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Chronic life stress and change in stress response functioning in urban youth: The role of social support and gender

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Chronic life stress and change in stress response functioning in urban youth: The role of social support and gender

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Biography

The author was born in Midland, Michigan in 1986. She graduated from East Kentwood High School and received her Bachelor of Arts degree in Psychology with honors from DePaul University in 2013.
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Abstract

Salivary alpha-amylase (sAA) has been established as a bio-marker of stress reactivity in acute stressor tasks. Much less research exists exploring whether sAA is bio-marker for chronic stress exposure, and therefore, allostatic load. Extant research is inconsistent with some evidence to support chronic stress exposure having an impact on sAA reactivity and diurnal levels of sAA, while other studies have failed to support this theory. Social support is regarded as a protective factor against stress, mental health, and physical health consequences. It has been suggested that gender may play a role in the utilization and benefits of social support networks. The aim of this study was to add to existing research in finding support for sAA reactivity as a bio indicator of exposure to chronic stress in adolescents, and further ascertain whether social support, in the form of family cohesion, moderated those effects. Additionally, this study aimed to explore whether gender would further moderate the moderation of family cohesion on the relationship between chronic stress and sAA. A sample of 130 public school children in 6th to 12th grade participated in an acute stressor task, during which saliva samples were taken using the passive drool method. sAA was measured from the saliva samples for reactivity during the task. The participants completed the Family Relationship Scale (FRS) questionnaire to assess for family cohesion and participated in a semi-structure Life Stress Interview to be coded by reliable raters for chronic stress scores. The present study did not find significant results for chronic stress as a predictor of sAA reactivity. The role of family cohesion and gender as moderators could not be explored due to lack of a significant relationship between chronic stress and sAA.

Keywords: Salivary alpha-amylase, acute stressor task, chronic stress, family cohesion, adolescents
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Adolescence can be a particularly stressful period in a child’s life. Many changes may occur in their environment during this time, such as the transition from middle school to high school, changes in groups of friends, and changes in relationship with parents as children grow independence. Furthermore, many physiological changes occur during adolescence; the body is maturing and hormone levels are increasing (Hostinar & Gunnar, 2013).

Youth living in urban environments not only experience typical stressors encountered by most adolescents, but they may also be exposed to many additional stressors, such as high rates of community violence (Foster, Kupermine, and Price, 2004; Eisman, Stoddard, Heinze, Caldwell, and Zimmerman, 2015) and economic disadvantage, with minority youth at an especially high risk for experiencing uncontrollable stressors (Landis et al., 2007). Chronic exposure to multiple stressors is associated with many negative mental health outcomes including depression, substance abuse, and anxiety (Low, Dugas, O’Loughlin, Rodriguez, Contreras, Chaiton, & O’Loughlin 2012; Stroud, Davila, Hammern, and Vrshek-Schallhorn, 2011). Additionally, chronic stress exposure also leads to adverse physical health outcomes (McEwen, 2008). Because youth in urban environments are at higher risk for experiencing chronic stressors, this is a particular concern for this population.
The Stress Response System, Salivary Alpha-Amylase, and Allostatic Load

Recent research has begun to explore how salivary alpha-amylase (sAA) serves as a bio indicator of stress and how it may help to identify exposure to chronic stress in particular. sAA is an enzyme found in the saliva which aids in the breakdown of food, the secretion levels of which are regulated by the sympathetic nervous system (Nater, Rohleder, Schlotz, Ehlert, & Kirschbaum, 2007), more specifically the sympa-tho-adrenal medullary system (SAM) (Vineetha, Pai, Vengal, Gopalakrishna, & Narayanakurup, 2014). Increased sAA levels occur after the stress response has been activated via the sympathetic nervous system (SNS) during autonomic nervous system (ANS) activation (Herman, Figueiredo, Mueller, Ulrich-Lai, Ostrander, Choi, & Cullinan, 2003). The ANS serves a large role in responding to stress through the activation of physiological arousal and further, facilitates the return to homeostasis (Herman et al., 2003).

When a person is a faced with an immediate stressor, the sympathetic nervous system of the ANS engages in what is commonly known as the fight or flight response. During SNS activation, epinephrine is released and blood pressure and heart rate increase (Rohleder, Wolf, Maldonado, & Kirschbaum, 2006; Juster, McEwen, Lupien, 2010). Further, the bladder is relaxed, digestion and secretion are inhibited, pupils are dilated, and sweat gland secretion increases (Sapolsky, 2004). All of these processes go into effect to aid the body in the ability to fight off whatever stressor is imminent or flee from the scene. This process in and of itself is an adaptive survival mechanism which aids humans in the ability to
handle an immediate threat or stressor. However, the allostatic load model suggests that exposure to chronic stress leads to over-activation of the stress response system. Those chronic stressors which are uncontrollable are particularly likely to lead to allostatic load.

Allostatic load may include subsequent permanent dysregulation of physiological responses during a resting state as well as dramatically increased or muted physiological responsiveness in several systems in response to acute stressors (Evans, Kim, Ting, Tesher, & Shannis, 2007). To illustrate, an animal model demonstrated how animals exposed to a novel stressor after having been exposed to a previous chronic stressor, showed elevated reactivity to the new stressor in comparison with those animals which had not experienced chronic stress (McCarty, Horwatt, & Konarska, 1988). In a more recent study conducted with human subjects, Nater and colleagues (2007) found that individuals who endorsed high chronic stress exhibited higher sAA reactivity in response to an acute stress task than those participants who did not endorse having experienced chronic stress exposure. This over-activation of the stress response system can add damaging strain on the physiological mechanisms involved (Sapolsky, 2004). This, in turn, can lead to a wide array of physical and mental health problems. For example, a study conducted by Kim and colleagues (2013) found that chronic stress exposure throughout childhood mediated the relationship between childhood poverty and emotion regulation in adulthood due to changes in activity in the prefrontal cortex. In addition, a longitudinal study completed by Gale and colleagues (2015), measured various biomarkers beginning in adolescence which
assessed for allostatic load. Through their research, they found allostatic load partially mediated depression later in adulthood. Further, Seeman and colleagues (2001) found cumulative allostatic load was predictive of declines in physical and cognitive functioning in older age. These studies illustrate the potential long-term consequences of chronic stress beginning in adolescence.

Furthering the impact of stress in adolescents, is the increase in basal as well as reactive levels of HPA functioning during this developmental stage (Gunnar, Wewerka, Frenn, Long, & Griggs, 2009). The physiological changes which occur in adolescence have been shown to increase susceptibility to maladaptive stress response patterns. To illustrate, a study completed with adolescent rats showed chronic stress exposure during this developmental period can lead to hypo-reactivity of the stress response system and also lead to depression like symptomatology (Wulsin, Wick-Carlson, Packard, Morano, & Herman, 2016). It is theorized that HPA axis dysregulation may lead to depression because elevated levels of cortisol disrupt functioning in brain areas important for emotion regulation, impacting the ability to cope with stress (Johnson, Joormann, LeMoult, & Miller, 2015).

A study by Nederhof and colleagues (2015) explored how ANS and HPA axis reactivity taken together may better explain associations between stress and externalizing symptoms in adolescents. Several biomarkers for SNS activation were recorded along with cortisol levels to examine these interactions. Increased reactivity of the ANS along with hypoactivation of the cortisol response during a social stress task were associated with reported externalizing problems in
participating adolescents. These findings suggest allostatic load may play a role in the development of externalizing problems in adolescents and elevated reactivity in the SNS may be a partial mediator of this risk.

Researchers utilize the measurement of sAA to measure reactivity of the SNS as an indicator of the stress response during lab induced stressor tasks. A non-invasive method for the measurement of these is through the collection of saliva samples, which typically involves the passive drool method (Rohleder, Wolf, Maldonado, & Kirschbaum, 2006; Nater & Rohleder, 2009). sAA is an enzyme found in the saliva which has been linked to Autonomic Nervous System (ANS) activation and therefore, higher sAA levels can be expected in saliva when measured during stressor exposure (Nater & Rohleder, 2009). Further, sAA has been credited as a fast responder to lab induced stressor tasks, as well as an indicator of chronic stress (Vineetha et al., 2014).

A gap in the literature exists for sAA as bio-indicator of exposure to chronic stress and allostatic load. Fewer studies exist on this topic in general, and only one research team has explored sAA as it relates to chronic stress in adolescent populations (Wolf, Nicholls, & Chen, 2008), which is discussed in depth within the discussion. Existing research examining chronic stress exposure in relation to diurnal levels of sAA and sAA reactivity, in general, contain inconsistencies, with some studies providing support for sAA as a bio-marker of chronic stress (Nater, Rohleder, Shlotz, Ehlert, & Kirschbaum, 2007; Berndt, Strahler, Kirschbaum, & Rohleder, 2012; Vineetha et al., 2014; Teixeira, Diaz, Silva Santos, Bernardes, Peixoto, Bocanegra, Neto, & Espinada, 2015), while
others do not provide support for this model (Wolf et al., 2008; Strahler, Berndt, Kirschbaum, & Rohleder, 2010).

**Social Support as a Buffer to Stress**

Previous research has explored the importance of social support as a buffer to the effects of stress (Cohen & Wills, 1985; Mossakowski and Zhang, 2014). A recent review of studies by Ditzen and Heinrichs (2014) exploring the buffering effect of social support on stress consolidated evidence to support the theory that social support may act as a buffer on reactivity in the ANS and HPA axis during stress. Specifically, they found that across studies, the strongest buffer effect has been found for perceived social support that is non-evaluative in nature.

Adolescence in particular is a time when social networks may be changing for better or worse. Adolescents tend to rely more on their peer relationships than younger children, because of their growing need for independence. The relationship between mental health related outcomes and multiple forms of stress exposure in youth has been shown to be buffered by social support in numerous studies. Studies exploring these outcomes have found that perceived peer support acts as a buffer for later depression symptoms (Cooley, Fite, Rubens, & Tunno, 2015; Licitra-Kleckler & Waas, 1993; Yang, Yao, Zhu, Zhang, Ling, Abela, Esseling, & McWhinnie, 2010). Further, social support has been shown to moderate the relationship between stress and quality and duration of sleep in adolescence (Van Schalkwijk, Belssinga, Willemen, Van der Werf, Schuengel, 2015).
Interestingly, a study conducted by Muller and colleagues (2000) found that for youth exposed to community violence, social support did not act as a buffer between the development of exposure related stress and trauma and subsequent psychopathology. They did, however, find that social support buffered the association between familial violence and psychopathology. These findings may be important to keep in mind when exploring social support as a buffer for stress in urban youth, given the increased risk of exposure to community violence in this population.

