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Social memory, social stress, and economic behaviors

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Abstract

Social memory plays a pivotal role in social behaviors, from mating behaviors to cooperative behaviors based on reciprocal altruism. More specifically, social/person recognition memory is supposed, by behavioral-economic and game-theoretic analysis, to be required for tit-for-tat like cooperative behaviors to evolve under the *N*-person iterated prisoner's dilemma game condition. Meanwhile, humans are known to show a social stress response during face-to-face social interactions, which might affect economic behaviors. Furthermore, it is known that there are individual differences in a social stress response, which might be reflected in individual differences in various types of economic behaviors, partially via different capacities of social memory. In the present study, we investigated the acute effects of social stress-induced free cortisol (a stress hormone) elevation on hippocampus-dependent social memory by utilizing the Trier social stress test (consisting of a public speech and a mental arithmetic task). We also examine the correlation between an economic behavior-related personality trait (i.e., general trust scale) and social stress-induced cortisol elevations. We found that (1) social stress acutely impairs social memory during social interaction and (2) interpersonal trust reduces social stress response. Together, interpersonal trust may modulate economic behaviors via stress hormone's action on social cognition-related brain regions.

1. Introduction

Social memory/person recognition memory plays a pivotal role in social cooperation. Specifically, social/person recognition memory is required for tit-for-tat like cooperative behaviors to evolve in *N*(>2)-person iterated prisoner's dilemma situations [1]. The most important type of human social memory might be a face-name association memory, which is mediated principally in the hippocampal subregions especially in the face-name encoding phase [16]. In addition, other types of person recognition memory, such as person-episode association memory, are also mediated via hippocampus-dependent neural circuits [8]. Meanwhile, animals including humans are known to show a social stress response during social interactions, a phenomenon that can be activated by a laboratory psychosocial stressor. The social stress response is indicated by an elevation in stress hormone levels via the activation of the hypothalamic–pituitary–adrenal (HPA) axis [5]. Furthermore, it is known that there are individual differences in the HPA reactivity in response to social stress exposure [5], which might be reflected in individual differences in various types of social behaviors, partially via different capacities of social memory.

Many studies have proposed that stress hormones (e.g., cortisol in humans and corticosterone in rodents) acutely and chronically modulate hippocampal neuronal functions in a concentration-dependent biphasic manner: low levels of a stress hormone elevation mainly activate mineralocorticoid receptor (MR, Type I receptor)-mediated neuronal pathways, which result in enhanced synaptic potentiation. Conversely, high levels of a stress hormone elevation strongly activate glucocorticoid receptor (GR, Type II receptor)-mediated neuronal pathways, which result in suppressed synaptic potentiation. Therefore, the relationship between stress hormones and synaptic plasticity might have an inverted-U shape [6,2].

In the emerging field of neuroeconomics [3], cooperative socioeconomic behaviors in game-theoretic experiments have recently been attracting much attention [9,7]. Studies in social psychology and experimental economics have revealed that individual differences in cooperative socioeconomic behaviors in game-theoretic experiments are strongly

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related to the personality trait of general trust scale, which measures individual's preference to trust and/or cooperate with other people in general [14]. Notably, cross-national differences in individual's interpersonal trust have been shown, by econometric analysis, to correlate with cross-national differences in income growth per capita [15].

This study was conducted to investigate (i) the acute effects of social stress-induced cortisol elevation on a face-name association memory (an important type of human social memory) in young healthy male subjects, by utilizing the Trier social stress test (TSST, consisting of a public speech and a mental arithmetic task) [5] and (ii) a correlation between interpersonal trust and social stress-induced cortisol elevation.

2. Methods

In the present study, 30 healthy male students aged 19–25 years (average: 20.4 years) participated. The participants were randomly assigned to either the control or the stress condition. The control condition consisted of social memory testing alone, while the stress condition consisted of both TSST and social memory testing. Smokers, drinkers, and subjects taking medicine, or suffering from acute or chronic hormonal dysregulations, atopic-, psychosomatic, or psychiatric diseases were excluded. To avoid the effects of a menstrual hormonal cycle, only male subjects were selected. The participating subjects were informed that the study involved the relationship between neuroendocrine measurements and cognitive performance. They were given instructions not to (i) drink anything containing alcohol or caffeine from 8.00 p.m. on the day before their participation, (ii) eat/drink anything except water, nor do physical exercises within 1 h prior to their participating in the experiment. The subjects had no prior experience with the TSST. They signed an informed consent form and received payment for participation. The effect of circadian hormone rhythms was minimized by conducting all sessions between 2.30 and 5.30 p.m.

