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Dietary Curcumin Promotes Resilience by Protecting Against Stress-Induced Anxiety in Mice

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Recent evidence suggests that curcumin, a compound found in the turmeric plant (Curcuma longa), prevents stress-induced depressive-like behavior in rodents and reduces symptoms of depression in patients. However, the use of curcumin for treating anxiety has not been examined. Here we tested whether dietary curcumin blocks the effects of a social stressor on innate fear. Adult male mice (129/EvEv) were given chow containing 1.5% curcumin or control chow for 5 days before being exposed to chronic social defeat stress or being housed in control conditions for 10 days. Social interaction was tested with an unfamiliar aggressive CD-1 mouse as the social target. One week after social defeat, innate fear in stressed and non-stressed controls was tested in the elevated plus maze, open field, and novelty suppressed feeding tests. One day after the last behavioral test, mice were restrained for 15 minutes, and were immediately sacrificed for serum/tissue collection. Mice remained on curcumin throughout behavioral testing. Stressed mice given control chow spent significantly less time interacting with the CD-1 social target relative to stressed mice given curcumin, and non-stressed mice fed control chow or curcumin. In the elevated plus maze, stressed mice given control chow spent significantly less time in the open arms than stressed mice given curcumin and non-stressed mice on control chow. Similarly in the open field, stressed mice given control chow spent significantly less time in the center of the arena than stressed mice on curcumin and control mice given control chow or curcumin. These results suggest that dietary curcumin blocks the effects of stress on social avoidance behavior and innate fear, suggesting that curcumin may effectively treat anxiety.
There is evidence that cumulative traumatic experiences over one’s lifetime can lead to negative behavioral and psychological effects such as poor parenting practices, which, in turn can lead to worse outcomes in affected offspring. The Developmental Origin of Adult Health and Disease Hypothesis (DoHaD) suggests that in utero maternal stress has long lasting effects on affected offspring. Socioemotional function, the ability to utilize emotions to communicate effectively with others, may be one such effect. There is also evidence that information processing capabilities, or cognitive function, plays a protective role against negative socioemotional outcomes in stress-affected children. However, this association has not been tested in toddlers, nor have the separate and combined effects of lifetime trauma and acute prenatal stressors been well explored. The Stress in Pregnancy Study follows women (N = 724, M age = 27.44, 24.5% Black, 8.3% Asia, 50.4% Hispanic, 12.5% White) during pregnancy and their children, through age four (N = 701, 48.4% female). Using a subset of this sample, we hypothesized that in the trauma and stress groups socioemotional function in offspring would be worse and that cognitive function would be protective against these effects. We compared children of mothers who had (1) experienced no trauma or prenatal stressor due to Superstorm Sandy (c.f., Sandy-exposure) (2) experienced only lifetime trauma (3) experienced only Sandy-exposure and (4) experienced both lifetime trauma and a Sandy-trauma exposure. At 24 (n=75) and 36 months (n = 72), information processing was measured using the Bayley-III Cognitive Scale and socioemotional function was measured using the Bayley-III Social-Emotional Scale. Maternal trauma was measured using a Traumatic Events Inventory specific to Superstorm Sandy and the PTSD Diagnostic Scale for DSM-5 (PDS-5). Linear Ordinary Least Squares regressions were run looking at moderating effects of cognition on the relation between trauma and stress and socioemotional function in 24 and 36-month-old children. At 24 months, no significant effects emerged. At 36 months, maternal lifetime trauma significantly interacted with cognitive function ($\beta = .49$, $p < .01$) to predict socioemotional functioning. This indicates cognitive function is positively related to socioemotional function, only in children whose mothers have experienced a lifetime trauma. Children in this group with higher cognitive function also had higher socioemotional function. These findings suggest that as cognitive functioning develops over the first three years of life, it enhances socioemotional functioning for children whose mother have experienced trauma over their lifetime but not for other children. The posttraumatic growth theory suggests that positive psychological changes can occur following challenging life events. This may explain why observed protective effects occurred only in children whose mothers had experienced a lifetime trauma. Mothers with a trauma history may have developed better coping and communication skills that, in turn, lead to better parenting practices, and therefore better child outcomes.
Curcumin Enhances the Extinction of a Pavlovian Fear Memory

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Background: Recent evidence from our lab has indicated that a diet enriched with the naturally-occurring polyphenol compound curcumin can impair the consolidation and reconsolidation of memories associated with Pavlovian fear conditioning, a widely studied animal model of traumatic memory formation in PTSD. In the present study, we explored the effect of curcumin on the extinction of a previously established Pavlovian fear memory.

Methods: Sprague-Dawley rats were fear conditioned with 3 tone-shock pairings in Context A, which consisted of a chamber with grid floors, clear plastic walls, and a house light. In the first experiment, rats were fed a curcumin-enriched (1.5%) or regular chow diet for 5 days before extinction training. In the second experiment, rats received infusion of curcumin (1μg/side) or vehicle directly into the infralimbic cortex (IL) immediately following extinction training. Extinction training and testing were performed in Context B, which consisted of a dark chamber, with a black plastic floor washed with peppermint soap.

Results: In the diet experiment, the curcumin-fed group showed significant facilitation of fear extinction learning and significant enhancement of extinction retention compared to chow-fed controls. In the IL infusion experiment, curcumin significantly enhanced extinction retention relative to vehicle controls, but had no effect on extinction learning.

Conclusions: Curcumin enhances the extinction of a Pavlovian fear memory, suggesting that it may be a potential adjunct to exposure-based treatments in patients with psychiatric disorders characterized by traumatic memory formation.
CK2 modulates serotonergic signaling and depression-related behaviors

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The serotonin (5-HT) neurotransmitter system has been widely implicated in the pathophysiology of mood disorders such as anxiety and major depression. In search of antidepressants exerting a fast onset of action, the 5-HT4 receptor (5-HT4) has emerged as a potential target: administration of partial agonists induce behavioral responses in rodents similar to those observed after chronic treatment with SSRIs, with onset of action after 3-7 days.

We have previously attributed a role for CK2 in the regulation of Gαs-coupled receptors, and thus far have shown that D1 and A2a receptor signaling are altered by CK2 in vivo. Here we describe that 5-HT4, another Gαs-coupled receptor, is also under regulation of CK2 on transcriptional and post-transcriptional levels. We have evidence that in the brain, there is a region-specific regulation of 5-HT4 expression dependent in CK2 activity. In two different conditional CK2 knockout mouse lines (Drd1a-Cre and Emx1-Cre), the 5-HT4 but not other 5-HT receptors are up-regulated in the prefrontal cortex but not in other regions, such as the striatum or the hippocampus, where CK2 is also ablated. Furthermore, these mice show a clear phenotype when tested in paradigms for mood and anxiety, reminiscent of antidepressant-treated animals, a finding which is easily reconcilable with elevated 5-HT4 levels/activity. We have been able to determine the specific brain region, namely, the prelimbic and infralimbic regions of the prefrontal cortex mediating the phenotype since focal virally-mediated knockdown of CK2 as well as overexpression of 5-HT4, rescue the phenotype seen in the conditional KO mice models. In addition, pharmacological inhibition of CK2 increases 5HT4-R activity in vitro and reduces endocytosis of the receptor. Our work clearly identifies CK2 as regulator of serotonergic signaling and adds a novel element to our understanding of the etiology of mood-related disorders and antidepressant function.
Sexual violence against young women is associated with symptoms of depression, anxiety, rumination, and vivid autobiographical memories

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Sexual violence is a global social and mental health concern affecting women of all ages, but most experiences occur during adolescence and young adulthood. A recent survey of United States universities indicates that more than 20% of women experience sexual violence during college (Cantor et al., 2015). In the present study, we examined mental health and memory outcomes in college-aged women with and without a history of sexual violence in their lifetime. After a structured interview with the SCID, women completed self-report questionnaires for depression (Beck Depression Inventory), anxiety (Beck Anxiety Inventory), rumination (Ruminative Responses Scale), posttraumatic cognitions (Post-Traumatic Cognitions Inventory), and autobiographical memories about a stressful life event (Autobiographical Memory Questionnaire), and a standard working memory task (Symmetry Span, Engle lab). Women with a history of sexual violence (n=28) reported significantly more depressive, anxious, and posttraumatic symptoms (p’s < 0.01), as well as more vivid autobiographical memories about a stressful life event and ruminative thoughts (p < 0.05), when compared to those measures in women without a history (n=81). Correlations among these measures were highly significant (p < 0.001). Working memory scores were not different between groups. These data suggest that sexual violence can increase the rehearsal of intense stressful life memories, which may contribute to or minimally interact with rumination, symptoms of depression, anxiety, and trauma-related thoughts.