Parent-child dynamics also play an important role in an adolescent’s life, and some studies have found support for the buffering effects of parental support on stress related outcomes in adolescence (Oliva, Jimenez, and Parra, 2009), including depression and delinquent behavior (Licitra-Kleckler & Waas, 1993). Maternal support specifically has been shown to moderate the effects of stress on sleep quality and duration (Van Schalkwijk et al., 2015). In addition, strong parental support has been shown to provide a buffering effect against stressful events related to family (Tsai et al., 2018). Further, previous research has suggested parental support is more stable over time while peer support can change with the recalibration of social networks (McMahon, Felix, Nagarajan, 2011). These findings indicate it is possible that parental support may play a stronger role in buffering the long-term effects of stress. With this in mind, it is essential to explore the potential influence that both peer and familial relationships have on how severe chronic stress can impact urban adolescent youth.
A meta-analytic review on the buffering hypothesis of social support on outcomes of stress proposed that additional factors should be explored to parse out how social support moderation may either be enhanced or diminished (Rueger, Malecki, Pyun, Aycock, & Coyle, 2016). One avenue to explore with adolescent populations is the impact of gender on deriving benefits from perceived social support. To illustrate, research has suggested gender may play an integral role in the buffering effect of peer relationships. Female adolescents, for example, may derive more benefits from social support as a buffer against stress because of their innate tendency to “tend and befriend” (Hostinar and Gunnar, 2013; Taylor, Klein, Lewis, Gruenewald, Gurung, & Updegraff, J. A. 2000). This theory suggests females may be more likely than their male counterparts to seek out social support in times of stress, and further, are more likely to provide support because of their innate tendencies towards nurturance.

**Social Support and Physiological Processes**

As discussed in the above sections, chronic stress has been associated with adverse physiological (McEwen, 2008) and mental health outcomes (Low et al., 2012; Stroud et al., 2011). Social support has been found to moderate the relationship between stress exposure and psychopathology (Cooley et al., 2015; Yang et al., 2010). These findings, from previous research, provide rationale for the hypothesis that social support may have a buffering effect on adverse outcomes related to chronic stress exposure. Because dysregulation of the stress response system is a consequence of chronic stress exposure (Evans et al., 2007; McCarty et al., 1988), it is hypothesized that dysregulation plays a role in the
development and onset of physical health abnormalities and mental health issues that have been related to chronic stress exposure. If social support is found to moderate the hypo-activation of sAA in those with chronic stress exposure, it may indicate an opportunity to prevent related negative physical and mental health outcomes. This is especially pertinent with regards to individuals residing in environments and contexts where exposure to chronic stress is uncontrollable. To illustrate, youth living in urban environments face greater risk of being exposed to community violence and poverty. If these are factors out of their own control, it can be posited the next best step to eliminating the source of chronic stress is in finding a buffer for the effects of stress. Social support may be a relatively accessible means to achieve this goal. Interventions targeted towards building and strengthening supportive social connections with peers and family members in this case, could provide a good means of maintaining mental and physical health despite chronic stress exposure.

**Rationale and Current Study Hypotheses**

Although many studies have explored stress reactivity, the HPA axis, and how social support may play a role as a buffer against harmful physiological effects associated with stress, there is a relative dearth of information in this area for urban, adolescent youth in particular. Further, less focus has been given to the role of ANS functioning and how sAA reactivity as a biomarker of SNS activation may indicate a history of exposure to chronic stress. Because of the high risk of chronic stress exposure associated with this population, it is an important area to explore further. Additionally, gender roles in urban adolescent
youth may influence the extent to which social support serves as a buffer against the effects of chronic stress exposure.

The current study will test the hypothesis that chronic life stress will predict hypoactivation of the sympathetic nervous system as evidenced by lower reactivity of salivary alpha-amylase in those participants who endorse high levels of chronic stress, compared with those who do not. In addition, it is predicted that family cohesion, as a measure of social support, will moderate this effect, with those who higher exposure to chronic stress but high family cohesion not manifesting down-regulation of the SNS stress response system, and therefore, also not exhibiting blunted sAA reactivity during a stress exposure task. Finally, it is expected that females will benefit more from the effects of social support or have higher levels of reported social support than males (see Figure 1 for visual representation of the full model).

**Research Question 1**

Does chronic stress predict dysregulation of the stress response system, specifically SNS functioning as evidenced by lower levels of sAA reactivity?

**Hypothesis 1.** Chronic life stress will predict lower levels of sAA in response to a laboratory stress task.

**Research Question 2**

Does family cohesion moderate the relationship between chronic stress and stress response dysregulation?
**Hypothesis 2.** The relationship between chronic stress and hyporeactivity will be moderated by perceived family cohesion. Such that high levels of family cohesion will reduce the strength of the association between chronic stress and low levels of sAA reactivity.

**Research Question 3**

Does gender moderate the moderating relationship of family cohesion on the relationship between chronic stress and stress response dysregulation, specifically hyporeactivity?

**Hypothesis 3.** Gender will moderate the moderating relationships of family cohesion on the relationship between chronic stress and hyporeactivity of the ANS demonstrated by low levels of sAA reactivity; such that, a three-way interaction will be present between chronic stress, family cohesion, and gender. It is hypothesized that being female will increase the reduction in strength of high levels of familial cohesion on the association between chronic stress and dampened sAA reactivity.
Participants

The current study utilized data from the larger Stress and Learning Project study. Participants who completed the three main measures of focus in the current study, the GPST-A (Hostinar, MCQuillin, Mirous, Grant, & Adam, 2014), UCLA Life Stress Interview (Rudolph & Hammen, 1999), and the Family Relationship Scale (Tolan, Gorman-Smith, Huesmann, & Zelli, 1997), were included in the present sample. One participant was removed from the sub sample due to abnormally high reactivity values, as measured in their salivary alpha-amylase change score. The total sub-sample included in the present study is comprised of 130 sixth to twelfth graders, who were recruited from urban public middle schools and an urban public high school. The sample is representative of almost equal
gender ratio with 53% female (n = 69), and 47% male (n = 61), as well as a diverse array of ethnic backgrounds (38% African American, 32% Latino, 12% Caucasian, 11% Asian, and 7% identified as other). Participants attended a full-day data collection event and were compensated for their time with gift cards to a store of their choosing.

**Procedure**

All of the measures and protocols used in this study were approved by the Institutional Review Board at DePaul University as well as Northwestern University. All adolescent participants signed written assent forms and signed consent forms were obtained from a parent/guardian of each participant. Participants came to DePaul University for a full day of data collection activities on one of five consecutive Saturdays in the fall of 2012. During the data collection day, participants were randomly assigned to one of three groups, determining the order in which they would participate in data collection tasks and other activities. Every group started off with a check-in, orientation, and breakfast then either went on to complete Life Stress Interviews, the Group Public Speaking Task for Adolescents (GPST-A), health and executive functioning measures, surveys, tours, and watched a film. Lunch was served after the initial activity. Each group then went on to complete the next two activities not yet participated in and attended a wrap-up and dinner directly after (see Figure 2).
Figure 2. Data Collection Procedure by Group

Measures

Group public speaking task for adolescents (GPST-A). The Group Public Speaking Task for Adolescents (GPST-A; Hostinar et al., 2014) was used to expose youth to a minor stressor in vivo. The GPST-A, is a modified version of the Trier Social Stress Test for Groups (TSST-G; Von Dawans, Kirschbaum, & Heinrichs, 2011). The GPST-A is an age appropriate version of the task, for adolescents, mimicking a classroom setting rather than a business or laboratory setting as in the TSST-G (Von Dawans et al., 2011). The overall point of the task is still the same, creating an environment of social-evaluative stress (Hostinar et al., 2014).

The GPST-A was administered in the following way: Baseline saliva samples were collected by research assistants using the passive drool method before the task start time, and mood surveys were completed by the participants. For a full visualization of the timeline see Figure 3. The passive drool method required participants to think of something that will cause them to produce more saliva (i.e. “Think about eating something sour”) and filling up a vial with as much drool as possible. The vials were labeled with the participant ID numbers and stored in a refrigerator after collection. The participants were then given three
minutes to prepare a brief speech introducing themselves to a new classroom of
students which they would present in the next phase of the protocol. Five to eight
participants were brought into a classroom and seated at a desk with dividers
between them. Each participant presented their speech for 1.25 minutes; saliva
samples were taken again before the participant gave the speech and immediately
after they gave the speech. The speech was videotaped and there was a 2-person
judge panel at the front of the room and a researcher and research assistant seated
at a table on the right side of the room (when facing front). The judges were
dressed in business attire in an effort to mimic school personnel. After the speech
was completed, participants were given mood surveys and debriefed; saliva
samples were taken at three ten-minute intervals during the debriefing and rest
period. The saliva samples were stored at −20 °C in a freezer until they were sent
by the research team at Northwestern University to the University of Trier in
Germany for time-resolved fluorescence immunoassay (Hostinar et al., 2014). For
the present study, the change in salivary alpha-amylase from saliva sample
collection two to saliva sample collection three was computed to produce a
change score. The difference in measurement at saliva sample collection two and
saliva sample collection three indicate stress reactivity from baseline to speech
time.
UCLA life stress interview. The UCLA Life Stress Interviews (Rudolph & Hammen, 1999) were administered to participants by graduate research assistants to assess chronicity and severity of life stress experienced by participants across multiple domains including: academic, behavioral, peer relationships, parent-child relationship, romantic relationships, health-self, health-other, finance, legal, neighborhood, exposure to violence, and acculturation/discrimination. The UCLA Life Stress Interview is a semi-structured interview that took about 40 minutes to administer on average. The interview begins with general prompts about each of the domains of functioning and more specific queries are given when more information is needed about something the child says. Questions probing the academic domain included “Have you had any problems with schoolwork in the past year?”, “In a typical week, how often do you have some kind of trouble with schoolwork?”, and “How long has it been this way?” (Rudolph & Hammen, 1999). To illustrate how further probing works, if in response to the general question, “How have your grades been?”, if the participant indicates their grades have dropped, questions which
query further information about the situation are asked. These include “How long have they been dropping?”, or “what kinds of grades did you get before?” and “what kinds of grades do you get now?”.

