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Research article

Visually guided equivalence learning in borderline personality disorder

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ABSTRACT

The hallmark symptoms of borderline personality disorder are maladaptive behavior and impulsive emotional reactions. However, the condition is occasionally associated with cognitive alterations. Recently, it has been found that the function of the basal ganglia and the hippocampi might also be affected. Hence, deterioration in learning and memory processes associated with these structures is expected. Thus, we sought to investigate visually guided associative learning, a type of conditioning associated with the basal ganglia and the hippocampi, in patients suffering from borderline personality disorder. In this study, the modified Rutgers Acquired Equivalence Test was used to assess associative learning in 23 patients and age-, sex-, and educational level-matched controls. The acquisition phase of the test, which is associated primarily with the frontostriatal loops, was altered in patients with borderline personality disorder: the patients exhibited poor performance in terms of building associations. However, the retrieval and generalization functions, which are primarily associated with the hippocampi and the medial temporal lobes, were not affected. These results corroborate that the basal ganglia are affected in borderline personality disorder. However, maintained retrieval and generalization do not support the assumption that the hippocampi are affected too.

1. Introduction

Borderline personality disorder (BPD) is a mental health disorder characterized by maladaptive behavior (long-term pattern of unstable interpersonal relationships and distorted sense of self) and impulsive emotional reactions. Its prevalence rate is 2–3% in the adult population, making it is the most common personality disorder [1]. The disorder usually begins during young adulthood and it has a significantly higher prevalence in women than in men [2].

The neural correlates of BPD have not been fully elucidated. Based on neuroimaging studies, the basal ganglia, the orbitofrontal cortex, the amygdalae, and probably the hippocampi are affected [3, 4, 5, 6, 7]. Due to dysfunction of these brain structures, patients with borderline personality disorder may exhibit altered cognition as compared to persons free of the disorder [8, 9, 10]. However, no information is available about the associative learning abilities of these patients.

Associative learning is an ancient learning function, which is associated with the function of the frontal cortex, the basal ganglia and the hippocampi. The visually guided Rutgers Acquired Equivalence Test [11] assesses this specific type of learning. The test is divided into two main phases: the acquisition phase and the test phase. The acquisition phase relies on the function of the basal ganglia. Hence, association building between two different visual stimuli with the help of feedback about the correctness of responses can be evaluated. Meanwhile, the test phase, which does not involve feedback, mainly depends on the function of the hippocampi and the medial temporal lobe. The test phase is further divided into two parts: retrieval and generalization. During the retrieval part, previously learned associations are presented, while during the generalization part, the task of the test subject is to make hitherto not learned associations which are predictable from what has already been learned.

Equivalence learning was investigated in several psychiatric and neurological disorders, including Parkinson's disease, Alzheimer's disease, schizophrenia, obsessive compulsive disorder, and migraine without aura. These conditions are characterized by the dysfunction of the basal ganglia and hippocampi [11, 12, 13, 14, 15, 16]. As mentioned before, these structures are thought to be dysfunctional in BPD too [11, 12, 13, 14, 15, 16]. However, it is not known whether this also shows in

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the related cognitive processes of BPD patients, such as associative learning. This is what we sought to investigate in this study with the Rutgers Acquired Equivalence Test. As the literature considers both the basal ganglia and the hippocampi to be affected in BPD, we hypothesized that our patients would underperform matched controls in both the acquisition and test phases.

2. Results

In the present study, we analyzed the associative learning abilities of BPD patients without otological, opthalmological, neurological, or psychiatric comorbidities (n = 23). All participants could complete the applied Rutgers Acquired Equivalence Test.

2.1. Comparison of BPD patients and controls

In the acquisition phase of the paradigm, the patient group's performance was significantly inferior to that of the control group (Figure 1).

The median NATs of the patient and control groups were 71.0 (range: 45.0–185.0) and 50.0 (range: 42.0–95.0), respectively (Mann–Whitney U test = 146.5, p = 0.0098, effect size = 0.8077, power = 0.9525). The median ALERs were 0.084 (range: 0.0–0.227) and 0.043 (range: 0.0–0.15) for the patients and controls, respectively (Mann–Whitney U test = 162.5, p = 0.0255, effect size = 0.7845, power = 0.9520). The reaction times of the two groups in the acquisition phase did not differ. The median reaction times were 1565.44 (range: 1034.97–3402.02) ms and 1674.51 (range: 1069.54–3489.21) ms for the patients and controls, respectively (Mann–Whitney U test = 256, p = 0.860).

In the test phase of the paradigm, the performance of the patients and controls did not differ significantly, either in terms of retrieval or generalization (Figure 2).

