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Acculturation, Childhood Trauma and the Cortisol Awakening Response in Mexican American Adults

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Abstract

Exposure to chronic and traumatic stress has been associated with the dysregulation of crucial stress response systems. Acculturation has been associated with unique forms of chronic psychosocial stress. The purpose of this study was to examine the effects of exposure to early traumatic stress and acculturation on dysregulation of the cortisol awakening response (CAR) in Mexican-American adults. Salivary cortisol samples were collected at awakening and 30, 45, and 60 minutes thereafter, on two consecutive weekdays from 59, healthy Mexican American adult males (26) and females (33), ages 18-38 years. Participants were assessed for level of acculturation and exposure to early trauma. Data were analyzed using a mixed effects regression model with repeated measures at four time points. Mixed effects regression results indicated a significant Early Trauma x Time interaction (p=.0029) and a significant Acculturation x Time interaction (p=.0015), after controlling for age and sex. Subsequent analyses of the interaction of Trauma x Acculturation x Time showed that more than minimal exposure to either risk factor was associated with attenuation of the awakening cortisol response (p=.0002). Higher levels of acculturation with greater Anglo-orientation were associated with attenuation of the CAR in Mexican-American adults. Both moderate and higher levels of exposure to early trauma were associated with an attenuated CAR. However, greater exposure to both risk factors was only incrementally worse than exposure to either one.

Keywords

Acculturation; Childhood Trauma; HPA axis; Cortisol; Mexican-Americans

Introduction

Childhood trauma is a significant problem in the United States and is associated with mental and physical health problems in adulthood (Heim et al., 2000a, 2001a; Gunnar and Vazquez, 2001, 2006; Dong et al., 2004; Nicolaidis et al., 2009), that lead to increased health care utilization and costs (Felitti, 1991; Felitti et al., 1998; Newman et al., 2000). Early exposure

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to trauma is a robust risk factor for the development of anxiety and depressive disorders in adulthood (Kessler and Magee, 1993; Kessler et al.,1997; Young et al., 1997; Kendler et al., 2001; Safren et al., 2002), and is associated with increased reports of emotional distress (Spertus et al., 2003), risk for suicidal behaviors (McCauley et al., 1997), greater behavior problems (Messina and Grella 2006), greater psychiatric symptom severity (Yehuda et al., 2001), higher risk for substance use disorders (Epstein et al., 1998; Fleming et al., 1999), greater risk of ischemic heart disease (Dong et al., 2004), and more overall long-term physical health consequences (Lechner et al., 1993; Bendixen et al., 1994; McCauley et al., 1997; Moeller et al., 1993; Irish et al., 2009). Findings from the National Child Abuse and Neglect Data System (NCANDS; 2002) and the National Child Traumatic Stress Network (NCTSN; 2005), show that a significant percentage of children exposed to childhood trauma from maltreatment are Latino/Hispanic. When compared to Caucasian children, Latino/Hispanic children experience higher rates of domestic violence, community violence, and family-perpetrated abuse (Sanders-Phillips et al., 1995; Shaw et al., 2001). Latino/Hispanic children are also at greater risk for exposure to certain forms of early trauma including: general or complex traumas (72%), domestic violence (53%), caregiver impairment (47%), emotional abuse (42%), and traumatic loss (42%), (NCTSN; 2005). Latino/Hispanic victims of early trauma report higher rates of post-abuse depression (Sanders-Phillips et al., 1995; Roosa et al., 1999), and more psychiatric symptoms associated with anxiety, depression and dissociation compared to their trauma-free counterparts (Mennen, 2000; Martinez-Taboas and Bernal, 2000). In addition, childhood trauma is associated with overall poorer subjective health in Latinos/Hispanic men, and more muscular skeletal and gastrointestinal symptoms in females (Baker et al., 2009). Moreover, depression appears to mediate the relationship between childhood trauma and negative health outcomes for Latino/Hispanic men and women (Baker et al., 2009). Clearly, early exposure to traumatic stress represents a significant, pre-existing risk factor for the onset of certain mental and physical health problems in the Latino/Hispanic community.

In addition to pre-existing exposure to early traumatic stress, some high risk Latinos/Hispanics may be exposed to culturally unique sources of chronic stress as adults, including the stress associated with the challenges of acculturation (Cervantes and Castro, 1985; Mena et al., 1987; NCTSN, 2005; Bernal and Santiago, 2006). Acculturation is the process that involves adaptation into a host culture (Mena et al., 1987). The combination of psychological, somatic, and social stressors that may be associated with the process of adaptation into a host culture is acculturative stress (Berry, 1980; Mendoza, 1984; Sodowsky et al., 1991). The stressors associated with acculturation increase risk for the onset of negative health outcomes in high risk Mexican-Americans, and the challenges associated with acculturation modulate the relationship between childhood trauma and negative health outcomes (Matheson et al., 2008). However, the specific physiological mechanisms contributing to this relationship remain to be clarified (Williams and Berry, 1991; Hovey and King, 1996; Aranda et al., 2001). Given these findings, an examination of the interplay between early traumatic stress and unique cultural risk factors (i.e., acculturation) on crucial biological stress response systems may be of particular importance in this understudied and underserved minority group.

Stress-induced dysregulation of the Hypothalamic-Pituitary-Adrenal (HPA) axis is one neurobiological pathway that may link childhood trauma and acculturation to negative health outcomes (McEwen et al., 1998; Danese et al., 2007). HPA axis activation during early development following childhood trauma increases risk for mood and anxiety disorders (Goodwin and Stein, 2004; Heim et al., 2006). A series of seminal studies conducted by Heim and colleagues (1999, 2000a, 2000b, 2001a, 2001b) suggest that exposure to childhood trauma (sexual/physical abuse), during critical periods of neural plasticity, may alter the set point of the stress response system. This may result in adaptive counter-regulation of the adrenal cortex with HPA activation during early development leading to adult hypocortisolism. It is possible that for Hispanic children, exposure to childhood trauma during critical periods of neural

plasticity, coupled with the stressors associated with acculturation as adults, may alter the set point of the stress response system. This may lead to adaptations that result in aberrant cortisol secretion and poor psychological and physical health outcomes.

