



Effects of childhood trauma on cortisol levels in suicide attempters and ideators



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ABSTRACT

Objectives: Suicide is a global health issue. Dysregulated hypothalamic-pituitary-adrenal (HPA) axis activity, as measured by cortisol levels, has been identified as one potential risk factor for suicide. Recent evidence has indicated that blunted cortisol reactivity to stress is associated with suicidal behavior. The current study investigated whether childhood trauma was associated with blunted cortisol reactivity to a laboratory stressor and resting cortisol levels in suicide attempters and ideators.

Methods: 160 Participants were recruited and grouped according to history of previous suicidal attempt, suicidal ideation or as control participants. Participants completed background questionnaires, including the Childhood Trauma Questionnaire, before completing a laboratory stress task. Cortisol levels were assessed at rest and during the stress task.

Results: The highest levels of childhood trauma were reported in those who had attempted suicide (78.7%), followed by those who thought about suicide (37.7%) and then those with no suicidal history (17.8%). Moreover, regression analyses showed that childhood trauma was a significant predictor of blunted cortisol reactivity to stress and resting cortisol levels, such that higher levels of trauma were associated with lower cortisol levels in those with a suicidal history. Family history of suicide did not interact with the effects of childhood trauma on cortisol levels.

Conclusions: These results indicate that childhood trauma is associated with blunted HPA axis activity in vulnerable populations adulthood. The challenge for researchers is to elucidate the precise causal mechanisms linking trauma, cortisol and suicide risk and to investigate whether the effects of childhood trauma on cortisol levels are amendable to psychological intervention.

1. Introduction

Suicide is a global health issue accounting for 1.5% of all mortality. Researchers have been investigating the causes of suicidal behavior for decades to identify risk factors in order to inform interventions (e.g. Mann et al., 1999; O'Connor, 2011; O'Connor and Nock, 2014; van Heeringen and Mann, 2014; van Orden et al., 2010; World Health Organization, 2014). Recently, a small number of studies has explored whether cortisol reactivity to laboratory stressors may be associated with suicide attempt and ideation. For example, McGirr et al. (2010) investigated whether dysregulation of the HPA axis to a stressor was a heritable risk factor for suicidal behavior. In this study, a sample of first-degree relatives of those who died by suicide and matched controls

were compared on their cortisol reactivity to an acute laboratory stressor. The results showed that the first-degree relatives exhibited a blunted cortisol response to the stressor. Another study of adult offspring of parents with mood disorder (Melhem et al., 2016) found the lowest levels of total cortisol output in the offspring who had attempted suicide (compared to the offspring with suicide-related behavior but who had never attempted suicide, a non-suicidal offspring group and a healthy control group). However, they did not find a significant difference between the groups on a measure of cortisol reactivity to stress.

Most recently, O'Connor et al. (2017) investigated whether cortisol reactivity to a laboratory stressor differentiated individuals who had previously made a suicide attempt from those who had thought about suicide (suicide ideators) and control participants. The results showed

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that participants who had made a previous suicide attempt exhibited significantly lower cortisol levels in response to the stressor compared to participants in the control group. Suicide ideators were intermediate. Moreover, participants who made a suicide attempt and had a family history of suicide exhibited the lowest levels of cortisol in response to stress. In addition, Melhem et al. (2016) also found lower cortisol output in response to stress in offspring of parents with mood disorder who also had a parent with a history of suicide attempt. Taken together, the evidence is converging to suggest that low (or blunted) cortisol responsiveness to stress is associated with aspects of suicide behavior in adults and that aspects of the dysregulation of the stress response system may be a heritable risk factor for suicide risk.

An important next step for research in this area is to understand the factors that may contribute to HPA axis dysregulation in individuals vulnerable to suicide. A key candidate is likely to be childhood trauma. Numerous studies have linked childhood trauma to suicide risk, as well as to depression and psychopathology in adulthood (e.g., Carr et al., 2013; Heim et al., 2008; Marshall et al., 2013). For example, a large retrospective cohort study showed that adverse childhood experiences (e.g., abuse, neglect) increased risk of attempted suicide 2- to 5-fold throughout the lifespan (Dube et al., 2001). A systematic review found that childhood trauma, including sexual abuse, physical abuse, emotional abuse, physical neglect, and emotional neglect, triggers, maintains and increases the recurrence of psychiatric disorders (Carr et al., 2013). More recently, Marshall et al. (2013), in a prospective cohort study, found that severe sexual, physical and emotional childhood abuse conferred a substantial increased risk of suicide risk in illicit drug users.

