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Hydrocortisone accelerates the decay of iconic memory traces: On the modulation of executive and stimulus-driven constituents of sensory information maintenance

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Summary A substantial amount of research documents the impact of glucocorticoids on higher-order cognitive functioning. By contrast, surprisingly little is known about the susceptibility of basic sensory processes to glucocorticoid exposure given that the glucocorticoid receptor density in the human visual cortex exceeds those observed in prefrontal and most hippocampal brain regions. As executive tasks also rely on these sensory processes, the present study investigates the impact of glucocorticoid exposure on different performance parameters characterizing the maintenance and transfer of sensory information from iconic memory (IM; the sensory buffer of the visual system) to working memory (WM).

Using a crossover factorial design, we administered one out of three doses of hydrocortisone (0.06, 0.12, or 0.24 mg/kg bodyweight) and a placebo to 18 healthy young men. Thereafter participants performed a partial report task, which was used to assess their individual ability to process sensory information. Blood samples were concurrently drawn to determine free and total cortisol concentrations. The compiled pharmacokinetic and psychophysical data demonstrates that free cortisol specifically accelerated the decay of sensory information ($r = 0.46$) without significantly affecting the selective information transfer from IM to WM or the capacity limit of WM. Specifically, nonparametric regression revealed a sigmoid dose—response relationship between free cortisol levels during the testing period and the IM decay rates.

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Our findings highlight that glucocorticoid exposure may not only impact on the recruitment of top-down control for an active maintenance of sensory information, but alter their passive (stimulus-driven) maintenance thereby changing the availability of information prior to subsequent executive processing.

1. Introduction

One of the most consistent findings in neuroendocrine stress research is the severe impairment of memory retrieval by glucocorticoid exposure (Het et al., 2005). Animal studies suggest that such impairments are likely to be mediated by a high density of glucocorticoid receptors (GR) in the underlying (e.g. hippocampal) brain regions (Joëls, 2006; see Pryce, 2008, for review). Many recent studies adopted this line of reasoning taking the somewhat lower GR density in the prefrontal cortex as motivation to investigate the effects of pharmacological and stress-related glucocorticoid exposure on executive (or top-down) functioning by computer-based tasks (e.g. Lupien et al., 1999; Schoofs et al., 2008; Plessow et al., 2011). However, such tasks yield inconsistent evidence for decreased (e.g. Lupien et al., 1999; Schoofs et al., 2008), increased (e.g. Oei et al., 2009; Plessow et al., 2011), or unaffected executive functioning (e.g. Young et al., 1999) due to glucocorticoid exposure. While some ambiguity of these findings may be attributable to time-dependent interactions of catecholamine and glucocorticoid secretion in real-life stress situations (Hermans et al., 2014), this may also highlight an issue that is commonly referred to as task impurity (Miyake et al., 2000): Any frontal lobe task that is supposed to assess the selective processing of task-relevant stimulus features and/or their shielding from distracting stimulus features also relies on other cognitive processes, such as the ability to maintain the stimulus' sensory representations (Stelzel et al., 2009). Consequently, many claims about glucocorticoid effects on executive functioning might be as well interpretable as glucocorticoid effects on such residual components of task performance, which in turn constitute a building block for higher-order cognition (Öğmen et al., 2013).

The presumed codependence of executive and sensory processing has recently received support by neuroimaging studies. For example, Egner and Hirsch (2005) were able to demonstrate that a concordant activity change in prefrontal and visual brain regions was associated with a reduced interference of distracting stimulus features and an improved task performance (see also Stelzel et al., 2009).

Using biophysical modeling approach, Zylberberg et al. (2009) also confirmed the crucial interaction of executive and sensory processing: They demonstrated that the ability to maintain stimulus features does not only rely on their (active) top-down amplification but also depends on the (passive) decay of the transient neuronal response that encodes their information on a sensory level. Both constituents of active and passive stimulus feature maintenance were mechanistically well elaborated for the *partial report* task (e.g. Lu et al., 2005), which was originally developed to assess the main characteristics of iconic memory (IM), that is, the huge capacity and rapid decay of information stored in this sensory buffer of the visual system (see Coltheart, 1980, for review). Based on this biophysical network model, we derived two competing hypotheses about the primary way of glucocorticoid action on the active as compared to the passive constituent of stimulus feature maintenance. The passive constituent of the maintenance is supposed to strongly depend on the N-Methyl-D-Aspartat (NMDA) receptor trafficking (Zylberberg et al., 2009), which is known to be impaired by glucocorticoid exposure (Coussens et al., 1997; Pavlides et al., 1995). In consequence, the transient neuronal responses should decay faster in response to glucocorticoid exposure. If, by contrast, the amount of actively recruited top-down control is increased in response to isolated glucocorticoid exposure (i.e., in the absence of catecholamine secretion; see Hermans et al., 2014, for review), the stimulus feature maintenance should be enhanced.

Intriguingly, both model-based predictions are supposed to be behaviorally distinguishable using the *partial report* task. Any stimulus feature is initially stored in IM (as a trace of sensory information), which then decays after the stimulus has faded unless it is cued for retrieval. Thus, a feature that is cued directly after stimulus fading can be reported with large accuracy, whereas a feature that is cued with a long delay can only be reported with a chance that corresponds to the proportion of the number of stimulus features to the capacity limit of working memory (WM; Coltheart, 1980; Cowan, 2000). This decline of *partial report* performance across time can be modeled as an exponential decay function (cf. Gegenfurtner and Sperling, 1993) that is determined by three different performance parameters. These parameters index (1) the WM capacity limit (i.e., the amount of sensory information that is non-selectively transferred from IM to WM at extremely delayed and/or omitted cue presentation), (2) the *partial report* superiority effect (PRSE), that is, the performance advantage (over the WM capacity limit) directly after stimulus fading, and (3) the temporal maintenance of sensory information (represented as the decay rate of IM traces).

According to the above-mentioned hypotheses about glucocorticoid effects on stimulus feature maintenance, a specific modulation of its active and passive constituents should then manifest as (A) a reduced decay rate of IM traces and/or a shifted limit of WM capacity, or (B) an increased decay rate of IM traces but an unaffected limit of WM capacity, respectively. In order to test these hypotheses, 18 healthy young men accomplished the *partial report* task after hydrocortisone (i.e., a dose of 0.06, 0.12, or 0.24 mg/kg bodyweight) and placebo administration.