Neurobiology of Learning and Memory 132 (2016) 29–39

Contents lists available at ScienceDirect

Neurobiology of Learning and Memory

journal homepage: www.elsevier.com/locate/ynlme

Acute stress switches spatial navigation strategy from egocentric to allocentric in a virtual Morris water maze ☆

Dustin J.H. van Gerven, Thomas Ferguson, Ronald W. Skelton *

Department of Psychology, University of Victoria, Canada

ARTICLE INFO

Article history:
Received 14 October 2015
Revised 4 May 2016
Accepted 8 May 2016
Available online 9 May 2016

Keywords:
Acute stress
Stress hormones
Spatial cognition
Navigational strategy selection
Virtual Morris water maze
Gender

ABSTRACT

Stress and stress hormones are known to influence the function of the hippocampus, a brain structure critical for cognitive-map-based, allocentric spatial navigation. The caudate nucleus, a brain structure critical for stimulus–response-based, egocentric navigation, is not as sensitive to stress. Evidence for this comes from rodent studies, which show that acute stress or stress hormones impair allocentric, but not egocentric navigation. However, there have been few studies investigating the effect of acute stress on human spatial navigation, and the results of these have been equivocal. To date, no study has investigated whether acute stress can shift human navigational strategy selection between allocentric and egocentric navigation. The present study investigated this question by exposing participants to an acute psychological stressor (the Paced Auditory Serial Addition Task, PASAT), before testing navigational strategy selection in the Dual-Strategy Maze, a modified virtual Morris water maze. In the Dual-Strategy maze, participants can chose to navigate using a constellation of extra-maze cues (allocentrically) or using a single cue proximal to the goal platform (egocentrically). Surprisingly, PASAT stress biased participants to solve the maze allocentrically significantly more, rather than less, often. These findings have implications for understanding the effects of acute stress on cognitive function in general, and the function of the hippocampus in particular.

1. Introduction

Stress is an important and ever-present aspect of our daily lives. Thus, whether, and how, stress influences cognition is an important area of study. Stress is usually considered to be caused by perceived environmental challenges which lead to a physiological response, often accompanied by emotional distress and associated with elevations in stress hormones (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007; Lupien & McEwen, 1997). So far, acute stress or stress hormones have been shown to impact many cognitive functions, including attention (Kopell, Wittner, Lunde, Warrick, & Edwards, 1970), working memory (Elzinga, Bakker, & Bremner, 2005; Lupien, Gillin, & Hauger, 1999; Oei, Everaerd, Elzinga, van Well, & Bermond, 2006), and decision making (Porcelli & Delgado, 2009; Putman, Antypa, Crysovergi, & van der Does, 2010). There have also been many studies on the effects of stress on declarative memory (for reviews see McGaugh & Roozendaal, 2002; Schwabe, Joëls, Roozendaal, Wolf, & Oitzl, 2012).

It is not surprising that stress affects declarative memory, because declarative memory is known to rely on the hippocampus (Squire, 1982), which is known to be sensitive to stress hormones (Joels & De Kloet, 1989; Lupien et al., 2002; Newcomer, Craft, Hershey, Askins, & Bardgett, 1994; see Joëls, Pu, Wiegert, Oitzl, & Krugers, 2006; Lupien & McEwen, 1997 for reviews). Stress influences hippocampal function by one or both of two routes. First, the rapid sympathetic-adrenal-medullary axis (SAM) system is engaged, activating the sympathetic nervous system, which causes adrenaline to be released, stimulating the release of norepinephrine (NE) in the brain (Joëls, Fernandez, & Roozendaal, 2011; McGaugh & Roozendaal, 2002). NE activation of the amygdala leads to the modulation of activity in a number of other neural structures, notably the hippocampus, caudate nucleus, and frontal lobes (Packard, Cahill, & McGaugh, 1994; Roozendaal, McReynolds, & McGaugh, 2004). The second, slower, hypothalamic–pituitary–adrenal (HPA) involves adrenocorticotropin-induced release of glucocorticoids (cortisol in humans, corticosterone in rats) (Tsigos & Chrousos, 2002) which in turn modulate brain activity in many areas, again including the hippocampus, caudate nucleus, and frontal lobes (Packard et al., 1994; Roozendaal et al., 2004).

