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Fluctuations in spatial recognition memory across the menstrual cycle in female rhesus monkeys

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Abstract

Findings are inconsistent regarding whether women's cognitive performance fluctuates across phases of the menstrual cycle, but differences in methodology and the use of reported cycle phase rather than precise hormonal measures may underlie these disparities. Studies in monkeys may help resolve these discrepant findings, since hormonal status can be reliably determined. We tested four young (5–7 years old) female rhesus monkeys daily during one entire menstrual cycle on three cognitive tasks displayed on a computerized touch-screen system: a Matching to Sample task with a 30 s delay (MTS-30s), a Matching to Sample task without delay (MTS-no delay) and the spatial condition of the Delayed Recognition Span Test (spatial-DRST). Blood samples were collected at specific time intervals throughout the cycle and assayed for estradiol and progesterone in order to identify hormonal status. There was a nonsignificant trend for the MTS-30s scores to be better during the follicular and luteal phases, when estradiol levels were low, than during the peri-ovulatory phase, when estradiol levels were at their highest. MTS-no delay performance did not vary as a function of hormonal status. Spatial-DRST scores were significantly better during the follicular and luteal phases than during the peri-ovulatory phase of the cycle. These data in the female rhesus monkey support the hypothesis that spatial memory performance is sensitive to estradiol variations across the menstrual cycle, with better performance associated with low estradiol levels. © 2001 Elsevier Science Ltd. All rights reserved.

Keywords: Estrogen; Cognition; *Macaca mulatta*

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1. Introduction

Pre- and perinatal, or organizational, influences of gonadal hormones permanently affect brain organization and a variety of reproductive and nonreproductive sexually dimorphic behaviors (Goy and McEwen, 1980), including some cognitive abilities (Collaer and Hines, 1995; Williams and Meck, 1991). There is ample evidence that sex differences in cognition are largely controlled by hormonal factors (Halpern, 1992; Kimura, 1999). For example, animal studies (Williams and Meck, 1991) have shown that organizational effects of androgens and their metabolites underlie the well documented male advantage in spatial ability that characterizes many species including our own. Indirect evidence corroborating these findings in humans arises from studies of people with endocrine abnormalities. Girls with congenital adrenal hyperplasia, for example, a condition that exposes the fetus to high levels of androgens prenatally, tend to have higher spatial abilities than normal girls (Hampson et al., 1998).

In addition, sex hormones exert a modulatory or activational influence on previously organized brain patterns in adulthood (Arnold and Gorski, 1984) and include some effects on cognitive functioning. For example, changes in circulating testosterone levels modulate spatial performance in both sexes (Christiansen and Knussmann, 1987; Gouchie and Kimura, 1991; Moffat and Hampson, 1996; Neave et al., 1999; Silverman et al., 1999) and testosterone administration results in enhanced spatial ability in aged men (Janowsky et al., 1994; but see Wolf et al., 1999) and female-to-male transsexuals (Van Goozen et al., 1994, 1995; Slabbekoorn et al., 1999). Several lines of evidence indicate that estrogens may also have an activational influence on cognitive function in women. Estrogen deficiency in young women after ovariectomy (Sherwin, 1988) or temporary suppression of ovarian function (Sherwin and Tulandi, 1996) results in verbal memory deficits that can be reversed by estrogen replacement therapy (ERT). In addition, estrogen deficiency at menopause is associated with an exacerbation of age-related cognitive decline in a variety of domains (Halbreich et al., 1995) and some of the deficits can be reduced or even reversed by ERT (Sherwin, 1997; Resnick et al., 1997). ERT may also reduce a woman's risk of developing Alzheimer's Disease (Henderson, 1997) but does not prevent the cognitive decline in women affected by the disease (Mulnard et al., 2000).

Studies of the effects of estrogens on the brain have uncovered several plausible mechanisms by which estrogens could affect cognition (reviewed by McEwen and Alves, 1999; McEwen et al., 1997; Miller and Franklin, 1999). Estrogen receptors are found in nonreproductive brain regions, such as the hippocampus, cerebral cortex or amygdala (Blurton-Jones et al., 1999; Gundlah et al., 2000; Osterlund et al., 2000a,b; Register et al., 1998), which are typically involved in memory processes. Moreover, estrogens rapidly increase the number of dendritic spines and synapses in CA1 of the rat hippocampus (Woolley, 1998, 1999), enhance long-term potentiation in CA1 (Cordoba Montoya and Carrer, 1997), enhance the production of new cells in the dentate gyrus (Tanapat et al., 1999), enhance cholinergic function (Gibbs, 1998) and have a number of neurotrophic and neuroprotective effects (Brinton et al., 2000; Green and Simpkins, 2000; Wise et al., 1999). Neuroimaging studies have

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