For the subjects to be exposed to a social stressor, the TSST procedure, consisting of both a public speech (5 min, a serious-minded self-introduction) and a mental arithmetic task (5 min, serial subtractions of the number 13 from 1022) in front of both an audience (consisting of three male experimenters wearing white lab-coats, having pencils and paper for noting the evaluation of subjects' speech/arithmetic ability, sitting in front of the subject) and a video camera was employed [5]. Twenty subjects (belonging to the "stress group") participated in this procedure, while the remaining 10 subjects served as controls. The control condition consisted of social memory testing alone, immediately after saliva sampling, without TSST. We assessed the participants' perceived stress scores, employing a standard questionnaire method, with a visual analogue scale (VAS, 0–100%). On arriving, there was no significant difference in the VAS scores between the control (45 ± 6%, $n = 10$) and the stress group (43.3 ± 3.9%, $n = 20$). The stress group's perceived stress significantly increased after TSST (77.5 ± 3.7%, $n = 20$). Saliva samples for the assessment of free salivary cortisol were collected immediately before the onset of the social stress sessions as well as 13 min after the cessation of TSST, when cortisol levels peak [5]. In order to additionally examine the relation between resting cortisol levels and social memory performance of the controls, saliva samples were also collected from the control group, on arriving, immediately before the memory testing.

To test the subjects' social memory performance, a face-name association task was employed [16]. A face list was compiled of the pictures of 20 Japanese faces (10 male and 10 female faces), which were unfamiliar to subjects. A name list of 20 fictional Japanese family names was compiled.

After the subjects were given explicit instructions to try to remember which face was associated with which name for later testing, the 20 face-name pairs were sequentially displayed on a computer monitor (5 s for each pair). This face-name presentation procedure was repeated once again, in order to help the subjects memorize the face-name pairs. After 90 s display of the instruction regarding the following memory testing task on the computer monitor, the previously presented 20 face pictures were again randomly and sequentially displayed, without the paired name (15 s for each face) and the subjects were required to match each presented face and each name on a prepared form. The performance on this task ("social memory performance") was expressed in terms of the percentage of their correct answers [=100 × (the number of correct answers of the face-name association task)/(the number of the totally presented face-name pairs (=20))]. For the subjects belonging to the stress group, the social memory test was conducted three minutes (the shortest time interval with experimental feasibility) after the termination of the social stress sessions. For the controls, the social memory test was conducted immediately after the saliva sampling, without TSST.

Saliva was collected from the subjects using Salivette (Sarstedt, Rommelsdorf, Germany) collection devices. All procedures determining salivary cortisol levels were conducted using the standard protocols [4]. It should be noted that two participants had the resting cortisol levels higher than the upper limit of a normal concentration range for resting cortisol levels [4]. Therefore, their data were excluded from further analysis, following the standard analysis criterion [4]. Consequently, the data from a total of 28 subjects (consisting of the10 subjects for the controls and the 18 subjects for the stress group) were analyzed.

Statistical analysis was performed as described below. First of all, an unpaired *t*-test, not based on the assumption of equal variances, was utilized to test the significance of observed differences between the conditions. Second, we divided the 18 subjects (the stress group) on a post hoc basis into three groups:

Table 1
Characteristics of cortisol levels

	Basal cortisol level (nmol/L)	Post-stress cortisol (nmol/L)	Cortisol change (nmol/L)
Stress group ($n = 18$)	7.3 ± 2	11.4 ± 1.9*	4.1 ± 1.6
Non-responders ($n = 8$)	9.7 ± 3	6.3 ± 2	−3.4 ± 1.2
Responders ($n = 10$)	5.4 ± 1.6	15.7 ± 2.1*	10.2 ± 2
Low responders	4.7 ± 1.9	9.4 ± 1.8	4.7 ± 0.8
High responders	6.1 ± 1	21.8 ± 2.4*	15.7 ± 3

Data are expressed as mean ± S.E.M.
* Significantly higher than the controls' basal cortisol (6.1 ± 1 nmol/L, $n = 10$) ($P < 0.05$).

(i) "Non-responders"; those who did not respond to the acute socially stressful condition (no change or a decrease (<0) in cortisol levels).
(ii) "Low responders"; those who ranked in lower 50% in terms of cortisol elevations (>0), within responders.
(iii) "High responders"; those who ranked in upper 50% in terms of cortisol elevations (>0), within responders.