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Executive Control Training Enhances Emotion Regulation

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Adaptive behavior depends on the ability to effectively regulate emotional responses. Failure in the regulation of emotional arousal can result in heightened physiological reactions and disruptive behavioral performance. In turn, these behavioral and physiological alternations can lead to various psychopathologies. In several studies we demonstrated that training executive control, an attentional mechanism that enables goal-directed behavior, lead to reduced emotional interference by aversive pictures and to a lower amygdala activation to these pictures. Moreover, we showed that training individuals to recruit executive control prior to the presentation of unpleasant pictures enhances their ability to regulate an upsetting personal event using reappraisal. These findings suggest that the interplay between emotion and executive control is essential for maintaining adaptive behavior and may be impaired in individuals with emotion regulation difficulties.
Salutary Effects of an Attention Bias Modification Mobile Application on Biobehavioral Measures of Stress and Anxiety during Pregnancy

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Stress and anxiety during pregnancy are associated with a range of adverse health outcomes, thus there is an unmet need for low-barrier treatments that target stress and anxiety. One such treatment approach, attention bias modification training (ABMT), reduces the anxiety-related attentional threat bias, which is also associated with disrupted neural processing of threat. It remains unclear, however, whether reducing treatment barriers via mobile delivery of ABMT is effective and whether ABMT efficacy varies depending on individual differences in neural processing of threat. The present study tested whether mobile, gamified ABMT reduced prenatal threat bias, anxiety and stress, and whether ABMT efficacy varied with individual differences in neural responses to threat. Participants were 29 women in their 19th – 29th week of pregnancy, randomized to four weeks of ABMT versus placebo training (PT) versions of the mobile app using a double-blind design. Self-report of anxiety, depression, and stress were obtained, and salivary cortisol was collected at home and in lab in response to stressors to index biological stress reactivity. Threat bias was measured using a computerized attention assay during which EEG was recorded to generate event-related potentials (ERPs) to threat cues. Results showed lower levels of threat bias (1-tailed) and lab cortisol following ABMT versus PT. Although the main effect of ABMT on subjective anxiety was not significant, the magnitude of cortisol reduction was correlated with lower levels of subjective anxiety and threat bias. Those receiving ABMT also reported less anxiety when showing smaller ERPs to threat (P1, P2) prior to training, but, conversely reported more anxiety when showing larger ERPs to threat. Use of gamified, mobile ABMT reduced biobehavioral indices of prenatal stress and anxiety, but effects on anxiety varied with individual differences in cortisol response and neurocognitive indices of early attention to threat.
Impact of Maternal Depression and Exposure to Superstorm Sandy on Placental HPA Gene Expression

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Background: Prenatal stress can alter HPA axis functioning in offspring via the placenta, which regulates the exchange of hormones between mother and fetus. Key genes involved in this exchange include HSD11B2, SLC6A4 and CRHBP, whose functioning have long-term health implications for offspring neurobehavioral development.

Methods: The present study utilized 293 placentas collected by the Stress in Pregnancy (SIP) study, an on-going longitudinal birth cohort study at the Icahn School of Medicine at Mount Sinai and New York Presbyterian Queens. Participants were recruited during their 2nd trimester (i.e., baseline), and followed throughout their pregnancy and as their children develop. A subset of the cohort was exposed to Superstorm Sandy during pregnancy. Maternal depressive symptomatology was assessed via self-report (Edinburgh Postnatal Depressive Scale) at baseline. Placenta tissue samples were collected following participant births, and subsequently, gene expression of HSD11B2, SLC6A4, and CRHBP was quantified using qPCR.

Results: Results of an Analysis of Co-Variance (ANCOVA) adjusted for maternal age, education, and race/ethnicity examined the main effects and interaction of depression and Sandy exposure during pregnancy on placental gene expression. CRHBP was hypoexpressed among those depressed (p=.05), whereas HSD11B2 and SLC6A4 was hyperexpressed in those exposed to Sandy (p=.001; p=.002, respectively). There is no notable interaction effects, yet pairwise comparisons revealed that both the unaffected and comorbid groups had lower levels of HSD11B2 and SLC6A4 expression (p<.001, p=.001, respectively) as compared to the depressed only group. We also found that the depressed only group had a higher level of CRHBP expression when compared to the disaster only group (p=.048).

Conclusions: Lower levels of HSD11B2 and SLCA4 expression may expose the developing fetus to higher levels of cortisol and serotonin, which has been linked with suboptimal neurobehavioral trajectories. Higher expression of CRHBP may expose the fetus to lower levels of CRH, which has been associated with negative temperament outcomes postpartum. These findings indicate that observed stress (i.e., Superstorm Sandy exposure) and perceived stress (i.e., depressive symptomatology) might differentially influence underlying biological mechanisms. Enhanced understanding of the underlying biological pathways by which maternal stress (objective and subjective) impacts fetal/child development can help improve identification and intervention for affected mothers and their offspring.
Assessing the Impact of Maternal Smoking During Pregnancy and Gene Expression of MAO-A and MAO-B On Child Neurodevelopment

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Maternal smoking during pregnancy (MSP) is associated with adverse child neurodevelopment with significant gender differences (Suzuki et al., 2011). Monoamine oxidase (MAO-A) and Monoamine oxidase (MAO-B) are neurodevelopmental genes that modulate child development. The current study investigated the roles of MSP, MAO-A/B gene expression and the MSP x MAO-A/B interaction in predicting child outcomes separately for boys and girls. In a subsample of 84 women and their offspring (boys, n = 38) participating in a larger birth cohort (SIP Study, PI Nomura), participants were recruited at the OB/GYN clinics of Mount Sinai Hospital and New York Presbyterian Queens, and followed through pregnancy and as their children develop. MSP was ascertained by self-report during the 2nd trimester, while their offspring's cognitive, language, motor, social-emotional, and general development were evaluated using the Bayley Scales of Infant and Toddler Intelligence (Bayley III) between ages 18 and 42 months. The mRNA levels of MAO-A/B gene expression were analyzed from the stored maternal placenta tissues. Our results showed that in boys: (1) MSP was linked with poorer social-emotional (p=.040) and general functioning (p=.001); (2) Higher MAO-A was linked to poorer general functioning in MSP exposure (p < .001). In girls, it was found that (1) MSP was linked with poorer language (p=.020) and cognitive abilities (p=.001), but better general functioning (p = .085) (2) MAO-B was negatively linked with cognitive abilities (p=.020), but positively with social-emotional (p=.067); (3) Lower MAO-A was linked to poorer language capabilities in MSP exposure (p=.031); (4) Higher MAO-B was linked to poorer cognitive ability in MSP exposure (p < .001). Taken together, our findings suggested the importance of examining MSP and MAO-A/B gene expressions as well as their interaction to better understand the etiologies of suboptimal neurodevelopment in offspring and to explain significant gender differences. These findings could be critical factors for identifying high-risk populations and informing pregnant women in the interest of optimal child development.
Translational profiling of CA3 neurons in BDNF Va66Met mice exposed to early life stress

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Early life stress (ELS) has been associated with an increased risk of developing a mental health disorder later in life. Rodent models of ELS show impaired learning and memory in adulthood that is coincident with persistent changes in hypothalamic-pituitary adrenal (HPA) axis reactivity and neuronal morphology. Importantly, decreases in dendritic spine density in the CA3 neurons of the hippocampus (a region important for learning and memory) have been observed in adult mice exposed to bedding deprivation as pups. Several genetic mutations have also been identified that can increase the risk of developing a mood disorder. Among them is a single nucleotide polymorphism in the brain derived neurotrophic factor (Bdnf) gene that results in the substitution of a methionine (Met) for a valine (Val) at position 66. These mice exhibit increased sensitivity to the effects of stress in adulthood. However, the effects of ELS on BDNF Val66Met mice remain unknown. In this study, the effects of ELS on the translational profile of CA3 pyramidal neurons were examined in the BDNF Val66Met mice.

Isolation of in vivo translating RNA fractions from a genetically homogenous population of CA3 pyramidal neurons was accomplished using transgenic mice expressing an EGFP fused to the L10a ribosomal subunit that is under the control of a cell-type specific promoter. These reporter mice were crossed with BDNF Val66Met allele carriers to generate double transgenic animals, which were then subjected to bedding and nesting deprivation from P2-P12 (Rice…Baram, 2008). Afterwards, mice were given standard housing conditions until 4mos of age. Mice were rapidly decapitated and the hippocampus was dissected for RNA isolation by Translating Ribosomal Affinity Purification (TRAP). TRAP and unbound mRNA fractions were subjected to RNA-sequencing. Differentially expressed genes were grouped into pathways using the DAVID tool. Comparisons of the TRAP fractions from unstressed BDNFMet/+ mice with unstressed wild type (WT) mice identified changes in the levels of 1,420 genes. WT mice subjected to ELS compared with unstressed controls revealed that 1,553 genes remain changed even in adulthood. Over half of the genes changed after ELS in WT mice were identical to those changed in the unstressed BDNFMet/Met mice. Pathway analysis of the genes changed in Met allele carriers and mice exposed to ELS revealed common molecular mechanisms that might underlie the altered neuroanatomy and behavior, such as changes in synaptic signaling proteins. Slightly more genes (1,682) were changed in response to ELS exposure in BDNFMet/+ mice compared with unstressed BDNFMet/+ mice. The substantial overlap in gene pathways changed in BDNF Met allele carriers or WT mice exposed to ELS identifies common mechanisms of stress susceptibility derived from either genetic or environmental factors. Together, this data not only reveals mechanisms underlying mood disorder susceptibility in CA3 neurons, but also lays the groundwork for developing novel treatments to reverse the changes induced by ELS or resulting from a genetic predisposition to mood disorders.