The median RERs were 0.028 (range: 0.0–0.25) for the patient and 0.028 (0.0–0.22) for the control group (Mann–Whitney U test = 216, p = 0.275). The median GERs were 0.00 (range: 0.0–1.0) and 0.083 (range: 0.0–1.0) for the patient and control groups, respectively (Mann–Whitney U test = 220, p = 0.307). The reaction times of the two groups in the retrieval and generalization parts did not differ. During the retrieval part of the test phase, the median reaction times of the patient and control groups were 1934.37 (range: 1106.66–3728.59) ms and 1693.50 (range: 1145.46–2838.23) ms, respectively (Mann–Whitney U test = 258, p = 0.895). In the generalization part, the median reaction times were 2213.21 (range: 1223.82–8549.00) ms for the patient group and 2309.68 (range: 1158.50–10883.36) ms for the control group (Mann–Whitney U test = 235, p = 0.879).

In the patient group, we also calculated the correlation (Pearson's r) between test performance and the time elapsed since the diagnosis. At the time of testing, the patients had been diagnosed with BPD for a mean of 12.9 years (±9.9 years). None of the investigated parameters (NAT, ALER, RER, GER and RTs) showed significant correlation with the time elapsed since the diagnosis (p > 0.05).

2.2. Performance of BPD patients according to medication status

In the acquisition phase of the paradigm, the performance of the medicated subgroup did not differ from that of the non-medicated subgroup. The median NATs of the medicated and non-medicated subgroups were 69.0 (range: 45.0–133.0) and 80.0 (range: 46.0–185.0), respectively (Mann–Whitney U test = 47, p = 0.4196). The median ALERs were 0.077 (range: 0.0–0.218) for the medicated subgroup and 0.117 (range: 0.043–0.227) for the non-medicated subgroup (Mann–Whitney U test = 36, p = 0.129).

Medication status did not make a significant difference in the test phase either. The median RERs were 0.028 (range: 0.0–0.167) for the medicated subgroup and 0.056 (range: 0.0–0.250) for the non-medicated subgroup (Mann–Whitney U test = 48.5, p = 0.468). The median GERs were 0.00 (range: 0.0–1.0) and 0.00 (range: 0.0–0.750) for the medicated and non-medicated subgroups, respectively (Mann–Whitney U test = 56.5, p = 0.830).

3. Discussion

To our knowledge, this study has been the first to investigate the visually guided associative learning abilities of BPD patients. The results suggest that BPD patients experience difficulties with equivalence learning (making pairs by association), but not with the retrieval of already learned associations or even making inferences from them regarding previously not learned associations.

The results of studies about the impairment of implicit and explicit learning functions in BPD patients are controversial. Earlier studies found no alterations in implicit statistical learning [17] and procedural memory consolidation in patients with BPD [18]. However, primary implicit acquired equivalence learning, which is mainly correlated with the function of the basal ganglia, was found to be significantly altered in the BPD group of our study. Similarly, a comprehensive clinical and neuropsychological study revealed deficits in this patient group in visually guided functions such as visual memory, visuospatial abilities, and executive functions [19]. In contrast, the psychophysical part of a functional magnetic resonance imaging (fMRI) study found no significant difference in visually guided episodic and semantic memory retrieval between BPD patients and controls. However, a stronger cortical activation was required for the same performance in the patient group [20]. Verbal and visual episodic memory appear to be spared in BPD [21], but these patients exhibit poor performance in the go/no-go task, which indicates response inhibition impairment [22].

In the current study, we applied the modified version [14] of the original Rutgers Acquired Equivalence Test [11, 23]. The test was developed to investigate the visually guided associative learning in healthy humans and those with various psychiatric and neurological disorders. Its acquisition phase tests association learning between two independent visual stimuli, which is considered to depend on the intact function of the basal ganglia [11, 23]. Therefore, this phase is assumed to

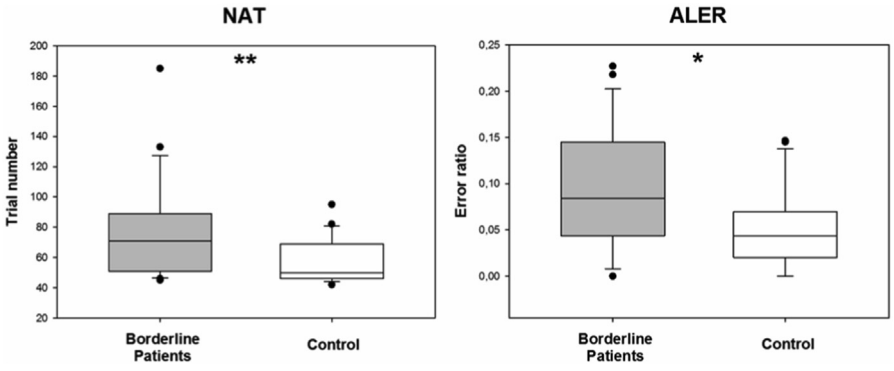

Figure 1. Performance in the acquisition phase. NAT: the number of trials necessary for the completion of the acquisition phase. ALER: the ratio of incorrect choices during the acquisition trials. The lower margin of the boxes represents the 25th percentile. The line within the boxes marks the median, and the upper margin of the boxes indicates the 75th percentile. The error bars (whiskers) above and below the boxes denote the 90th and 10th percentiles, respectively. The dots over and under the whiskers indicate the extreme outliers. *: p < 0.05, **: p < 0.01.

