

Stress and Memory: Opposing Effects of Glucocorticoids on Memory Consolidation and Memory Retrieval

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It is well established that glucocorticoid hormones, secreted by the adrenal cortex after a stressful event, influence cognitive performance. Some studies have found glucocorticoid-induced memory enhancement. However, many studies have reported impairing effects of glucocorticoids on memory function. This paper reviews recent findings from this laboratory on the acute effects of glucocorticoids in rats on specific memory phases, i.e., memory consolidation and memory retrieval. The evidence suggests that the consequences of glucocorticoid activation on cognition depend largely on the different memory phases investigated. Posttraining activation of glucocorticoid-sensitive pathways involving glucocorticoid receptors enhances memory consolidation in a pattern highly similar to that previously described for adrenal catecholamines. Also, similar to catecholamine effects on memory consolidation, glucocorticoid influences on memory consolidation depend on noradrenergic activation of the basolateral complex of the amygdala and interactions with other brain regions. By contrast, memory retrieval processes are usually impaired with high circulating levels of glucocorticoids or following infusions of glucocorticoid receptor agonists into the hippocampus. The hypothesis is proposed that these apparently dual effects of glucocorticoids on memory consolidation and memory retrieval might be related and that the basolateral complex of the amygdala is a key structure in a memory-modulatory system that regulates, in concert with other brain regions, stress and glucocorticoid effects on both memory consolidation and memory retrieval. © 2002 Elsevier Science (USA)

Key Words: amygdala; corticosterone; dexamethasone; emotional arousal; hippocampus; memory storage; norepinephrine.

This paper is dedicated to the memory of my mentor and friend, Dr. Béla Bohus.

The author thanks Dr. James L. McGaugh for comments on an earlier version of the manuscript and continuous support. The research was supported by USPHS Grant MH12526 from NIMH (to James L. McGaugh).

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INTRODUCTION

Adrenal hormones (i.e., catecholamines and glucocorticoids) are secreted during stressful events and influence, together with other components of the stress system, the organism's ability to cope with stress. These hormones also affect memory function by influences on limbic brain structures. It is well established that adrenal catecholamines promote consolidation and/or storage of novel information (Bohus, 1994; McGaugh, Cahill, & Roozendaal, 1996; McGaugh & Roozendaal, 2002). Immediate posttraining systemic injections of epinephrine or norepinephrine to rats enhance memory of aversively motivated inhibitory avoidance training (Gold & van Buskirk, 1975; Borrell, de Kloet, Versteeg, & Bohus, 1983; Liang, Juler, & McGaugh, 1986; Cahill & McGaugh, 1991; Roozendaal, Carmi, & McGaugh, 1996a). These findings, which have been confirmed by experiments using many different types of training tasks, support the hypothesis that endogenously released epinephrine modulates memory consolidation (Izquierdo & Diaz, 1985; Sternberg, Isaacs, Gold, & McGaugh, 1985; McGaugh, Ferry, Vazdarjanova, & Roozendaal, 2000; Liang, 2001). In contrast, there are conflicting findings concerning glucocorticoid effects on cognition (Lupien & McEwen, 1997; de Kloet, Oitzl, & Joëls, 1999; Roozendaal, 2000). Although some studies have found glucocorticoid-induced memory enhancement (Cottrell & Nakajima, 1977; Sandi & Rose, 1994a; Sandi, Loscertales, & Guanza, 1997; Buchanan & Lovallo, 2001), the potentially disruptive effects of glucocorticoids on memory have recently received much attention (Newcomer, Craft, Hershey, Askins, & Bardgett, 1994; Kirschbaum, Wolf, May, Wippick, & Hellhammer, 1996; Lupien et al., 1997; Diamond, Park, Heman, & Rose 1999). Such evidence raises the question whether glucocorticoids differ fundamentally from catecholamines in their effects on memory function.

The evidence reviewed in this paper suggests that the consequences of glucocorticoid activation on cognition depend largely on the different memory phases investigated. In recent studies, we have examined the acute effects of glucocorticoids on different memory phases, i.e., memory consolidation and memory retrieval. Glucocorticoids, or their specific receptor agonists or antagonists, were administered to rats either immediately after training on several tasks to examine their effects on memory consolidation or shortly before retention testing to examine effects on memory retrieval. The findings indicate that memory consolidation is enhanced by posttraining activation of glucocorticoid-sensitive pathways in a manner similar to that described for catecholamines. As epinephrine effects on memory consolidation require activation of the amygdala (McGaugh et al., 1996), this paper will also give an overview of the amygdala's role in mediating glucocorticoid effects on memory consolidation. The evidence strongly suggests that epinephrine and glucocorticoids influence memory consolidation through activation of similar neural systems. Memory retrieval is usually impaired when glucocorticoids are administered shortly before retention testing. I will address the issue of whether these apparently dual effects of glucocorticoids on memory consolidation and memory retrieval might be related and serve to enable appropriate cognitive responses.

GLUCOCORTICOID EFFECTS ON MEMORY CONSOLIDATION

Extensive evidence from both animal and human studies indicates that memory traces are initially fragile after training and become consolidated only over time (McGaugh,

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