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Keywords: Hippocampus, Gene environment interactions, Early life stress, BDNF, RNA-Sequencing
Aberrant prelimbic-amygdala stress-induced activation contributes to susceptibility to acute social defeat stress in mouse

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Depression is a prevalent and debilitating neuropsychiatric disorder with lifetime prevalence of 20% in the developed world. Treatment for depression remains largely ineffective. Only 30-50% of those suffering from depression respond to currently available therapies. There is therefore a clear and immediate need to expand our understanding of the neurobiology that contributes to depression. Recent neurobiological advances are increasingly demonstrating that depression is a whole brain disorder, caused by dysfunctional communication between multiple regions. However very little is understood about the state of these circuits prior to stress, and how these predispose to susceptibility and resilience to depression. Chronic social defeat is a well-validated mouse model capable of inducing depressive-like behavior in a subset of animals. A proportion of mice that undergo chronic social defeat exhibit depressive-like behavior, termed “susceptible”, while a separate group exposed to the same experience do no not, termed “resilient”. This is therefore an excellent model for examining the circuits that contribute to susceptible and resilient response to stress. Using an acute model of social defeat stress that allows for distinction of susceptibility and resilience one hour after being exposed to six minutes of social stress, we explored differences in activation of the prelimbic-amygdala circuit between resilient and susceptible animals. Our experiments revealed that susceptible animals had a greater recruitment of this circuit in response to acute stress. To probe the structural substrate for this difference in activation, we analyzed the synaptic structure of stress-activated prelimbic cells that project to the amygdala in susceptible and resilient animals. We found that activated cells from susceptible mice had greater density of mushroom spines on basal dendrites compared to activated cells from resilient animals. These results may indicate stronger prelimbic-amygdala stress-induced connectivity predating exposure to social stress in susceptible mice. To test the functional relevance of the observed differences in circuit connectivity to divergent behavioral responses, we chemogenetically inhibited the activation of the prelimbic-amygdala circuit during acute social defeat stress and found an increase in resilient behavior. Together, these results indicate that differences in prelimbic-amygdala connectivity prior to stress exposure are functionally involved in the establishment of divergent behavioral responses to acute stress.
Recent literature has reported that both psychosocial risk factors and neurobiological deficits are associated with antisocial tendencies. This research is part of a longitudinal study looking at psychological and physiological influences on antisocial behavior. Our prior work has shown that prenatal maternal stress and autonomic arousal interacted to predict conduct problems and psychopathic traits in youth. The present study examines the longitudinal relationship between prenatal maternal stress, autonomic arousal, and antisocial behavior from the same cohort. At Time 1 prenatal maternal stress was assessed in 253 8-to-10 year old children through caregiver’s retrospective report, and children’s heart rate (HR) and respiratory sinus arrhythmia (RSA) were acquired during rest periods. Both caregiver and child-reported measures of antisocial behavior and psychopathic traits were collected at Time 1 and again one year later at Time 2. Our results indicated that low levels of resting HR during Time 1 predicted higher levels of delinquency at Time 2. Prenatal maternal stress was positively correlated with impulsivity at Time 2. A significant interaction effect was found; children with lower resting HR and higher prenatal maternal stress had higher impulsivity scores during Time 2. We saw no such relationship with RSA. Our findings suggest that resting HR may be more robust than RSA in predicting antisocial behavior and psychopathic traits, and that psychopathic traits (i.e., impulsivity) but not antisocial behaviors are more stable and likely to be influenced by epigenetic factors.
Modulation of Fear Memory by Dietary Curcumin in Chronically Stressed Rats

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Chronic stress has been strongly implicated in the development of a number of psychiatric disorders, such as post-traumatic stress disorder (PTSD). One prominent symptom of PTSD is robust and extinction-resistant traumatic memory formation. It is hypothesized that the susceptibility to enhanced fear memory formation characteristic of disorders like PTSD is due to maladaptive changes in the hypothalamic-pituitary axis after chronic stress exposure. The release of glucocorticoid hormones (e.g. cortisol) in response to chronic stress has been found to have contrasting effects on both the morphology and physiology within different learning and memory-related regions of the brain enriched with glucocorticoid receptors. The ‘fear memory circuit’ of the brain, which includes the lateral amygdala (LA), hippocampus (HIP), and infralimbic cortex (IL), plays a critical role in the formation of fear memories (LA), fear generalization (HIP) and fear extinction (IL). Interestingly, exposure to chronic stress has been shown to impair hippocampal and prefrontal dependent memories while enhancing amygdala-dependent memories. Recent findings in our lab have shown that chronic oral exposure to corticosterone (CORT), a stress-associated adrenal steroid, persistently enhances the expression of the synaptically-localized proteins GluR1 and synaptophysin within the LA of rats. This effect is consistent with studies that have observed long-lasting dendritic hypertrophy and increased spine density in LA neurons of chronically stressed rats. Further, we have observed that these CORT-induced synaptic changes in the LA are prevented if rats are fed a diet enriched with the polyphenol compound curcumin (derived from the rhizome of Curcuma longa) during the CORT exposure period. In the present study we have asked whether chronic stress, as modeled by a 10-day restraint paradigm, is associated with changes in synaptically-localized proteins within the fear memory circuit and to enhanced formation of Pavlovian fear memories and impaired fear memory extinction. Further, we have asked whether a dietary source of curcumin provided during the period of chronic stress can prevent these molecular and behavioral effects. In separate experiments, we have also asked whether these chronic stress-induced molecular and behavioral changes persist after a period of recovery. Preliminary findings reveal that chronically stressed rats gain significantly less weight and have significantly larger adrenal glands compared to non-stressed controls following stress exposure, effects which are not prevented by curcumin. Rats that received chronic stress followed by a 10-day recovery period similarly exhibited significantly larger adrenal glands, but this effect was prevented if they were fed a curcumin-enriched diet during the period of chronic stress. Rats that were fear conditioned with tone-shock pairings immediately following the 10-day restraint stress protocol exhibited higher levels of fear memory to both the training context and the tone that persisted after a period of recovery, and curcumin was observed to partially mitigate this effect. We are currently using Western blotting to assay quantitative changes in synaptically localized proteins like GluR1, GluR2/3, synaptophysin, BDNF and PSD95 in the wider fear memory circuit following chronic stress in rats fed a diet of normal chow or curcumin-enriched chow.
Resting connectivity between the amygdala and the ventral anterior cingulate cortex is associated with sympathetic reactivity to a trauma reminder

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Background
Trauma exposure can have enduring effects on a person's mental and physical health, such as symptoms of post-traumatic stress disorder (PTSD) and hyperactivity of the neural and endocrine stress systems (e.g., Pitman et al. 2012).

For example, trauma-exposed people show heightened sympathetic reactivity to affective information, such as trauma-related cues (e.g., McTeague et al. 2010), which is associated with PTSD symptoms (e.g., Libezon et al. 1999).

Trauma-exposed people also show heightened reactivity and abnormal resting connectivity between the amygdala, a key region involved in stress response initiation, and other stress-relevant brain regions, such as the salience network and prefrontal regulatory regions (e.g., Patel et al. 2012).

However, the relation between heightened sympathetic reactivity and abnormal amygdala connectivity in trauma-exposed people has yet to be clarified. Therefore, we used resting-state fMRI to test trauma-related differences in functional amygdala connectivity and their association with sympathetic reactivity in trauma-exposed women.

Hypotheses
We hypothesized that trauma-related symptoms would be associated with sympathetic reactivity to a trauma reminder, indexed by salivary alpha-amylase (sAA). We also hypothesized that, compared to controls, trauma-exposed women would show greater resting amygdala connectivity with the salience network and weaker resting amygdala connectivity with prefrontal regulatory regions. Finally, we hypothesized that resting amygdala connectivity would be associated with sAA reactivity to a trauma reminder.

Method
Twenty-four trauma-exposed women and 20 no-trauma controls completed a resting-state fMRI scan and a clinical interview that covered traumatic events. To measure sAA reactivity to the trauma report/reminder, we collected saliva pre-interview and immediately post trauma report.

Neuroimaging data were analyzed using whole-brain mass univariate GLM with anatomically-defined amygdalae as seed regions. Associations with sAA reactivity were tested within a functionally-defined region of interest.

Results
SAA reactivity to the trauma reminder was associated with number of PTSD symptoms (total: $b=0.45$, $p=0.003$; re-experiencing: $b=0.34$, $p=0.028$; avoidance: $b=0.47$, $p=0.001$; and hyperarousal: $b=0.40$, $p=0.013$).
Trauma-exposed women showed greater resting connectivity between the amygdala and the ventral anterior cingulate cortex (vACC). Finally, resting amygdala-vACC connectivity was associated with sAA reactivity to the trauma reminder.

**Discussion**

Trauma-related symptoms were associated with sympathetic reactivity to a trauma reminder. This result is consistent with previous research that has implicated sympathetic hyperactivity in trauma-related psychopathology (e.g., Bedi & Arora 2007).

Trauma-exposed women showed greater resting connectivity between the amygdala and the vACC, a region implicated in affect regulation. Successful affect regulation has been previously linked to *negative* cortico-limbic connectivity indicative of inhibition of the amygdala by the vACC. Therefore, greater *positive* amygdala-vACC connectivity in trauma-exposed women might indicate diminished inhibition of the amygdala by the vACC.