Example questions from the behavior domain include: “In a typical week how often do you get in trouble at school?”, “How do you get along with the adults at your school?”, and “Have you ever had a suspension?”. The peer section of the interview asks questions such as, “About how many close friends do you have?”, “Do you have a friend you feel you can trust?”, “How often do you do something with friends outside of school?”, to name a few. The parent-child section of the interview asks “How are things going at home this year?”, “Do you feel your parents are around when you need them?”, “Are they supportive?”. From the parent-child domain, the interview leads into the marital section asking questions that get at the relationship between caregivers by asking questions such as, “How do your parents or those who live at your home get along with each other?”, “How often do your parents argue or fight?”, and “How do your parents handle disagreements?”. The body image section includes questions such as the following, “Has anything changed about your appearance that you like or don’t like?”, “When people say bad things about the way you look what do they say?”, and “Who are the people that make you feel good about the way you look?”. After the body-image domain, questions are posed in the romantic domain such as, “Have you been in a relationship or dated anyone this year?”, “Have you started any new relationships?”, “Have you had any breakups?”, and “Have you felt any pressure related to sex and dating?”. In the neighborhood domain, the interviewer
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asks “How would you describe your neighborhood?”, “Have there been any changes in your neighborhood?”,”How do you get along with your neighbors?”, and “Are there any stressors associated with travelling through different areas of your neighborhood?” The self-health domain asks questions about the health of the participant such as, “How has your health been?”, “Have you been to the doctor?”, and “Have you experienced any illnesses or injuries?”. The health-other domain focuses on information about the health of friends, family, and acquaintances of the participant and questions are asked such as, “Have there been any illnesses or hospitalizations of a family member or friend?”, “Have there been any deaths of a family member or friend?”, and “Have you experienced the death of a pet?”. The financial domain includes questions such as, “Do you ever feel embarrassed about not having enough money or other things, like clothes?”, “Do your parents ever talk about worries about money?”, and “Do you ever feel pressure to do something illegal to get more money?”. The legal section covers any legal problems that either the participant or their family have experience by asking questions such as, “Have you ever been picked up by the police or had a bad interaction with the police?”, and “Anyone else in your family have problems with the law?”. The violence domain addresses whether the participant has been exposed to any type of violence, for instance, “Have you had any experiences with violence during the past year?”, “Has someone close to you hurt or threatened you?”, “Have you ever seen someone else get hurt or threatened?”, and “Have you heard about someone getting hurt or threatened?”. The final domain of the interview asks about stressors related to acculturation and discrimination and
include the following questions, “Have you ever been treated differently because of your religion, gender, skin color, or sexual orientation?”, “Have you ever had a hard time speaking with others because your English wasn’t fluent?”, “and “Anything specific that’s happened in terms of being treated differently because of your race, ethnicity, religion, gender, sexual orientation that’s been stressful or been a really big change?”.

Each of the domains in the Life Stress Interview include a question at the end of the section designed to assess the chronicity of the events talked about within that domain. The question is “Related to everything we just talked about, how long have things been this way for you?”. Further, when specific events are recounted by the participant, the interviewer asks when those specific events occurred.

**Coding for Chronic Life Stress.** A team of undergraduate research assistants were recruited and trained by the first author to reliably code each interview domain to assess the severity and chronicity of stress for each respectively. Research assistants were assigned to coding team pairs and trained to effectively code the Life Stress Interviews utilizing the developers’ codebook (Rudolph & Hammen, 1999), which outlines criteria for severity scores in each domain on a scale from 1 (severe stress) to 5 (no to low stress). Chronicity scores refer to the length of time the participant has been exposed to the stressors coded within each respective domain and were assessed on a five-point scale using the following criteria as outlined in the codebook: “1” = 1 mo. to 6 mo., “2” = > 6 mo. and ≤ 1 yr., “3” = > 1 yr. and ≤ 2 yrs., “4” = > 2 yrs. and ≤ 5 yrs., and “5” = >
5 yrs. Practice sessions with the full coding team were be conducted on the same practice interviews (about ten interviews) until team members reached consistency with their coding. Once training was complete, the pair members independently coded the same participant interviews and then met for consensus coding. The pairs came to agreement on a “master code” for each domain of the interview, and the codes from each individual coder were entered into a dataset along with the “master code”. The individual codes for each domain of the interview were assessed for inter-rater reliability utilizing IBM SPSS Statistics software for Windows, Version 22.0 (2013) to obtain an intra-class correlation coefficient (ICC) for each domain. The purpose of completing consensus agreement as well as inter-rater reliability with the initial individual scores was to ensure as stringent a method as possible for coding agreement. Eighty-six percent of the interviews were coded by coding dyads who achieved reliability their coding partner and overall team, and fourteen percent were coded by an individual coder who achieved reliability with the rest of the coding team and previous coding partners. A two-way mixed model was utilized to assess for absolute agreement. The Intraclass Correlation Coefficients for ten of the thirteen domains of the life stress interview range from .64 to .82. The domain of Discrimination/Acculturation was removed before calculation of the participant’s mean stress score due to low reliability of the chronicity rating (.22). The domain of parent-child was left out of the cumulative stress average to control for overlap between parent-child stress and family cohesion, and the peer domain was left out
of the cumulative stress average to control for peer evaluative stress in the acute stressor task.

**Family cohesion.** Participants in the current completed four subscales (Cohesion, Organization, Communication, and Support) of the Family Relationship Scale (FRS; Tolan, Gorman-Smith, Huesmann & Zelli, 1997) during the survey portion of the data collection day. The FRS measures 35 self-report items on a 5-point Likert scale and includes six domains of family relationships, including, Cohesion, Beliefs About the Family, Deviant Beliefs, Support, Organization, and Communication. The current study utilized the domain of Cohesion which has been shown to have good reliability by the developers ($\alpha = .72$; Tolan et al., 1997). Internal consistencies were calculated for all four subscales measured on the current sample utilizing IBM SPSS Statistics software for Windows, Version 22.0 (2013). On the current sample, internal consistencies ranged from low to very good ($\alpha .33$ to $\alpha = .85$), with the cohesion subscale demonstrating a very good internal consistency statistic ($\alpha = .85$).

**Analytic Plan**

**Statistical Analyses**

A series of multiple regression models and moderated multiple regression models were computed utilizing the Rockchalk (Johnson, 2018) regression functions package in R Studio, Version 1.01.36 (R Studio Team, 2016). A hierarchical linear regression approach was used for model comparison between multiple regression and moderated multiple regression models, to test for improvement of fit. Preliminary analyses and descriptive statistics were calculated.
prior to computing regression models employing the Psych (Revelle, 2017) package in R Studio, Version 1.01.36 (R Studio Team, 2016). Diagnostic calculations were applied to the regression models with the Car (Fox & Weisberg, 2011) package in R Studio, Version 1.03.36 (R Studio Team, 2016).

**Descriptive statistics and preliminary analyses.** A variance inflation factor (VIF) and tolerance were calculated utilizing the Car (Fox & Weisberg, 2011) package in R Studio, Version 1.01.36 (R Studio Team, 2016) to assess for multicollinearity of continuous predictor and control variables. Statistics for VIF and tolerance all fell within the acceptable range; VIF <10 and tolerance >.10 (Hair, Anderson, Tatham, & Black, 1995). Histograms were plotted to assess for distribution skewness and kurtosis for all continuous variables. A slight positive skew was detected for both salivary alpha amylase and chronic stress and a slight negative skew was detected for age, thus, the Moments (Komsta & Novomestky, 2015) package was employed to calculate the Agostino skewness test (D’Agostino & Belanger, 1990) and D’Agostino and Pearson’s (1973) measure of kurtosis. All variables returned a skewness of less than |2|, which has been identified as the threshold value for non-normally skewed distributions. All variables displayed kurtosis values less than the accepted cutoff level of 7. Kernel density plots for the residuals were produced for the full moderated regression model to test for heteroscedasticity and followed up with a Breusch-Pagan test for non-constant variance, for which the calculated conditional probability was greater than .05 ($\chi^2 = 0.00, p = 0.98$). In addition, Cohen’s (2003) quantification of heteroscedasticity was utilized to determine residual variance values for each
group, given their groupings on X. The largest variance divided by the smallest variance value produced for all models was <10. The distribution of X in relation to the variance of residuals in the kernel density plot was unclear, however, both the Breusch-Pagan test and Cohen’s quantification of heteroscedasticity suggest the models meet assumptions of homoscedasticity.

Missing data were assessed for patterns to assist in determination of values missing completely at random, at random, or missing not at random. Visualized patterns did not suggest data was missing as a factor of the outcome variable, but indicate patterns of missing demographic data, suggesting some participants may have skipped questions during the beginning of the survey. Due to these factors, data were determined to be missing at random (Rubin, 1976). A predictive mean matching (pmm) multiple imputation method, with five imputed data sets and fifty iterations, was utilized to fill in construct-level missingness for the cohesion variable utilizing the MICE (Van Buuren & Groothuis-Oudshoorn, 2011) package in R Studio, Version 1.01.36 (R Studio Team, 2016). Construct-level missingness for the cohesion variable was present for less than ten percent of participants. Pmm has been explored in simulation models and shown to be a more robust option of imputation in comparison with parametric imputation techniques, even in cases of imputation model misspecification (Morris, White, & Royston, 2014). All variables utilized in the regression analyses, along with additional variables being tested for covariation were included in the imputation model. The inclusion of these variables was completed in an effort to comply with best practices in utilizing multiple imputation models by including variables associated with the
variable being imputed, accounting for variables which may assist in explaining missingness of values in the variable targeted for imputation, and inclusion of the criterion variable of the main statistical model (Morris, White, & Royston, 2014). Item-level missingness was two percent in the LSI domains and was corrected utilizing individual mean imputation.

Bivariate correlations (Table 1) revealed the association between chronic stress and sAA reactivity was not significant \((r = -0.10, \rho = 0.87)\). The relationship between family cohesion and sAA reactivity was also not significant \((r = 0.09, \rho = 0.16)\). Additionally, the relationship between family cohesion and chronic stress was not significant \((r = -0.17, \rho = 0.08)\). Age and start time of stressor task are two common covariates for sAA and were assessed for bivariate correlation. There was no significant correlation between sAA and age \((r = -0.08, \rho = 0.36)\) or start time of acute stressor task \((r = 0.04, \rho = 0.65)\). Welch’s Two-Sample T-tests were conducted to test for significant differences between means for males and females on the main variables of interest. Results of the tests suggest differences between the two samples are not significant for sAA reactivity \(t(123.24) = -0.76, \rho = 0.45\), chronic stress \(t(121.17) = 0.62, \rho = 0.54\), or family cohesion \(t(123.99) = 1.35, \rho = 0.18\). Additionally, bivariate correlations were assessed on all variables grouped by gender to assess for differences in direction of association between main variables accounting for gender. The direction of the association remained negative and not significant for chronic stress and sAA reactivity for both genders (see Table 1.2 in the appendix for statistics). The relationship between family cohesion and sAA reactivity remained positive and
not significant for both males and females. The relationship between family cohesion and chronic stress remained negative, though the strength of the association was stronger and significant for males (r = -0.28, p < .05) and not significant for females (r = -0.09, p = 0.46).

