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Chronicity of depressive problems and the cortisol response to psychosocial stress in adolescents: The TRAILS study

Sanne H. Booij*, Esther M.C. Bouma, Peter de Jonge, Johan Ormel, Albertine J. Oldehinkel

Interdisciplinary Center Psychopathology and Emotion regulation and Groningen Graduate School of Medical Sciences, Department of Psychiatry, University of Groningen, University Medical Center Groningen, The Netherlands

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Summary Clinical and epidemiological studies, further supported by meta-analytic studies, indicate a possible association between chronicity (i.e., persistence or recurrence) of depression and hypothalamic—pituitary—adrenal (HPA) axis responsiveness to psychosocial stress. In the present study, we examined whether and how chronicity of depressive problems predicts cortisol responses to a standardized social stress test in adolescents. Data were collected in a high-risk focus sample (n = 351) of the Tracking Adolescents' Individual Lives Survey (TRAILS) cohort, a large prospective population study with bi- to triennial measurements. Depressive problems were assessed around age 11, 13.5, and 16. Cortisol levels were measured in saliva, sampled before, during, and after the Groningen Social Stress Test (GSST), to determine the cortisol response to psychosocial stress. The area under the curve with respect to the increase (AUCi) (i.e., change from baseline) of the cortisol response was used as a measure of HPA axis response. By means of linear regression analysis and repeated-measures analysis of variance, it was examined whether chronicity of depressive problems predicted the cortisol response to the GSST around the age of 16. Chronicity of depressive problems was significantly associated with cortisol stress responses. The relationship was curvilinear, with recent-onset depressive problems predicting an increased cortisol response, and more chronic depressive problems a blunted response. The results of this study suggest that depressive problems initially increase cortisol responses to stress, but that this pattern reverses when depressive problems persist over prolonged periods of time.

Introduction

One of the prime factors in the precipitation of depression is the experience of psychosocial stress (Kendler et al., 1999; Ormel et al., 2001). Psychosocial stressors are capable of

* Corresponding author at: Interdisciplinary Center Psychopathology and Emotion regulation (ICPE), University Medical Center Groningen, CC72, P.O. Box 30.001, 9700 RB Groningen, The Netherlands. Tel.: +31 50 3615733; fax: +31 50 3619722.
E-mail address: s.h.booij@umcg.nl (S.H. Booij).

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activating a major player of the human stress system: the hypothalamic–pituitary–adrenal (HPA) axis (Holsboer and Ising, 2010). Abnormal HPA axis functioning has been a much studied facet of the pathophysiology of depression over the last years. Despite this, the exact role of the HPA axis response to psychosocial stressors in depressed persons is unclear. This is, at least in part, due to inconsistent findings: some studies found that individuals suffering from depression displayed increased cortisol responses to psychosocial stress, while others found blunted cortisol responses (Burke et al., 2005). A meta-analysis in adults' samples revealed that, overall, depressed persons had blunted cortisol responses to psychosocial stress (i.e., change from baseline), but the results were heterogeneous. This heterogeneity was partly caused by the fact that blunted responses were seen particularly in severely depressed and in older subjects (Burke et al., 2005). To the best of our knowledge, only one study examined the association between depression and HPA axis responses to stress in adolescents. In this study, depressed adolescents displayed exaggerated cortisol responses to a psychosocial stress test (Rao et al., 2008).