☆ This research was supported in part by a scholarship to van Gerven from the Natural Sciences and Engineering Research Council of Canada (NSERC).

* Corresponding author at: Department of Psychology, University of Victoria, Box 3050, Victoria, BC V8W 3P5, Canada.
E-mail address: skelton@uvic.ca (R.W. Skelton).

http://dx.doi.org/10.1016/j.nlm.2016.05.003
1074-7427/

The link between stress and hippocampal function has led multiple researchers to investigate the possibility that hippocampal functions in particular may be affected by stress. In rats, acute stress prior to learning generally leads to impairment in hippocampal function, which is usually measured using spatial navigation tasks, especially in the classic Morris water maze (MWM); (Morris, 1981; see Cazakoff, Johnson, & Howland, 2010, for a review). In humans, much of the research has focussed on declarative memory, for which the hippocampus is critical (Squire, 1982). However, when stress is given prior to learning, some studies have found that declarative memory is enhanced by stress (e,g., Domes, Heinrichs, Reichwald, & Hautzinger, 2002; Nater et al., 2007; Payne et al., 2007; Schwabe, Bohringer, Chatterjee, & Schachinger, 2008; Smeets, Giesbrecht, Jelicic, & Merckelbach, 2007), while others have found that it is impaired by stress (e.g., Elzinga et al., 2005; Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Payne et al., 2007; Wolf, Schommer, Hellhammer, McEwen, & Kirschbaum, 2001; Zoladz et al., 2011).

Perhaps what is surprising is that there have only been a few studies investigating the effects of stress on another well-known hippocampal function – spatial navigation. The key hippocampal function in spatial navigation is allocentric (world-centred, cognitive map) navigation (O'Keefe & Nadel, 1978). The alternate strategy for spatial navigation is egocentric (cue or response based) navigation, mediated primarily by the caudate nucleus (Iaria, Petrides, Dagher, Pike, & Bohbot, 2003; Maguire et al., 1998).

To date, only four studies have been conducted on the effects of stress on spatial navigation in humans. Their results have been confusing and have not always mirrored findings in rats. Three used allocentric tasks. One study found that stress impaired female navigational efficiency (Thomas, Laurance, Nadel, & Jacobs, 2010), while another found that stress enhanced male efficiency (Duncko, Cornwell, Cui, Merikangas, & Grillon, 2007). A third study found no effect whatsoever (for either sex) (Klopp, Garcia, Schulman, Ward, & Tartar, 2012). A fourth study recently tested participants in a forced allocentric task and also in a forced egocentric task (Guenzel, Wolf, & Schwabe, 2014). They found that, contrary to findings with rodents, stress did not impact navigational efficiency in either task, although it impaired later memory for the goal locations in the egocentric task, but not the allocentric task. One problem with all tasks that force one strategy or another is that although they measure efficiency in either egocentric or allocentric navigation, they cannot tell us about strategy selection or preference. Furthermore, they cannot show whether stress shifted navigation from one strategy to another.

