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Getting better, but not well: A 1.5 year follow-up of cognitive performance and cortisol levels in clinical and non-Clinical burnout

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ABSTRACT

The purpose was to reexamine cognitive performance and cortisol levels of initial clinical burnout patients, non-clinical burnout individuals, and healthy controls. After 1.5-years of the initial measurement, clinical burnout patients showed a reduction of burnout symptoms and general physical and psychological complaints, but these were still elevated compared with controls. Nonetheless, they continued to report cognitive problems and still showed a minor impaired cognitive test performance. However, they no longer reported larger subjective costs associated with cognitive test performance and their cortisol awakening response (CAR) returned to a normal level. Compared with controls, non-clinical burnout individuals still reported the same, elevated, level of burnout symptoms, general physical and psychological complaints, and cognitive problems. Their cognitive test performance and associated subjective costs remained normal. However, they seemed to continue to display a lowered CAR. To conclude, after 1.5-years, clinical burnout patients got better, but not 'well', and non-clinical burnout individuals remained not 'well'.

1. Introduction

Employees with burnout frequently report cognitive problems, such as difficulties with concentration and memory (e.g., Weber & Jaekel-Reinhard, 2000). Research has shown that these self-reported cognitive problems are accompanied by actual cognitive impairments as measured with neuropsychological tests (Diestel, Cosmar, & Schmidt, 2013; Jonsdottir et al., 2013; Oosterholt, Van der Linden, Maes, Verbraak, & Kompier, 2012; Österberg, Karlson, & Hansen, 2009; Sandström, Rhodin, Lundberg, Olsson, & Nyberg, 2005; Van Dam, Keijsers, Eling, & Becker, 2011; Van der Linden, Keijsers, Eling, & Van Schaijk, 2005). Nevertheless, the actual burden of these impairments is still not clear, as they range from relatively mild (e.g., Österberg et al., 2009) to profound impairments (e.g., Sandström et al., 2005).

It has been hypothesized that the cognitive deficits in burnout are related to stress (e.g., Österberg et al., 2009; Sandström et al., 2011). This hypothesis is plausible as burnout is generally considered to be a stress-related condition (e.g., Cordes & Dougherty, 1993; Maslach, Schaufeli, & Leiter, 2001) and there is substantial evidence that stress can have a detrimental impact on the brain, for example, on the hippocampus (e.g., Lupien & Lepage, 2001) and the prefrontal cortex (e.g., Arnsten, 2009). These brain structures are, among others, responsible for memory consolidation and executive functioning, respectively. The mechanism underlying the relationship between stress and cognition is assumed to involve the hypothalamic-pituitary-adrenal axis (HPA axis), a part of the neuroendocrine system that plays a role in the regulation of stress reactions. Specifically, the hormone cortisol, the release of which is regulated by the HPA axis and which is considered to be the main stress hormone, is believed to be involved in mediating the stress-cognition relation, whereby both high and low levels of cortisol can have detrimental effects on cognition (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007).

Cortisol levels in relation to burnout have been examined in several studies. The results of these studies are mixed (Danhof-Pont,

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Van Veen, & Zitman, 2011). For example, in some studies burnout was found to be related to lower levels of cortisol (e.g., Marchand, Juster, Durand, & Lupien, 2014; Sonnenschein et al., 2007), whereas in other studies higher cortisol levels were found (e.g., De Vente, Olff, Van Amsterdam, Kamphuis, & Emmelkamp, 2003; Melamed et al., 1999). Moreover, there are also studies in which burnout did not relate to any cortisol deviations (Grossi, Perski, Evengård, Blomkvist, & Orth-Gomér, 2003; Mommersteeg, Keijsers, Heijnen, Verbraak, & Van Doornen, 2006c).

To gain further insight into the burnout-cognition and burnout-cortisol relationships, we recently examined cognitive performance (Oosterholt, Maes, Van der Linden, Verbraak, & Kompier, 2014) as well as cortisol levels (Oosterholt, Maes, Van der Linden, Verbraak, & Kompier, 2015) in a sample of clinical burnout patients (employees seeking treatment for their burnout symptoms and diagnosed as such), non-clinical burnout individuals (employees reporting symptoms of a burnout, but neither diagnosed as such nor seeking help for these symptoms), and healthy control individuals. An asset of these studies was that we examined burnout by including both a clinical and a non-clinical burnout group (and a healthy control group). Furthermore, compared to the majority of other studies in this area, we used relatively large samples, and we used well-validated and extensive measures to assess both cognitive performance and cortisol levels. With regard to cognitive performance, we found that, although both the clinical burnout patients and the non-clinical burnout individuals reported cognitive problems, only clinical burnout patients showed a relatively mild impaired cognitive test performance. Compared with the healthy controls, they also reported larger subjective costs associated with their cognitive test performance. Specifically, they invested more effort in completing the tests, and rated the tests as more demanding. As regards cortisol levels, we found the cortisol awakening response to be lower in both clinical burnout patients and non-clinical burnout individuals compared with healthy individuals. In addition, some evidence was found indicating that the decline of cortisol during the day was smaller in individuals with a non-clinical burnout than in healthy controls. These results suggested a hypoactive HPA axis in both our clinical and non-clinical burnout group.

