life Adversity

Measurement Model: Latent Trait Cortisol. Preliminary analyses indicated that the waking and 30-min post-waking samples were significantly correlated, with the exception of sample 1 from day 1 and sample 2 from day 3 (See Table 5). No moment-level correlates were significantly associated with corresponding cortisol samples, thus excluding moment-level covariates from the model. Oral contraceptive and medication usage were retained as predictors of the LTC factor for theoretical purposes.

Model fit of the *theoretical model* (i.e., within-day sample covariation) was adequate (χ^2 (16) = 32.59, p =.008; RMSEA = .07; CFI = .92). Table 5 presents factor loading estimates, all of which were greater than |.5| except Day 1 Sample 1 (λ = .437, SE = .082, p<.001). Error covariance estimates between samples in Day 1 (c = .08, SE = .12, p = .508) and Day 2 (c = -.20, SE = .26, p = .440) were not significant, but the error covariance between samples in Day 3 (c = .36, SE = .11, p = .001) was significant and positive.

In the *data model*, model fit was very good: χ^2 (16) = 20.14, p =.21; RMSEA = .035; CFI = .98. As indicated in Table 6, all factor loadings were significant and above |.5| (ps < .001) except sample 1 from day 1 (λ = .466, SE = .078, p<.001). Error covariances between Day 3 Sample 2 (D3S2) and D2S1 (c = -.49, SE = .20, p = .015), D3S2 and D3S1 (c = .26, SE = .15, p = .082), and D2S2 and D1S2 (c = .39, SE = .11, p < .001) were freely correlated according to suggested modification indices.

The bi-factor model indicated adequate fit: χ^2 (14) = 25.06, p = .034; RMSEA = .062; CFI = .95. Table 6 presents factor loading estimates for the bi-factor model, all of which were above |.5| except D3S2 (λ = .450, SE = .124, p<.001) in the LTC factor, and

D1S2 (λ = -.466, SE = .127, p<.001) and D3S2 (λ = -.425, SE = .159, p<.001) in the nuisance factor.

Although the data model demonstrated the best model fit, the theoretical model demonstrated adequate fit and is recommended to establish a measure of latent trait cortisol based on theory rather than sample differences. It is also closer in model complexity and structure to established LTC methods from prior literature than the bi-factor method, thus offering a valid means of comparison to previously proposed LTC constructs. The theoretical model of LTC was thus used in the proceeding path models. Additional sensitivity analyses examined path models using the bi-factor method.

Path Model: Early Life Adversity. Table 7 includes fully standardized (STDYX standardization in MPlus) coefficients, standard deviations, and statistical significance for all exogenous variables (i.e., primary predictors, covariates) predicting the latent trait cortisol factor. Model fit for the model including ACEs and relevant person-level covariates (i.e., immigrant generation, parent education, sex) was good: $\chi^2(46) = 53.51$, $p = .21$; RMSEA = .03; CFI = .96. There was a significant negative association between ACEs sum score and the LTC factor ($b = -.21$, SE = .08, $p = .02$).

Aim 2: Ethnic Racial Identity

Model fit including ERI and relevant person-level covariates (i.e., immigrant generation, parent education, sex, high school ethnic composition) was good: $\chi^2(51)=65.22$, $p =.09$; RMSEA = .04; CFI = .93. Total ERI did not significantly predict the LTC factor ($b = .12$, SE = .08, $p = .16$) when added to the model. When replacing total ERI with respective ERI subscales, model fit was good for exploration: $\chi^2(51) = 65.86$, $p =.08$; RMSEA = .04; CFI = .93; resolution: $\chi^2(51) = 65.71$, $p =.08$; RMSEA =

.04; CFI = .93; and affirmation: $\chi^2(51) = 68.07$, p =.06; RMSEA = .04; CFI = .92. None of the ERI subscales nor covariates had significant main effects on LTC (see Table 7 for exact estimates).

Aim 3: Moderation analyses

Model fit when adding the interaction term between early adversity and total ERI was excellent, $\chi^2(61)=76.09$, p =.47; RMSEA = .035; CFI = .93. The interaction term did not significantly predict the LTC factor (b = -.05, SE = .08, p = .50), nor did the interactions between early adversity and ERI exploration (b = -.05, SE = .08, p = .53), resolution (b = -.01, SE = .08, p = .86), or affirmation (b = -.02, SE = .09, p = .85) in exploratory analyses. Model fit statistics for all exploratory analyses were good (i.e., χ^2 p-values > .05, RMSEA < .05, CFI > .90).

Sensitivity analyses

All results were consistent when non-significant person-level covariates were trimmed from model. In addition, the measurement model (i.e., LTC single-factor theoretically-driven CFA with oral contraceptive and medication usage as covariates) with the *full* cortisol sample (i.e., including all cortisol samples, regardless of compliance criteria) was adequate ($\chi^2(16)=62.07$, p <.001; RMSEA = .12; CFI = .93), but exhibited poorer fit compared to analyses with the strict compliance criteria, likely due to extreme deviations in cortisol levels due to external factors (e.g., corticosteroid usage, exercising prior to sampling). All standardized (STDYX) factor loadings were greater than |.5|. When ACEs and relevant covariates were added to the model as predictors of the LTC factor, model fit was good (χ^2 (46) = 86.92, p <.001; RMSEA = .07; CFI = .93), but ACEs was not a significant predictor of LTC (b = -.12, SE = .07, p = .136), deviating

from primary findings using strict-criteria compliance. When total ERI and relevant covariates were included as predictors of LTC, model fit was adequate ($\chi^2(51)=104.01$, p <.001; RMSEA = .07; CFI = .92), and total ERI was not a significant predictor of the LTC factor (b = -.01, SE = .05, p = .775). Model fit indices and path estimates were consistent in direction, magnitude, and significance when ERI subscales replaced total ERI in separate models. In the model with an interaction term between ACEs and total ERI ($\chi^2(61) = 109.90$, p <.001; RMSEA = .06; CFI = .92), the interaction term was also not a significant predictor of LTC (b = -.01, SE = .03, p = .727). Model fit and path estimates were consistent in direction, magnitude, and significance when ERI subscales replaced total ERI in separate models.