Determination of the Cortisol Awakening Response (CAR) is increasingly being utilized as a means to interrogate stress-induced injury to the HPA Axis (Pruessner et al., 1999). Changes in the CAR can yield important information regarding the relationship between altered stress responsivity and injury to the awakening portion of the cortisol circadian rhythm. The sensitivity and capacity of the adrenal cortex is suggested to play a crucial role in the magnitude of the CAR (Pruessner et al., 1997, 1999; Schulz et al., 1998; Schmidt-Reinwald et al., 1999; Wust et al., 2000a; Kudielka and Kirschbaum, 2003). A lower CAR has been reported in adults with a history of childhood trauma, with psychopathology (Stein et al., 1997; Meinslschmidt and Heim, 2005), in patients with a history of childhood trauma and borderline personality disorder (Flory et al., 2009), and in women with a history of intimate partner violence (Johnson et al., 2008). However, to date, no studies have examined the effects of childhood trauma on the CAR in healthy subjects, while adequately screening out depression, which is known to alter the HPA axis and the CAR (Heim and Nemeroff, 2001a; Pruessner et al., 2003; Huber et al., 2006; Stetler and Miller, 2005). Therefore, it has been difficult to differentiate the effects of depression from the effects of trauma on the CAR. Moreover, to our knowledge, there has never been an examination of the effects of acculturation on the CAR. Therefore, it has been impossible to unravel the effects of childhood trauma from the effects of the psychosocial stress that is often associated with the challenges of acculturation.

The aims of the present investigation were to examine: 1) the effects of early traumatic stress on the CAR in Mexican-American adults screened for depression; 2) the effects of acculturation on the CAR; and 3) the effects of a trauma x acculturation interaction on the CAR. We hypothesized that childhood trauma and acculturation would be associated with an attenuated CAR.

MATERIALS AND METHODS

Participants and Study Design

The University of Texas Institutional Review Board approved the study, and all participants gave written, informed consent prior to participation. Participants of Mexican descent (n=59), aged 18 to 38, were recruited from the San Antonio metropolitan area, through advertisements in the community and local college campuses. During an initial visit to the laboratory, participants underwent an in-person screening interview and a battery of self-report assessments designed to identify and exclude factors known to potentially affect the HPA axis including: lifetime depression (Bhagwager et al., 2005; Shea et al., 2007), use of oral contraceptives in the past 60 days (Meulenberg and Hofman et al., 1990; Pruessner et al., 1997,1999), current pregnancy (Meulenberg and Hofman et al., 1990), menstrual cycle abnormalities in the past 60 days (Suh et al., 1988; Bao et al., 2003, 2004), strenuous aerobic exercise (more than 2 hours per day for 4 or more days per week in the past 60 days (Kanaley et al., 2001; Hansen et al., 2008; Kelly et al., 2008), reported medical conditions and previous head trauma, use of medications, severe obesity (defined as a body mass index of > 30.0 kg/m^2) and, alcohol or other drug use disorders (Wand and Dobs, 1991; Huizink et al., 2006; Hansen et al., 2008). In addition, participants were excluded from the study who reported abnormal sleeping patterns, including disrupted sleep for more than one night per week, in the past 60 days, as determined by a 60-day sleep diary (Lasikiewicz et al., 2008), shift work including night shifts for the past week, overtime work for more than 8 hours per week in the past 30 days (Lundberg and Hellstrom, 2002; Clow et al., 2004; for review, see Hanrahan et al., 2006). To determine subjects' economic status information regarding family income and parental occupational status was collected.

Psychometric Assessment

Depression—Participants were screened for depression using the Hamilton Depression Inventory Short Form (HDI-SF; Reynolds and Kobak, 1995), and excluded from participation based on a score of 10 or greater as recommended by Reynolds and Kobak (1995) indicating a strong likelihood of depression. The HDI-SF has strong psychometric properties and the reliability for the current Mexican-American sample was acceptable (α=.78).

Substance Abuse/Dependence—Participants were screened and excluded for alcohol and other drug use disorders using the World Health Organization Alcohol, Smoking and Substance Involvement Screening Test (WHO ASSIST; Humeniuk et al., 2008). The ASSIST assesses the frequency of lifetime and recent use (within the past 3 months) and problems associated with the use of alcohol and other drugs of abuse (e.g., cannabis, cocaine, stimulants, etc.). The ASSIST has strong psychometric properties and the reliability for the current subject sample was acceptable (α=.80).

Menstrual Irregularities, Premenstrual Dysphoric Disorder (PMDD)—The Calendar of Premenstrual Experiences (CPE; Mortola et al., 1990) was used to assess and exclude participants with PMDD, irregular menstrual cycles and premenopausal symptoms. The test-retest reliability of the calendar given in the same phase of two consecutive menstrual cycles was high (r=0.78, p <.0001).

Ethnicity/Nativity—Participants reporting Hispanic origin other than Mexican or Mexican-American were excluded from the study. Participants were determined to be of Mexican descent if they reported that both biological parents and both maternal and paternal grandparents were of Mexican descent.

Acculturation—The revised Acculturation Rating Scale for Mexican Americans (ARSMA-II; Cuéllar et al., 1995) was used to assess acculturation by measuring subjects' orientation towards Anglo culture and Mexican culture. Scores on the ARSMA Linear Acculturation Scale (LAS) are based on the extent to which participants endorse Anglo and Mexican orientations. The ARSMA-II has strong psychometric properties with good reliability for the current sample (α = .89).

Childhood Trauma—The current study assessed exposure to severe childhood stress prior to the age of 16, using the Early Trauma Inventory–Short Form adapted from the clinician administered Early Trauma Inventory (ETISR-SF; Bremner et al., 2007). The ETI-SF is a 27 item questionnaire that is comprised of "yes/no" items designed to assess whether someone has been exposed to potential traumatic experiences. The inventory includes four subscales: General Trauma, Physical Punishment, Emotional Abuse and Sexual Events, and a total trauma score is calculated by summing the total number of endorsed items across subscales. Scores are a reflection of degree of diversity of early exposure to severe childhood stress, rather than severity or frequency of exposure to any single stressor. The ETISR-SF has strong psychometric properties (Hyman et al., 2005; Bremner et al., 2007) with good reliability for the current sample (α = .82).

Measurement of the CAR—Participants were free to wake up as usual (using an alarm or spontaneously), and instructed to start sampling immediately at awakening (T_0), and T_{30}, T_{45} and T_{60} minutes following awakening. Sampling was completed on two consecutive weekdays. Participants were instructed to log wake-up times along with sampling times for comparison with electronic monitoring data. Participants were instructed to refrain from strenuous exercise and from brushing their teeth, eating or drinking, and use of alcohol, caffeine and nicotine during sampling to avoid contamination of cortisol samples (Clow et al., 2004;

Badrick et al., 2007). Participants placed a roll of cotton in their mouths, chewed on it until it became saturated, and then returned it to the salivette (Sarstedt; Rommelsdorft, Germany). Participants remained sitting upright in bed until the second saliva sample was obtained, when they were free to follow their normal weekday routine during the sampling period. To employ a conservative approach to control for possible effects of menstrual cycle phase on the CAR (Bao et al., 2003, 2004), cortisol was assessed in females during the early follicular phase of their menstrual cycle (determined by menstrual diary).