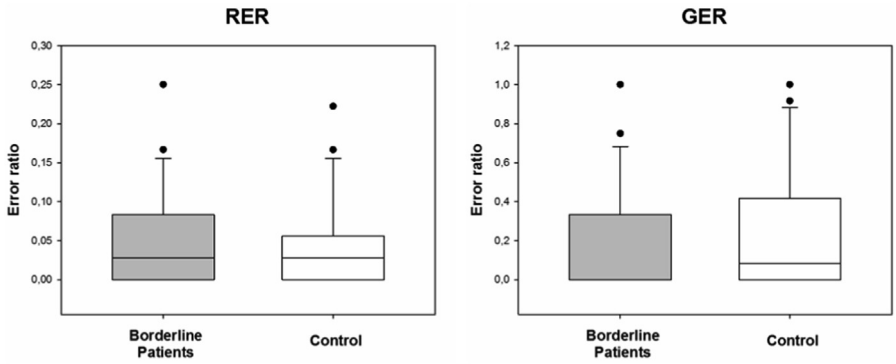

Figure 2. Performance in the test phase. RER: error ratios in the retrieval phase. GER: error ratios in the generalization phase. The conventions are the same as in Figure 1.

test the adequate functioning of the basal ganglia. Accordingly, poor performance has been reported in Parkinson's disease [11], Tourette syndrome [24], and adult migraine [14]. The test phase focuses on functions that are assumed to be mediated by the hippocampi and the medial temporal lobes: retrieval and generalization based on the retrieved information [23, 25]. Poor performance in the test phase has been reported in hippocampus–medial temporal lobe injury [23, 26], Alzheimer's disease [12], and adult migraine [14].

In this study, we have described the cognitive performance of a group of BPD patients without any neurological and/or psychiatric comorbidity (including substance abuse), as assessed by the Rutgers Acquired Equivalence Test.

The results of the acquisition phase (NAT, ALER) indicate that BPD patients found it more difficult to build associations between independent visual stimulus pairs. This finding may be interpreted as a behavioral indicator of suboptimal basal ganglia function in BPD, and so it corroborates the results of studies that suggest that the basal ganglia are affected in this personality disorder [6, 7, 8, 9].

A comparison of reaction times did not reveal differences between patients and controls, which suggests that the difference found in the acquisition phase did not stem from impaired response inhibition in the patient group (which would be indicated by significantly shorter reaction times). This is not necessarily evidence against the presence of impaired response inhibition in this patient population as suggested by Rentrop and colleagues [23], but even if it is a stable feature of BPD patients, it did not influence their performance in this task.

In contrast to the acquisition phase, however, no significant difference was found between patients and controls in the test phase. That is, BPD patients retrieved the previously learned associations and generalized the previously acquired rule of association to new stimulus pairs just as efficiently as controls. In fact, the generalization performance of BPD patients was slightly superior to that of controls, which raises the possibility that in BPD and in this specific task, the hippocampi may function somewhat more efficiently. While the difference was not significant, and we definitely do not have enough data to draw a firm conclusion regarding this issue, it must be noted that such a compensatory function of the hippocampi has been reported in other studies regarding learning [27, 28]. Therefore, as this phase of the test is assumed to depend on the hippocampi, the results may be interpreted as evidence against the hippocampi being affected in BPD - at least, if there is hippocampal involvement, it does not interfere with retrieval and generalization in the context of equivalence learning.

We also considered some potential limitations when interpreting the results of this study. The first one is the relatively low number of participants. This, however, is only a prima facie limitation and we argue that it did not interfere with the generalizability of the results. On one hand, this low number of participants was the result of the strict application of the diagnostic criteria and that substance abuse and neurological/psychiatric comorbidity were exclusion criteria. This way, it became possible to focus on the effect of BPD itself. On the other hand, the statistical tests that returned significant results had a high statistical power (and also a large effect size) so, in this case, a seemingly small sample size did not result in poor statistical power. All in all, the small size of the sample in itself cannot put the validity and generalizability of our results in question.