**Primary analyses.** To test hypothesis 1, a simple linear regression model was conducted where level of chronic stress (X₁) predicts sAA change score (Y) to test the association between chronic stress and SNS reactivity in the sample. In an attempt to ascertain model fit, a hierarchical approach was taken to assess whether variables of interest, when added to the model, assisted in explanation of variance in sAA change. Family cohesion was added to form a multiple regression model at step two, where chronic stress (X₁) and family cohesion (X₂) performed as predictors of sAA change (Y). To test hypothesis two, the interaction effect of chronic stress and family cohesion was added at step three, forming a moderated multiple regression model including the main effects for chronic stress (X₁) and family cohesion (X₂), and the interaction effect of chronic stress by family cohesion (X₁ * X₂). Both chronic stress and family cohesion were grand mean centered to control for multicollinearity.

Hypothesis 2 was assessed utilizing a multiple regression model, and a moderated multiple regression models, with a hierarchical approach for model fit. Chronic stress (X₁), family cohesion (X₂), and gender (X₃) comprised the independent variables regressed onto sAA change (Y) in the first step. The interaction effect of chronic stress by family cohesion (X₁ * X₂) was added to the multiple regression model in the second step to form the equation
\[ Y = \beta_0 + \beta_1 X_1 + \beta_2 X_2 + \beta_3 X_3 + \beta_4 X_1 \times X_2 + \epsilon. \]

To test hypothesis three, three interaction terms were added to the multiple regression model along with a three-way interaction term to assess the final equation

\[ Y = \beta_0 + \beta_1 X_1 + \beta_2 X_2 + \beta_3 X_3 + \beta_4 X_1 \times X_2 + \beta_5 X_2 \times X_3 + \beta_6 X_1 \times X_3 + \beta_7 X_1 \times X_2 \times X_3 + \epsilon, \]

in an effort to explore whether a moderated moderation effect exists where gender moderates the association of chronic stress and cohesion on sAA change. The steps for reaching the final equation testing hypothesis three, are as follows:

gender was dummy coded with female as the reference code, with a value of 0, and male as the dummy code, with a value of 1. In step three, the second interaction term, of family cohesion (\(X_2\)) by gender (\(X_3\)) was added to the equation

\[ Y = \beta_0 + \beta_1 X_1 + \beta_2 X_2 + \beta_3 X_3 + \beta_4 X_1 \times X_2 + \beta_5 X_2 \times X_3 + \beta_6 X_1 \times X_3 + \epsilon. \]

The final two-way interaction term, chronic stress (\(X_1\)) by gender (\(X_3\)) was added during the fourth step to form equation

\[ Y = \beta_0 + \beta_1 X_1 + \beta_2 X_2 + \beta_3 X_3 + \beta_4 X_1 \times X_2 + \beta_5 X_2 \times X_3 + \beta_6 X_1 \times X_3 + \epsilon. \]

For models included in a hierarchical method of investigation, ANOVAs would typically be run to compare the fit for each model in comparison to the subsequent model for each addition of a new term. This was not completed in the current study as regression coefficients were not significant. Common covariates for sAA, age and time of stressor task, were not correlated with sAA reactivity in
this sample. Because no significant correlations were found, as controls, these variables were not included in the analyses.

Results

Hypothesis 1. First, the relationship between chronic stress and sAA change in response to a stressor task was assessed with a simple linear regression model. The results for the analysis were insignificant ($\beta = -0.10, \rho = 0.26$; see Table 2 in the appendix), therefore, the null hypothesis was not rejected.

Hypothesis 2. As a first step to exploring whether there is a relationship between chronic stress, cohesion and sAA reactivity in our sample, and whether family cohesion moderates the relationship, cohesion was added to our first model, to create model 2 (see Table 3 in the appendix). The overall multiple regression model of sAA reactivity predicted by chronic stress and family cohesion was not significant ($F(2, 123) = 0.98, \rho = 0.38, \text{adj. } R^2 = 0$), nor were the main effects ($\beta = -0.10, \rho = 0.26$). The interaction of chronic stress and cohesion on sAA reactivity was examined in model 3, and values were not significant ($F(3, 122) = 1.22, \rho = .31, \text{adj. } R^2 = .005; \beta = -0.12, \rho = 0.20$; see Table 4 in the appendix), indicating the null hypothesis was not rejected.

Hypothesis 3. In model 4, chronic stress, family cohesion, and gender were set as predictors for sAA reactivity. The overall model was not significant ($F(3, 122) = 0.87, \rho = 0.46, \text{adj. } R^2 = 0$; see Tables 5 through 8 in the appendix for full steps and corresponding statistics). The interaction of stress, cohesion, and gender was tested in model 9, the overall model was not significant ($F(7, 118) = 0.89, \rho = .52, \text{adj. } R^2 = 0$; see Table 9 in the appendix), nor were the interaction
effects ($\beta = -0.27$, $\rho = 0.18$; see Table 9 in the appendix), indicating the null hypothesis was not rejected.

**Discussion**

The goal of the current study was to identify whether a relationship exists between chronic stress and autonomic nervous system functioning as measured by sAA reactivity. This information would be a useful addition to extant research on allostatic load. Further, we wished to explore whether family cohesion moderates this relationship, in general, and whether differences exist in the moderating relationship according to adolescent gender. The purpose of hypothesis 1 was to explore whether chronic stress in the current adolescent population is related to sAA reactivity. More specifically, we tested whether higher levels of chronic stress are related to stress response dysregulation in the SNS. It was hypothesized that higher levels of chronic stress would predict lower sAA reactivity in male and female adolescents. Though sAA has been established as a biomarker that adequately measures reactivity during acute stressor tasks (Nater, Rohleder, Gaab, Berger, Jud, Kirschabum, & Ehlert, 2005; Rohleder, Wolf, Maldonado, & Kirschbaum, 2006; van den Bos, Rooij, Miers, Bokhorst, & Westenberg, 2014; Katz & Peckins, 2017), research regarding the reactivity of sAA in adolescents after exposure to an acute stressor task in relation to chronic stress remains relatively unexplored. One study completed by Wolf and colleagues (2008), with children and adolescents, reports lower daily sAA output in children with asthma who have experienced relatively higher chronic stress compared with control participants without asthma and exposure to relatively high levels of chronic
stress. The same study did not reveal a significant relationship between daily sAA output and chronic stress exposure in children without asthma. Research studies exist exploring sAA functioning and chronic stress exposure in adult populations but report inconsistent findings regarding whether chronic stress exposure is associated with, or effects a change in diurnal sAA or sAA reactivity. To illustrate, Teixeria and colleagues (2015) found blunted sAA reactivity in male adults with chronic stress exposure in response to an acute stressor task. Berndt, Strahler, Kirschbaum, and Rohleder (2012) found daily values of sAA are lower in adult competitive ballroom dancers who have experienced chronic exposure to social/evaluative stress compared with a control group. Conversely, Nater and colleagues (2007) found a significant positive relationship between chronic stress exposure and diurnal levels of sAA, such that participants who reported experiencing higher levels of chronic stress, exhibited a pattern of elevated sAA across the day. Finally, Strahler and colleagues (2010) compared young and older adults and did not find support for chronic stress predicting a change in diurnal sAA. The varying results of these studies depict a necessity for further research into how chronic stress may or may not impact the bio-marker, sAA.

The results of the present study were not significant and fail to support previous research indicating chronic stress exposure leads to change in sAA levels during acute stressor exposure. The lack of significant findings reflects the inconsistencies in existing published studies and supports the need for additional research to be conducted and made available to better understand factors contributing to these inconsistencies. Additionally, studies exploring this topic
with adolescent populations is scarce. Exploration of sAA functioning in adolescents related to chronic stress exposure is critical given the variability of stress bio-markers across the developmental period.

The purpose of hypothesis 2 was to explore whether family cohesion reduces the strength of the effect of chronic stress on sAA reactivity. Significant moderation effects were not discovered ($\beta = -0.12, \rho = 0.20$; see Table 4 in the appendix for full results).

The purpose of hypothesis 3 was to ascertain whether the moderating effect of family cohesion on chronic stress exposure and sAA reactivity is further moderated by gender. Specifically, if a significant relationship between chronic stress and sAA were present, and family cohesion moderated the relationship, we expected to see further moderation by gender. The results of the moderated moderation were not significant ($\beta = -10.06, \rho = 0.18$; see Table 9 in the appendix for full results).

The insignificant results in this study, are not completely surprising given the varied findings in extant research exploring the effects of chronic stress exposure on SNS functioning, and more specifically, sAA reactivity (Wolf et al., 2008; Nater & Rohleder, 2009; Strahler et al., 2010; Berndt et al., 2012; Teixeria et al., 2015). As mentioned previously, much less research has focused on exploring effects of chronic stress on sAA reactivity and functioning in comparison with cortisol reactivity, and only one study (Wolf et al., 2008) was found which explored sAA functioning in relation to chronic stress exposure in adolescent populations. Given most non-significant findings from studies are not
published in empirical journals, it is conceivable unknown studies on this topic exist which also failed to find significance between the relationship of chronic stress exposure and sAA reactivity in human populations.

It is possible sAA does not reflect the effects of chronic stress and allostatic load as well as cortisol, heart rate variability, or other measures. A research study by Ali and Pruessner (2012) provided evidence for a ratio of sAA and cortisol which they reported as being a better indicator of allostatic load than cortisol or sAA alone. Given the sympathetic nervous system and the parasympathetic nervous system work together in homeostatic processes, replication of the findings reported by Ali and Pruessner (2012) would support a more holistic measure of allostatic load in stress research. This may be especially impactful given much research is dichotomized where exploration of sAA, cortisol and other bio indicators of stress and allostatic load are concerned, with findings indicating these markers do not always parallel one another (Schommer, Hellhammer, & Kirschbaum, 2003; Nater, La Marca, Florin, Moses, Langhans, Koller, & Ehlert, 2006; Nater et al., 2007; Nederhof et al., 2015). Additionally, future studies seeking to replicate these findings may explore potential protective effects of variables including familial support and peer support to help better define a model whereby adolescents may reduce the risk of future physical and mental health outcomes related to allostatic load.