We utilized electronic monitoring devices together with self-reported time of sampling to determine concordance between monitored times and self-reported times, detect deviations from the protocol and maximize accuracy in the documentation of sample times (Jacobs et al., 2005). To further address accuracy, participants collected saliva samples on two separate, consecutive days. Salivettes were stored in a vial equipped with an electronic medication event monitoring system (MEMS; Aardex; Zug, Switzerland), as recommended for use in an ambulatory setting by (Kudielka et al., 2003). The MEMS is designed to compile the dosing/sampling-compliance histories of participants. This system includes a standard plastic vial with cap that contains a threaded opening and a closure that contains a micro-electronic circuit that registers times when opened and closed. Time-stamped medication events are stored in the unit for later software analyses to maximize accuracy of recorded awakening and sampling times included in later analyses.

Hormonal Assays—Participants stored the samples in their home freezers until they were returned to the laboratory the next day and stored at −80 C until analyzed, when they were thawed at room temperature and centrifuged at 2500g for 15 minutes at −4 C. The saliva samples were kept frozen until the start of the assay. Salivary cortisol levels were assayed in duplicate using high sensitivity enzyme immunoassays (Diagnostic Systems Laboratories; Webster, Texas), with a mean lower sensitivity limit of 0.11 ug/dL, standard curve range from 0.1 to 10ug/dL. The intra-assay and inter-assay coefficients of variation were less than 5% at all levels of the calibrator curve. The concentration of cortisol in saliva was expressed as ug/dL. To minimize the potential effects of exposure to stressful events during the sampling period, participants who were currently students were not sampled the week prior to scheduled class examinations. In addition, participants indicating daily hassles, or exposure to stressful daily events during the sampling period (van Eck et al., 1996), sleep disturbances (Lasikiewicz et al., 2008) and other unusual events or protocol noncompliance (teeth brushing, eating, etc), during sampling periods were excluded from the final analyses.

Statistical Analyses

Data were available from 59 participants. Descriptive statistics are reported to characterize the sample. Cortisol data were log-transformed to correct positive skew. The primary statistical design was a mixed effects regression model with repeated measures at four time points (awakening and 30, 45, and 60 minutes thereafter). Fixed design effects were self-reported Early Trauma (ET), used as a continuous, dimensional variable (scores ranged from 0-18), Time (a fixed classification factor with four levels), and their interaction. The ET x Time interaction was the primary focus as it tests the association between level of trauma and changes in cortisol over time. The same analysis design was used to assess effects of Acculturation (ARSMA-II; LAS) on the cortisol response. First, we looked at Acculturation, substituting the continuous measure of acculturation for the measure of early trauma in a mixed effects regression model with repeated measures as above. Then, early trauma and acculturation were examined together in a mixed regression model that included main effects of both factors and their interaction. Primary interest was again on the interactions of these terms with time, testing unique direct (main) and moderating (interaction) effects on the cortisol response. Continuous

variables were centered by subtracting the mean to reduce confounding of lower and higher order effects.

Modeling time as a fixed classification factor simplified specification of the covariance structure and presentation of the results, and was supported by the observation of extremely little intra-individual variability in the timing of cortisol sampling: 93% of the 339 samples were obtained within five minutes of the scheduled time, and 97% within 10 minutes of protocol. Two samples were obtained 61 and 75 minutes later than scheduled. These were both from the same participant, and could have been recording errors as they are almost exactly one hour off. Omitting them had no meaningful effect on the analyses, so they were retained as recorded. We specified the Kenward-Rogers adjustment for degrees of freedom and a spatial power structure for the covariance matrix with measures at 0, 30, 45 and 60 minutes. This is a generalization of first-degree autoregressive structure to the case of unequally spaced measures, and all of the likelihood-criteria provided by SAS MIXED (AIC, AICC, BIC) indicated a better fit than with compound symmetry, autoregressive, or a fully unstructured matrix (Littell, et al., 1996, pp. 126-130).

All but three participants provided cortisol data following the same measurement protocol on two weekdays. Summary cortisol statistics are presented separately for days one and two to enable comparison with previous data in other samples, but for the regression analyses we used the average of the day one and day two data. Averaging over a period from two to six days and weekday sampling minimizes the effects of state factors, increases trait specificity (Clow et al., 2004; Kunz-Ebrecht et al., 2004, Scholtz et al., 2004; Hellhammer et al., 2007; Thorn et al., 2006, 2009), and considerably simplifies the covariance structure and analysis. We had no expectation of meaningful day-to-day variability, but confirmed this judgment by performing preliminary mixed effects analyses including Day as an additional design factor. These did not reveal any significant effects involving Day (all $p>0.10$) or meaningfully change the results, and are not reported further. Statistical analyses were done using the SAS 9.2 statistical library. Tests were at unadjusted two-tailed $p=.05$. Inferences are based on the results of the analyses using the continuous measures, but analyses were also done grouping both scales into classes (e.g., low, high) for the purpose of graphing results to aid interpretation.

RESULTS

Participant Characteristics

Table 1 presents demographic characteristics of the sample. Participants were single (98%), adult males (44%) and females (56%). Most were first or second year college students, ranging in age from 18-38 years (median=20 years, $M = 22$, $SD = 5.4$) with a normal Body Mass Index (BMI). A little over half the sample (56%) reported a family income below $40,000. Just over one-fourth (28%) of the sample were first generation Mexican-Americans (born in Mexico), 28% were born in the United States, and 44% were third generation (parents and self were born in the United States). The majority were bilingual. Participants were healthy, with mean scores on the General Health scale of the RAND-36 for the current sample ($M = 79.90$, $SD = 20.10$) in line with those previously reported for healthy, young adults ($M = 77.60$, $SD = 20.10$, Vander Zee et al., 1996). They reported no current psychiatric diagnoses or current use of psychotropic medication. Individuals with HDI scores higher than 10 were excluded from the study. The mean score on the HDI for the sample was lower than normative means reported for college-aged participants ($M = 5.16$, $SD = 4.48$), (Reynolds and Kobak, 1995;Vander Zee et al., 1996), most likely due to exclusion based on a diagnosis of depression. However, scores on the HDI ranged from 0 to 9.50 ($M = 2.84$, $SD = 2.39$.) for the sample, suggesting subjects endorsed a range of depressive symptoms.

Exposure to Early Trauma

The prevalence of exposure to trauma in this sample was high. Ninety-five percent of the participants reported exposure to at least one childhood trauma item. However epidemiological evidence suggests that over 56% of Americans experience a lifetime trauma, and risk of exposure to general or complex traumas are reported to be as high as 72% among Latino/Hispanic children (NCTSN; 2005). Consistent with this evidence, participants, on average, reported exposure to more than five different trauma items across all trauma dimensions. Table 2 depicts mean exposures to the dimensions of trauma (ETISR-SF; Bremner et al., 2007) for the male and female participants in the sample.