Analyses with the Mexican-origin only sample (n = 175) revealed similar patterns as in the full sample. Prior to running sensitivity analyses, preliminary exploratory independent samples t-tests were conducted to determine differences in variables depending on Mexican heritage versus non-Mexican heritage group affiliation (1 = Mexican heritage, 0 = not Mexican heritage). Early adversity, average waking cortisol, average waking + 30 min. cortisol, total ERI, ERI exploration, and ERI resolution were not significantly different between groups. ERI affirmation was significantly different between groups (equal variances not assumed; $t(104.8) = -2.12$, p = .036), but after correcting for multiple comparisons using a Bonferroni correction (adjusted p = .007), this difference was no longer significant. Thus, this sample did not demonstrate significant individual variable differences depending on Mexican-heritage status.

The measurement model (i.e., theoretical model) with the Mexican-origin only subsample revealed adequate model fit ($\chi^2(16)=37.89$, p < .015; RMSEA = .09; CFI =

.88), with all standardized factor loadings equal to or above |.5| and significantly loading onto the LTC factor. The model fit with ACEs and relevant covariates in the model was good ($\chi^2(46)=59.06$, $p = .09$; RMSEA = .04; CFI = .93), and ACEs significantly predicted the LTC factor in the expected direction ($b = -.20$, SE = .09, $p = .021$). When total ERI served as predictor of LTC (along with relevant covariates), model fit was good ($\chi^2(51)=69.14$, $p = .046$; RMSEA = .05; CFI = .90), but total ERI did not significantly predict the LTC factor ($b = .12$, SE = .09, $p = .189$). Model fit and path estimates in separate models with ERI subscales as predictors were consistent in direction, magnitude, and significance.

When an interaction term between ACEs and ERI was added to the model, model fit was still good ($\chi^2(61)=77.294$, $p = .08$; RMSEA = .04; CFI = .91), but the interaction term was still not significant ($b = -.02$, SE = .08, $p = .84$). Model fit and path estimates in separate models with ERI subscales replacing total ERI as predictors were consistent in direction, magnitude, and significance.

Findings were partially consistent when the bi-factor model was used in path models (i.e., both the LTC and group factor were regressed on the predictors). With ACEs added as the predictor, model fit was excellent ($\chi^2(40)=43.711$, $p = .317$; RMSEA = .02; CFI = .98), but ACEs was no longer a significant predictor of the LTC factor ($b = -.12$, SE = .09, $p = .207$). However, ACEs was a significant predictor of the group factor ($b = -.23$, SE = .12, $p = .031$). Total ERI, ERI subscales, and interaction terms between ACEs and ERI (total and subscales) in separate models were not significant predictors of LTC ($ps > .05$), despite good-to-excellent model fit among all models (i.e., χ^2 test p-value > .05; CFI >.90; RMSEA <.05).

CHAPTER 4

DISCUSSION

Identifying protective factors that attenuate the deleterious health effects of cumulative adversity among adolescents is critical. Due to social stratification forces in the United States, youth of color may experience disproportionate risk of exposure to adversity (Llabre et al., 2017; Loria & Caughy, 2018), while simultaneously practicing culturally-unique resilience processes (García Coll et al., 1996) that may buffer against later allostatic load. However, there is a dearth of literature using a cultural neurobiological approach (Doane, Sladek, & Adam, 2018; Doane, Sladek, Breitenstein, et al., 2018) to delineate whether specific cultural promotive factors, such as ethnic racial identity, may mitigate the impacts of cumulative adversity on trait-level HPA axis functioning, a potential indicator of allostasis (Stroud et al., 2018). This study aimed to extend foundational work establishing latent trait cortisol (LTC) among adolescents (Doane et al., 2015; Stroud et al., 2016), identify a theoretically-driven model of LTC in Latinx older adolescents, and examine distal risk and resilience factors contributing to individual differences in LTC.

The present study found that a theoretically-driven approach to modeling the LTC factor was an adequate metric of Latinx adolescents' trait cortisol output, and that greater cumulative early adversity was associated with lower LTC. I did not find that adolescents' total ethnic racial identity – or exploration, resolution, or affirmation of ethnic racial identity – significantly predicted LTC, nor did ERI moderate the effects of cumulative early adversity in this sample. This study contributes to the emerging body of work on LTC by testing this novel construct of HPA axis functioning and its association

with adversity in a diverse sample of Latinx adolescents. Further, I add a strengths-based and culturally-focused approach to the study of allostatic processes.