Finally, resting amygdala-vACC connectivity was associated with sympathetic reactivity to the trauma reminder. This result suggests that enhanced connectivity between regions of the stress-relevant cortico-limbic neurocircuitry might underlie stress system hyperactivity in trauma-exposed people.

Together, these results suggest that abnormal connectivity of the stress-relevant neurocircuitry, possibly indicative of inefficient amygdala inhibition that persists even in the absence of overt stressors, might be a stable marker of trauma exposure and might underlie trauma-related stress system hyperactivity and psychopathology.
INVESTIGATION OF THE BASAL FOREBRAIN - AMYGDALA CIRCUIT DURING FEAR RECALL

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The amygdala is an integral structure for processing learning about threat and anxiety. Previous studies have shown that amygdala communication is synchronized via theta frequency (4-12 Hz) oscillations with the prefrontal cortex during discrimination of threat and safety. In particular, during safety, the prefrontal cortex activates inhibitory circuits in the amygdala, decreasing amygdala output and the behavioral manifestations of fear. It is not clear however, which inputs amplify the prominent theta oscillation seen in the amygdala during fear and anxiety. One candidate structure is the substantia innominata/ventral pallidum (SI/VP) region of the basal forebrain. Cholinergic and inhibitory projections from the SI/VP to the amygdala have been characterized, but it is not well understood whether these inputs partake in encoding aversion in the amygdala. To address this question, we are using the immediate early gene cfos to identify the SI/VP cell populations that become active during recall of fear conditioned stimuli. Furthermore, we are recording local field potentials in the SI/VP and the amygdala during conditioned fear recall. Together, these experiments will identify the dynamic patterns of SI/VP-amygdala communication and the SI/VP cell populations that become active during threat processing. This work contributes to our understanding of the fear learning mechanism by answering the question of whether there is a specific cell population in the basal forebrain that acts as an important input to the amygdala at the time of predictive threat.
Antidepressant and prophylactic ketamine administration differentially impact adult hippocampal neurogenesis

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Ketamine, an N-methyl-D-aspartic acid receptor (NMDAR) antagonist, has been shown to have rapid, long-lasting antidepressant effects in treatment-resistant patients with major depressive disorder (MDD). We have recently shown that ketamine acts as a prophylactic and protects against the development of stress-induced depressive-like behavior in mice, indicating that preventative treatment for mental illness is possible. While there is significant investigation into ketamine’s antidepressant mechanism of action, little research has been done to elucidate ketamine’s underlying prophylactic mechanism. More specifically, whether the prophylactic actions of ketamine are similar or divergent from its antidepressant actions are entirely unknown. Therefore, the goal of the current study is to determine age-dependent molecular signatures of cell-populations governing ketamine’s antidepressant and prophylactic effects. Mice were administered a number of behavioral paradigms as described in Brachman et al., 2016. Post-fixed brains underwent an immunohistochemistry protocol for age-dependent neuronal markers including doublecortin (DCX), a marker of proliferating neurons; calretinin (CR), a marker for immature granule cells; and calbindin (CB), a marker for mature granule cells. The number of DCX⁺ neurons in the DG, including those with tertiary dendrites, were not affected by prophylactic or antidepressant ketamine treatment, nor were they altered by SD. While prophylactic ketamine had no effect on the number of CR⁺ cells in the hilus, antidepressant ketamine treatment increased the number of CR⁺ cells in SD mice to that of control levels. Lastly, antidepressant, but not prophylactic ketamine administration altered CR and CB expression in the HPC. These data suggest that prophylactic and antidepressant ketamine treatment differentially mediate the expression of age-dependent markers of adult hippocampal neurogenesis. The findings further elucidate the neurogenesis based changes associated with MDD pathology and can be used to develop a more targeted therapeutic approach when using ketamine to treat MDD.
A history of major early life stress and social discrimination has been linked to altered acute social stress responses in adulthood. However, there are important individual differences in the size and direction of these effects. Some of this variation can be explained by chronic and developmental stress history and genetic variants of HPA-related genes, including FKBP5. We explored developmental and contextual sources of individual differences in the relationship between stress history and adult responses: perceived status, discrimination experiences, social support, background activity of HPA axis, and genetic variants related to the stress response system. In keeping with other reports, it was predicted that both childhood adversity (ACE) and perceived social discrimination (Lifetime and Daily) would be negatively related to salivary cortisol response and recovery in the Trier Social Stress Test (TSST) of adults. Given recent evidence of interaction between the glucocorticoid receptor and the mitochondrial genome during stress, we investigated the association between methylation of the mitochondrial gene ND6 in buccal cells and ACE score as an exploratory measure. Participants (N= 90; ages 18-66) were recruited from the greater Boston area. Percent change in cortisol from baseline in response to the TSST was blunted in the High ACE group ($p<.05$, $\eta^2 = .22$), an effect facilitated by elevated baseline cortisol in the same group ($p< .01$, $\eta^p = .24$). There was a significant association between baseline cortisol and Lifetime Discrimination ($p <.02$, $r_s = .22$), Daily Discrimination ($p = .044$, $r_s = .23$), and FKBP5 rs1630807 genotype ($p<.01$, $r_s = .28$). Principal component analysis was used to further explore the relationship between these variables and two factors emerged (eigenvalues above 1): Factor 1 consisted of ACE scores and both discrimination measures (.4 or above) and Factor 2 consisted of FKBP5 genotype. Stepwise multiple regression analysis indicated that Factor 1 and FKBP5 genotype predicted baseline cortisol levels, (F(2,86)= 8.52, $p<.001$), $R^2 = .43$. Average methylation levels of ND6 in the Low ACE group ($Mdn = 1.22$) were significantly lower than the High ACE group ($Mdn = 1.40$), $U = 670.50$, $z = -2.05$, $p <.05$, $r = .23$. These findings suggest multiple contributions across psychological, genetic, and social domains to vulnerability and resilience in hypothalamic-pituitary-adrenal axis regulation which should be explored further. This study extends recent findings that environmental pollutants are associated with mitochondrial genome methylation to include associations with social stress experiences.

Childhood Adversity, Discrimination Experiences, and FKBP5 rs1630807 Genotype in HPA Axis Regulation and ND6 Methylation
Chronic social defeat stress induces dynamic changes in myelination and persists after cessation of stress

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The importance of glial cells in clinical depression is being increasingly recognized in humans, due to advances in brain imaging and histological studies on postmortem tissue and to studies in animal models. Reduced oligodendrocyte numbers and downregulated oligodendrocyte transcripts have been reported in specific brain regions of postmortem tissue from patients with major depressive disorder. However, it remains unclear whether depression induces transient or long-lasting adaptations in myelination and oligodendrocyte function. Here we examine the ultrastructural and transcriptional changes occurring in murine oligodendrocytes after chronic social defeat stress, an experimental model of depression. Mice displayed depressive-like phenotype (i.e. susceptible) following chronic social defeat stress. Transcriptomic analysis of differentially expressed genes in the medial prefrontal cortex (mPFC) of defeated mice compared to controls, identified the functional category “myelination” as the top functional ontology term in susceptible- but not in resilient mice (i.e. defeated mice that did not show any behavioral response to stress). These findings were paralleled by the different levels of myelination and oligodendrocyte-specific transcripts detected in two critical regions involved in cognitive function and the reward response: the mPFC and nucleus accumbens (NAc) in susceptible and resilient mice. Impaired myelination was associated with aberrant chromatin structures of oligodendrocytes in defeated mice and was consistent with the persistence of transcriptional changes after cessation of social defeat stress. Together, these results indicate that social stress induces dynamic changes in myelination in the mPFC and NAc that are long-lasting after the withdrawal of stressful stimuli.
Anxiety is associated with an attentional bias towards threatening information in the environment (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & Van Ijzendoom, 2007; Cisler & Koster, 2010). This anxiety-related threat bias (TB) is commonly measured using the reaction time-based dot probe task that tracks the speed of responding to cues replacing either a neutral or threatening facial expression. A simple modification of the dot probe task, termed Attention Bias Modification Training (ABMT), is a promising new treatment for anxiety (Dennis, Egan, Babkirk & Denefrio, 2016; Hakamata et al., 2010). However, recent inconsistencies and null findings in ABMT studies have highlighted the role of individual differences in ABMT efficacy (Clarke, Notebaert, & MacLeod, 2014; MacLeod & Clarke, 2015). One such individual difference is engagement or a person’s level of interest in and motivation to complete a specific task (Matthews et al., 2002). In the present study, anxiety and positive and negative affect were assessed to examine their impact on engagement, then TB was measured at three timepoints: arrival, following a brief stressor, and again following one session of ABMT in a large college-aged sample (N= 109). Self-reported engagement was assessed following each TB assessment and post-ABMT to test the hypotheses that 1) less anxiety would predict increased engagement and that 2) increased engagement would predict better ABMT efficacy. Engagement decreased across all timepoints, \( F (3, 306) = 39.699, p = .001 \). However, as predicted, increased engagement at each timepoint was associated with reduced trait anxiety (\( p’s < .02 \)), state anxiety (\( p’s < .002 \)), and increased positive affect (\( p’s < .001 \)) prior to ABMT. Furthermore, increased engagement in ABMT was related to reduced difficulty disengaging from threat post-ABMT \( r(108) = -.194, p = .045 \). The current findings suggest the importance of boosting engagement in order to optimize ABMT efficacy, and the potential impact of mood on engagement even in a non-anxious sample. Future research should focus on developing methods aimed at improving mood and engagement in order to bolster ABMT efficacy.
Future-Oriented Fantasies & Depressive Symptoms: Indirect Relationship Through Brooding

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**Background.** A positive outlook about the future is generally associated with psychological well-being. However, there is increasing evidence that not all types of positive future-oriented thinking are beneficial. One form, in particular – indulging in positive fantasies about a desired future outcome – has been found to exacerbate negative mood-related outcomes such as depressive symptoms. The present study examined rumination as a cognitive mechanism in this relationship.