Characterization of the Cortisol Awakening Response

Salivary cortisol response for the overall sample was characterized with respect to overall cortisol secretory activity measured in the period after awakening and the dynamic of the response (change in concentration from awakening to peak levels) as recommended by Clow et al. (2004). Cortisol concentrations were assessed over 4 time points, and were averaged over two days for each person. Calculation of the changes in cortisol concentration from awakening to 30 min post-awakening for trial one (11.90 ± 8 nmol/l) and trial two (10.09 ± 9 nmol/l) were comparable to what is previously reported (9.3 ± 3.1 nmol/l) (Clow et al., 2004). The correlation between delta cortisol for days one and two was r = 0.47 (df=43, p=.0003; Spearman rho=0.34, p=.01). The simple correlation across all data points (i.e., using 4 values per person with N = 59) was r = 0.53 for raw cortisol, and r=0.50 for log (cort), both p<.0001, df=57.

Early Trauma Exposure and Awakening Cortisol Response

The mixed effects regression results indicated a significant Trauma x Time interaction (F=4.85, df=3, 170, p=.003) when the Trauma scale was included as a dimensional covariate. To clarify this interaction, the sample was divided into approximate thirds based on the Trauma Score (0-2=Low, N=17; 3-6=Middle, N=23; 7-18=High, N=19). This grouping (29%, 39%, 32% of the sample respectively) was the closest to equal possible. The mixed effects regression using this trichotomy in place of the dimensional score also yielded a significant Trauma x Time interaction (F=3.13, df=6,168, p=.0062). The finding is depicted in Figure 1. The analysis was based on log-transformed data, but the figure presents antilogs to display the data in its original scale and an easily recognizable metric (a graph of the logged data looks almost identical). Addition of Age (F=0.00, df=1, 59, p=0.99) and Sex (F=1.88, df=1, 59, p=0.18), as covariates had no effect on the Trauma x Time interaction (F=4.84, df=3, 170, p=.0029).

The three groups defined by level of Trauma exposure show attenuated cortisol response in direct relation to the level of trauma. Follow-up analyses revealed that the significant interaction depicted in Figure 1 was largely due to the greater rise in CAR seen in the lowest Trauma group (0-2) 30 minutes after awakening. A contrast comparing the slope in the lowest Trauma group with the average of the other two was highly significant (t=3.55, df=184, p=.0005), while the rising slopes of the other two groups did not differ (t=1.02, df=184, p=0.31). Furthermore, the differences in cortisol levels observed by the 30 minute time point remained significant thereafter. A mixed effects model that included only the three post 30 minute-awakening measures showed a significant main effect of Trauma group (F=3.86, df=2, 59.2, p=0.027) and a non-significant Trauma x Time interaction (F=1.22, df=4,113, p=0.305). There was a significant difference, averaged across time, between the Low trauma and High trauma groups (t=2.76, p=0.008). The middle group fell between the others, and did not differ significantly from the low (t=1.76, p=.084) or high Trauma groups (t=1.16, p=0.250, all df=59.2).

Acculturation

The mixed effects regression using Acculturation as the independent variable also yielded a highly significant interaction with time (F=5.36, df=3, 169, p=.0015). Again, addition of Age and Sex as covariates had no effect (F=5.35, df=3, 169, p=.0015). Inspection of the distribution of acculturation scores (ARSMA-II; LAS), indicated a bimodal distribution with a natural split at zero, with scores less than zero generally indicating greater Mexican orientation, and scores greater than zero, indicating greater Anglo orientation. Figure 2 displays the cortisol response in the two groups defined by splitting scores on the ARSMA-II (LAS) at zero (Lower N=24 (41%), Higher N=35 (59%). Again, the analysis was based on log-transformed data, but Figure 2 presents antilogs to display an easily recognizable metric.

Combined effects of Early Trauma and Acculturation

The results with Acculturation were strikingly similar to those obtained with Early Trauma. This raises the question, were these same or distinct effects? The Trauma and Acculturation measures were not significantly correlated (r=0.20, df=57, p=0.13), suggesting the latter. The combined effects were assessed in a fully crossed mixed effects regression. Interest focused on the 2-way interactions of Trauma and Acculturation with Time, which tests the impact of these variables on the awakening cortisol response, and the three-factor interaction (Trauma x Acculturation x Time), which tests for moderating effects. Because of the presence of multiple interactions involving continuous measures in this model, the Trauma and Acculturation measures were both centered at zero (Kraemer and Blasey, 2004).

Both the Early Trauma x Time interaction (F=2.72, df=3, 164, p=.046) and the Acculturation x Time interaction (F=3.95, df=3, 164, p=.009) were again significant. The three-factor interaction (Acculturation x Trauma x Time) was not significant (F=1.27, df=3, 164, p=0.29), suggesting that these two effects were independent and additive (i.e., cumulative).

These results are displayed in Figure 3 which is based on a mixed effects regression model analysis that used the categorical versions of the Early Trauma (3 groups) and Acculturation (2 groups) variables described above. Early Trauma (F=2.29, df=6, 159 p=0.04) and Acculturation interactions with Time (F=3.97, df=3, 158, p=0.01) were again significant.

The three-factor interaction was not significant, but Figure 3 gives the impression of larger differences between the Low Acculturation-Low Trauma classification and the other five groups than a simple additive model would suggest. The non-significant interaction (a complex effect with df=6) is probably due to the fact that almost all of this effect is in a single df=1 contrast, namely, the larger rise between 0 and 30 minutes in the Low Acculturation-Low Trauma group than in the other five groups, which accounts for almost all of the group differences. An omnibus test of the differences in the cortisol rise, during the first interval, among the other five groups yielded F=1.66, df=4, p=0.16, indicating that the rise in cortisol did not differ among these groups. Therefore, these groups were combined and then compared to the Low Acculturation-Low Trauma group. A test of the change in cortisol during the first 30 minutes in the Low Acculturation-Low Trauma group showed that it was significantly greater compared to the average change in the same time interval in the other five groups combined.(t=3.80, df=174, p=.0002). Figure 3 presents antilogs of the estimated means of the logged data, and there is no standard error for these exponentiated values. However, to estimate the standard errors of those exponentiated values we bootstrapped the mixed effects regression using 1000 replications, sampling cases with replacement. We saved the estimated means of the logged data in each of the 6 groups at each of the 4 time points. We exponentiated the estimated means to achieve values like those depicted in the graphic, and calculated their standard deviations over bootstrap 1000 replications sampling cases with replacement to get a

bootstrap estimate of their standard errors. Table 3 presents these estimates to provide an idea of the variability of the means depicted in the graphic.