The lack of change in sAA functioning could also be specific to this sample. For example, it may be indicative of developed resiliency in response to the experience of chronic stressors. The allostatic load model posits individual
differences in allostatic functioning of the stress response system exist based on a host of factors including adaptation, coping, resiliency, and genetic factors (McEwen, 1998). It is possible, therefore, that individual level and system level differences exist in this sample that protect against physiological effects of repeated stress exposure. Given the current sample of adolescents reside in an urban setting, where exposure to community violence and other stressors unique to urban settings exist, it is possible the youth have developed adaptive coping strategies and gained resilience in response to chronic stressors. An animal model conducted by Suo and colleagues (2013), illustrates chronic predictable stress exposure in adolescence predicting increased resiliency in adulthood. Another animal study demonstrated utilization of adaptive coping strategies in response to a novel stressor after chronic stress exposure in adolescence (Kendig, Bowen, Kemp, & McGregor, 2011). Studies in humans have also found that community cohesion, sense of belonging in neighborhood, a strong parent-child relationship, attendance of religious services, and finding meaningfulness in life are protective factors against ill effects commonly associated with acute and chronic stress (Gelkopf, Berger, Bleich, & Silver, 2012; Juster, McEwen, & Lupien, 2010; Grote, Bledsoe, Larkin, Lemay, & Brown, 2007). Therefore, it is possible adolescents in this sample may generally possess one or more of the aforementioned protective factors. Further exploration would need to be conducted on the sample to see if this is the case. Further, differences in sAA reactivity within the sample may exist such that we were not able to adequately
parse out the effects of chronic stress exposure on sAA reactivity. These limitations are discussed in the section below.

**Limitations and Future Directions**

There are several limitations that could have influenced the results. In particular, the subsample used for this study was relatively small, making representation of adolescents with lower sAA reactivity compared to those exhibiting expected sAA reactivity relatively difficult to explore. Multiple variables shown to be covariates with sAA reactivity were not included in the models of this study, including hours of sleep the night before the study, monitoring of physical activity the day of the study, and BMI. Some research has exhibited results supporting these factors do not affect sAA reactivity levels (Nater et al., 2007), however, additional research supporting these findings should be accumulated before such variables are eliminated as covariates in future studies.

The impact of chronic stress on diurnal patterns of sAA reactivity were not explored in this study and should be included in future studies to learn more about the impact of chronic stress on ANS functioning along with other outcomes (e.g. heart rate variability, respiration, and blood pressure) to gain a better understanding of ANS response to stress and return to homeostasis (Rohleder et al., 2006; Juster et al., 2010). Previous research has explored the utilization of sAA and cortisol reactivity together as a more holistic measure of allostatic load of the stress response system and provided results that indicate it is a better measure of stress response system functioning in relation to chronic stress than
sAA or cortisol alone (Ali & Pruessner, 2012). Further, research indicates the
various biomarkers of stress are measurements of varying systems (Schommer et
al., 2003) and possibly reflect different types of stress making them useful as
independent parameters explored together (Nater et al., 2006).

It is also possible that, instruments were not adequate in exploring the
constructs of focus. For example, chronic stress was measured utilizing coded
values from a semi-structured interview which requires the reliance on self-report.
It is possible under-reporting of stressful experiences occurred if participants felt
discomfort in discussing topics of life stress with the interviewer. Further, the
interviews were one task out of many during a data collection day, which may
have been motivation for participants to report less to get through the interview
faster. Under-reporting of stressful events across domains would impact the
strength and possibly the direction of the associations explored in the current
study. For example, if a percentage of adolescents falsely denied experience of
any stressful events but had, in fact, experienced moderate to high levels of stress
across domains the direction between the relationship of chronic stress exposure
and sAA could be misrepresented, the strength of the association weakened, or
both.

Relatedly, it is possible our measurement of chronic stress was not a
complete or accurate representation of chronic stress experienced by our sample.
Because the goal of this study was to look at overall chronic stress exposure
across multiple domains, scores were averaged across domains for each
participant to obtain one, overall, score representing chronic stress. It is possible
each domain potentially holds different weight, in general, or for each individual, which would become indiscernible once collapsed into an averaged value. Another option would be to include several measures of chronic stress or variables known to affect allostatic load, individually, such as, community violence, income, and exposure to racism or discrimination, among others (Juster et al., 2010). Measures using objective data (i.e. geo-mapping for rates of community violence, family income level, etc.) lessening reliance on self-report.

Finally, if there are differences in sAA functioning to be discerned in adolescent human populations exposed to chronic stressors, it may be there was not enough variety in our sample, or a big enough sample size to view these differences. To explicate, the range of reported chronic stress for this sample was 1 to 3.08, with possible values ranging from 1 to 5. The mean of this sample is 1.86, with the majority of the values falling between 1.5 and 2.5. If chronic stress does indeed influence sAA, it may be stress exposure for this sample, is generally too low to see any effects of allostatic load. Participants reporting more exposure to chronic stress may, additionally, have been too few for any relationship to be seen. If the same study was replicated in a larger sample, and perhaps, representative of a larger geographical region of the city, more variability in chronic stress exposure may be seen.

Finally, the present study was cross-sectional, and therefore, included only associations between the variables of interest at one time-point. Perhaps, significant findings would emerge from a longitudinal, mediational study examining causal effects of chronic stress and stress reactivity over time.
Summary & Conclusion

Adolescence is a developmental period fraught with physiological and psychosocial changes and can be a stressful period for those in this stage. Adolescents in urban environments may experience additional stressors unique to their urban setting including community violence, low-income, lack of access to adequate resources, and over-crowding (Foster et al., 2004; Eisman et al., 2015). Previous research has shown exposure to chronic stress to be deleterious to physical (McEwen, 2008) and mental health outcomes (Low, 2012; Stroud et al., 2011).

Allostatic load describes the change in homeostatic physiological processes in which the human body engages to maintain important regulatory functions triggered by exposure to stressors over time (McEwen & Wingfield, 2003). Extant research posits cortisol as being a reliable biomarker for measuring the stress response and somewhat less reliable measure of allostatic-load given variation in findings (Miller, Chen, & Zhou, 2007; Agbedia et al., 2011; Slodek, Doane, & Stroud, 2017; Kwak, Taylor, Anaya, Feng, Erich, & Jones, 2017). Heart rate variability, blood pressure, respiration, and sAA have been identified as biomarkers for stress in the ANS (Rohleder et al., 2006; Juster et al., 2010). Less research has explored whether the effects of chronic stress and allostatic load are visible in samples of sAA. Social support has been shown to have protective effects against stress (Cohen & Willis, 1985; Oliva et al., 2009; Mossakowski & Zhang, 2014; Ditzen & Heinrichs, 2014; Van Schalkwijk et al., 2015; Tsai et al., 2018) and various adverse mental health risks in previous research (Cooley et al.,
Additional differences have been explored that suggest males and females may rely differentially on social support networks when faced with stressful experiences (Hostinar & Gunnar, 2013; Taylor et al., 2000).

The present study endeavored to add to the literature on these topics by exploring the effects of chronic stress in adolescents, as measured through a LSI, on sAA change in response to an acute stressor task. Exploring whether social support moderated the effects of chronic stress exposure on allostatic load and whether gender changed the strength of that relationship was an additional goal of the current study.

The present study did not find a significant relationship between chronic stress exposure and sAA change in response to an acute stressor task. These findings do not lend support to sAA as a biomarker for allostatic load. Unfortunately, because the relationship between chronic stress exposure and sAA was not significant, in this study, familial cohesion and related differences of possible buffering effects by gender, could not be explored.

Limitations for this study included measurements that may not have fully captured variables of interest and limited sample size. The scope of this study was relatively small given the limited number of variables explored. Future research could include several biological measures for SNS and ANS functioning and incorporate a ratio of cortisol and sAA, as discussed in a previous section. Further, baseline and diurnal patterns could be explored, in addition to reactivity, for each of these measures. This would aid in identifying whether allostatic load
can be measured in daily patterns, reactivity, both, or neither for specific bio-
markers. Additionally, self-report and objective measures of chronic stress should
be included in future studies, and specificity of different types of chronic stress
should be explored. This study highlighted the inconsistent findings in present
research and posits a need for additional specificity and comparison of measures
to elucidate the complex mechanisms of physiological processes as they relate to
chronic stress exposure.
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Appendix A
Study Models

Model 1: sAA predicted by chronic stress exposure
sAA reactivity ~ Chronic Life Stress

Model 2: Moderating relationship of social support
sAA reactivity ~ Chronic Life Stress + Family Cohesion + chronic life stress * Family Cohesion

Model 3: Gender moderating the moderating relationship of family cohesion on chronic stress and sAA
sAA reactivity ~ Chronic life stress + family cohesion + gender + chronic life stress * family cohesion * gender

Figure 1. Moderated Moderation Model
## Appendix B

### Family Relationship Scale

1. Below are a list of things that describe families. We want to know how much each one is true about your family. Using the scale below, pick the best answer that best says how true each one is about your family in your opinion.

<table>
<thead>
<tr>
<th>Statement</th>
<th>Not At All True</th>
<th>Hardly Ever True</th>
<th>True A Lot</th>
<th>Almost Always or Always True</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. My family expects too much of me.</td>
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<tr>
<td>2. My family knows what I mean when I say something.</td>
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<td>3. My family doesn't care about me.</td>
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<td>4. I often understand what other family members are saying.</td>
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<td>5. If someone in the family has upset me, I keep it to myself.</td>
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<tr>
<td>6. I have trouble accepting someone else’s answer to a family problem.</td>
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<td>7. If I’m upset with another family member, I let someone else tell them about it.</td>
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<td>8. My family doesn’t let me be myself.</td>
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<td>9. My family and I have the same views about what is right and wrong.</td>
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<td>10. I keep on trying when things don’t work out in the family.</td>
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</table>

2. Using the scale below, pick the best answer that best says how true each one is about your family in your opinion.

<table>
<thead>
<tr>
<th>Statement</th>
<th>Not At All True</th>
<th>Hardly Ever True</th>
<th>True A Lot</th>
<th>Almost Always or Always True</th>
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</thead>
<tbody>
<tr>
<td>11. I am tired of being blamed for family problems.</td>
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<td>12. I am able to let others in the family know how I really feel.</td>
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<td>13. My family and I have the same views about being successful.</td>
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<td>14. I’m available when others in the family want to talk to me.</td>
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<td>15. I listen to what other family members have to say, even when I disagree.</td>
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<tr>
<td>16. I worry too much about the rest of my family.</td>
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<td>17. Family members ask each other for help.</td>
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<td>18. In solving problems, the family follows the children’s suggestions.</td>
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<td>19. Family members like to spend time with each other.</td>
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<tr>
<td>20. Family members feel very close to each other.</td>
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Note: Items from the family cohesion subscale (14, 15, 17, 19, 20, 22) were utilized in this study
Appendix C
Stress Taxonomy Codebook

Stress Taxonomy Codebook

Chronicity Initial:

<table>
<thead>
<tr>
<th></th>
<th>1 mo.- 6 mo.</th>
<th>&gt;6 mo. and ≤ 1 yr.</th>
<th>&gt;1 yr. and ≤ 2 yrs.</th>
<th>&gt;2 yrs. and ≤ 5 yrs.</th>
<th>&gt;5 yrs.</th>
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Chronicity Follow-Up (Past Year):

<table>
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<th></th>
<th>1 mo.- 3 mo.</th>
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<th>&gt;6 mo. and ≤ 9 mo.</th>
<th>&gt;9 mo. and &lt; 12 mo.</th>
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Chronicity ratings should be made for each category

A. Academic Chronic Stress

1. Severe academic stress:
   - Failure in 3 or more subjects in 1 year; or
     - Failure of a grade; or
     - Full-time placement in a learning disabilities class; or
     - Extreme disparity between parent(s)’ (or other central figures’) expectations and adolescent’s academic functioning or his/her perceptions of academic ability (e.g., adolescent experiences extreme pressure to “get all A’s” and believes he/she is not capable of achieving at this level) with significant perceived negative consequences for failure to meet expectations (e.g., loss of place on important sports team if expectations are not met, perception that parents will no longer love the adolescent if expectations are not met).