**Methods.** Undergraduates from a public college in the northeastern U.S. took part in two study sessions, averaging six weeks apart. At baseline, participants completed the Future Experiences Questionnaire (FEQ), in which they were asked to imagine a desired future event, and self-report measures of depressive symptoms (BDI-II), brooding and reflective rumination (RRS). At follow-up, participants again completed the FEQ and the BDI-II. Independent raters coded participant narratives on the FEQ for valence and degree of engaging in positive future-oriented fantasies.

**Results.** Engaging in a positive fantasy about the future was associated with the brooding subtype of rumination but not with reflection at baseline. There was an indirect relationship between fantasies at baseline and depressive symptoms at 6-week follow-up through brooding at average and high levels of independently rated positivity of the fantasy when levels of fantasizing were consistent or increased over time, but not when they were not consistent or when positivity was low. Engaging in future-oriented fantasies was indirectly associated with perceived difficulty anticipating likely positive future outcomes when indulging was consistent, or inconsistent and level of fantasy positivity was high or average.

**Conclusion.** Consistent engagement in positive fantasies about the future may be maladaptive to the degree that it resembles brooding.
Background: Standard antidepressant treatments often take weeks to reach efficacy, and for many patients, do not work. Ketamine, an N-methyl-D-aspartate (NMDA) antagonist, has been shown to be a rapid-acting antidepressant. Here, we investigated whether ketamine’s efficacy is dependent upon adult hippocampal neurogenesis in concert with the GluN2B subunit of the NMDA receptor (NMDAR).

Methods: We utilized a transgenic strategy in which we ablated the GluN2B subunit of the NMDAR in 6-week-old adult-born hippocampal neurons in mice. Mice with or without the GluN2B subunit were administered either saline or ketamine (n=5-6 mice per group). We also used two additional models to test the effects of ablation of 6-week-old adult born hippocampal neurons: a GFAP-thymidine-kinase (TK) genetic model and x-irradiation. We assessed the antidepressant effects of ketamine in the forced swim test (FST), contextual fear conditioning (CFC), and novelty suppressed feeding (NSF) paradigms. The effects of drug treatment and context were analyzed using an ANOVA, using repeated measures where appropriate.

Results: Consistent with previous reports, we found that ketamine had antidepressant effects in the FST \( [p < 0.04] \), pro-cognitive effects in CFC \( [p < 0.01] \), and anxiolytic effects in NSF \( [p < 0.03] \) in mice with
GluN2B-containing NMDARs. Deletion of GluN2B from 6-week-old adult-born neurons occluded all of these effects [FST: $p = 0.15$, CFC: $p = 0.89$, NSF: $p = 0.84$]. Whole cell ablation in the genetic or x-irradiation models prevented some of ketamine’s effects.

**Conclusion:** These results suggest that 6-week-old adult-born hippocampal neurons expressing GluN2B modulate ketamine’s rapid-acting antidepressant properties.
Ginkgolide Treatment Enhances Schwann Cell Myelination and Differentiation

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Myelin sheath is a membrane that surrounds neuronal axons, facilitating the transmission of nerve impulses. In the peripheral nervous system (PNS), the specialized type of glial cell that insulate nerve fibers is known as a schwann cell (SC). Although the exact mechanism behind myelination remains illusive, our lab suspects that the cytoskeleton plays an important role in myelination processes. Microtubules (MT), a component of the cytoskeleton, have been shown to go through changes during SC maturation and play an important role in myelin protein trafficking. MTs are heterodimer proteins composed of α-tubulin and β-tubulin, which polymerize together forming long cylindrical tubes. In order to understand how MT dynamics affects myelination, we used a MT modifying drug (GA) that inhibits the removal of the amino acid tyrosine from α-tubulin (detyrosination). This inhibition is known to promote instability in MTs. Using several cell culture techniques, we have found that the GA drug increases the amount of myelin segments in SC neuronal co-cultures, while also promoting myelin protein expression in mature SCs.
Physiological Correlates of Infant Memory for a Stressful Social Event at 4-Months.

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Research on infant memory is typically based on non-stressful stimuli such as novelty- or imitation-paradigms, but our understanding of infant memory for a social stressor is limited. To fill this gap, the present study uses the Face-to-Face Still Face (FFSF) procedure, a paradigm that elicits a well documented behavioral and physiological stress response in infants.

Infants in the experimental condition (n=35) were exposed to the FFSF on two consecutive days, while the control group (n=33) completed a time-matched play-session on day 1 and the FFSF on day 2.

Changes in behavior, heart rate (HR) and salivary cortisol on both days were evaluated.

Infants in the experimental condition showed a significant increase in HR on day 2, compared to controls. Additional regression analysis indicated that for both conditions overall arousal during day 1 (FFSF procedure or control play-session) predicted baseline HR on day 2. The change in infant HR was independent from maternal HR which did not differ between day 1 and day 2 or between groups. The groups did not differ in behavior or salivary cortisol on day 2.

Findings suggest that a previous stressful experience may elicit a physiological response in infants 24 hours later. The results could have implications for further research on stressful and traumatic events in early childhood.
Neurocognitive Assessment of Emotional Context Sensitivity

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Sensitivity to emotional context is an emerging construct for characterizing adaptive emotion regulation, an ability that promotes resilience in the face of stress, but few measurement approaches exist. The current study combined behavioral and neurocognitive measures to assess context sensitivity in relation to self-report measures of adaptive emotional flexibility and well-being. Sixty-six adults completed an emotional go/no-go task using happy, fearful, and neutral faces as go and no-go cues, while EEG was recorded to generate event-related potentials (ERPs) reflecting attentional selection and discrimination (N170) and cognitive control (N2). Context sensitivity was measured as the degree of emotional facilitation or disruption in the go/no-go task and magnitude of ERP response to emotion cues. Participants self-reported on emotional flexibility, anxiety, and depression. All participants evidenced emotional context sensitivity, such that when happy faces were go stimuli, accuracy improved (greater behavioral facilitation), whereas when fearful faces were no-go stimuli, errors increased (disrupted behavioral inhibition). These indices predicted emotional flexibility and well-being: greater behavioral facilitation following happy cues was associated with lower depression and anxiety, whereas greater disruption in behavioral inhibition following fearful cues was associated with lower flexibility. ERP indices of context sensitivity revealed additional associations: greater N2 to fear go cues was associated with less anxiety and depression, and greater N2 and N170 to happy and fear no-go cues, respectively, were associated with greater emotional flexibility and well-being. Results suggest that pleasant and unpleasant emotions selectively enhance and disrupt components of context sensitivity, and that behavioral and ERP indices of context sensitivity predict flexibility and well-being.
Fetal programming by exposure to Superstorm Sandy and stress diathesis on infant temperament at 6 months old

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Background & Significance: The prenatal environment is thought to prepare the developing fetus for conditions in the postnatal world. Animal models show that prenatally stress was associated with HPA-axis reactivity and anxiety behavior. However, less is known about how these findings translate to human models. Capitalizing on Superstorm Sandy disaster, the study attempts to examine the role of fetal programming in infant temperament at 6 months of age.

Methods: The study (n=380) examined the role of fetal programming in relation to infant temperament at 6 months of age. It further evaluated the stress-diathesis theory by evaluating the direct effects of prenatal exposure to Superstorm Sandy and mothers’ history of prior trauma on infant temperament. Twelve areas of trauma consistent with the Diagnostic and Statistical Manual (DSM-IV) criteria for PTSD from the Posttraumatic Diagnostic Scale (Foa, et al., 1997) were used to measure mother’s pre-pregnancy history of trauma. The index of in-utero Superstorm Sandy exposure was generated based on the child’s date of birth and the date of Superstorm Sandy. Infant temperament was measured by the Infant Behavior Questionnaire-Revised (Gartstein and Rothbart 2003), which produces 14 subscales of temperament. To account for potential correlations among those subscales, we used a multivariable general linear model (GLM) analysis. First, we tested the main effects of in-utero exposure to Superstorm Sandy and mother’s pre-pregnancy history of trauma on infant temperament. This multivariate GLM was followed by the same model with an additional interaction term between the two on infant temperament. In all models, a priori determined potential confounders, including sex of the child, maternal age, education, marital status, race, parity, and smoking during pregnancy, were included for statistical adjustment.