We examined a summary measure of the rising limb of the CAR, namely Delta cortisol, (defined as the Peak cortisol concentration minus the cortisol concentration at time point zero), to further clarify the result. We examined the six groups formed by crossing the 3 (Trauma) x 2 (Acculturation) groups and performed a one-way analysis of variance on Delta cortisol. There was a significant interaction of Trauma x Acculturation on Delta cortisol, with higher Delta cortisol in the Low Trauma/Low Acculturation group ($F=6.21$, $df=5, 53$, $p=.0001$). The 5 pair wise t-tests comparing this group with the others were all significant at $p<.001$. None of the 10 pairwise t-tests between the other groups were significant (all $p>.15$).

DISCUSSION

To our knowledge this is the first examination of the effects of childhood trauma and acculturation on the CAR in Mexican-Americans. In addition, this study is distinguished by its examination of the effects of childhood trauma in healthy adults, without depression, while utilizing a carefully constructed monitoring system to determine sampling compliance. Findings from the current study show that greater exposure to early trauma is associated with attenuation of the CAR, even after controlling for age and sex. Higher levels of acculturation (greater Angloorientation) were also associated with attenuation of the CAR. This effect was largely due to group differences in the slope of the CAR from awakening to thirty minutes post-awakening. Although the initial interaction (Trauma x Acculturation x Time) was not significant, subsequent analyses showed that the Low Acculturation/Low Trauma group demonstrated a significantly greater rise in cortisol during the first 30 minutes compared to the average change for the same time interval in the other groups. Apparently, anything more than minimal exposure to either risk factor was associated with attenuation of the awakening cortisol response. High exposure to both risk factors appeared to be only incrementally worse than exposure to either one.

Our findings have an additional important novel observation. It has long been recognized that depression alters HPA axis dynamics including the CAR (Heim and Nemeroff, 2001a; Pruessner et al., 2003; Huber et al., 2006; Stetler and Miller, 2005). It has also been reported that early life trauma increases the risk for depression in adulthood (Heim and Nemeroff, 2001a; Weiss et al., 1999; Duncan et al., 1996; Bernet and Stein, 1999). However, the subjects in this study were carefully screened and excluded if they met diagnostic criteria for depression. Therefore, our findings show that both early life trauma and the stress of acculturation can attenuate the CAR independent of the influences of depression.

The findings in this study are not attributable to major methodological sources of variability and sampling error observed in some previous studies. For example, our findings cannot be attributed to the effects of depression or antidepressant use on the CAR, given that both conditions were carefully screened and excluded (Aihara et al., 2007). It is unlikely that our findings are attributable to the known effects of substance abuse on the CAR, since we assessed and excluded participants for current alcohol, nicotine and other drug use disorders to minimize the potential influence of these confounds. The HPA axis response to stress is affected by levels of estrogen and progesterone that vary due to gender (Kudielka and Kirschbaum, 2005), the use of oral contraceptives, and menstrual phase cycle (Kirschbaum et al., 1999), producing potential sources of variability and error in previous findings. In the current study, female participants were screened and excluded for use of oral contraceptives prior to enrollment, and sampled during the early follicular phase. We also performed our measurements with strict reference to time of awakening (Wust et al., 2000b; Federenko et al., 2004; Williams et al., 2005), to minimize the influence of time on measurement of the CAR (Edwards et al., 2001;

Federenko et al, 2004). We minimized sampling error through the use of electronic monitoring devices (Kudielka et al., 2003; Broderick et al., 2004; review in Hansen et al., 2008). Moreover, the current study measured the CAR across two separate sampling sessions to minimize the influence of fluctuating situational factors on the CAR, and to maximize trait specificity (Clow et al., 2004; Kunz-Ebrecht et al., 2004; Scholtz et al., 2004; Thorn et al., 2006; Hellhammer et al., 2006, 2007).

Our findings show an unusually robust rise in cortisol in the lowest risk group (low acculturation/low exposure to childhood trauma), with an averaged, percent difference of 238%, compared to a relatively normal cortisol excursion in the other five subject groups. We are uncertain why the CAR was more robust in this sample. The design was carefully crafted, sampling compliance was monitored through the use of MEMS caps, and subjects were carefully trained in in-home salivary collection techniques. Therefore, we do not have any reason to suspect technical issues related to sample acquisition or cortisol measurement. However, few studies have measured the CAR in Mexican-American samples, and to our knowledge the CAR has never been examined in a similar subgroup of healthy subjects free from lifetime mood disorders and history of childhood trauma.

Our finding that greater exposure to early trauma is associated with an attenuated CAR in Mexican Americans lends support to an expanding body of preclinical and clinical evidence showing that early exposure to adversity during a time of high neuronal plasticity, may contribute to changes in several brain systems including the HPA axis, leading to persistent behavioral and neuroendocrine dysregulation in adulthood (Plotsky and Meaney, 1993; Caldji et al., 2000; Sanchez et al., 1998, 2001, 2005, 2006). Results are consistent with findings demonstrating reduced adrenocortical secretion in female survivors of sexual and physical abuse with and without major depressive disorder (Heim et al., 2001b), in pregnant females with a history of childhood abuse (Shea et al., 2007), in a sample of adult offspring of holocaust survivors (Yehuda et al., 2001), in PTSD patients (Rohleder et al., 2004; Wessa et al., 2006), and in adults with early loss experience (Meinslschmidt and Heim, 2005).

Although the majority of evidence suggests severe childhood stress is associated with diminished cortisol concentrations and a lower CAR, it should be noted that two studies report an enhanced CAR associated with early life adversity in postpartum females (Gonzalez et al., 2009) and enhanced cortisol levels throughout the day in adult men exposed to early parental loss (Nicolson, 2004). However, in one of these studies the CAR was calculated based on a limited time course, without respect to time of awakening, in the absence of a systematic monitoring of protocol compliance, while the other study relied on a small subject sample, did not monitor protocol compliance and did not screen for previous exposures to other forms of childhood adversities.

Our finding that a higher degree of acculturation (greater Anglo-orientation) is associated with an attenuation of the CAR, is of particular interest. A number of studies suggest many Mexican-Americans face unique stressors associated with some aspects of acculturation (Cervantes and Castro, 1985; Mena, et al., 1987; Hovey and King, 1996; Finch, et al., 2000; Miranda and Matheny, 2000; Huebner, et al., 2005), that can lead to greater stresses, psychological disorders and health problems (Salgado de Snyder, 1987; Hovey and King, 1996; Escobar et al., 2000; Finch, et al., 2000; Hovey, 2000a, 2000b; Aranda, et al., 2001; Gonzalez et al., 2001; Finch and Vega, 2003; Thoman and Suris, 2004). Some have argued that greater acculturation with more Anglo-orientation may diminish protective factors associated with the culture of origin (familism). As protective factors erode, individuals may become more vulnerable to the effects of continuous challenges and chronic psychosocial stressors associated with acculturation (Kaplan et al., 1990; Rogler et al., 1991; Gil et al., 1994; Vega and Amaro, 1994; Finch et al., 2001; Finch and Vega, 2003).