2. Serious academic stress:
   - Failure of 1-2 subjects in 1 year; or
     - Near failure in greater than 2 subjects; or
     - Threat of grade failure; or
     - Part-time placement in a learning disabilities class without significant strengths in other areas; or substantial change in functioning (e.g., dropping from an “A” student to a “C” student); or
     - Substantial “under-functioning” relative to goals set by significant others (e.g., parents/teachers/coaches) with significant negative feedback from parents/teachers that improvement must be made.

3. Moderate academic stress:
   - Near failure (i.e. D grade) in 1-2 subjects; or
     - failure for limited time in a single subject, which was later resolved; or...
• Significant variability in performance across academic subjects and/or time.
• Noticeable change in functioning (e.g., dropped from a “B” to a “C” student);
• Some negative feedback from parents/teacher that improvement must be made.

4. **Average academic stress:**
   • B to C average
   • No significant problems or changes
   • Significant others may wish for improvement, but substantial pressure to improve is not present
   • There is little negative feedback about performance and/or negative feedback is balanced by positive feedback.
   • Student may report that they do not have enough time to finish homework and/or fulfill responsibilities in numerous extracurricular activities

5. **Little to no academic stress:**
   • Mostly A’s; or
     o In accelerated classes and doing above average work.
   • Not below average in any classes.
   • Parents/teachers/other important figures are pleased with adolescent’s performance.

B. Behavioral Chronic Stress (at school)

1. **Severe behavioral stress:**
   • Expelled from school; or
     o In special school due to behavior problems; or
     o Dropped out of school.

2. **Serious behavioral stress:**
   • Repeated suspensions or repeated truancy (>25% of the time); or
     o Significant trouble at school related to behavior problems.

3. **Moderate behavioral stress:**
   • Single suspension for greater than 1 day; or
     o Repeated trouble at school related to behavior problems for a short period; or
     o Significant behavioral conflicts with teachers (whether initiated by self or others).

4. **Average behavioral stress:**
   • No significant problems (e.g., may have minor infractions).

5. **Little to no behavioral stress:**
   • No problems reported during any time period.
C. Peer Chronic Stress

*Peer stress is evaluated on the basis of the presence of friends, quality of friendships, instrumental and emotional support, conflict/conflict resolution, rejection, and loneliness/social isolation.*

1. **Severe peer stress:**
   - Adolescent has no friends/is completely isolated from peers; or
     - Adolescent is frequently engaged in fights and conflicts/is rejected by peers.
   - Adolescent consistently experiences intense pressure to conform to peers; or
     - Adolescent consistently experiences intense teasing from peers
   - Adolescent is a member of a gang; or
     - Adolescent consistently experiences intense pressure to join a gang; or
     - Adolescent reports having two or more very close friends that are involved in gangs

2. **Serious peer stress:**
   - Adolescent is somewhat isolated from peers and spends much time alone, but has some acquaintances; or
     - Adolescent has 1 or 2 friends but has frequent conflicts with peers and friendships are of very low quality (e.g., low on trust and supportiveness).
   - Adolescent often experiences moderate to intense pressure (or somewhat moderate and frequent pressure) to conform to peers; or
     - Adolescent often experiences moderate to intense teasing from peers
   - Adolescent often experiences moderate to intense pressure (or somewhat moderate and frequent pressure) to join a gang; or
     - Adolescent reports a moderately close friend is a member of a gang

3. **Moderate peer stress:**
   - Adolescent is average in popularity, but has no close friends; or
     - Adolescent has some close friends but is engaged in conflicts with peers every now and then or has inadequate social support.
   - Adolescent experiences occasional intense pressure (or frequent mild pressure) to conform; or
     - Adolescent experiences occasional intense (or frequent mild) teasing from peers
   - Adolescent experiences occasional intense (or frequent mild) pressure to join a gang; or
     - Adolescent reports one or more acquaintances that are members of a gang

4. **Average peer stress:**
• Adolescent has some close friends and engages in some social activities outside of school
• Adolescent has no significant problems with peers at school
• Adolescent experiences occasional mild pressure to conform; or
  o Occasional mild teasing from peers

5. **Little to no peer stress:**
• Adolescent has many close friends, is well-liked, and engages in frequent social activities outside of school
• Adolescent has no problems with peers at school.

D. Family Chronic Stress

**Parent-Adolescent Stress**

*Parent-adolescent stress is evaluated on the basis of parent time availability, trust, provision of tangible and emotional support, and conflict/conflict resolution.*

1. **Severe parent-adolescent stress:**
• Adolescent has very poor quality of relationship with parents, as evidenced by several significant problems without significant positive aspects of the relationship (e.g., lack of communication or trust; parents not available; can’t turn to parents with problems; frequent arguments; arguments poorly resolved; anger persists after argument is over).

2. **Serious parent-adolescent stress:**
• Adolescent has some significant ongoing problems with parents, but relationship has some positive aspects as well (e.g., parent is available, but there are frequent, poorly resolved conflicts; there are frequent conflicts, but adolescent still feels he/she can turn to parent with problems).

3. **Moderate parent-adolescent stress:**
• Adolescent has good quality relationship with either mom or dad, some problems in relationship with 1 parent; or
  o If just one parent, adolescent has inconsistent relationship with parent, sometimes good and sometimes poor.

4. **Average parent-adolescent stress:**
• Adolescent has consistently good quality relationship with at least one parent and no significant problems with other parent; or
  o If just one parent, adolescent as consistently good relationship with parent

5. **Little to no parent-adolescent stress:**
• Adolescent has exceptional quality relationships with both parents.

**Marital Stress**
1. **Severe marital stress:**
   - Parents or other parental figures have substantial conflict (e.g., conflict is intense, frequent, poorly resolved; few positive qualities in the relationship); and/or
     - There is evidence of one or more incidents of marital violence.

2. **Serious marital stress:**
   - Parents or other parental figures have some conflict (e.g., conflict is somewhat intense, frequent, and poorly resolved)
   - Relationship may have some positive qualities
   - Parents do not argue; but overt tension exists in relationship

3. **Moderate marital stress:**
   - There is conflict between parents or other parental figures, but it is of moderate extremity (e.g., frequent, but not intense)
   - Some positive qualities in the relationship.
   - Parents do not argue, but tension is evident; or
     - Parents are divorced

4. **Average marital stress:**
   - Parents for the most part get along fine, with occasional minor conflicts.
   - There is no contact between parental figures (i.e. single-parent)

5. **Little to no marital stress:**
   - Parents have an exceptional relationship with each other.
   - Infrequent conflicts are resolved maturely.

E. **Body Image Chronic Stress**

1. **Severe body image stress:**
   - Adolescent dislikes 5 or more aspects of appearance; or
     - Wants to change 5 or more aspects of appearance; or
     - Dislikes one or more aspects of appearance intensely; or
     - Has tried many methods or several more severe methods (e.g., exercises excessively, vomits to control weight, refuses to eat, etc.) to change appearance; or
     - Adolescent receives frequent global negative feedback about appearance (e.g., central figures, such as parents, describe the adolescent as “fat”, and/ or peers describe the adolescent as “ugly”) and little or no positive feedback.

2. **Serious body image stress:**
   - Adolescent dislikes 3-4 aspects of appearance; or
     - Wants to change 3-4 aspects of appearance; or
     - Substantially dislikes one or more aspects of appearance; or
     - Has tried several methods (3 or more) to change appearance; or
1. Receives frequent negative feedback about appearance that is not
global (e.g., comments about unattractive hair or pimples on face)
or global negative feedback balanced with some positive feedback
about appearance.

3. **Moderate body image stress:**
   - Adolescent dislikes 1-2 aspects of appearance; or
     - tries to change 1-2 aspects of appearance
   - Adolescent has engaged in several less serious methods to change
     appearance, such as occasional dieting and/or exercise; or
     - Occasionally receives negative feedback about appearance.

4. **Average body image stress:**
   - No significant problems with body image
   - May have dieted or exercised to modify appearance but reports overall
     satisfaction with appearance; or
     - Dislikes 1-2 aspects of appearance but has not tried to change
     appearance OR
       - Wants to change one or more aspects of appearance
   - Has experienced infrequent negative comments about appearance.

5. **Little to no body image stress:**
   - No body image problems reported
   - Adolescent expresses satisfaction with appearance and appears to
     appreciate appearance.
   - Adolescent reports that he/ she has not experienced any negative feedback
     about appearance.

F. Romantic Relationships Chronic Stress

1. **Severe romantic relationship stress:**
   - Adolescent has had several important relationships that have ended badly; or
     - Adolescent has experienced rejection/ betrayal in a highly
       significant relationship (e.g., romantic partner was primary source
       of support); or
     - Adolescent has been a part of a significant relationship
       characterized by frequent and intense conflict (or one or more
       incidents of dating violence); or
     - Adolescent reports a significantly distressing experience(s) related
       to sexuality (e.g., date rape, pregnancy, abortion, miscarriage, giving
       up baby for adoption, etc.).

2. **Serious romantic relationship stress:**
   - Adolescent has had a few significant romantic relationships that have ended
     badly; or
     - Has experienced rejection/ betrayal in a significant relationship; or
3. **Moderate romantic relationship stress:**
   - Adolescent may have had relationships that ended badly but they were not significant relationships; or
   - Adolescent occasionally has problems or gets into arguments with partner.

4. **Average romantic relationship stress:**
   - No significant problems
   - Romantic relationships are either not that serious or no significant problems are reported.