Results: Infants with in-utero exposure to Superstorm Sandy, as compared to infants without, displayed greater fear (3.48 vs. 3.00, p=.04), lower soothability (5.02 vs. 5.51, p=.01), slower recovery from distress (4.77 vs. 5.11, p=.05), greater perceptual sensitivity (4.78 vs. 4.29, p=.06), and greater impulsivity (5.61, vs. 5.17, p=.03). Infants of mothers with prior trauma compared to those without exhibited greater sadness (3.67 vs. 3.28, p=.04) and greater soothability (5.42 vs. 5.11, p=.04). Furthermore, there were significant interaction effects between in-utero exposure to Superstorm Sandy and mother’s pre-pregnant trauma on fear (p=.05), smiling and laughter (p=.05), high pleasure-seeking (p=.004), low pleasure-seeking (p=.05), soothability (p<.0001), perceptual sensitivity (p=.04), impulsivity (p=.08), and vocal reactivity (p=.03). Notably, when mothers had pre-pregnancy trauma, in-utero exposure to Superstorm Sandy had greater negative impacts on infant temperament.

Conclusion: Prenatal exposure to maternal stress, caused by Superstorm Sandy devastation had adverse influences on infant temperament, particularly in relation to decreased emotion regulation and lower effortful control. Although fetal programing by in-utero exposure to stress on infant temperament is evident, maternal history of pre-pregnancy trauma exacerbates the negative impact the exposure on
temperament, providing further evidence for the stress-diathesis model in action. While the stress-diathesis model is a psychological theory, further biological understanding of the underlying mechanism by which fetal programming by prenatal stress exposure could magnify its tax on the offspring is needed.
Amygdala PKMζ involvement in the emergence of threat learning in infant rodents

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Early childhood attachment to a caregiver is critical for the insurance of long-term infant care and protection. Previous studies in rodent models have identified a sensitive period prior to post-natal day 10 (PN10), during which pups display heightened preference learning accompanied by decreased aversion learning (Sullivan et al., 2000). Exposure to a fear-conditioning paradigm using paired odor-shock results in persistent preference for the paired odor in pups trained during this sensitive period while older pups show an aversion. This switch in the learned response to an aversive stimulus is mediated through activation of the amygdala by corticosterone (CORT), which is typically very low in pups during the sensitive period and increases at PN10 (Moriceau & Sullivan, 2006). Consolidation of aversive learning in adults requires the protein PKMζ (Serrano et al., 2005; 2008). Through a combination of pharmacological and naturalistic approaches, here we show that the infant’s environment can regulate the emergence of adult-like amygdala consolidation processes through changes in CORT. Specifically, we show that amygdala PKMζ is increased in older pups that can learn threat (>PN10). In addition, experimentally-induced increases in CORT through CORT injections and exposure to maternal alarm odor during conditioning have been shown to prematurely engage amygdala-dependent threat learning in pups younger than PN10 (Debiec & Sullivan, 2014). Here we show that these same groups show increases in amygdala PKMζ. In all groups that learned threat, intra-amygdala infusions of the PKMζ inhibitor ZIP prevented expression of learning. Additional markers associated with consolidation, including GluA1, GluA2, PSD-95, and BDNF were also modulated in the amygdalae of rats that learned threat. A causal role for CORT in these effects was further demonstrated through pharmacological inhibition of CORT in PN12 rats during learning; these rats showed no evidence of threat learning or concomitant changes in molecules involved in memory consolidation in the amygdala. Taken together, these data indicate that environmental control of threat learning in infants involves changes in stress hormones to produce adult-like amygdala function.
Placental MAOA Expression Mediates Prenatal Stress Effects on Child Temperament

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Prenatal stress has been linked to suboptimal child temperament development. Stress in utero has been linked to down regulation of monoamine oxidase A (MAOA) gene expression. MAOA breaks down key neurotransmitters in the brain and lower expression of MAOA gene contributes to emotional and behavioral problems. Capitalizing on ongoing longitudinal study, data from ninety-five dyads were analyzed to evaluate whether MAOA expression mediates the association between prenatal stress and infant temperament. Prenatal stress was defined by exposure to Superstorm Sandy during pregnancy. MAOA expression was analyzed from the placental tissues. 14 domains of temperament were measured by Infant Behavior Questionnaire (IBQ-R). Independent sample t-tests revealed that children whose mothers were exposed to Superstorm Sandy had lower MAOA expression, \( t(93) = 2.55, p = .01 \), and lower Smile and Laughter domain scores, \( t(93) = 2.96, p = .004 \). MAOA expression was positively associated with Smile and Laughter, \( r = .29, p = 004 \). Finally, mediation analysis using bootstrapping procedure confirmed that MAOA expression partially mediated the relationship between Superstorm Sandy exposure and Smile and Laughter (\( \beta = -.11, 95\% \text{ CI} = [-.37, -.003] \)). Sandy exposure in utero was a significant predictor for Smile and Laughter (\( \beta = -.54, p = .004 \)) and remained significant after controlling for MAOA expression (\( \beta = -.43, p = .02 \)). These findings suggest that stress experienced in utero could shape the temperamental development epigenetically through alteration of MAOA gene expression.
Individual differences in trait emotionality, emotion regulation, and myoelectrical gastric reactivity

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Background: Among individuals with affective disorders, emotional distress is frequently associated with gastrointestinal symptoms, suggesting that emotion-related deficits in reactivity and regulation may be linked to altered gastric activity. Yet the role of emotionality in gastrointestinal symptoms has not yet been well characterized using psychophysiological measures. In the present study, we used electrogastrography (EGG), a non-invasive technique for recording smooth muscle activity of the stomach, to investigate associations between individual differences in trait emotionality/regulation and gastric reactivity to emotional stimuli.

Methods: Participants (n = 41) were part of a larger multimodal study of emotion regulation in an unselected sample of young adults. Participants completed trait self-report measures of emotionality (responses to threat and reward) and emotion dysregulation (worry; rumination; avoidance). They also viewed neutral and emotionally salient film clips designed to elicit fear, sadness, and disgust during a lab-based stressor task. EGG was recorded via two electrodes placed on the abdomen, and analyzed for perturbed gastric activity (bradygastria; tachygastria) or normal gastric rhythm (normogastria).

Results: At baseline, tachygastria (i.e., overly rapid activity) was moderately correlated with threat sensitivity (r’s = .35 to .37) and rumination (r = .32), p’s < .05. Tachygastria during neutral was positively associated with reward sensitivity (r = .31), but negatively associated with threat sensitivity (r = -.32). Bradygastria (i.e., slowed activity) during fear was negatively correlated with trait sensitivity to both threat and reward (r’s = -.32 to -.43). No other significant relationships were observed between self-report and dysrhythmia during fear, sadness, or disgust. However, participants who reported higher sensitivity to reward showed more normogastria during sadness (r = .35).

Conclusion: Overall, trait emotionality, rather than emotion dysregulation, appeared more closely tied to perturbed gastric activity during fear and sadness conditions, as well as during neutral and baseline conditions. The present findings suggest that trait-level individual differences in threat and reward sensitivity are associated with gastric activity, both at baseline and during negative emotional contexts. We next plan to incorporate state-level emotion measures and other psychophysiology (heart rate, skin conductance), to further delineate core processes that may contribute to common somatic symptoms in affective disorders.
PTSD symptoms and daily affect vary with menstrual phase in trauma-exposed women

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Introduction: More than 50% of the US population will experience a traumatic event in their lifetime (Kessler et al., 2005), and some will develop enduring trauma-related symptoms (e.g., re-experiencing the traumatic event, heightened physiological arousal, negative alterations in cognitions and mood). Given that women are twice as likely to be diagnosed with PTSD following trauma compared to men, it is important to learn more about physiological processes unique to women that might contribute to increased vulnerability for post-trauma symptoms. One potential risk factor for symptoms is fluctuations in the hormone estradiol. Throughout the lifespan, changes in circulating estradiol levels are associated with changes in affect, and risk for affective disorders increases when estradiol levels drop during the perimenopause, menopause and postpartum periods (e.g., Vesga-Lopez et al., 2008). In naturally-cycling, premenopausal women, regular fluctuations in estradiol across the menstrual cycle are also associated with variation in stress and affect, as well as a range of psychological symptoms (e.g., Walder et al., 2012). In women who have experienced trauma, cyclic variation in estradiol might therefore be associated with aversive affective experiences, including trauma-related symptoms. This pilot study explores daily affective experiences during a 10-day period spanning the early follicular (low estradiol) and late follicular (high estradiol) phases.

Hypothesis: We hypothesized that trauma-exposed women would report more aversive daily affective experiences and greater PTSD symptom severity during the early follicular versus late follicular phase.

Method: Ten naturally-cycling women completed a clinical interview to assess trauma exposure and menstrual cycle regularity. Following this lab session, participants completed daily questionnaires during a 10-day ecological momentary assessment (EMA) period spanning the early follicular (cycle days 2-6) and late follicular (cycle days 7-11) phases. Participants rated current affect on the dimensions of valence (unpleasant to pleasant) and arousal (low to high) at five daily assessment points: after waking, before bed and at three variable daytime points. At each daytime assessment point, participants received a text message instructing them to complete a questionnaire. Evening questionnaires also assessed PTSD symptoms experienced that day.