Theoretically-Driven Latent Trait Cortisol

Prior innovative studies expanded widely-used metrics of the diurnal cortisol rhythm by identifying a stable construct of daily cortisol, using confirmatory factor analysis (among other statistical approaches) to derive commonalities of morning salivary cortisol samples across three days (Doane et al., 2015). While these studies demonstrated long-term stability and reliability of confirmatory factor analysis LTC models, such approaches were potentially limited by sample-specific methods, such as using modification indices to determine data-driven error covariances between salivary cortisol samples in the LTC factor. Further, to my knowledge, the use of single-factor confirmatory factor analysis to model LTC has only been examined in four study samples (Doane et al., 2015; Giesbrecht et al., 2015; Stroud et al., 2016; Vergara-Lopez et al., 2021), two of which used adolescent or young adult samples, two of which included only adolescent girls, and the majority of which used predominantly non-Hispanic White samples. As members of minoritized ethnic and racial groups in the U.S. are often underrepresented or excluded from psychological and developmental sciences (Causadias, Vitriol, et al., 2018; Syed et al., 2018), it is necessary that future studies replicate neurobiological processes and statistical approaches that may reciprocally inform psychological functioning (e.g., LTC) with more diverse groups.

The present study tested data-driven and theoretically-driven models of LTC, in addition to a bi-factor model, and found that all models demonstrated adequate fit. While the data-driven model demonstrated the best fit, it was considered to be sample-specific

given its use of modification indices to determine error covariances, and as such revealed unexpected associations between daily cortisol samples (Adam, 2012; Ross et al., 2014). For example, the covariance between the first sample of the second day and the second sample of the third day was statistically significant and negative, but this association over and above other sample correlations was deemed theoretically arbitrary. By estimating the error covariance between these two samples in the model and thus removing the negative covariance between these samples (and therefore removing potential error due to unknown yet potentially important influences) from the latent factor, this process likely increased the factor loadings and thus artificially improved the fit of the model. Given such inexplicable associations based on suggested modification indices, I consider the data-driven model to be unlikely to replicate in other samples. The bi-factor model also offered adequate fit, but factor loadings were low (i.e., < .5) in the group factor, and this method was not as consistent with prior work, nor has prior work established its long-term stability. Future studies should examine whether the bi-factor model of LTC holds across waves and developmental stages to determine the construct validity and reliability of measuring LTC in this way.

Importantly, modeling LTC with error correlations derived from theory (i.e., correlating morning salivary cortisol samples within the same day to account for daily state-level influences; Ross et al., 2014) was an adequate measure of LTC based on model fit indices, and its single-factor CFA procedures were largely consistent with prior literature (e.g., Doane et al., 2015; Stroud et al., 2016). As such, the theoretically driven model was determined to be the most parsimonious measure of LTC in this study, as its factor structure both resembled prior work and may be replicated by design in future

studies. Indeed, future work should aim to reproduce the theory-driven model of LTC, ideally through longitudinal and multi-wave designs, in order to confirm that this model holds as a stable indicator of LTC across samples, demographics, and developmental stages. Future studies may also compare this model with other theoretically derived models of LTC, such as a different single factor CFA in which every waking sample is correlated and every 30-minutes post-waking sample is correlated.

Early Adversity is a Predictor of Latent Trait Cortisol

Following the allostatic load model (McEwen, 1998, 2004), the present study found that cumulative (i.e., greater count of) exposure to early adversity negatively predicted LTC. This finding supported my hypotheses and extended prior work examining longitudinal links between adversity and HPA axis functioning (Miller et al., 2007; Repetti et al., 2002). These results may be explained by cumulative risk models of allostatic theory, such that cumulative, prolonged, and/or chronic elevations in HPA axis activity may alter thresholds that regulate the HPA axis and potentially weaken the availability of neuroendocrine resources over time, leading to later hypocortisolism (Miller et al., 2007). Indeed, attenuation theories suggest that the HPA axis may downregulate cortisol secretion following sustained periods of hypersecretion (such as during prolonged exposure to adversity; Gunnar & Vazquez, 2001; Heim et al., 2008; Susman, 2006; Trickett et al., 2010). This process of cortisol downregulation is hypothesized to be adaptive in chronically adverse contexts, as prolonged exposure to cortisol may adversely impact brain structures (e.g., hippocampus, frontal cortex), immunological wellbeing, and cardiovascular functioning (McEwen, 2007; Raison & Miller, 2003). The present study provides further evidence that the accumulation of

exposure to early adversity contributes to individual differences in adolescents' trait-like HPA axis activity and suggests that early adversity may be linked with stable hypocortisolism in older adolescence. Contrasting LTC literature (e.g., Doane et al., 2015, Essex et al., 2011) may be attributed to the timing with which participants experienced adversities (Miller et al., 2007), developmental differences in HPA axis activity (Gunnar & Quevedo, 2007; Shirtcliff et al., 2012), or type and severity of adversity experienced (e.g., multiple deaths of family members vs. legal problems of family members; Stroud et al., 2016a). For example, prior work by Essex and colleagues (2011) found that exposure to high levels of adversity predicted higher trait morning cortisol at age 9, but these same youth's trait cortisol levels did not significantly differ from other youth by age 15. Thus, time since onset of stressor or developmental stage likely play a role in the direction of trait-level cortisol output after adversity (Essex et al., 2011; Miller et al., 2007).

Importantly, this is the first study, to my knowledge, to reproduce previously identified patterns of lower LTC after adversity in a sample of Latinx older adolescents, and I demonstrate that findings are directionally consistent with much of the literature examining early adversity and LTC levels in predominantly White or ethno-racially diverse samples of adolescents (e.g., Stroud et al., 2016a, 2016b; Vergara-Lopez et al., 2021). In line with socioecological theories of cultural adaptation (García Coll et al., 1996) and cultural neurobiology (Doane et al., 2017), I examined within-group heterogeneity of derivatives of social stratification (i.e., cumulative early adversity) and neurobiological outcomes among a sample of minoritized U.S. youth, a particularly salient concern given that Latinx youth experience higher rates of adversity than White

counterparts (Llabre et al., 2017). The present study's replication of prior work de-emphasizes White-minority differences and reinforces the "cultural similarities" hypotheses (Causadias et al., 2018), indicating that early adversity similarly "gets under the skin" of Latinx adolescents and manifests in alterations of trait-like HPA axis downregulation (i.e., LTC).