5. **Little to no romantic relationship stress:**
   - No problems reported; or
   - No romantic relationships present.

G. **Neighborhood Chronic Stress**

1. **Severe neighborhood stress:**
   - Adolescent reports many problems with neighbors, as evidenced by several significant arguments and/or altercations; or
   - Significant changes in the neighborhood have taken place (e.g., marked increase in crime or gentrification and/or flight of lower-income or higher-income families); or
   - Adolescent rarely feels safe in neighborhood; or
   - Adolescent has been a victim of a serious crime or has witnessed a serious crime take place (e.g., physical assault, rape, murder, etc); or
   - Adolescent does not feel he/she can trust any neighbors; or
   - There have been one or more significant instances in which the adolescents’ family was made to feel unwelcome in the neighborhood (e.g., cross-burning on lawn).

2. **Serious neighborhood stress:**
   - Adolescent reports some problems with neighbors, as evidenced by a few significant arguments and/or altercations; or
   - There have been a few changes to the neighborhood (e.g., moderate increase in non-violent crime, such as vandalism, drug sales, etc.); or
   - Adolescent expresses a moderate amount of concern about his/her safety in the neighborhood; or
   - Adolescent has been a victim of a non-violent crime (e.g., mugging, theft, etc.); or
3. **Moderate neighborhood stress:**
   - Adolescent reports a few Moderate problems with neighbors that were resolved or were not significant in nature; or
     - Adolescent has witnessed a non-violent crime (e.g., mugging, theft, vandalism, drug sales)
   - Adolescent feels safe in his/her neighborhood for the most part; or
     - Adolescent has a positive relationship with some neighbors.

4. **Average neighborhood stress:**
   - Adolescent feels comfortable and safe in his/her neighborhood
   - He/ she may have heard about a non-violent crime committed in the neighborhood
   - Adolescent feels he/ she can trust most neighbors (not required to know most neighbors)

5. **Little to no neighborhood stress:**
   - No problems reported during any time
   - Adolescent feels comfortable and safe in his/her neighborhood all of the time.
   - Adolescent reports good relationships with neighbors.

**H. Health-Self Chronic Stress**

1. **Severe health-self stress:**
   - Adolescent reports a severe (i.e., potentially life-threatening or chronically debilitating) health problem or accident/ injury.
     - Chronic seizures, terminal illness, severe chronic disease/illness

2. **Serious health-self stress:**
   - Adolescent reports one or more serious (i.e., temporarily debilitating) health problem(s) or accident(s)/ injuries.
     - One lifetime seizure, non-chronic severe illness (i.e. pneumonia)

3. **Moderate health-self stress:**
   - Adolescent has experienced health problems but they did not result in significant debilitation (i.e., adolescent was able to return to school and participate in most activities within a few weeks).
     - Broken bones
4. **Average health-self stress:**
   - Adolescent reports a few minor illnesses and/or injuries.
     - Sprain
   - Adolescent had to visit the doctor/urgent care

5. **Little to no health-self stress:**
   - Adolescent reports being very healthy with few if any illnesses or injuries.

I. Health-Other Chronic Stress

1. **Severe health-other stress:**
   - Adolescent reports that a very close family member or friend has experienced a severe (i.e., potentially life-threatening or chronically debilitating) health problem(s) (including mental health) or accident/injury and/or has died.
     - Parent/primary care-giver death only; may apply to family member who is a parental figure to the child (e.g. grandparent who is like a parent); or
     - Death of close sibling

2. **Serious health-other stress:**
   - Adolescent reports that a very close family member or friend has experienced one or more serious (i.e., temporarily debilitating) health problem(s) (including mental health) or accident(s)/injuries; or
     - A moderately close family member or friend has experienced a severe (i.e., potentially life-threatening or chronically debilitating) health problem(s) or accident/injury and/or has died.
     - Grandparent death

3. **Moderate health-other stress:**
   - Close family member(s) or friend(s) have experienced some health problems (mental or physical) but they did not result in significant debilitation; or
     - Moderately close family member(s) or friend(s) have experienced more serious (i.e., temporarily debilitating) health problem(s) or accident(s)/injuries.
       - Aunt/Uncle, Cousin death - use contextual information to change rating depending on closeness of relationship

4. **Average health-other stress:**
   - Adolescent reports a few incidents of family and/or friends suffering from minor illnesses and/or injuries.

5. **Little to no health-other stress:**
   - Adolescent reports that family and friends have been very healthy with few if any minor illnesses/injuries.

J. Finance-Related Chronic Stress
1. **Severe finance-related stress:**
   - Adolescent reports that family has experienced a sharp decrease in financial income; or
     - Adolescent reports chronic lack of funds for necessities and/or adolescent reports chronic embarrassment about not having enough money/things relative to others; or
     - Adolescent has engaged on several occasions in dangerous activities (e.g., non-trivial theft, drug sales, prostitution, etc.) to make money.

2. **Serious finance-related stress:**
   - Adolescent and family have experienced a moderate decrease in family income; or
     - Adolescent reports frequent lack of funds for necessities and/or adolescent reports frequent embarrassment about not having enough money/things relative to others; or
     - Adolescent has engaged at least once in a dangerous activity to make money.

3. **Moderate finance-related stress:**
   - Adolescent and family have experienced a slight decrease in family income; or
     - Adolescent occasionally feels like he/she or family does not have enough money for necessities and/or adolescent feels somewhat embarrassed about not having enough money/things relative to others.

4. **Average finance-related stress:**
   - Adolescent and family appear to have enough money for the most part, with occasional financial problems.

5. **Little to no financial stress:**
   - No financial problems reported.
   - Family appears to be comfortable financially.

K. Legal Chronic Stress

1. **Severe legal stress:**
   - Adolescent and/or close family member(s) have frequently had serious problems with the law or other authorities (e.g., been arrested or gotten into trouble with police) or have had a single severe problem (e.g., imprisoned for a significant period of time).

2. **Serious legal stress:**
   - Adolescent and/or close family member(s) have had between one and a few serious problems with the law or other authorities; or
o Moderately close family members have had frequent serious problems with the law or a single severe problem.

3. **Moderate legal stress:**
   - Adolescent and/or close family member(s) have had a few problems with the law or other authorities but these problems have not been significant (searched by police and let go, curfew violations, etc.); or
   - Moderately close family member(s) have had between one and a few serious problems with the law.

4. **Average legal stress:**
   - Adolescent and/or family member have had one or more legal problems that were minor in nature (e.g., minor traffic violation).

5. **Little to no legal stress:**
   - No problems reported during time period.

L. **Exposure to Violence Chronic Stress**

1. **Severe exposure to violence stress:**
   - Adolescent has been the victim of a significant violent act (e.g., physical and/or sexual abuse, injury-sustaining assault by peers); or
   - Adolescent has seen someone else get seriously hurt; or
   - Adolescent knows of a close family member or friend who was the victim of a life-threatening violent act (e.g., murder, attempted murder, severe assault).

2. **Serious exposure to violence stress:**
   - Adolescent has frequently been threatened with serious violence; or
   - Adolescent has witnessed violence involving important attachment figures (e.g., domestic violence) that did not result in physical injury; or
   - Adolescent knows of a close family member or friend who was the victim of a serious violent act that was not life-threatening; or
   - Adolescent knows of a moderately close family member or friend who was the victim of a life-threatening violent act.

3. **Moderate exposure to violence stress:**
   - Adolescent or close family member or friend has been threatened between one and a few times with serious violence; or
   - Adolescent knows of a moderately close family member or friend who has been the victim of a serious violent act that was not life-threatening; or
CHRONIC STRESS AND SAA

4. **Average exposure to violence:**
   - Adolescent may have experienced some forms of physical punishment (e.g., “spankings” that did not leave any bruises or lasting injuries) or minor threats of a non-serious nature; or
   - Adolescent may have heard about one or more acquaintances that have been victims of non-life-threatening violent acts; or
   - Adolescent may have heard about someone they know has been threatened

5. **Little to no exposure to violence stress:**
   - Adolescent has never been hurt or threatened with violence
   - Adolescent is not aware of any family, friends, or acquaintances that have experienced violent victimization.

M. Exposure to Discrimination and Acculturation Chronic Stress

1. **Severe exposure to discrimination or acculturation stress:**
   - Adolescent (or close family) has consistently experienced being treated differently/unfairly due to race/ethnicity/religion/gender/sexual orientation etc; or
   - Adolescent (or close family) has experienced one or more severe and blatant acts of discrimination (e.g., cross-burning on lawn, racial epithets scrawled across garage door, life-threatening mail, severe assault based on sexual orientation, severe sexual harassment); or
   - Adolescent consistently experiences intense pressure to fit in with people who have a different background; or
   - Adolescent consistently experiences intense conflict between his/her values and others’ values; or
   - Adolescent constantly experiences intense conflict with parents because he/she prefers “U.S. ways” or because his/her parents do not know “U.S. ways”; or
   - Adolescent (or family) has avoided government agencies (i.e., the police, hospitals, social agencies, etc.) frequently, for fear of being deported.

2. **Serious exposure to discrimination or acculturation stress:**
   - Adolescent has had many experiences in which he/she (or close family) was treated differently/unfairly due to race/ethnicity/religion/gender/sexual orientation; or
   - Adolescent often experiences moderate to intense pressures to fit in with people who have a different background; or
3. **Moderate exposure to discrimination or acculturation stress:**
   - Adolescent (or family) has had a few experiences in which he/she was treated differently/unfairly due to race/ethnicity/religion/gender/sexual orientation; or
   - Adolescent experiences occasional intense pressure (or frequent mild pressure) to fit in with people who have a different background; or
   - Adolescent experiences occasional intense conflict (or frequent mild conflict) between his/her values and others’ values; or
   - Adolescent experiences occasional intense conflict (or frequent mild conflict) between his/her parents because adolescent prefers U.S. ways; or
   - Adolescent must occasionally act as a translator for parents because they do not speak English well.

4. **Average exposure to discrimination or acculturation stress:**
   - Adolescent (or family) has experienced little exposure to discrimination; any incidents experienced have been minor and/or not clearly tied to race/ethnicity/religion/gender/sexual orientation; or
   - Adolescent for the most part has not experienced acculturation pressures or conflicts, with only occasional minor incidents.