Our findings suggest that for some individuals, acculturation with greater Anglo-orientation may lead to injury in crucial physiological stress response systems. It is possible that greater Anglo-orientation, may contribute to allostatic load, and over time, lead to injury of the circadian rhythm and decreased adrenal capacity, similar to that observed with other chronic stressors (Pruessner et al., 1999). Similar effects have been observed in teachers with burnout symptoms who report living under chronic stress (Pruessner et al., 1999), in individuals reporting job stress (Caplan et al., 1979) and in Romanian orphans with a history of social deprivation (Carlson and Earls, 1997).

Determination of the precise developmental mechanisms that contribute to the association between a lower CAR and early exposure to trauma or greater acculturation (Anglo-orientation) is beyond the scope of the current study. However, it has been hypothesized that early exposure to stress alters the development of the hypothalamic CRF system, leading to enhanced stress reactivity (Meaney et al., 1993; Plotsky and Meaney, 1993; van Oers et al., 1998; Wigger and Neuman, 1999; Caldji et al., 2000; Ladd et al., 2000) which can then undergo desensitization with subsequent exposure to chronic stress. The model proposes that the HPA axis transitions from a system with increased stress responsivity to a system with a lower CAR (Heim and Nemeroff, 1999; Ehlert et al., 2001; Heim et al., 2001b; Meinslschmidt and Heim, 2005). The mechanism for this transition is unclear. Alternatively it is also plausible that early childhood stress or persistent chronic stress produces a blunted CAR with no antecedent HPA axis hyperactivity.

There are some limitations of the current study worth noting. The current study used a linear assessment of acculturation that assesses levels of acculturation based on patterns and mastery of language, demographic factors, and participation in either ethnic group or host culture (Marin and Marin, 1991; Cuellar et al., 1995). This approach assumes that as an individual increases their orientation in one culture there is a corresponding reduction in orientation in the other culture. It is more likely that some bicultural individuals may embrace aspects of both cultures. The effects of a highly bicultural adaptation to acculturation on the CAR will need to be examined in future studies.

It is also possible that unreported or undiagnosed PTSD in our subject sample influenced our findings because we did not specifically assess for the presence of a diagnosis of PTSD. We believe this is unlikely given that while 56% of Americans experience a lifetime trauma, only 8% subsequently develop Post Traumatic Stress Disorder (PTSD), (Perkonigg et al., 2000). Moreover, the odds of an abused and neglected child developing PTSD are only 1.75 times higher than the odds for a matched comparison subject (Widom, 1999). In addition, the conditional risk for PTSD is highest for females exposed to assault-related violence (Breslau et al., 1998, 2002), while in our sample, mean number of exposures to assault-related violence for female participants was less than one, decreasing the probability that PTSD influenced our findings. Also, depression is one of the most commonly co-occurring disorders with PTSD, and among individuals who have or have had a diagnosis of PTSD; approximately 48% also have current or past depression (Breslau, 2002). The assessment and exclusion of participants for depression in our sample, further minimizes the potential influence of unreported or undiagnosed PTSD on current findings.

About half of the sample reported a relatively low family income; therefore it is possible that a lower socioeconomic status contributed to a diminished CAR, possibly due to material hardship, and increased burden (Anderson and Armstead, 1995; Ranjit et al., 2005). However, we believe it is unlikely that our findings are due to the overall lower income in our sample because there were no differences in parental income among trauma groups ($F=0.58$, $df=2, 54$, $p=.56$) or between acculturation groups ($F=0.41$, $df=1, 55$, $p=.53$). Moreover, the effects of socioeconomic parameters on cortisol secretion and the CAR generally indicate an inverse

relationship (Lupien et al., 2000, 2001; Rosmond and Bjorntorp, 2000; Kapuku et al., 2002; Bennett et al., 2004; Kunz-Ebrecht et al., 2004; Wright and Steptoe, 2005). However, it is possible that prolonged exposure to the increased material hardship and burden associated with low SES could result in an enhanced HPA axis response to stress transitioning to a system with a lower CAR.

Taken together, these findings show that both moderate and extreme exposure to early traumatic stress is associated with attenuation of the CAR in a sample of healthy, adult Mexican-Americans. These findings also show that higher levels of acculturation (greater Anglo-orientation) may be associated with culturally unique, chronic stressors that lead to injury of the HPA axis and attenuation of the CAR. The effects of early childhood trauma and acculturation on the CAR were not additive. Early childhood trauma and higher levels of acculturation modulate the CAR independent of the influences of depression on cortisol dynamics. It will be important to determine if injury to the HPA axis induced by early childhood trauma and the stress of acculturation is one possible mechanism accounting for the reported higher incidence of negative health outcomes in this population.

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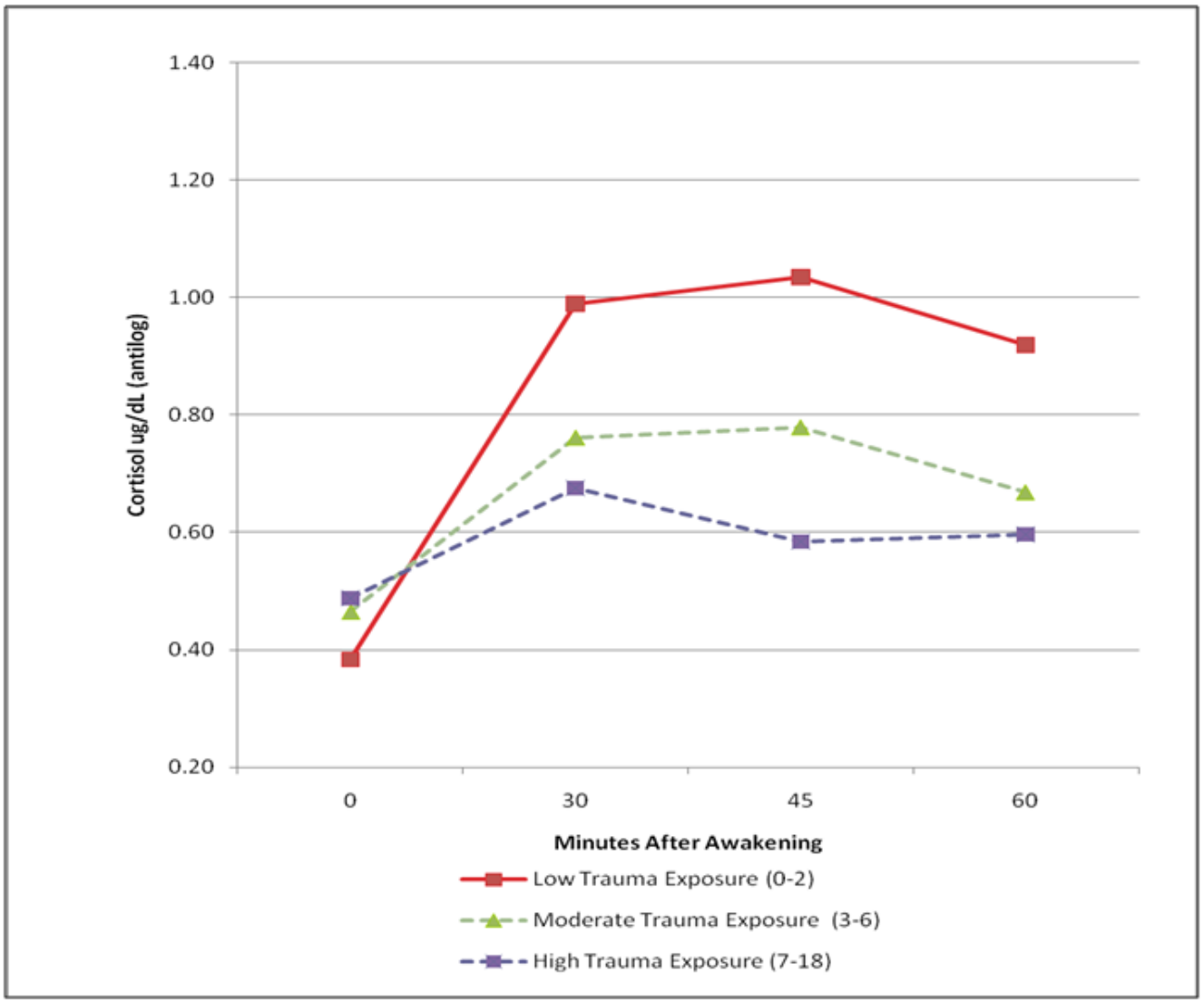