Of note, sensitivity analyses using the bi-factor method as a measure of LTC revealed that early adversity was not significantly associated with the common factor of LTC, but it was negatively associated with the nuisance factor, which captured common variance attributed to the second sample of the day (waking + 30 minutes samples). This was an unexpected association, and may reflect a targeted effect of compounded adverse exposures on patterns of cortisol 30-minutes after awakening, rather than morning cortisol as a whole. This differentiation aligns with some associations between adversity and the cortisol awakening response (CAR), which models the increase in cortisol levels in the morning, such that chronic or cumulative adversity may lead to a blunted increase in morning cortisol over time (Miller et al., 2007). However, given the relative instability of CAR (Doane et al., 2015; Ross et al., 2014), differentiating latent patterns among and predictors of waking vs. 30-minutes post-waking samples using confirmatory bi-factor analysis may further elucidate the origins of adversity's effects on trait-level cortisol. For example, a study of children found that heritability is highest for samples taken around 45 minutes after awakening, compared to waking samples and afternoon samples (Bartels et al., 2003), indicating potential differences in the trait-like tendencies of morning samples. Researchers should aim to replicate the associations evident in the bi-factor model to

determine the mechanisms through which early adversity may impact the nuisance factor of 30-minute post-waking samples.

Promotive and Protective Features of Ethnic Racial Identity

Contrary to my hypotheses, neither total ERI nor its subconstructs demonstrated significant associations with LTC in this sample. This lack of findings partially contrasts with preliminary literature linking ERI and ERI subconstructs with diurnal cortisol output in Mexican American adolescents (Zeiders & Causadias, 2018) and may be attributed to a number of factors. First, ethnic racial identity affirmation, in particular, revealed significant skewness and kurtosis and little variability among study participants; for example, the majority of participants (76.2%) endorsed the highest possible amount of affirmation, thus potentially reducing the availability of adequate sample variability for ERI affirmation and/or total ERI. The present study's use of a pan-ethnic sample (i.e., Latinx) rather than a Mexican-heritage only sample, in addition, may have contributed to disproportionate culture-specific distributions, as variability within pan-ethnic U.S. minority groups is significant (e.g., in psychopathology; Causadias et al., 2018). However, sensitivity analyses using only Mexican-heritage participants revealed equivalent findings to the pan-ethnic full sample, suggesting that the sample demographics likely did not contribute to the present study's dearth of findings related to ERI. Notably, although the brief version of the Ethnic Identity Scale demonstrates construct validity and reliability among similar samples and age demographics (Douglass & Umaña-Taylor, 2015) and ethnic racial identity has been shown to protective against negative proximal stressors (Yip et al., 2019), many studies of ERI use alternative measures of ERI (e.g., multigroup ethnic identity measure [MEIM]; Phinney, 1992).

Perhaps the promotive and/or protective features of ERI in the context of early adversity are better captured by alternative measures such as the MEIM. Future studies should compare differences in measurement of ERI and their associations with HPA axis functioning.

Importantly, the present study examined a stable indicator of cortisol as the outcome, but prior work (e.g., Zeiders et al., 2018) found effects of ERI constructs on diurnal cortisol slope as opposed to LTC, suggesting that ERI may be associated with environmental, daily, or state-level fluctuations of HPA axis functioning, rather than trait-level differences. Other recent literature similarly failed to find significant main effects of ERI constructs (i.e., individual or community regard, racial centrality, or ethnic behaviors; constructs were identified via exploratory factor analysis of a study-specific questionnaire) on total cortisol output (AUC) or cortisol slopes among Black and White adolescents, nor did they find interactive effects of ERI with racial discrimination (Adam et al., 2020). However, individual regard averaged across adolescence and young adulthood was associated with lower total cortisol among Whites and higher total cortisol among Black individuals, and higher community regard across adolescence and young adulthood was linked with higher adult total cortisol for Black and White participants combined (Adam et al., 2020). Given that the effects of ERI on diurnal cortisol indicators were more robust during young adulthood, developmental stage might further qualify the impact of ERI on HPA axis functioning. Although Adam and colleagues (2020) used a study-specific measure of ERI, their findings reify the importance of replicating studies of cortisol indicators in multiple developmental stages. Future studies of ERI and LTC, in particular, should examine whether ERI plays a more salient role in LTC later in the

lifespan (e.g., in young adulthood), perhaps as the cognitive and affective processes involved in ERI mature (Umaña-Taylor et al., 2014).

Importantly, the lack of significant direct and interactive effects of ERI might indicate that adversity and HPA axis functioning represent orthogonal mechanisms of cumulative stress processes that are distinct from those potentially informed by ethnic racial identity. Indeed, the present study did not account for the social stratification and social position factors (e.g., discrimination) that likely qualify the ways in which youth derive psychosocial benefits from their social identities (Tajfel, 1974; García Coll et al., 1996). Recent work found that other related ERI constructs (e.g., private and public regard) moderated the associations between racial discrimination and same-day and next-day overall diurnal cortisol patterns in Black adults (ages 17-56; Seaton & Zeiders, 2021), and meta-analyses identify ERI as an effective buffer of the effects of discrimination on general youth adjustment (Yip et al., 2019). These studies suggest that exploration of, commitment to, and positive beliefs about one's ethnic racial identity and group indeed exhibit protective effects, but these mechanisms are perhaps specific to adversity related to social position and ethno-racial specific domains, rather than broad or distal adversity exposure. Future research should consider mechanisms and derivatives of social stratification (e.g., discrimination, racism) when examining the protective factors of ethnic racial identity and its implications for latent trait cortisol levels.

