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Review

Early life adversity reduces stress reactivity and enhances impulsive behavior: Implications for health behaviors

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ABSTRACT

Altered reactivity to stress, either in the direction of exaggerated reactivity or diminished reactivity, may signal a dysregulation of systems intended to maintain homeostasis and a state of good health. Evidence has accumulated that diminished reactivity to psychosocial stress may signal poor health outcomes. One source of diminished cortisol and autonomic reactivity is the experience of adverse rearing during childhood and adolescence. The Oklahoma Family Health Patterns Project has examined a cohort of 426 healthy young adults with and without a family history of alcoholism. Regardless of family history, persons who had experienced high degrees of adversity prior to age 16 had a constellation of changes including reduced cortisol and heart rate reactivity, diminished cognitive capacity, and unstable regulation of affect, leading to behavioral impulsivity and antisocial tendencies. We present a model whereby this constellation of physiological, cognitive, and affective tendencies is consistent with altered central dopaminergic activity leading to changes in brain function that may foster impulsive and risky behaviors. These in turn may promote greater use of alcohol other drugs along with adopting poor health behaviors. This model provides a pathway from early life adversity to low stress reactivity that forms a basis for risky behaviors and poor health outcomes.

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1. Introduction

Most models of stress reactivity and health outcomes assume that large stress reactions are harmful and that smaller responses are by definition better for the individual (Lovallo, 2005; Lovallo and Gerin, 2003). We have recently advanced the alternative hypothesis that both exaggerated and diminished stress reactivities indicate systems dysregulation with negative health implications (Carroll et al., 2009; Lovallo, 2011). There has been little consideration of the pathways by which individuals become more or less stress reactive than normal. We will review data from our studies and others suggesting that one pathway to low stress reactivity is the experience of stressful or adverse circumstances in childhood and adolescence. Ultimately, this pathway may lead to disinhibited behavior that can increase risk for alcoholism and other substance use disorders.

This review will focus on studies of persons whose adverse experiences occurred in childhood and adolescence and who were studied as adolescents and young adults. We exclude studies of persons prenatally exposed to stress or those studied as infants, children, or in old age. With minor exceptions the review is confined to persons lacking serious psychiatric comorbidities. Although some studies have examined hypothalamic–pituitary–adrenocortical axis (HPA) reactivity using pharmacological challenges, we primarily confine this review to cortisol responses to behavioral and psychosocial stressors. We also exclude studies of recent but transient life stressors (Chida and Hamer, 2008; Luecken and Lemery, 2004) and touch only briefly on studies of resting or basal levels of cortisol secretion.

2. Adversity and stress reactivity in the Oklahoma Family Health Patterns Project

In a series of earlier studies conducted with patients undergoing alcoholism treatment at the VA Medical Center in Oklahoma City, we had observed that the alcohol treatment groups had lower cortisol and heart rate stress responses than matched controls (Bernardy et al., 1996; Errico et al., 1993; Lovallo et al., 2000; Panknin et al., 2002). Because these patients had an average daily alcohol consumption of approximately one fifth of hard liquor for 8-years, it was impossible to determine if the blunted stress reactivity of these patients was due to heavy drinking or some preexisting difference. Therefore, with the goal of exploring premorbid characteristics of persons at risk for alcoholism, we designed the Oklahoma Family Health Patterns Project (OFHP) to study healthy young adults with

Abbreviations: FH+, positive family history of alcoholism; FH−, negative family history of alcoholism; CPI-So, California Personality Inventory Sociability Scale; SES, socioeconomic status; OFHP, Oklahoma Family Health Patterns; HPA, hypothalamic–pituitary–adrenocortical axis; ASPD, antisocial personality disorder; COMT, catechol-o-methyltransferase; MAOA, monoamineoxidase A; 5-HT, serotonin.

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and without a family history of alcoholism (FH+ and FH−) who would therefore reflect either elevated or reduced risk for the disorder. With this goal in mind, we have recruited over 400 volunteers with an average age of 24 years, 58% women, who are free of psychiatric disorders including current abuse of alcohol and other drugs, and are non-obese. Because of limited initial data on FH characteristics, our organizing principle was to focus broadly on the emotions and associated behaviors since substance use disorders represent a failure to regulate motivated behavior. Accordingly data collection encompassed domains of personality and temperament, affect, cognition, behavioral regulation, and stress reactivity.

Our first and most pervasive finding was that FH+ are much higher in antisocial tendencies than FH− based on the California Personality Inventory Socialization Scale (CPI-So) (Sorocco et al., 2006), indicating a pattern of risk taking and poor norm adherence (Sher et al., 1991; Tarter et al., 2004) with potential implications for risk for alcoholism. In our current sample, CPI-So sores are much lower for FH+ than for FH− persons (M ± SEM; 29.5 ± 0.37 vs 33.3 ± 0.31, respectively, $t = 7.77$, $p \ll .00001$), with low scores indicating low levels of socialization, norm adherence, and behavioral regulation reflecting a pattern of impulsive and disinhibited behaviors. The relevance of CPI-So scores for alcoholism risk is seen in a progressive relationship between low scores and a greater number of alcoholic relatives (Table 1).

