



Higher overcommitment to work is associated with lower norepinephrine secretion before and after acute psychosocial stress in men

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Summary

Background: Overcommitment (OC) is a pattern of excessive striving. In reaction to work stress, OC has been associated with higher sympathetic nervous system activation and cortisol release, but data on neuroendocrine reactivity to standardized stressors are scarce. We investigated whether OC is associated with differential levels of the stress hormones norepinephrine and cortisol in response to acute psychosocial stress.

Methods: Fifty-eight medication-free non-smoking men aged between 20 and 65 years (mean \pm S.E.M.: 36.3 ± 1.8) underwent an acute standardized psychosocial stress task combining public speaking and mental arithmetic in front of an audience. We assessed OC as well as a variety of psychological control variables including vital exhaustion, perfectionism, chronic stress, and cognitive stress appraisal. Moreover, we measured plasma norepinephrine as well as salivary cortisol before and after stress and several times up to 60 min thereafter.

Results: Higher OC was associated with lower baseline norepinephrine levels ($r = -0.37$, $p < 0.01$). General linear models controlling for age, BMI, and mean arterial blood pressure revealed that higher overcommitment was associated with lower norepinephrine and cortisol levels before and after stress (p 's < 0.02) as well as with lower norepinephrine stress reactivity ($p = 0.02$). Additional controlling for the potential psychological confounders vital exhaustion, perfectionism, chronic stress, and depression confirmed lower norepinephrine levels before and after stress ($p < 0.01$) as well lower norepinephrine stress reactivity ($p = 0.02$) with increasing OC. Higher OC independently explained 13% of the total norepinephrine stress response ($\beta = -0.46$, $p < 0.01$, R^2 change = 0.13).

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Conclusions: Our findings suggest blunted increases in norepinephrine following stress with increasing OC potentially mirroring blunted stress reactivity of the sympathetic nervous system.

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1. Introduction

“Overcommitment” (OC) has been defined as an enduring cognitive-motivational pattern of maladaptive coping with demands characterized by excessive striving and an inability to withdraw from obligations (Siegrist et al., 2004). Overcommitted persons are driven by their high need for control and approval, thereby repeatedly overtaxing their own resources and, thus, precipitating exhaustion and breakdown in the long run (Joksimovic et al., 1999). OC has been introduced as an intrinsic component to the model of effort-reward imbalance (ERI) at work. In this context, OC is thought to magnify stressful experience resulting from high cost/low gain conditions at work because it induces exaggerated efforts which are not met by extrinsic rewards (Siegrist, 1996).

Several prospective studies suggest that OC increases coronary heart disease risk (Siegrist et al., 1990; Joksimovic et al., 1999; Kuper et al., 2002). The biological pathways through which OC might increase cardiovascular disease risk are beginning to be understood. For instance, OC has been related to major physiological cardiovascular risk factors including elevated lipid levels and hemostatic risk factors (Peter et al., 1998; Vrijkotte et al., 1999). Moreover, OC is strongly associated with the concept of vital exhaustion, an independent psychological risk factor for coronary heart disease (CHD) (Appels, 1990; Cole et al., 1999; Preckel et al., 2005).

The concept of physiological hyperreactivity to stress posits that studying short-term cardiovascular responses to controlled physiological, cognitive, and emotional challenges serves as a window into complex psychological and physiological processes that are involved in the development of cardiovascular disease (Steptoe and Willemsen, 2002; Linden et al., 2003). Although not uniform (Vrijkotte et al., 2000), in reaction to work stress, OC has been associated with indicators of sympathetic nervous system activation including elevated blood pressure (Steptoe et al., 2004), shorter pre-ejection period (PEP) levels and reduced PEP variability (Vrijkotte et al., 2004). Moreover, associations between OC and elevated levels of the stress hormone cortisol over the workday have been reported (Eller et al., 2006). Yet, these findings result from ambulatory monitoring studies performed in everyday life where control of potential confounders is limited.

To the best of our knowledge, experimental studies on associations between OC and physiological reactivity to standardized psychological stressors have not yet been published. According to the concept of vital exhaustion (Appels, 1997), it might be assumed that prolonged stress first leads to heightened hormonal responsiveness. Later, chronic elevations of hormones overtax an exhausted system which then might respond to this load by down-regulating the sensitivity of target tissues (Wirtz, 2002; Wirtz et al.,

2003). As a result, reduced rather than increased responsiveness following stressful experience might occur in overcommitted persons. Indeed, the OC-related constructs vital exhaustion and ERI suggest blunted sympathetic and cortisol reactivity to acute standardized mental stressors. ERI was associated with blunted sympathetic reactivity in terms of lowered epinephrine, heart rate as well as cortisol elevations following a modified Stroop Test (Siegrist et al., 1997). Similarly, vitally exhausted persons were less likely to show large cortisol responses to a speech task (Nicolson and van Diest, 2000; Kristenson et al., 2004).

The aim of this study was to investigate associations between OC and neuroendocrine reactivity to a standardized psychosocial stress task in unmedicated non-smoking men. We measured the neuroendocrine parameters norepinephrine and cortisol as well as heart rate before and several times after stress. We favored norepinephrine over epinephrine because of its higher relevance with respect to cardiovascular disease risk (Goldstein, 1981; Rahn et al., 1999). In line with assumptions derived from the concept of vital exhaustion we hypothesized that higher OC would be associated with lower neuroendocrine stress reactivity. Moreover, to control for potential confounders, we additionally assessed psychological parameters including vital exhaustion, perfectionism, chronic stress, and cognitive stress appraisal, which are related to either OC or to neuroendocrine stress reactivity.

2. Methods

2.1. Study participants

The Ethics Committee of the State of Zurich, Switzerland, formally approved the research protocol. Of a total of 64 participants we obtained complete OC scores of 58 subjects representing the final study sample. All subjects provided written informed consent. Recruitment was carried out through advertisement of the study on pin boards at the University of Zurich and by members of the research team who accompanied the mobile blood donation units of the Swiss Cross of the State of Zurich. We intentionally recruited non-smoking middle-aged men who were in excellent physical and mental health confirmed by an extensive health questionnaire (Wirtz et al., 2003) and telephone interview. Specific exclusion criteria, obtained by subjects' self-report, were: regular strenuous exercise, alcohol and illicit drug abuse; any heart disease, varicosis or thrombotic diseases, elevated blood sugar and diabetes, elevated cholesterol, liver and renal diseases, chronic obstructive pulmonary disease, allergies and atopic diathesis, rheumatic diseases, and current infectious diseases. In addition, participants were included only if they reported taking no medication, either regularly or occasionally and if their

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