Strengths, Limitations, and Future Directions

By examining potential interactions between cultural adaptation (i.e., ERI) and exposure to early adversity, this study responded to the need to consider how cultural beliefs and identities affect physiological stress outcomes (Causadias, 2013; García Coll

et al., 1996) and leveraged a strengths-based approach to measuring risk and resilience among Latinx youth. The sample represented a heterogenous group of Latino/Hispanic youth from 92 different high schools in the surrounding metropolitan area, and reflected within-group variability with respect to family immigrant generation, national origin, college generation, and perceived social class. The sample was restricted, however, to youth who lived within a radius of 60 miles of the focal university during high school. This may have limited variability of the sample with regard to differential contextual and sociodemographic influences that may inform ethnic racial identity, in particular. Similarly, Latino adolescents of different ancestry and backgrounds (e.g., Mexican vs. Puerto Rican) likely differ on various cultural adaptation processes (e.g., ethnic identity, self-esteem, familial ethnic socialization; Causadias, Korous, et al., 2018; Umaña-Taylor & Fine, 2001). While the present study sought to account for these differences in sensitivity analyses by replicating findings in the largest unique nativity group (Mexican-origin) in the sample, future studies may consider testing adversity, ERI, and LTC interactions among larger single-nativity samples (e.g., Mexican-American adolescents only). Further, this study was cross-sectional by design, limiting the ability to determine whether additional factors (e.g., developmental change) may have influenced variables.

The present study's measure of self-reported retrospective accounts of early adversity would be enhanced by multiple informant reports (Cooley & Jackson, 2020; Newbury et al., 2018) and prospective data collection (Baldwin et al., 2019). In addition, recent literature calls for more expansive measures of adversity, given that the 10 items listen in the original ACEs study (Felitti et al., 1998) do not account for the full range of possible adversities, especially adversities experienced by youth of color in the United

States (e.g., discrimination) or peer influences (e.g., community violence, peer victimization; Finkelhor et al., 2015). Although the cumulative index of ACES is a widely established determinant of physical and mental health outcomes (Gilbert et al., 2015), it is important to consider additional sources of cumulative stress that may contribute to allostatic load. Studies seeking to replicate or extend the present study's findings may consider more expansive measures of early life exposure to adversity.

With regard to the present study's examination of latent trait cortisol, this is the first study, to my knowledge, to derive trait-level cortisol among a diverse group of Latinx adolescents, which is especially important given the dearth of representation of youth of color in developmental and health psychology study samples (Causadias, 2013; Causadias, Vitriol, et al., 2018). I also add to prior findings that identified trait-level cortisol during the older adolescent developmental stage (Doane et al., 2015). In addition, by employing hypotheses based on theoretical underpinnings of the diurnal cortisol rhythm (Adam, 2012; Ross et al., 2014), the present study advances methods to construct LTC and produces a replicable model for future studies examining trait-level HPA axis functioning. I further compared the theoretically-derived model of LTC with prior models (e.g., data-driven models) and preliminarily proposed a new method (e.g., the bi-factor method) for further examination.

These findings were complemented by the study's high rates of saliva sample participation (n = 206 participants) and our use of gold-standard procedures for deriving reliable indicators of cortisol (Granger et al., 2012; Stalder et al., 2016), including the use of track cap compliance devices to derive the exact timing of saliva samples and actigraph watches to more precisely identify participants' wake times. In addition, the

present study used six separate morning samples from participants, thus potentially strengthening the present study's analytic power (Von Ende, 2001) and the reliability of the LTC indicator. However, only 73% of samples (n = 197 participants) were used in the final set of analyses due to participants' lack of compliance with procedures, an unfortunate yet expected by-product of collecting salivary biosamples from adolescents and young adults (Granger et al., 2012). For reference, one study measuring LTC *without* the use of track cap compliance devices to assess exact timing excluded 13.8% of samples (Yeung et al., 2016), whereas a study using similar methods to the present study (e.g., track cap) excluded 28.7% of their sample due to compliance issues (Stroud et al., 2016). Sensitivity analyses indicated that the present study's method of accounting for compliance deviations in main analyses likely improved model fit of LTC, and path models largely remained consistent when running analyses using only samples that met strict compliance criteria versus including all samples, regardless of whether they met compliance criteria. Future studies should continue to strictly account for deviations in compliance and follow established guidelines for salivary cortisol collection and processing, despite potential reductions in sample size (Granger et al., 2012).

Conclusions

Emerging literature demonstrates that LTC mediates associations between adversity and later internalizing symptoms (Stroud et al., 2018), that LTC levels after adversity may be conditional on serotonergic genetic variation (Chen et al., 2017), and lower LTC may predicate cardiovascular risk factors (e.g., higher blood pressure; Yeung et al., 2016). As such, constructing a replicable measure of LTC may aid in identifying adolescents at-risk for developing psychopathology or health risks after cumulative

exposure early adversity, a considerable allostatic vulnerability for many youth of color. As Latino/Hispanic individuals are projected to make up 30% of the U.S. population by 2060 (Colby & Ortman, 2014), and Latino youth experience significant exposure to adversity relative to non-Latino peers (Llabre et al., 2017; Loria & Caughy, 2018), beginning to isolate sources of proximal cultural resilience and adaptation among Latinx youth - such as youth's positive ethnic racial identity – may inform interventions and contribute, partially, to reducing health inequity in the United States.