Childhood trauma has been linked clearly to altered dynamics of the HPA axis and to persistent sensitization of the stress response system within the context of major depression (Heim et al., 2000; Heim et al., 2008). In addition, the effect of childhood trauma on depression has also been explained by changes in glucocorticoid resistance, increased central corticotropin-releasing factor (CRF) activity, immune activation, and reduced hippocampal volume. However, the results are mixed in the context of childhood trauma and cortisol reactivity. For example, Heim et al. (2000) showed that women who had a history of childhood abuse, with and without major depression, exhibited *increased* cortisol to an acute laboratory stressor (i.e., the Trier Social Stress Test; TSST). Whereas, a study by Carpenter et al. (2007) showed *decreased* cortisol levels in response to a laboratory stressor in childhood maltreated men who were never depressed. In a later study, Carpenter et al. (2011) replicated this finding and showed that women reporting childhood physical abuse displayed a blunted cortisol response to the TSST compared to women without physical abuse. Other studies have begun to emerge suggesting that early life adversity is associated with blunted cortisol reactivity to stress (e.g., Lovallo et al., 2012; see also Lovallo, 2013 for a review). Data from the Oklahoma Family Health Patterns Project showed that experience of adversity predicted reduced cortisol response to laboratory stress challenge (Lovallo et al., 2012).

In addition to cortisol reactivity to stress, evidence is also converging to suggest that resting cortisol levels are also associated with suicide behavior (O'Connor et al., 2016). A study by Keilp et al. (2016) found evidence of low baseline cortisol levels in those who have attempted suicide compared to non-attempters (Keilp et al., 2016). Similarly, Lindqvist et al. (2008) showed that low cortisol activity was associated with suicidal behavior. More recently, Melham et al. (2017) reported that those with a suicide attempt history had lower hair cortisol concentrations compared to controls (as well as ideators). However, similar to the cortisol reactivity studies in this area, less is known about the factors that may contribute to HPA axis dysregulation in individuals vulnerable to suicide.

Therefore, using data from the recent O'Connor et al. (2017) study, the primary aim of the current paper was to investigate whether childhood trauma was associated with low levels of cortisol reactivity to a laboratory stressor and lower resting cortisol levels (not previously

reported) in individuals vulnerable to suicide. Moreover, we were interested in exploring whether any observed relationships held while controlling for recent levels of depression and hopelessness. The secondary aim of the current paper was to investigate whether family history of suicide interacted with childhood trauma in order to predict additional variability in levels of resting cortisol and cortisol reactivity to stress.

2. Method

2.1. Design and participants

Full details of the method have been reported elsewhere, therefore, only a brief summary is presented here (see O'Connor et al., 2017). One hundred and sixty participants (100 females) were recruited to a previous attempt ($n = 49$), a suicidal ideation but no attempt ($n = 55$) and a control group ($n = 48$) based upon established measures of suicidal behavior (see below). Participants were aged between 18 and 62 years ($M = 26.84$ years, $SD = 9.32$) with 73.8% identified as Caucasian. Participants were enrolled to the study in response to a local advertising campaign on websites (e.g., Gumtree, Twitter), via posters, flyers and emails. Eligible participants were required to be at least 18 years old and to understand English. Suicide ideation and attempt were assessed using the Self-Injurious Thoughts and Behaviors Interview (SITBI; Nock et al., 2007) and the Beck Scale for Suicide Ideation (Beck et al., 1979; Beck et al., 1988). Participants were allocated to the previous attempt group if they reported attempting to take their own life in the past or to the ideation group if they reported having thoughts of killing their self in the past 12 months. Participants were recruited to a control condition who reported no history of suicide attempt or ideation (and did not report any current psychiatric or psychological conditions). The current study was approved by the Research Ethics Committee of the School of Psychology, University of Leeds and the US Department of Defense Human Research Protections Office.

Of the 160 participants recruited to the study, 6 participants withdrew due to having a negative reaction to the Maastricht Acute Stress Test (e.g., felt faint, or did not want to take part in all of the stress test), 8 other participants were unable to be clearly allocated to any of the conditions (e.g., reporting an inconsistent suicide history or changing their suicide history between screening and commencing the study) and 1 participant who had extreme cortisol values (e.g., exhibiting values of 45.80 nmol/L and 52.42 nmol/L which remained outside the distribution after log transformation). Following removal of these participants, the statistical analysis was conducted on 145 participants (control group = 45, ideator group = 53, attempter group = 47; see Table 1 for demographics and Table 2 for descriptive statistics for the main study variables). In the attempter group, 14 reported an attempt within the previous 12 months and 33 reported an historical attempt. In terms of family history of suicide, 25 participants reported they had a first degree relative who had attempted or completed suicide (control group = 3 [6.7%], ideator group = 6 [11.3%], attempter group = 16 [34%]). At baseline, 26.2% ($n = 38$) of participants reported using prescribed medication (control group = 5 [11.1%], ideator group = 16 [30.2%], attempter group = 17 [36.2%]).

2.2. Child trauma questionnaire (CTQ; Bernstein et al., 2003)

A brief 28-item self-report inventory was used to assess for a history of abuse or neglect in childhood or adolescence. The CTQ has five subscales relating to types of maltreatment: emotional, physical and sexual abuse and emotional and physical neglect, with five items for each subscale (1 = never true, 5 = very often true). It also includes a 3-item minimisation/denial scale for detecting false-negative trauma reports. Each subscale has a cut-off score to indicate a level of severity of childhood trauma ranging from none (or minimal), low (to moderate), moderate (to severe) and severe (to extreme). In the current study, we

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