Results: Preliminary analyses show that, partially consistent with our hypothesis, valence ratings were less positive during the early versus late follicular phase, \( t(9) = 2.34, p = 0.044, d = 0.35 \), but arousal ratings did not differ by phase. There was also a trend towards greater variability (i.e., larger standard deviations) in valence ratings
during the early follicular phase, \( t(9) = 2.09, p = 0.066, d = 0.35 \). Additionally, although total PTSD symptom severity did not differ by menstrual cycle phase, participants reported greater severity of D Cluster PTSD symptoms (negative alterations in cognitions and mood) during the early versus late follicular phase, \( t(9) = 2.74, p = 0.023, d = 0.37 \).

**Conclusions:** These preliminary results suggest that low estradiol menstrual cycle phases might be associated with less pleasant and more variable affect, as well as more trauma-related negative cognitions. Additionally, our results support the utility of ecological momentary assessment in capturing menstrual cycle variability in affective fluctuations and trauma-related symptoms.
Mindfulness as resilience: buffering the deleterious effects of rumination on health behaviors

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Avoiding adaptive health behaviors such as a healthy diet and exercise contribute to the development of a number of chronic illnesses. Mindfulness, or focusing one’s awareness on the present with non-judgment, has consistently been linked to engaging in more adaptive health behavior patterns, and is being used increasingly in health behavior change interventions (e.g., Black, Johnson, Sussman, & Milam, 2012; see Grossman, Niemann, Schmidt, & Walach, 2004, for a review). However, very few studies have examined the potential mechanisms of change of how mindfulness increases adaptive health behaviors (Salmoirago-Blotcher et al., 2013). Decreased rumination (e.g., Riley, 2015), increased self compassion (e.g., Neff, 2010), and increased self distancing (e.g., Ayduk & Kross, 2010) have been implicated as possible mediators; this study aims to empirically explore these constructs as mediators of the mindfulness to health behavior link.

Method: 157 college students (67.6% female, mean age 18.9 years, 64.9% Caucasian) completed an 11 day daily diary study and completed a battery of questionnaires each evening that measured that day’s mindfulness (Cognitive and Affective Mindfulness Scale; Feldman et al., 2007), diet (Dietary Screener Questionnaire; NCI, 2014), exercise (Godin Leisure-Time Exercise Questionnaire; Godin & Shephard, 1985), rumination (The Ruminative Styles Questionnaire, Ruminative Responses Subscale; Nolen-Hoeksema & Morrow, 1991), self compassion (Self Compassion Scale; Neff, 2010), and self distancing (item, Ayduk & Kross, 2010).

Results and Discussion
Multilevel modeling in MPlus revealed a significant relationship between daily mindfulness and daily exercise (β = .097, p = .048) and a trending relationship between mindfulness and fruit (β = .085, p = .054) and vegetable (β = .060, p = .092) intake at the daily, or within person, level. Multilevel mediation analyses indicated that rumination was a trending mediator of the mindfulness to exercise relationship (β = .084, p = .092), but not the mindfulness to fruit and vegetable intake relationship. Self compassion was not a significant mediator of the mindfulness to health behavior relationship. Self distancing was a significant mediator of the mindfulness to exercise (β = .121, p = .032) and fruit (β = .103, p = .047) and vegetable (β = .094, p = .050) relationships. Mindfulness acts on health behaviors through increased self distancing on the daily level. Additionally, daily mindfulness may act on exercise through decreased daily rumination. Self compassion may not be a mediator of mindfulness to increased health behavior engagement, at least for exercise and fruit and vegetable intake on the daily level. Mindfulness is a multifaceted construct, which is utilized for health behavior change in intervention research (e.g., MBSR, Kabat-Zinn, 2003), and it is important to know more about the mechanisms of change,
so that targeted outcomes can be optimized. If self distancing is the most potent component of mindfulness for behavior change, as has been implicated in previous research (Ayduk & Kross, 2010), it may be important to examine how interventions can maximize self distancing. Limitations include a homogeneous sample of college students that may not be generalizable to the general population. More research is needed to further relationships among these variables and other possible mediators.
In humans, stress has been shown to precipitate psychological disorders such as major depressive disorder (MDD) and anxiety. MDD is a highly prevalent disorder creating a large economic burden. The effect of stress on the induction of depression and anxiety symptoms is not homogeneous—some people are resilient to the effects of stress and do not display long-term changes in mood, while others are more susceptible. Specifically, women are also more likely to be diagnosed with depression than men. Further, clinical evidence suggests that active coping strategies improve the individual response to stress and mitigate the development of depressive disorders. Despite these clinical findings there is no clear neurophysiological understanding of the influence of sex and coping strategy on stress susceptibility.

Chronic social defeat (CSD) in male mice has been used as a model for stress-induced depression and anxiety. This treatment induces two behavioral phenotypes: resilient mice, which do not develop long-term increases in depression measures in response to stress, and susceptible mice, which experience a long-lasting increase in anxiety and decrease in sociability.

Previous work using the CSD model has shown long-term changes in firing patterns in midbrain dopamine neurons, and that these effects are different depending on the stress-induced phenotype of the mouse. The resilient phenotype has lower, control-like firing rates in the ventral tegmental area (VTA), while susceptible have increased firing rates. Although the CSD model induces MDD-like symptoms in mice, the social defeat stressor has been criticized as being overly artificial; extreme social defeat is not always a precursor for development of MDD.

We have developed a model of stress-induced MDD called repeated variable social stress (RVSS), where mice are subjected to ten days of variable stressors, including a predator odor in their home cage, overcrowding, home cage instability, and witness-restraint. These stressors induce behavioral and physiological changes similar to those seen in the chronic social defeat model, but using more ethologically relevant stressors.

The RVSS model allows for characterization of individual coping strategies in response to the various stressors. We have found that active coping strategies, such as approaching and burying an aversive odor, correlate to the individual’s subsequent phenotype: active copers are more social and less anxious than passive copers.
Importantly, the RVSS model can be used in both male and female mice, allowing for the characterization of the contribution of sex and estrus cycle to resilience to stress and coping strategy. We found that females are more likely to adopt a passive coping strategy than males, and that female mice generally have increased baseline firing rates in the VTA. Together, these findings may indicate a potential mechanism for the increased susceptibility of females to depression.
Using Electronic Health Records Data to Evaluate the Association Among Biological, Social and Nutritional Status on Adolescent Pregnancy Rates, Physiology and Birth Outcomes

We created a community-academic partnership that included New York City Community Health Centers (n=4) and Hospitals (n=4), The Rockefeller University, The Sackler Institute for Nutrition Science and Clinical Directors Network (CDN). We used the Community-Engaged Research Navigation model to establish a multisite de-identified database extracted from electronic health records (EHRs) of female adolescents aged 12-21 years (January 2011 – December 2012) and their offspring through 24 months of age. These patients received their primary care between 2011-2015. Clinical data were used to explore possible associations among specific measures. We focused on the preconception, prenatal, postnatal periods, including pediatric visits up to 24 months of age. The de-identified database contains electronic health records (EHR) information from female adolescents aged 12-21 years (January 2011-December 2012) and their offspring through 24 months of age. These patients received their primary care at the 8 partnering Community Health Centers (CHCs) and Community Hospitals between 2011-2015. The preliminary analysis included all female adolescents (n=84,714) and a subset of pregnant adolescents with offspring data available (n=2,917). Patients were mostly from the Bronx; 45% of all adolescent females were overweight (22%) or obese (23%) and showed higher systolic and diastolic blood pressure, blood glucose levels, hemoglobin A1c, total cholesterol, and triglycerides levels compared to normal-weight adolescent females (p<0.05). There was a statistically significant association between the BMI status of mothers and infants’ birth weight, with underweight/normal-weight mothers having more low birth weight (LBW) babies and overweight/obese mothers having more large babies. The odds of having a LBW baby was 0.61 (95% CI: 0.41, 0.89) lower in obese compared to normal-weight adolescent mothers. The risk of having a preterm birth before 37 weeks was found to be neutral in obese compared to normal-weight adolescent mothers (OR=0.81, 95% CI: 0.53, 1.25). Preliminary associations are similar to those reported in the published literature. This EHR database uses available measures from routine clinical care as a “rapid assay” to explore potential associations, and may be more useful to detect the presence and direction of associations than the magnitude of effects. This partnership has engaged community clinicians, laboratory and clinical investigators, and funders in study design and analysis, as demonstrated by the collaborative development and testing of hypotheses relevant to service delivery.
Folic acid Supplementation alters the GABAergic system possible epigenetics modification to the stress circuitry.