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APPENDIX A

TABLES

Table 1. Demographic information and study variable descriptive statistics.

Demographic summary	***n***	***%***
Sex		
Female	134	65.0%
Male	72	35.0%
Family national origin		
Mexican	175	85.0%
South or Central American	18	8.7%
Cuban	11	5.3%
Other	5	2.4%
Immigrant generation		
1st generation	22	10.6%
2nd generation	127	62.0%
3rd generation or more	57	27.4%
Subjective family social class		
Upper/Upper-middle class	22	10.7%
Middle class	98	47.6%
Lower-middle/Working class	83	40.3%
Other/Unsure	2	1.4%
Health variables		
Caffeine consumption	108	52.4%
Smoke/take nicotine	4	1.9%
Birth control usage	15	7.3%
Medication usage	36	17.5%

Study variables	***M***	***SD***	**Range**
Waking cortisol (nmol/L)	6.88	.51	5.31 — 8.20
30-min post-waking cortisol (nmol/L)	7.48	.48	5.92 — 8.85
Early life adversity (ACEs count)	2.03	2.01	0.00 – 8.00
Ethnic racial identity (ERI total)	8.25	10.78	4.33 – 12.00
ERI exploration	2.43	2.43	1.00 – 4.00
ERI resolution	3.06	3.06	1.00 – 4.00
ERI affirmation	3.82	3.82	1.00 – 4.00

Note: N=197.

Table 2. Descriptive statistics from early life adversity.

Type of Adversity	**n**	**%** [a]
Emotional abuse	66	31.6%
Physical abuse	38	18.2%
Sexual abuse	20	9.6%
Emotional neglect	49	23.4%
Physical neglect	9	4.3%
Parental separation or divorce	81	38.8%
Witnessed domestic violence	16	7.7%
Household substance use	55	26.3%
Household mental illness or suicide attempt	49	23.4%
Household incarceration	32	15.3%
No ACEs	54	26.5%
1 ACE	47	23.0%
2 ACEs	38	18.6%
3 ACEs	24	11.8%
4 ACEs	15	7.4%
5+ ACEs	26	12.7%

Note: N=204. Derived from 10-item the Adverse Childhood Experiences (ACEs) Scale (Felitti et al., 1998; Wingenfeld et al., 2011), adapted for exposure up to 16 years of age. [a]Some participants skipped questions (19 total missing answers out of 2,060 possible responses); percentages based on valid responses.

Table 3. Count and proportions of excluded samples by exclusion criteria.

	Day 1 Sample 1	**Day 1 Sample 2**	**Day 2 Sample 1**	**Day 2 Sample 2**	**Day 3 Sample 1**	**Day 3 Sample 2**	**Total**
Total provided samples	205	206	201	198	202	198	**1210**
Missing (not provided out of n = 206 possible samples)	1	0	5	8	4	8	**26**
			n (% of all provided)				
Waking sample collected more than 15 min. after waking	28 (14%)	11 (5%)	40 (20%)	29 (15%)	54 (27%)	38 (19%)	**200 (17%)**
Second sample provided less than 23 min. or more than 37 min. after waking per track cap, or track cap data not provided	-	39 (19%)	-	36 (18%)	-	41 (21%)	**116 (10%)**
3 SD beyond mean	2 (1%)	2 (1%)	2 (1%)	2 (1%)	2(1%)	1 (1%)	**11 (1%)**
Excluded or missing, n (% of all provided)	31 (15%)	52 (25%)	47 (23%)	75 (38%)	60 (30%)	88 (44%)	**353 (29%)**
Total samples after exclusion	175 (85%)	154 (75%)	159 (79%)	131 (66%)	146 (72%)	118 (60%)	**883 (73%)**

Table 4. Bivariate correlations of primary study variables.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Waking cortisol[a]	--													
2. Waking +30 min. cortisol[a]	.41**	--												
3. Oral contraceptive usage	-.10	-.05	--											
4. Medication usage	-.01	-.06	.13	--										
5. Sex (Male = 1)	-.06	-.10	-.21**	.07	--									
6. Some College	-.08	.07	.10	.08	-.13	--								
7. Advanced Degree	.05	.05	.06	.14*	.17*	-.32*	--							
8. Second Generation	.05	.03	.03	-.02	-.14*	-.02	-.26**	--						
9. First Generation	.07	.06	-.10	-.10	.11	-.01	-.02	-.44**	--					
10. High school ethnic composition[b]	.00	-.08	-.13	-.09	.00	.03	-.45**	.23**	.02	--				
11. Early life adversity	-.12	-.20**	.08	.05	-.12	.14*	-.17*	-.05	.05	.14*	--			
12. Ethnic Racial Identity (Total ERI)	.03	-.01	.05	-.07	-.05	.05	-.16*	.15*	.03	.06	-.11	--		
13. ERI exploration	.07	.06	.17*	.04	-.11	.05	-.02	.17*	.03	-.01	-.07	.84**	--	
14. ERI resolution	.08	.13	.05	-.01	-.03	-.04	-.05	.15*	.04	.03	-.09	.84**	.51**	--
15. ERI affirmation	-.05	-.04	.06	-.11	-.06	-.10	.03	-.01	-.21**	-.01	-.09	.43**	.10	.19**

[a]Averaged from 3 samples across three days. [b]Proportion of high school with Hispanic/Latino/Latinx students. *, $p<.05$. **, $p<.01$.