Recalling our earlier studies showing blunted stress cortisol responses in alcoholic patients, we then focused on adversity as a potential predictor of low reactivity prompted by work showing diminished reactivity in women exposed to traumatic stressors in adolescence (Carpenter et al., 2007, 2011). This rationale was also shaped by the influential work of Michael Meaney and others showing that variations in maternal nurturing or postnatal stress exposure could influence adult behavior and stress reactivity in rat models (Gutman and Nemeroff, 2003; Meaney, 2001). To assess adversity we drew on our subjects' reports of socioeconomic status (SES) and their psychiatric data for reports of adverse experiences that were clearly not due to the subjects' own behaviors but occurred due to the actions of others. We identified five questions that fit those criteria:

Have you ever been mugged or threatened with a weapon, or experienced a break-in or robbery?
Have you ever been raped or sexually assaulted by a relative?
Have you ever been raped or sexually assaulted by someone not related to you?
Before you were 15, was there a time when you did not live with your biological mother for at least 6 months?
Before you were 15, was there a time when you did not live with your biological father for at least 6 months?

Adverse experiences before age 15 and low SES were combined to form a three-level scale of low, medium, and high lifetime adversity. This scale resembles the self-report items assessed in studies by Caspi of maltreatment in the Dunedin cohort (Caspi et al., 2002, 2003). We then examined our OFHP cohort of over 450 volunteers for stress reactivity, cognitive function and behavioral tendencies. The following summarizes our findings.

Table 1
Persons high and low in sociability as a function of number of alcoholic relatives.

		Percent in each CPI-So group	
		>30	≤30
FH−	0	64	36
FH+	1	35	65
	2	34	66
	3>	24	76

Note: A score of 30 is an empirically determined cutoff that separates relatively norm-abiding sample groups (>30) from those that are less so (≤30), with lower scores indicating more antisocial tendencies (Gough, 1994). $X^2 = 104$, $p = 2.6 \times 10^{-21}$.

2.1. Early life adversity and diminished stress reactivity

Men and women in our high adversity groups showed diminished cortisol and heart rate responses to psychosocial stress (public speaking plus mental arithmetic) despite having normal diurnal cortisol curves (Fig. 1) (Lovallo et al., 2012). Significantly, preliminary analyses showed that the two largest predictors of stress cortisol responses were the subject's sex followed by their experience of adversity. Fig. 1 shows that relative to the group with no adversity, men experiencing two or more lifetime adverse events have a 40% reduction in cortisol response to our stressors and women have a 92% reduction (Cohen's $d' = .38$, and .41, respectively, indicating moderately large effect sizes). These values from our study may not generalize to other studies since the extent to which adversity has an impact on stress response would vary with different subject samples, methods of documenting adversity, and the stressors used. Reduced stress reactivity due to adversity, in the face of normal diurnal HPA regulation, implicates the stress axis at and above the hypothalamus as the portion of the system that is dysregulated in the high adversity group. This implies that brain areas including the limbic system, the amygdala and bed nuclei of the stria terminalis, along with medial and lateral prefrontal cortex are potentially affected in persons exposed to adversity. As noted elsewhere, these are brain regions involved in stress appraisals and shaping outputs to the body during states of stress (Lovallo, 2007). See Van Voorhees for a recent review of the impact of maltreatment on the HPA (Van Voorhees and Scarpa, 2004).

2.2. Early life adversity and altered cognition and behavior

In accord with the above list of possible brain regions reflecting the effects of adversity, we next explored whether exposure to adversity may have an impact on cognitive functions and behavioral tendencies. We observed that greater levels of adversity predicted: (1) higher interference scores on the Stroop color–word test ($F = 3.07$, $p = .048$), a measure sensitive to working memory capacity; (2) faster discounting of delayed rewards ($F = 3.79$, $p = .024$), a measure indicating a relatively immediate orientation to obtaining rewards and reduced self regulation; (3) lower Shipley mental age scores ($F = 4.01$, $p = .019$), a test of general intelligence; and (4) higher body mass indexes, in FH+ persons exposed to adversity ($F = 3.40$, $p = .035$), indicating a difference in eating habits and health behaviors (Lovallo et al., in press). These effects were not explained by age, sex, race, education, or depression. Our results connecting adversity to poor working memory, impulsive behaviors, and lower general intelligence indicates that adversity during development has a long-term effect on central nervous system areas associated with decision-making and motivated behavior. Again, these would implicate lateral and medial prefrontal cortex and inputs from the septum and limbic system areas used in formulating motivations and adaptive responses.

2.3. Early life adversity and altered affect regulation

In keeping with our focus on emotions and motivated behavior, we next examined the impact of adverse experience on affect regulation and temperament. Persons higher in adversity were more likely to have antisocial tendencies as indexed by their CPI-So scores and Factor II (indexing antisocial and disinhibitory tendencies) from Lilienfeld's Psychopathic Personality Inventory ($Fs > 8.0$, $ps < .01$) (Patrick et al., 2006). Adversity was also associated with higher scores on the Eysenck Neuroticism scale and the Beck Depression Inventory ($Fs > 10.0$, $ps < .01$). Together these indicate that persons exposed to adversity during development are more disinhibited in their lifestyles, less socially connected, and have less stable mood regulation