Figure 1.
The effect of early trauma on the Cortisol Awakening Response (CAR)

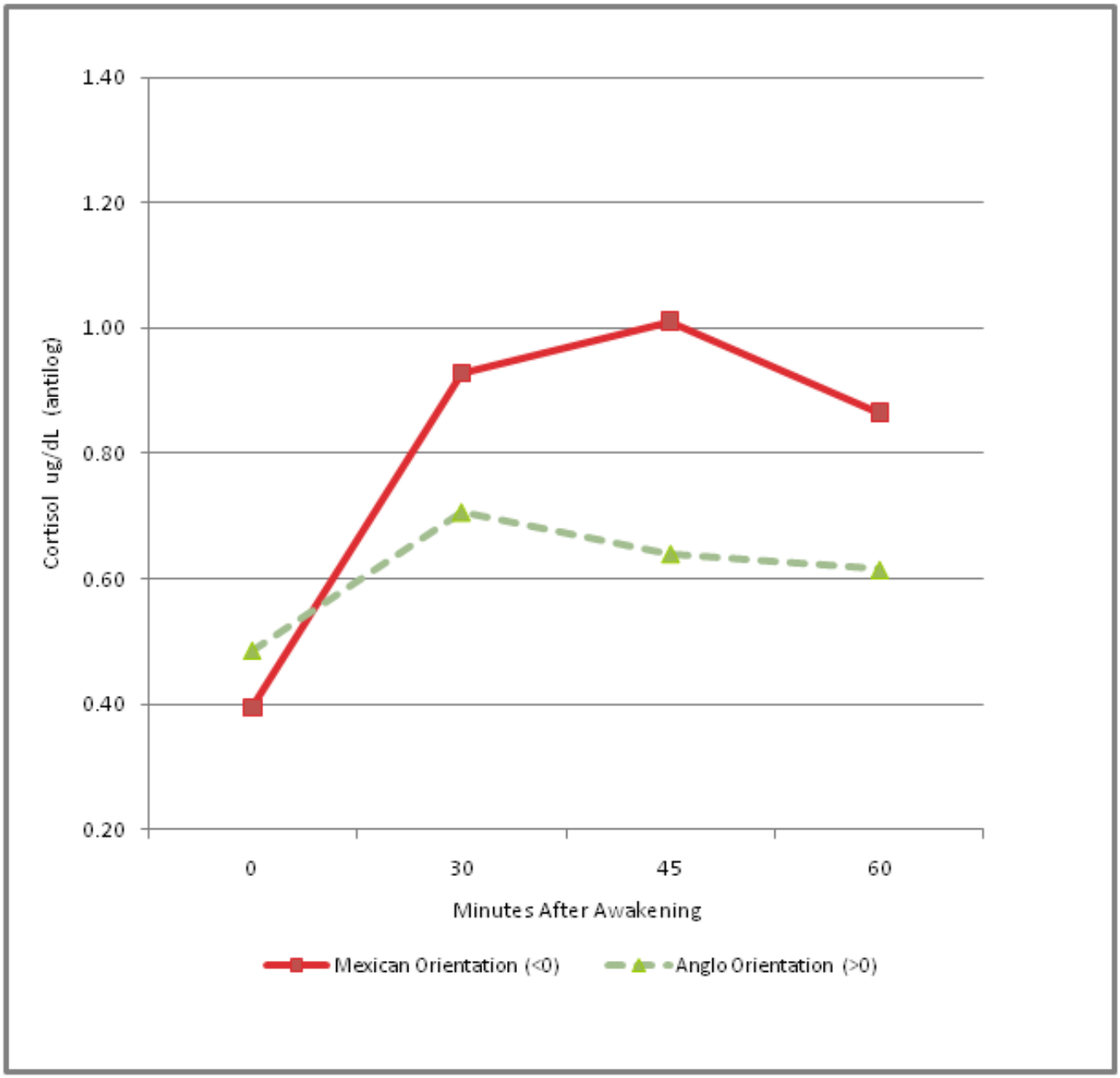

Figure 2.
The effect of acculturation on the Cortisol Awakening Response (CAR)