The finding that older depressed individuals are more likely to display blunted cortisol stress responses than younger ones could be explained by age differences, but older persons are also likely to have a persistent or recurrent depression (Burke et al., 2005). Unfortunately, studies looking into the relationship between HPA axis responses and depression have been mainly cross-sectional, and discriminated only between currently depressed and nondepressed individuals. This is in spite of evidence from several neuroimaging studies which suggests that persistent or recurrent depression coincides with changes in the brain that might affect HPA axis functioning, among which is reduced hippocampal size (Lorenzetti et al., 2009). In turn, these changes may lead to less effective inhibitory control of the hippocampus over the HPA axis, which has been thought to potentiate chronic release of cortisol and to be related to low HPA axis responses to stress (Jacobson and Sapolsky, 1991; Buchanan et al., 2009). In addition to evidence from neuroimaging studies, epidemiological studies have shown that the association between stressful life events and depression onset becomes weaker with subsequent depressive episodes (Kendler et al., 2000; Ormel et al., 2001). This so-called kindling effect (Post, 1992) indicates changes in reactivity to stress with subsequent depressive episodes.

In the present study, we addressed the role of chronicity, defined as persistent or recurrent depressive problems, with regard to the HPA axis response to psychosocial stress. Chronicity was defined as the presence of persistent or recurrent depressive problems, and operationalized as having depressive problems at consecutive assessment waves. We hypothesized that, compared to having no history of depressive problems (HDP), having recent-onset depressive problems would be related to an exaggerated cortisol response, whereas having more chronic depressive problems would be related to a blunted cortisol response. We studied the cortisol response to a psychosocial stressor under controlled laboratory conditions in a large sample of adolescents. Adolescents are an interesting group for studying first incidence and progression of depressive problems, because the prevalence of affective disorders starts to rise dramatically during adolescence, from an estimated 1% during preadolescence to rates of up to 25% at the end of adolescence (Kessler et al., 2005). An additional advantage is that the prevalence of potentially confounding somatic disorders is relatively low at this age.

Method

Participants

The present sample was selected from a focus sample of TRAILS (Tracking Adolescents' Individual Lives Survey). TRAILS is a large prospective population study of Dutch adolescents from the general population, which are followed from age 11 to at least age 21. The TRAILS study conducts measurements every two to three years. Three measurement waves (Ts) have been completed so far, while the fourth is currently being finalized. At T1, 2230 children were enrolled in the study (response rate 76.0), of whom 2149 (96.4%) participated at T2, and 1816 (81.4%) participated at T3. The mean age of the participants was 11.11 years (SD = 0.56) at T1, 13.57 years (SD = 0.53) at T2, and 16.28 years (SD = 0.71) at T3. A detailed description of this cohort is provided elsewhere (Huisman et al., 2008).

The focus sample consists of 715 adolescents who agreed to participate in a series of laboratory tasks additional to the usual assessments at T3 (response rate 96.1%). Adolescents with an increased risk of mental health problems had a greater chance of being selected for this experimental session. Increased risk was defined based upon temperament (high scores on frustration and fearfulness, low scores on effortful control), parental psychopathology (depression, anxiety, addiction, psychoses, or antisocial behavior), and environmental risk (living in a single-parent family), all measured at T1. In total, 66% of the focus sample had at least one risk factor, while the remaining 34% was randomly selected from the TRAILS cohort. Although adolescents with an increased risk of mental health problems were overrepresented, the focus sample still represented the whole range of problems seen in a normal population. This made it possible to use sampling weights in all analyses to reproduce the distribution in the total TRAILS sample. The experimental protocol was approved by the Dutch Central Committee on Research Involving Human Subjects (CCMO). Participants were treated in accordance with the Declaration of Helsinki, and experiments were carried out with adequate understanding and written consent of the participants. More information on the selection procedure can be obtained from the corresponding author.

From the focus sample, 24 adolescents were discarded because they had missing data on depressive symptoms at one of the three assessment waves, and 32 because their experimental session was more than 18 months before or 12 months after the assessment of depressive problems. In addition, we excluded girls who used oral contraceptives (OC) (n = 110) because a previous study in this sample showed that OC users displayed no significant cortisol response to the social stress test (Bouma et al., 2009) and habitual smokers (i.e., at least one cigarette a day, n = 76) because smoking attenuates the cortisol response to psychosocial stress (Rohleder and Kirschbaum, 2006). Four participants were excluded because they used corticosteroid medication or