Note that "responders" (=(ii) + (iii)) were all those who responded with an increase (>0) in cortisol levels. The rationale for this division is as follows: First, the existence of individual difference in coping with socially stressful situations does not predict a uniform neuroendocrine response to the acute social stressor via the HPA axis. Second, as noted earlier, there may be an involvement of two distinct types of stress hormone receptors, i.e., MR-dependent (low cortisol elevations) and GR-dependent (high cortisol elevations). Finally, Pearson's correlation analysis was utilized to examine the relationship between individual resting cortisol levels/cortisol elevations and social memory performance. In order to examine the relationship between subjects' interpersonal trust and social stress response, the general trust scale scores [14] were assessed before the social stress exposure.

Significance level was set at 5% throughout. Data are expressed as mean ± S.E.M. Furthermore, non-parametric statistical analysis was also performed, revealing essentially the same results. All statistical procedures were conducted with R language (R foundation for Statistical Computing) and SAS (SAS Institute, North Carolina, USA).

3. Results and discussion

Characteristics of salivary cortisol levels for each subgroup within the stress group are summarized in Table 1. Cortisol levels significantly higher than the controls' resting cortisol level (6.1 ± 1 nmol/L, $n = 10$) are denoted with asterisks. The average resting cortisol levels of all groups were within a normal concentration range [4]. There was no significant difference between salivary cortisol levels in the control group and the resting (pre-stress) salivary cortisol levels in the stress group. After the social stress exposure, the salivary cortisol level of the stress group significantly increased and reached 11.4 ± 1.9 nmol/L ($n = 18$). The social memory performance of the controls was 54.5 ± 9.4%, while the social memory performance of the stress group was 41 ± 5.4%. This difference in the social memory performance between the control and the stress group did not reach statistical significance ($P > 0.05$). These results are in line with the previous finding that differences in verbal memory performance between a control and a stress group did not reach statistical significance, because of the individual differences in HPA reactivity [13]. It should be noted that there was no significant correlation between the resting cortisol levels and social memory performance in the control group, implying that chronic cortisol levels did not significantly affect the social memory performance.

Next, as noted above, the 18 subjects belonging to the stress group were divided into three groups, according to their cortisol response: the high responders group ($n = 5$), the low responders group ($n = 5$), and non-responders group ($n = 8$). The social stress-induced salivary cortisol responses [=(salivary cortisol levels after the social stress exposure) − (salivary cortisol levels before the social stress exposure)] were 15.7 ± 3, 4.7 ± 0.8, and −3.4 ± 1.2 nmol/L, for high, low, and non-responders, respectively (Table 1). The social memory performances of the high and low responders were 25 ± 6.5%, and 58 ± 9.7%, respectively. The high responders' social memory performance was significantly lower than the controls' social memory performance ($P < 0.05$, Fig. 1). This indicates that the social stress-induced high cortisol elevation acutely impaired the subjects' social memory. On the other hand, the low responders' social

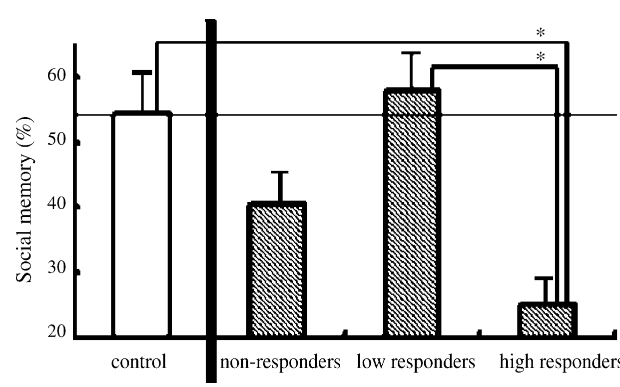

Fig. 1. Acute effect of social stress-induced cortisol elevation on social memory performance for each group. The vertical axis indicates social memory performance [=100 × (the number of correct answers of the face-name association task)/(the number of the totally presented face-name pairs (=20))]. Data are expressed as mean ± S.E.M. Asterisk (*) indicates significantly different ($P < 0.05$).