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The Hypothalamus-pituitary- adrenocortical (HPA) axis regulates stress. This fundamental function is believed to be in part modulated GABAergic interneurons. Dysfunctions in this mechanism leads to many neurological disorder. Glutamic acid decarboxylase (GAD) the enzyme requires to covert glutamate into GABA in the circuitry of stress. GAD is also a transcription factor that binds to the GABA promoter to increase transcription of the GABA gene. Micronutrients are essential vitamins and minerals that ensure normal metabolism and growth, and have occupied important position in human health and nutrition in recent years. A number of essential micronutrients were identified that are critical for the human health and growing newborns. Among this folate is considered as a public health success story in effectively reducing incidences of neural tube defects (NTDs) in newborns. In 1998, to decrease the incidence of NTDs a mandate to fortify all grain containing foods with folic acid (FA) was made. FA is necessary for proper neural tube closure and development. It is believed folic acid interacts with DNA methyltranferase to induce neuronal cell differentiation. However how much folic acid is required for proper neural tube closure and at what level it becomes toxic to a developing embryo has not been reported. In order to identify influence of FA in a neuronal system and possibly in the circuitry of stress, research we reported the alteration of the GABAergic system in SH-SY5Y cells. GABAA receptor subunit beta 1 was up-regulated with increasing FA concentration. We found glutamic acid decarboxylase (GAD) 65/67 and GABRB3 were down regulated with folic acid concentration at mRNA (qRT-PCR), protein levels Western blot and immunofluorescence analysis (confocal microscopy. All these proteins and respective genes play a crucial role in the regulation of stress and further behavioral research is warrant to determine if these changes at the molecular level affect response to stress.
The Effects of Maternal and Prenatal Stress and Trauma on HPA-Axis Gene Expression in Children

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Research has shown that women with a trauma history may continue to manifest disrupted physiological states during pregnancy, which could in turn disrupt the programming of their fetus’ developing HPA-axis system. Additionally, it has been found that maternal stress during pregnancy can put the child at risk for HPA-axis alterations in-utero, a concept known as fetal programming. However, little is known about the stress-diathesis model in action. There may be differences in this process for children of mothers with a trauma history compared to mothers who were exposed to adversity brought by natural disaster, while pregnant. The “Stress in Pregnancy” study has followed women ($M=27.54$ years; 88.7\% ethnic minorities) during pregnancy and as their children develop up to 4 years old ($N = 701$; 48.4\% female). A portion of these mothers were exposed to Hurricane Sandy during pregnancy, allowing for the unique opportunity to investigate the objective measure of stress brought by Sandy on offspring biological, behavioral and emotional development. Based on reported trauma history and Hurricane Sandy exposure, dyads were classified into one of four groups: (1) neither trauma history (c.f., trauma history) nor exposure to Hurricane Sandy in-utero (c.f., Sandy-exposure) ($n = 309$), (2) trauma history alone ($n = 124$), (3) Sandy-exposure alone ($n = 172$), and (4) both Sandy-exposure and trauma history ($n = 96$). We hypothesized that the four groups would differ in HPA-axis gene expression, as measured by epigenetic regulation of the HPA-axis genes ($MAOB$, $ATP1A1$, and $SLC6A4$), expressed on the placenta. Specifically, we expect to see synergistic increase in the HPA system gene alterations in children of mothers with a trauma history who also experienced Sandy during pregnancy. We also hypothesize that we will observe the HPA-axis dysregulation in a dose-response fashion where children of mothers with two types of traumas had the greatest dysregulation and children of mothers with neither had the lowest dysregulation. A one-way analysis of variance was conducted to evaluate if HPA-axis alterations differed among the four groups. Main effects were found between the groups on $MAOB$ ($p < .001$), $ATP1A1$ ($p < .001$), and $SLC6A4$ ($p < .001$) expression. Specifically, children of mothers exposed to Sandy and children of mothers with a trauma history and Sandy-exposure had lower $MAOB$ and $SLC6A4$ expression than children of mothers with a trauma history alone and than mothers with neither trauma history nor Sandy-exposure (all $p < .05$). Children of mothers who were exposed to Sandy and children of mothers with a trauma history and Sandy-exposure had lower $ATP1A1$ expression than children of mothers with neither trauma history nor Sandy-exposure (all $p < .05$). Prior research has demonstrated that such gene expression alterations can be viewed as biological resilience/vulnerability and can differentially impact individuals for their risk for subsequent onset of psychopathology. These results provide further evidence for the fetal programming model, specifically demonstrating a dose-response relationship between number of maternal traumas and offspring HPA-axis dysregulation, and may have implications for future neurodevelopmental consequences for these children.
The Role of Adverse Childhood Experiences and Traumatic Life Events in Reactions to Loss in a Diverse Sample of College Students

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BACKGROUND: Little is known about differences in reactions to grief among diverse young adult populations in urban settings, including the role trauma history may play in these reactions. Studies confirm that approximately 22% to 30% of college undergraduates in the U.S. are in the first twelve months of grieving a death of a family member or a friend (Balk, 2008). The research on effects of college students experiencing grief has found to impair immune system, sleep, concentration, memory and increase feelings of fear, confusion, anger, guilt or loneliness (Kris et al., 2006; Lindemann, 1944; Saldinger & Cain, 2005). Furthermore, trauma history, such as adverse childhood experiences, has been found to impact physical and mental health outcomes, such as autoimmune disease, increased risk of lung cancer, chronic obstructive pulmonary disease, depression or suicidality (Dube et al., 2009, 2001; Brown et al., 2010; Anda et al., 2008; Chapman et al., 2004). In this exploratory study, we were interested in understanding how reactions to grief may differ by demographic characteristics and trauma history among recently bereaved college students in an urban population.

METHODS: In this New York City-based study, 489 undergraduate students at a diverse urban campus completed an online survey that included sociodemographic items, items about social support, measures of trauma exposure (Life Events Checklist, Adverse Childhood Experiences) and reactions to grief (Hogan Grief Reaction Checklist) among those who had experienced a death of a loved one in the past two years.

RESULTS: A statistically larger proportion of students who identified as Black reported a recent death (37.9%) as compared to non-Black students (25.4%), $X^2(1, N = 489) = 6.28, p < .05$. A total of 137 individuals (28% of the sample) reported having experienced the death of a loved one within the past two years. Those who experienced a recent death had significantly higher scores on the traumatic life events scale ($M = 3.12, SD = 2.22$) than those who had not, ($M = 2.64, SD = 2.05$), $t(232) = -2.16, p = .032$. When looking at students’ reactions to grief on the six dimensions measured by the Hogan Grief Reaction Checklist (despair, panic behavior, personal growth, blame and anger, detachment, and disorganization), there were no significant differences by race/ethnicity. Among the participants who had experienced loss, gender, social
support, a close relationship with the deceased, recentness of the death, history of traumatic life events and adverse childhood experiences were significantly associated with grief reactions.

**CONCLUSION:** These findings suggest that, while prevalence of bereavement may vary by racial/ethnic group, reactions to death may be more likely correlated with adverse childhood experiences and traumatic life events. Though trauma exposure was generally associated to poorer outcomes, traumatic life events was also associated with greater personal growth as a result of bereavement. The finding that social support impacts grief reactions suggest implications for intervention strategies for this population. Further research with larger sample necessary to establish relationship between adverse childhood experiences and traumatic life events on grief reactions.
Mapping the Prefrontal Cortex- Basal Forebrain- Amygdala Circuit

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A comprehensive understanding of circuit connectivity between structures that partake in aversive learning and memory is crucial for developing targeted treatments for anxiety disorders. Much work has described strong reciprocal connections between the prefrontal cortex and the amygdala, as well as the basal forebrain and the amygdala. Direct projections from the infralimbic portion (IL) of the prefrontal cortex to the amygdala are functionally important during discrimination of safety. However, the IL may influence amygdala activity during safety discrimination indirectly, via its projections to the basal forebrain. Basal forebrain inputs to the amygdala are thought to underlie stimulus salience and attentional processing. Thus, the IL could encode safety in the amygdala indirectly, via silencing the basal forebrain. To investigate this possibility, we are using retrograde and anterograde tracing methods to map out IL projections to cells in the basal forebrain that project to the amygdala. To determine the functional significance of this circuit, we will combine tracing with immunohistochemistry for the immediate early gene cfos. After exposure to recall of aversive stimuli, we will identify the active subpopulations of cells in the basal forebrain as well as their mPFC input and BLA output structures. Given the important role of the prefrontal cortex in anxiety disorders, unveiling these connections is crucial to understanding how it contributes to amygdala activity via direct and indirect routes.
[Parental Interpersonal Competence Moderates Behavioral Problems in Previously Institutionalized Youth: A Longitudinal Study]

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Abstract:
Early adverse caregiving is a major risk factor for mental health problems later in development across multiple species. Institutional caregiving, an extreme form of caregiving neglect, is a severely stressful environment for humans, such that exposure to institutional care has been associated with an elevated risk for behavioral problems in children and adolescents. Being adopted and raised by psychologically committed parents has been associated with significant rescue of behaviors in many children (Zeanah et al., 2003; Hodges & Tizard, 1989), however, whether parental interpersonal competence plays a significant role in moderating children’s behaviors remains unknown. In the present study, we collected data three times across a 3-year period from 167 youth (Previously Institutionalized n = 72, Comparison n = 95) and their parents, who completed both the Child Behavior Checklist (CBCL/4-18; Achenbach, 1991) and 2-minute videotaped observational sessions. A factor analysis and a three-way ANOVA were conducted to test whether individual differences in youth behavior outcomes could be predicted by parental interpersonal competence (defined as warm, interpersonally connected, and comfortable/relaxed demeanor during interaction), and a mixed linear model analysis was performed to investigate whether this parenting benefit would be observed more as children matured toward adolescence. The study found that high parental interpersonal competence was associated with lower total problems in adolescents, and when examined longitudinally, it predicted children’s behavioral problems 3 years later as they transitioned into adolescence. These results suggest that (1) good parental interpersonal competence can buffer against risks for child behavior problems, and (2) childhood is a sensitive period of increased plasticity for the development of regulatory processes.

Keywords: early life stress/adversity, behavior problems, parental interpersonal competence