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Review

Impact of early adversity on glucocorticoid regulation and later mental disorders

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ABSTRACT

Early adverse experiences such as abuse or neglect can influence brain development and consequently bring forth a predisposition toward mental and behavioral disorders. Many authors suggest that long-term changes in the functionality of the HPA axis might be involved in mediating this relationship. The direction of change and its consequences have not been clarified though: Do early adverse experiences yield a stable glucocorticoid hyperfunction or a long-term glucocorticoid hypofunction, and how is this change of functionality associated with mental or behavioral disorders? This review summarizes correlative findings and illustrates inconsistencies of current research literature. It focuses on the specific neurochemical milieu accompanying early adverse experiences and discusses possible interactions of the glucocorticoid system with oxytocin and components of the serotonergic system. On the basis of this physiological view, a novel two-pathway model is presented, according to which specific early experiences are associated with characteristic early changes in the functionality of these systems and result in a predisposition to distinct mental and behavioral disorders.

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1. Introduction

Early adverse experiences can result in stable basal and stress-related changes of glucocorticoid secretion (for reviews, see e.g., Gunnar and Vazquez, 2001; McCrory et al., 2010). These changes likewise accompany various affective or personality disorders (for reviews see Heim et al., 1997; Loney et al., 2006; O'Leary et al., 2010; Steckler et al., 1999), and a functional relationship could be assumed, particularly if the large number of publications about the negative impact of early adverse experience on mental health is taken into account (for reviews see Afifi, 2012; Agid et al., 1999; Heim and Nemeroff, 2001; Maas et al., 2008; Poythress et al., 2006; Shi et al., 2012).

The direction of change is not clear though: Do adverse experiences result in a glucocorticoid *hyper*function or *hypo*function (for review see Gunnar and Vazquez, 2001; Hunter et al., 2011; Loman and Gunnar, 2010)? Does the change of function concern *basal* or *stress-related* activity of glucocorticoids (for review see Hunter et al., 2011)? Which state, hyperfunction or hypofunction, is related to which mental disorder (for review see Gold and Chrousos, 2002; Kasckow et al., 2001)?

We will first introduce the glucocorticoid system and review correlative findings about the relationship between early adverse experiences and long-term changes of the glucocorticoid system in humans and animals. Subsequently, we will turn to a physiological view of underlying mechanisms. Two molecular pathways that may lead to a glucocorticoid hyperfunction or hypofunction, respectively, are deduced as potential outcomes of specific early experiences. We will then compare these physiological assumptions with reviewed correlative findings. Furthermore, research on the association of stable changes of the glucocorticoid systems and later mental health problems in humans will be reviewed. Thereafter, we will propose physiological mechanisms mediating this association. Among these, an interaction of glucocorticoids with the serotonin system, will be highlighted. It will be concluded that specific early experiences may facilitate – via a characteristic pattern of glucocorticoid secretion – the development of distinct affective or personality disorders. Hereby, the presentation of two pathways which involve a glucocorticoid hyperfunction and a glucocorticoid hypofunction, respectively, and lead to a predisposition toward different mental disorders, will be continued.

2. The HPA axis

If the organism encounters a stressful stimulus, cells in the paraventricular nucleus of the hypothalamus are activated and, via their projections to other brain areas, generate reactions of various stress-systems, e.g., the sympathetic nervous system. One of the projections indirectly targets the pituitary as an important component of the hypothalamus-pituitary-adrenal axis (HPA axis, for review see Joëls et al., 2011). For an activation of the HPA axis to occur, parvocellular neurons of the paraventricular nucleus send their axons to the portal capillary plexus of the median eminence of the hypothalamus and secrete corticotropin-releasing factor (CRF) as well as vasopressin, which reach, via the portal capillaries, the anterior pituitary. Here, they stimulate the synthesis and release of adrenocorticotropic hormone (ACTH) into systemic circulation. ACTH, in turn, reaches the adrenal cortex and leads to a synthesis and release of glucocorticoid hormones into systemic circulation (for review see Whitnall, 1993) and, via the passage of the blood-brain barrier, into the brain (Fig. 1; for review see de Kloet et al., 1998).

3. Glucocorticoids

Glucocorticoids, predominantly cortisol in humans and corticosterone in the rodent, are secreted not only under stress, but also under basal conditions. Basal secretion follows a circadian rhythm: Release is low during inactive phases of sleep (nighttime in humans, daytime in rodents), increases in anticipation of waking, and peaks in the morning in humans and in the evening in rodents. These basally secreted glucocorticoids are released in a pulsatile, ultradian manner. The amplitude of the pulses is low at the beginning of the circadian period of inactivity, increases toward the onset of the active period and gradually declines thereafter, generating the characteristic circadian profile. It is assumed that these basal pulses coordinate and synchronize daily activities and sleep-related events (for reviews see Joëls et al., 2008; Walker et al., 2010). A frequently investigated parameter of basal glucocorticoid release is the *cortisol-awakening response* (CAR), a large pulse that accompanies awakening in the morning. The CAR is of high intraindividual stability and can serve as an index of HPA axis activity (for review see Wüst et al., 2000). Superimposed on this basal profile of secretion is the glucocorticoid *response to various stressors*. The stress-related increase in glucocorticoid secretion mobilizes resources of energy, promotes consolidation of experiences for future use and controls the initial reaction to stress via a feedback projection (for review see de Kloet and Sarabdjitsingh, 2008).

The HPA axis does not respond *indiscriminately* to any challenge of the organism, but preferably to those involving a social-evaluative threat or an uncontrollable outcome (Del Giudice et al., 2011), such as delivering a speech in front of a judging audience (Gunnar et al., 2009; Kirschbaum et al., 1993). For infants, a short-term separation of the child from its mother is a powerful activator of the HPA axis (Gunnar et al., 2009). In children, conflict, rejection or familial instability lead to increases of HPA axis activity – even

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