There is some evidence that acute stress can cause a shift in navigational strategies. Schwabe, Schächinger, de Kloet, and Oitzl (2010) showed that when given a choice between solving a spatial task egocentrically or allocentrically, stress caused rodents to preferentially solve the task egocentrically. Furthermore, those mice that switched to an egocentric strategy showed no performance impairment, whereas those mice that continued to navigate allocentrically showed deficits. In other words, when the mice were able to switch from a navigational system that was impaired by stress to one that was functioning normally, navigation was normal. This is consistent with the idea that stress may lead to a switch from hippocampus-based to caudate-based navigation. The same lab found consistent results in humans. Schwabe et al. (2007) tested human participants in a task that required participants to learn the correct card of 4 on a doll-sized table in a model room (1 cubic foot). By moving the one proximal cue on the table, they determined whether the participants had adopted a cue-based ("stimulus–response") or a configuration-based ("spatial") strategy. They found that stress increased the likelihood that participants would choose a stimulus–response strategy. In the same paradigm, Schwabe, Oitzl, Richter, and Schächinger (2009) found that exogenous cortisol that led to high salivary cortisol levels produced the opposite effect; i.e., an increased (rather than decreased) the usage of an allocentric strategy. Although the first finding was consistent with the rodent findings, their task did not require navigation, and it is not clear whether it required the formation or use of a cognitive map. Indeed, acquisition and use of static spatial relations such as these have been more often attributed to the parietal lobe (Colby & Goldberg, 1999; Karnath, 1997). To date, no study has directly investigated whether stress can affect human navigational strategy selection in large-scale space.

The main purpose of the present study is to determine whether acute stress influences spatial navigation strategy selection. The stressor was an extra-stressful version of the clinical neuropsychological tool, the Paced Auditory Serial Addition Task (PASAT; Gronwall, 1977). Similar modified versions of the PASAT have been successfully used to induce a physiological stress response (Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2010; Lejuez, Kahler, & Brown, 2003; Mathias, Stanford, & Houston, 2004; McHugh, Behar, Gutner, Geem, & Otto, 2010) and affect (Holdwick Jr. & Wingenfeld, 1999). Navigational strategy selection and navigational efficiency were tested using a virtual, dual-strategy version of the Morris water maze (Morris, 1981) which we previously showed to be able to detect differences in navigational strategy (van Gerven, Schneider, Wuitchik, & Skelton, 2012). Based largely on Schwabe et al.'s (2010) study on strategy selection under stress in rodents, we expected that acute stress from the PASAT, and the associated stress response, would cause an impairment in hippocampal function, and that this would be reflected in a bias away from allocentric navigation and towards egocentric navigation in our Dual-Strategy maze.

The second purpose was to examine whether changes in navigational strategy selection could be tied to the physiological stress response. To do so we measured HPA axis activation directly using salivary cortisol (Kirschbaum & Hellhammer, 1989), and SAM axis activation indirectly using heart rate (e.g. Espin et al., 2013; Meyer, Smeets, Giesbrecht, Quaedflieg, & Merckelbach, 2013; Zoladz et al., 2014), blood pressure (e.g. Elzinga et al., 2005; Zoladz et al., 2011), and skin conductance (Duncko et al., 2007).

The third purpose of the present study was to investigate whether two other biological factors may modulate the effects of stress on navigation. One factor was sex. Men exhibit a higher cortisol response to acute stress than women (Kajantie & Phillips, 2006; Kudielka & Kirschbaum, 2005; Sauro, Jorgensen, & Teal Pedlow, 2003). Furthermore, female sex hormones may protect against the influence of glucocorticoids on the hippocampus (Wolf et al., 2001). The other factor is time of day (TOD). One previous study found that the effects of stress on a hippocampal function (declarative memory) differed between morning and afternoon (Maheu, Collicutt, Kornik, Moszkowski, & Lupien, 2005). The authors proposed that this was mediated by the natural, circadian fluctuations in cortisol which manifests as a high level in the morning (shortly after rising) and a steady decline for several hours (Edwards, Clow, Evans, & Hucklebridge, 2001; Haus, 2007; Kirschbaum & Hellhammer, 2000). In other words, they speculated that the stress induced cortisol sums with the natural cortisol to produce a different response in the morning than in the afternoon. In the present study, we tested at two times of day (8 am and 9:30 am) where the cortisol levels were expected to be quite different (Edwards et al., 2001) but other circadian factors would be relatively equal. A TOD effect was also observed with exogenous cortisol (Het, Ramlow, & Wolf, 2005). In sum, we expected that the influence of stress on strategy selection would be stronger for male participants than female participants, and stronger earlier in the morning (when circadian cortisol levels are higher; Edwards et al., 2001) than later in the morning.