Almost all previous research on both the relationship between burnout and cognition and burnout and cortisol has been cross-sectional, and has been performed in individuals with acute burnout symptoms. However, relatively little is known about the longitudinal course of cognitive performance in burnout (Beck, Gerber, Brand, Pühse, & Holsboer-Trachsler, 2013; Oosterholt et al., 2012; Österberg, Skogsliden, & Karlson, 2014; Van Dam, Keijsers, Eling, & Becker, 2012; Wahlberg et al., 2009) as well as about the longitudinal course of cortisol levels in burnout (Moch, Panz, Joffe, Havlik, & Moch, 2003; Mommersteeg, Heijnen, Verbraak, & Van Doornen, 2006b; Mommersteeg et al. 2006c; Österberg, Karlson, Malmberg, & Hansen, 2012; Wahlberg et al., 2009). Moreover, both with regard to the burnout-cognition and burnout-cortisol relationship, the results of these previous studies are inconsistent (see Discussion for a more detailed review of the existing literature). The aim of the present study was to get more insight into the time course of cognitive performance and cortisol levels in burnout. To this end, we reexamined the initial clinical burnout group, non-clinical burnout group, and healthy control group that we reported on previously (Oosterholt et al., 2014, 2015) after a 1.5 year period. As recovery from burnout is a slow process (e.g., Sonnenschein et al., 2008), and previous longitudinal studies on the relationship between burnout and cognition (e.g., Oosterholt et al., 2012) as well on the relationship between burnout and cortisol (e.g., Moch et al., 2003) have shown that a relatively short follow-up period did not result in any positive changes, we chose to reexamine our groups after a rather long period of approximately 1,5 years. In-between the first examination (T1) and the second examination (T2), the patients in the clinical burnout group received psychological therapy aimed at reducing burnout symptoms. Although we did not have specific expectations as regards the non-clinical burnout group, we expected burnout symptoms and physical and mental complaints of the clinical burnout group to improve in the course of the treatment period. However, the question of interest was whether or not cognitive performance would also show any improvements and whether cortisol would return to a normal level. If reduced cognitive performance and cortisol deviation would result from burnout, it is possible that when burnout symptoms decrease this will be accompanied with a return to pre-burnout cognitive functioning and cortisol levels. Such changes are to be expected only if the prolonged stress, held to underlie the burnout symptoms, did not result in any permanent damage (McEwen, 2000).

In sum, the purpose of the present study was to answer two research questions, next to assessing the time course of burnout symptoms and general physical and psychological complaints. First, what is the course (from T1 to T2) of cognitive performance (self-reported cognitive problems, cognitive test performance, and subjective costs associated with cognitive test performance) in both clinical burnout and non-clinical burnout? Second, what is the course (from T1 to T2) of cortisol levels in both clinical and non-clinical burnout?

2. Method

2.1. Participants

The participants in the present study had been examined previously on both cognitive performance and cortisol levels, see Oosterholt et al. (2014), and Oosterholt et al. (2015), respectively. Of the 93 participants examined during the first examination (T1), 85 (91%) agreed to participate in the second examination (T2), approximately 1.5 years later. Of these participants, 31 (out of 33) belonged to the clinical burnout group, 27 (out of 30) to the non-clinical burnout group, and 27 (out of the 30) to the healthy control group. The difference between the clinical and non-clinical burnout group was that, at T1, the clinical burnout group comprised patients with a clinical burnout diagnosis whereas the non-clinical burnout group consisted of individuals who reported symptoms of a burnout, but were neither diagnosed as such nor seeking help for these symptoms and all still worked. From the reexamined healthy control group, one participant was excluded because he was treated for a burnout during the time between T1 and T2. The reasons for not participating at T2 ranged from an inability to get in contact with the participant, the participant working abroad, being unwilling to take off from work, or just being unwilling to participate again. At T2 the three groups were still matched on several demographical characteristics (see Table 1 for more detailed information) and had various occupational backgrounds. Furthermore, all participants were actively employed, except for one individual in the non-clinical burnout group and one in the control group.

In-between T1 and T2, the patients of the clinical burnout group received psychological treatment for their burnout symptoms. Treatment was provided by professional clinical psychologists according to a treatment protocol for burnout (Keijsers et al., 2004) that is commonly used in the Netherlands. Basic modules of this treatment include: reduction of complaints, cognitive-behavioral therapy, and relapse prevention. Additional therapy modules can be chosen if necessary. Although this treatment was aimed at reducing burnout symptoms and was not specifically directed at improving cognitive performance or changing cortisol levels, it gave us the opportunity to establish any possible changes in