Table 5. Sample sizes, means, and correlations for morning cortisol samples.

	All Samples (no exclusion)		Strict compliance criteria		Bivariate Correlations				
	n	*Mean*	*n*	*Mean*	1.	2.	3.	4.	5.
1. Day 1 Sample 1	205	6.73	175	6.77	-				
2. Day 1 Sample 2	206	7.40	154	7.51	.374**	-			
3. Day 2 Sample 1	201	6.92	159	6.94	.431**	.354**	-		
4. Day 2 Sample 2	198	7.42	131	7.52	.266**	.637**	.425**	-	
5. Day 3 Sample 1	202	6.89	146	6.95	.281**	.375**	.438**	.422**	-
6. Day 3 Sample 2	198	7.38	118	7.48	.073	.467**	.197*	.510**	.552**
Total	**206**		**197**						

Note: Strict compliance criteria sub-sample based on strict exclusion criteria during data cleaning process. Bivariate correlations based on strict compliance criteria sub-sample. *, $p<.05$. **, $p<.01$.

Table 6. Factor loading estimates for latent trait cortisol models.

	Data model		*Theoretical model*		*Bi-factor model*			
	LTC Factor Loadings		LTC Factor Loadings		General (LTC) Factor Loadings		Group factor loadings	
	λ	SE	λ	SE	λ	SE	λ	SE
1. Day 1 Sample 1	.466**	.078	.437**	.082	.520**	.073	--	--
2. Day 1 Sample 2	.617**	.074	.718**	.066	.569**	.081	-.466**	.127
3. Day 2 Sample 1	.735**	.072	.661**	.084	.756**	.072	--	--
4. Day 2 Sample 2	.699**	.070	.858**	.067	.613**	.080	-.660**	.129
5. Day 3 Sample 1	.614**	.072	.542**	.073	.643**	.076	--	--
6. Day 3 Sample 2	.655**	.099	.554**	.083	.450**	.124	-.425**	.159

Note: N=197. Number of samples based on strict exclusion criteria during data cleaning process. Estimates based on conditional model with birth control and medication usage as covariates. Model fit for data model was excellent: $\chi^2(16)=20.14$, $p=.21$; RMSEA = .035; CFI = .98. Model fit for theoretical model (in which samples within days covaried) was acceptable: $\chi^2(16)=32.59$, $p < .01$; RMSEA = .071; CFI = .92. Model fit for bi-factor model was acceptable: $\chi^2(14)=25.6$, $p=.034$; RMSEA = .062; CFI = .95. Completely standardized factor loadings reported (STDYX standardization in MPlus). LTC, latent trait cortisol. *, $p<.05$. **, $p<.01$.

Table 7. Estimates of effects of predictors and covariates on latent trait cortisol.

	Model 1	Model 2	Model 3	Model 4	Model 5
	β(SE)	β(SE)	β(SE)	β(SE)	β(SE)
Early Adversity	-.214(.080)**	-	-	-	-
Ethnic Racial Identity (Total)	-	.116(.083)	-	-	-
Exploration	-	-	.078(.085)	-	-
Resolution	-	-	-	.148(.081)	-
Affirmation	-	-	-	-	-.012(.085)
Birth Control Usage	-.086(.086)	-.120(.087)	-.117(.088)	-.114(.087)	-.106(.088)
Medication Usage	-.047(.085)	-.064(.085)	-.066(.085)	-.068(.084)	-.064(.086)
Some College	.121(.087)	.110(.087)	.103(.088)	.113(.087)	.102(.089)
Advanced Degree	.129(.092)	.151(.101)	.148(.101)	.160(.100)	.151(.101)
Second Generation	.108(.092)	.112(.094)	.116(.096)	.104(.094)	.132(.095)
First Generation	.164(.090)	.139(.091)	.135(.093)	.128(.092)	.145(.095)
Sex (1=M, 0=F)	-.155(.083)	-.130(.084)	-.130(.084)	-.136(.083)	-.134(.085)
High School Ethnic Composition	-	-.022(.092)	-.022(.093)	-.018(.092)	-.024(.0953