5. **Little to no exposure to discrimination or acculturation stress:**
   - Adolescent reports that neither he/she (nor anyone in family) has experienced any discrimination; or
   - Adolescent reports no experiences of acculturation pressures or conflicts for self or family.
Appendix D
Results Tables

Table 1
Pearson’s Correlations, Means, SDs for study variables (N=126)

<table>
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Note. * p < .10, * * p < .05, * * * p < .01

Table 1.2
Pearson’s Correlations, Means, SDs, by gender

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Male

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<tr>
<td>SD</td>
<td>61.92</td>
<td>0.45</td>
<td>3.62</td>
<td>--</td>
</tr>
</tbody>
</table>

Note. N(Females) = 67, N(Males) = 59, * p < .05

Table 2
Summary of simple regression model testing H1

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>β</th>
<th>SE_B</th>
<th>SE_β</th>
<th>r</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>29.78</td>
<td>0.00</td>
<td>5.65</td>
<td>0.09</td>
<td></td>
</tr>
<tr>
<td>Chronic Stress</td>
<td>-14.65</td>
<td>-0.1016</td>
<td>12.89</td>
<td>0.09</td>
<td>0.26</td>
</tr>
</tbody>
</table>

Note. NS; B = unstandardized regression coefficient on centered variable, β = standardized regression coefficient, SEB = Standard error of the
unstandardized regression coefficient, $SE_β$ = Standard error of the standardized regression coefficient

Table 3
Summary of step 1 in testing $H_2$

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>$β$</th>
<th>$SE_B$</th>
<th>$SE_β$</th>
<th>$ρ$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>29.78</td>
<td>0.00</td>
<td>5.66</td>
<td>0.09</td>
<td></td>
</tr>
<tr>
<td>Chronic Stress</td>
<td>-12.88</td>
<td>-0.09</td>
<td>13.09</td>
<td>-0.98</td>
<td>0.33</td>
</tr>
<tr>
<td>Family Cohesion</td>
<td>1.19</td>
<td>0.07</td>
<td>1.47</td>
<td>0.81</td>
<td>0.42</td>
</tr>
</tbody>
</table>

Note. NS; $B$ = unstandardized regression coefficient on centered variable, $β$ = standardized regression coefficient, $SE_B$ = Standard error of the unstandardized regression coefficient, $SE_β$ = Standard error of the standardized regression coefficient

Table 4
Summary of moderated multiple regression model testing $H_2$

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>$β$</th>
<th>$SE_B$</th>
<th>$SE_β$</th>
<th>$ρ$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>28.50</td>
<td>-0.02</td>
<td>5.73</td>
<td>0.09</td>
<td></td>
</tr>
<tr>
<td>Chronic Stress</td>
<td>-15.13</td>
<td>-0.10</td>
<td>13.16</td>
<td>0.09</td>
<td>0.25</td>
</tr>
<tr>
<td>Family Cohesion</td>
<td>1.20</td>
<td>0.07</td>
<td>1.46</td>
<td>0.09</td>
<td>0.41</td>
</tr>
<tr>
<td>Stress x Cohesion</td>
<td>-4.46</td>
<td>-0.12</td>
<td>3.42</td>
<td>0.09</td>
<td>0.20</td>
</tr>
</tbody>
</table>

Note. NS; $B$ = unstandardized regression coefficient on centered variable, $β$ = standardized regression coefficient, $SE_B$ = Standard error of the unstandardized regression coefficient, $SE_β$ = Standard error of the standardized regression coefficient

Table 5
Summary of step 1 in testing $H_3$

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>$β$</th>
<th>$SE_B$</th>
<th>$SE_β$</th>
<th>$ρ$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>25.41</td>
<td>-0.07</td>
<td>7.81</td>
<td>0.12</td>
<td></td>
</tr>
<tr>
<td>Chronic Stress</td>
<td>-12.06</td>
<td>-0.08</td>
<td>13.14</td>
<td>0.09</td>
<td>0.36</td>
</tr>
<tr>
<td>Family Cohesion</td>
<td>1.35</td>
<td>0.08</td>
<td>1.48</td>
<td>0.09</td>
<td>0.36</td>
</tr>
<tr>
<td>Gender</td>
<td>9.32</td>
<td>0.15</td>
<td>11.47</td>
<td>0.18</td>
<td>0.42</td>
</tr>
</tbody>
</table>

Note. NS; $B$ = unstandardized regression coefficient on centered variable, $β$ = standardized regression coefficient, $SE_B$ = Standard error of the
unstandardized regression coefficient, \( SE_\beta \) = Standard error of the standardized regression coefficient

Table 6
**Summary of step 2 in testing H3**

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>( \beta )</th>
<th>( SE_B )</th>
<th>( SE_\beta )</th>
<th>( \rho )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>24.82</td>
<td>-0.08</td>
<td>7.81</td>
<td>0.12</td>
<td></td>
</tr>
<tr>
<td>Chronic Stress</td>
<td>-14.31</td>
<td>-0.10</td>
<td>13.24</td>
<td>0.09</td>
<td>0.28</td>
</tr>
<tr>
<td>Family Cohesion</td>
<td>1.34</td>
<td>0.08</td>
<td>1.48</td>
<td>0.09</td>
<td>0.37</td>
</tr>
<tr>
<td>Gender</td>
<td>8.01</td>
<td>0.13</td>
<td>11.50</td>
<td>0.18</td>
<td>0.49</td>
</tr>
<tr>
<td>Stress x Cohesion</td>
<td>-4.24</td>
<td>-0.12</td>
<td>3.45</td>
<td>0.09</td>
<td>0.22</td>
</tr>
</tbody>
</table>

**Note.** NS; \( B \) = unstandardized regression coefficient on centered variable, \( \beta \) = standardized regression coefficient, \( SE_B \) = Standard error of the unstandardized regression coefficient, \( SE_\beta \) = Standard error of the standardized regression coefficient

Table 7
**Summary of step 3 in testing H3**

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>( \beta )</th>
<th>( SE_B )</th>
<th>( SE_\beta )</th>
<th>( \rho )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>24.78</td>
<td>-0.08</td>
<td>7.80</td>
<td>0.12</td>
<td></td>
</tr>
<tr>
<td>Chronic Stress</td>
<td>-14.43</td>
<td>-0.10</td>
<td>13.40</td>
<td>0.09</td>
<td>0.28</td>
</tr>
<tr>
<td>Family Cohesion</td>
<td>1.42</td>
<td>0.09</td>
<td>1.90</td>
<td>0.12</td>
<td>0.45</td>
</tr>
<tr>
<td>Gender</td>
<td>7.96</td>
<td>0.13</td>
<td>11.56</td>
<td>0.18</td>
<td>0.49</td>
</tr>
<tr>
<td>Stress x Cohesion</td>
<td>-4.27</td>
<td>-0.12</td>
<td>3.49</td>
<td>0.09</td>
<td>0.22</td>
</tr>
<tr>
<td>Cohesion x Gender</td>
<td>0.14</td>
<td>0.01</td>
<td>3.11</td>
<td>0.19</td>
<td>0.96</td>
</tr>
</tbody>
</table>

**Note.** NS; \( B \) = unstandardized regression coefficient on centered variable, \( \beta \) = standardized regression coefficient, \( SE_B \) = Standard error of the unstandardized regression coefficient, \( SE_\beta \) = Standard error of the standardized regression coefficient

Table 8
**Summary of step 4 in testing H3**

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>( \beta )</th>
<th>( SE_B )</th>
<th>( SE_\beta )</th>
<th>( \rho )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>25.02</td>
<td>-0.07</td>
<td>7.90</td>
<td>0.12</td>
<td></td>
</tr>
<tr>
<td>Chronic Stress</td>
<td>-20.64</td>
<td>-0.14</td>
<td>18.12</td>
<td>0.13</td>
<td>0.25</td>
</tr>
<tr>
<td>Family Cohesion</td>
<td>1.35</td>
<td>0.08</td>
<td>1.91</td>
<td>0.12</td>
<td>0.48</td>
</tr>
<tr>
<td>Gender</td>
<td>8.24</td>
<td>0.13</td>
<td>11.61</td>
<td>0.18</td>
<td>0.48</td>
</tr>
<tr>
<td>Stress x Cohesion</td>
<td>-3.88</td>
<td>-0.11</td>
<td>3.58</td>
<td>0.10</td>
<td>0.28</td>
</tr>
<tr>
<td>Cohesion x Gender</td>
<td>0.14</td>
<td>0.01</td>
<td>3.11</td>
<td>0.19</td>
<td>0.96</td>
</tr>
<tr>
<td>Stress x Gender</td>
<td>13.94</td>
<td>0.10</td>
<td>27.29</td>
<td>0.19</td>
<td>0.61</td>
</tr>
</tbody>
</table>
Note. NS; $B =$ unstandardized regression coefficient on centered variable, $\beta =$ standardized regression coefficient, $\text{SE}_B =$ Standard error of the unstandardized regression coefficient, $\text{SE}_\beta =$ Standard error of the standardized regression coefficient

Table 9

*Summary of three-way interaction multiple regression model testing $H_3*$

<table>
<thead>
<tr>
<th>Variable</th>
<th>$B$</th>
<th>$\beta$</th>
<th>$\text{SE}_B$</th>
<th>$\text{SE}_\beta$</th>
<th>$\rho$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>25.67</td>
<td>-0.06</td>
<td>7.88</td>
<td>0.12</td>
<td></td>
</tr>
<tr>
<td>Chronic Stress</td>
<td>-21.27</td>
<td>-0.15</td>
<td>18.07</td>
<td>0.13</td>
<td>0.24</td>
</tr>
<tr>
<td>Family Cohesion</td>
<td>1.18</td>
<td>0.07</td>
<td>1.91</td>
<td>0.12</td>
<td>0.54</td>
</tr>
<tr>
<td>Gender</td>
<td>4.27</td>
<td>0.07</td>
<td>11.93</td>
<td>0.19</td>
<td>0.72</td>
</tr>
<tr>
<td>Stress x Cohesion</td>
<td>-0.18</td>
<td>0.00</td>
<td>4.48</td>
<td>0.12</td>
<td>0.97</td>
</tr>
<tr>
<td>Cohesion x Gender</td>
<td>-0.31</td>
<td>-0.02</td>
<td>3.12</td>
<td>0.19</td>
<td>0.92</td>
</tr>
<tr>
<td>Stress x Gender</td>
<td>5.30</td>
<td>0.04</td>
<td>27.93</td>
<td>0.19</td>
<td>0.85</td>
</tr>
<tr>
<td>Stress x Cohesion x Gender</td>
<td>-10.06</td>
<td>-0.27</td>
<td>7.40</td>
<td>0.20</td>
<td>0.18</td>
</tr>
</tbody>
</table>

Note. NS; $B =$ unstandardized regression coefficient on centered variable, $\beta =$ standardized regression coefficient, $\text{SE}_B =$ Standard error of the unstandardized regression coefficient, $\text{SE}_\beta =$ Standard error of the standardized regression coefficient