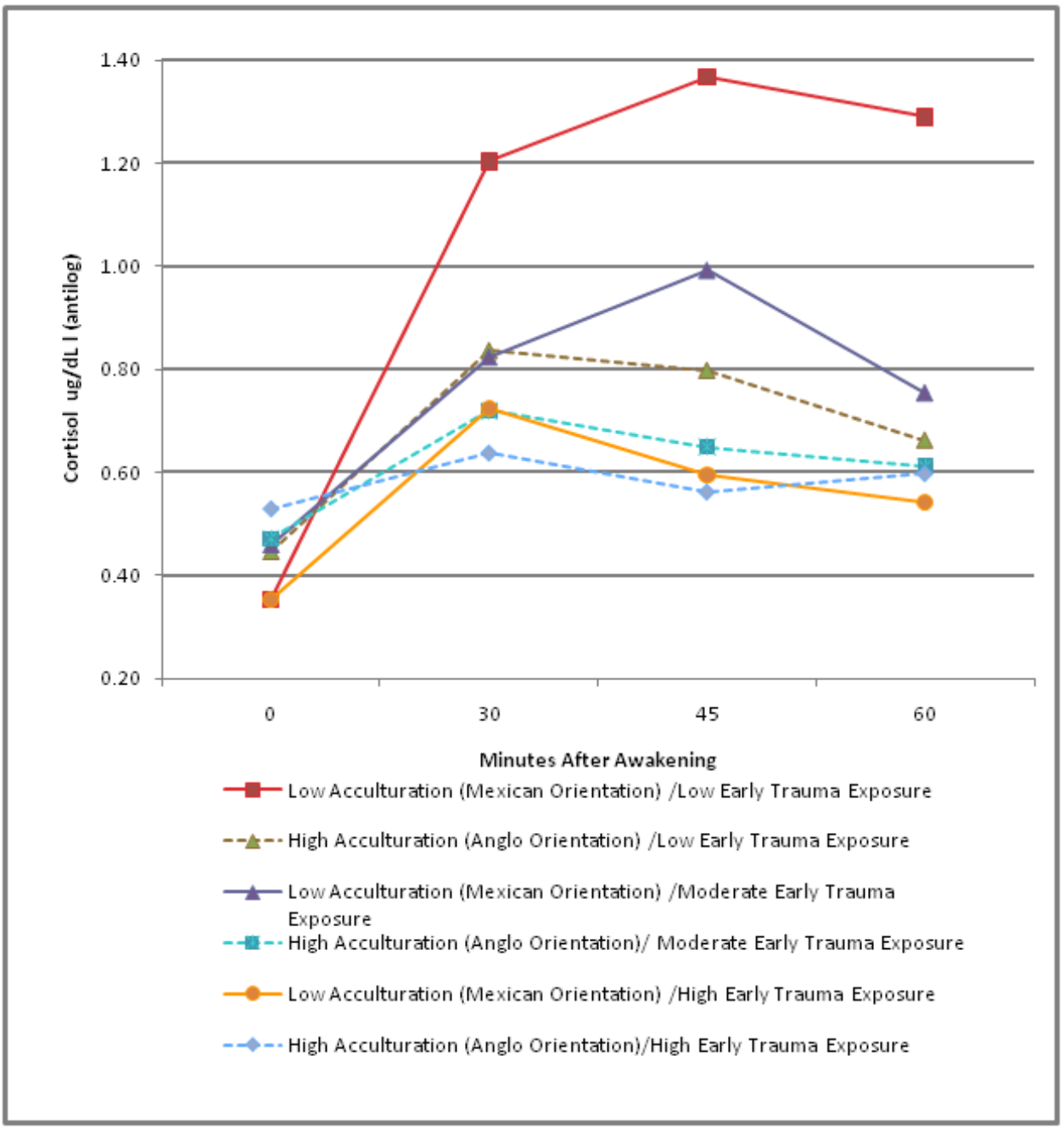

Figure 3.
The effects of acculturation and trauma on the Cortisol Awakening Response (CAR).

Table 1

Sample demographics for Mexican-American adult, males and females

	Overall Sample	Females	Males
Gender	(n=59)	33 (56%)	26 (44%)
Age (Median and Range)	20 (18-38)	21(18-38)	19 (18-37)
Body Mass Index (Mean and SD)	24.39 ± 4.19	24.54 ± 4.5	24.20 ± 3.8
Parental/Participant Income			
Below 40K	33 (55.9%)	22 (66.7%)	11 (42.3%)
40K-80K	16 (27.1%)	7 (21.2%)	9 (34.6%)
80K and above	10(17.0%)	4 (12.1%)	6 (23.1%)
Parental Occupational Status			
Executive	10 (16.9%)	5(15.2%)	5 (19.2%)
Administrative/Management	18 (30.5%)	11 (33.3%)	7 (27.0%)
Clerical/Skilled Manual Labor	20 (34.0%)	11 (33.3%)	9 (34.6%)
Semi-skilled or Unskilled Labor	11 (18.6%)	6 (18.2%)	5 (19.2%)
Participant Education Level			
≤12 years	4 (6.8%)	2 (6.1%)	2(7.7%)
13 years	23 (39.0%)	11 (33.3%)	12 (46.1%)
14 years	13 (22.0%)	11 (33.3%)	2 (7.7%)
15 years	6 (10.2%)	2 (6.1%)	4 (15.4%)
≥16 years	13 (22.0%)	7 (21.2%)	6 (23.1%)

Table 2

Number of exposures to different dimensions of trauma in adult, Mexican-Americans (Means and SD)

Trauma	Overall Sample (59)	Males (26)	Females (33)	*P*
Total Traumas	5.32 (4.25)	6.73(4.79)	4.21(3.45)	$p<.05$
General Traumas	2.42 (1.78)	2.96(1.89)	2.00(1.60)	$p<.05$
Physical Punishment	1.29 (1.33)	1.70(1.44)	0.97(1.16)	$p<.05$
Emotional Abuse	1.19 (1.58)	1.42(1.75)	1.00(1.44)	NS
Sexual Events	.42 (0.86)	0.65(1.02)	0.24(0.66)	NS

* Males tended to report greater exposure to all dimensions of early trauma compared with females, with significantly greater total trauma exposures ($F\ (1, 57) = 5.51$, $p<.05$), greater exposure to general traumas ($F\ (1, 57) = 4.48$, $p<.05$), and physical punishment ($F\ (1, 57) = 4.58$, $p<.05$). Gender differences in exposure to sexual events approached significance, although was not statistically significant ($F\ (1, 57) = 3.51$, $p = .066$).

Table 3

Estimated mean log cortisol by time after awakening in the six trauma-acculturation groups

				Minutes after awakening			
Trauma	**Acculturation**	**N**	**0**	**30**	**45**	**60**	**ML SEs**
Low	Low	9	−1.04 (0.19)	0.19 (0.20)	0.31 (0.19)	0.25 (0.22)	0.20
	High	8	−0.81 (0.19)	−0.18 (0.16)	−0.23 (0.18)	−0.41 (0.16)	0.21
Mid	Low	10	−0.78 (0.17)	−0.19 (0.07)	−0.01 (0.12)	−0.28 (0.12)	0.19
	High	12	−0.75 (0.15)	−0.33 (0.18)	−0.43 (0.20)	−0.49 (0.19)	0.17
High	Low	5	−1.04 (0.29)	−0.32 (0.16)	−0.52 (0.26)	−0.61 (0.23)	0.27
	High	15	−0.64 (0.18)	−0.45 (0.20)	−0.58 (0.21)	−0.51 (0.18)	0.15

Note: Bootstrap standard errors in body of table based on 1000 replications sampling cases with replacement. Figure 3 depicts antilogs of these values. Rightmost column presents ML estimated standard errors by group (SAS MIXED reports one SE for all four data points per group).