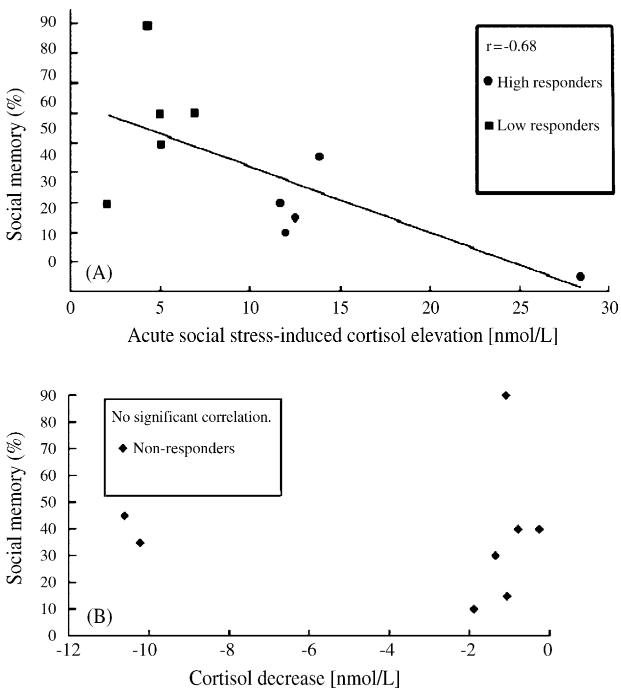

Fig. 2. (A) Scatter plot of acute social stress-induced cortisol elevations [=(salivary cortisol levels after the social stress exposure) − (salivary cortisol levels before the social stress exposure)] (>0) and social memory performance [=100 × (the number of correct answers of the face-name association task)/(the number of the totally presented face-name pairs (=20))] of the responders. A significant negative relation was observed ($r = -0.68$, $n = 10$, $P < 0.05$). (B) Scatter plot of cortisol decrease [=(salivary cortisol levels after the social stress exposure) − (salivary cortisol levels before the social stress exposure)] (<0) and social memory performance of the non-responders. No significant correlation was observed ($n = 8$, $P > 0.05$).

memory performance appeared slightly enhanced by the social stress exposure, compared to the controls; however, this enhancement was not statistically significant. This indicates that the acute memory-enhancing action of cortisol via MRs did not significantly affect social memory performance. Additionally, the non-responders' social memory performance was 40.5 ± 7.9%, which was not significantly different from the controls' social memory performance, also supporting this indication. Additionally, there was no significant difference in VAS after TSST, between high, low, and non-responders.

Because the low responders' social memory performance was not significantly enhanced by the cortisol elevations, it is supposed that GRs, which were activated by the social stress-induced cortisol elevations, mainly affected all the responders' (including both low and high responders) social memory performance. For all the responders, we therefore utilized a simple linear regression analysis (Pearson's correlation analysis) to examine the correlation between the individual cortisol elevations and social memory performance. A significant negative correlation was observed ($r = -0.68$, $P < 0.05$, $n = 10$, Fig. 2A), implying that social stress-induced cortisol elevation (>0) acutely impaired social memory in a negative, linear dose-dependent manner. A non-parametric correlation analysis, Spearman's rank correlation test, also revealed the significant negative correlation ($r = -0.67$, $n = 10$). On the contrary, in the non-responders, no significant correlation was observed ($P > 0.05$, $n = 8$, Fig. 2B). This implies that neuronal mechanisms, underlying individual differences in the non-responders' social memory performance, might be independent of glucocorticoid-dependent neuronal pathways. Additionally, for the entire stress group ($n = 18$), if not divided into responders and non-responders, neither linear nor non-linear (quadratic) regression analysis revealed significant correlation between cortisol change (both negative and positive) and social memory performance, also supporting this conclusion. Moreover, no significant correlation between the resting cortisol levels and social memory performance in the stress group ($n = 18$, $P > 0.05$) was observed, again implying that chronic cortisol levels did not significantly affect the present type of social memory. These findings are in line with other studies on the acute effect of stress on non-social, hippocampus-dependent memory in young male subjects [13].

Taken together, we observed that (i) the high responders demonstrated significantly impaired social memory performance, and (ii) in the responders, there was a negative correlation between the individual cortisol elevations and social memory performance. Our results indicate that social stress-induced cortisol elevation acutely impaired social memory performance in men [11]. Observed individual differences in the resting cortisol levels and social memory performance under the social stress condition may be due to individual differences in HPA reactivity, personality traits, and the level of GR/MR expression in the hippocampus [10].

Furthermore, we observed that there was a negative correlation between subjects' interpersonal trust [14] and social stress-induced cortisol elevation in the responders ($r_s = -0.64$), indicating that subjects with high levels of interpersonal trust had reduced social stress response during a social stress exposure. Collectively, interpersonal trust might possibly enhance social cooperation via better social memory due to lowered acute social stress actions [12] during a face-to-face social interaction, which would result in high levels of an economic growth [15].

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