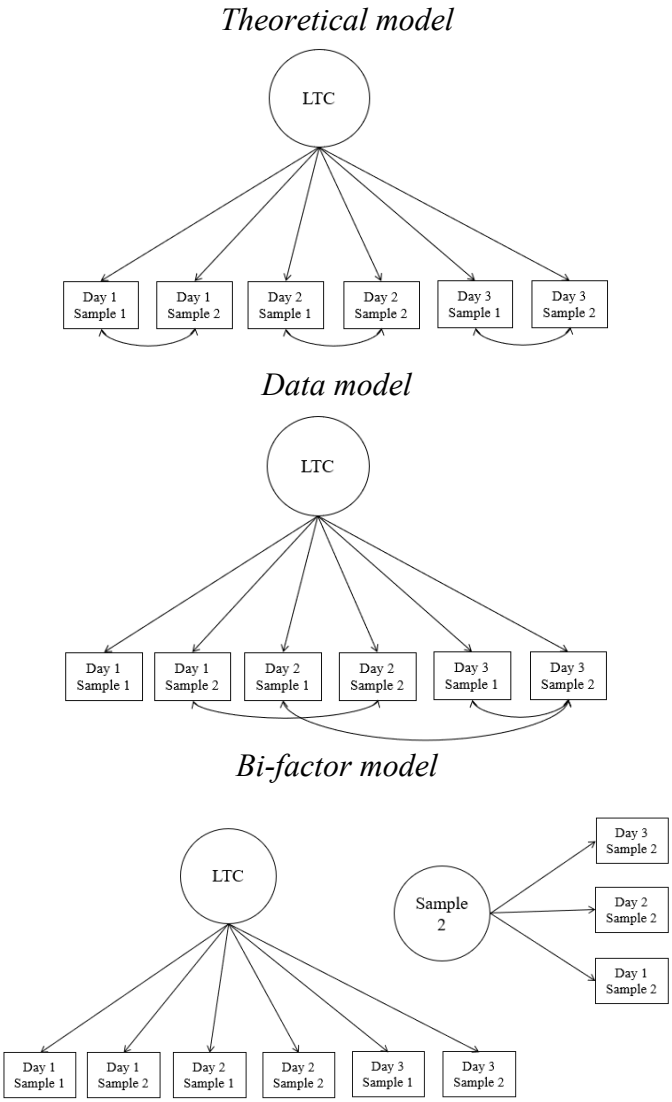
	Model 6	Model 7	Model 8	Model 9
	β(SE)	β(SE)	β(SE)	β(SE)
Early Adversity	-.206(.081)*	-.206(.081)*	-.203(.081)*	-.226(.082)*
Ethnic Racial Identity (Total)	.088(.082)	-	-	-
Exploration	-	.053(.085)	-	-
Resolution	-	-	.129(.080)	-
Affirmation	-	-	-	-.017(.086)
Early Adversity x				
Total ERI	-.053(.079)	-	-	-
Exploration	-	-.051(.081)	-	-
Resolution	-	-	-.014(.078)	-
Affirmation	-	-	-	-.052(.080)
Birth Control Usage	-.099(.086)	-.094(.087)	-.096(.086)	-.084(.087)
Medication Usage	-.046(.084)	-.046(.085)	-.053(.084)	-.047(.086)
Some College	.128(.086)	.122(.086)	.129(.086)	.117(.087)
Advanced Degree	.138(.100)	.135(.100)	.141(.099)	.130(.100)
Second Generation	.089(.094)	.097(.095)	.082(.094)	.100(.094)
First Generation	.143(.092)	.148(.092)	.142(.092)	.147(.095)
Sex (1=M, 0=F)	-.145(.083)	-.145(.084)	-.155(.083)	-.153(.084)
High School Ethnic Composition	.003(.092)	.003(.092)	.005(.091)	.004(.092)

Note: N=197. *, $p<.05$. Estimates based on STDYX standardization. See text for model fit for each model. Theoretical model of LTC served as the outcome for all models.

APPENDIX B

FIGURES

Figure 1. Comparisons of measurement models.



Note: Unique variances were estimated for each factor but are excluded from figure for visual simplicity. Correlations between factors correlated unique variance for each factor. LTC factor variance was constrained to 1.0 for each model (as was the group factor in the bi-factor model). Oral contraceptive and specific medication usage were including as covariates predicting factors.

Figure 2. Conceptual model of Aim 1 path model.

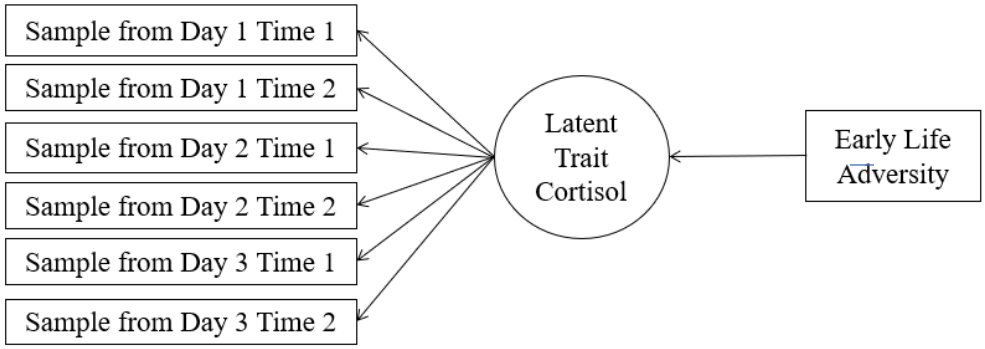


Figure 3. Conceptual model of Aim 3.

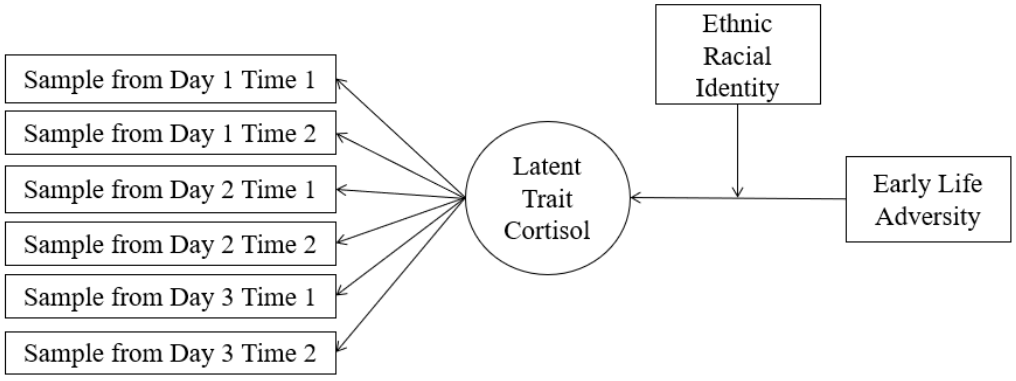


Figure 4. Descriptive scatterplots of average cortisol levels and early life adversity.