Another concern was that medications might affect the performance of BPD patients. In an attempt to exclude this possibility, we compared the performance of medicated and non-medicated patients. No difference was found between these subgroups in any of the studied parameters. However, it must be added that splitting the patient group into two subgroups resulted in quite small subsamples, so we advise against interpreting this result as evidence for the complete absence of such an effect.

4. Materials and methods

4.1. Participants

In total, 23 patients with borderline personality disorder (18 women and 5 men, mean age: 28.9 ± 9.6 [range: 18–55] years, median education level: 3.0 [range: 1.0–4.0]) were enrolled in this study. The educational levels were as follows: 1–elementary school, 2–secondary school, 3–high school, 4–university. Inpatients and outpatients from the Department of Psychiatry (Faculty of Medicine, University of Szeged) were recruited. The patients were diagnosed by psychiatrists at the hospital according to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition [2]. All patients were diagnosed with borderline personality disorder. Patients with otological, ophthalmological, neurological, or psychiatric comorbidities (including substance abuse) were not eligible for the study. In total, 15 patients received medications (see below). From our database, 23 matched healthy controls (18 women and five men, mean age: 28.7 ± 9.2 [range: 18–53] years, median education level: 3.0 [range: 2.0–4.0]) were identified and individually matched to the patients based on sex, age (difference $\leq$2 years), and education level. The comparison of the demographic data revealed no differences between the patient and control groups (Kruskal–Wallis test, $p > 0.05$) (Table 1).

Prior to testing, the Ishihara plates were used to rule out color blindness in both groups. Only subjects with normal color vision were included in the study.

None of the participants received financial compensation for their participation, and all patients provided written informed consent prior to the start of the study. This research was performed in accordance with the tenets of the Declaration of Helsinki, and it was approved by the Regional Ethics Committee for Medical Research at the University of Szeged, Hungary (50/2015-SZTE).

Fifteen of the 23 patients received several types of medication. Seven of the fifteen patients received monotherapy as follows: two patients received H1 antihistamine (hiroxizine), three patients received either of

Table 1. Demographic data of the patients and controls.

Group	Number of cases	Female/male	Age, mean (years)	Age, range: (years)	Educational level median (range)
All patients	23	18/5	28.9 ± 9.6	18–55	3.0 (1.0–4.0)
All controls	23	18/5	28.7 ± 9.2	18–53	3.0 (2.0–4.0)
Medicated patients	15	12/3	31.2 ± 10.7	18–55	3.0 (1.0–4.0)
Controls matched to medicated patients	15	11/4	30.7 ± 10.1	18–53	3.0 (2.0–4.0)
Unmedicated patients	8	6/2	24.6 ± 5.4	18–34	3.0 (3.0–4.0)
Controls matched to unmedicated patients	8	7/1	24.9 ± 5.6	18–35	3.5 (3.0–4.0)

three serotonergic medications (escitalopram, fluoxetine, vortioxetine) and two patients received antipsychotics (olanzapine, aripiprazole). Further five of the fifteen patients received a combination of two agents: two of them received the combination of an SSNRI (venlafaxine) and a GABA agonist (benzodiazepine or alprazolam); one of them received a combination of two types of GABA agonists (benzodiazepine and imidazopiridine); one of them received a combination of a GABA agonist (alprazolam) and a mood stabilizer (lamotrigine); and one of them received the combination of an SSRI (escitalopram) and an atypical antipsychotic (quetiapine). Finally, three of the fifteen medicated patients received three agents in combination: a GABA agonist (benzodiazepine, imidazopiridine or alprazolam), an SSNRI (duloxetine or venlafaxine) and a third agent, which was either a melatonine receptor agonist (agomelatine) or an atypical antipsychotic (olanzapine).

At the time of testing, the patients have taken their medications for a mean of 9.21 (±6.29) years (median: 9 years, range: 1–20 years, lower quartile: 4 years, upper quartile: 14 years).

4.2. The learning paradigm

Testing was carried out according to Myers and co-workers, according to the method known as the Rutgers Acquired Equivalence Test [11]. The testing software, which was originally prepared for iOS was rewritten in Assembly (for Windows). Stimuli were presented and responses were recorded with a desktop computer with a CRT screen. The testing sessions took place in a quiet room with the subjects sitting at a standard distance from the computer screen (114 cm). One subject was tested at a time and no time limit was set. Figure 3 shows a schematic representation of the paradigm.

The visual stimuli referred to as antecedents were cartoon faces of a woman (A1), a girl (A2), a man (B1) and a boy (B2). The consequents were yellow (X1), red (X2), green (Y1) and blue (Y2) fish.

During a trial, the participant was shown an antecedent (a face) and two consequents (a pair of fish of different color) and asked to choose one of the latter by pressing one of two buttons on the keyboard marked as

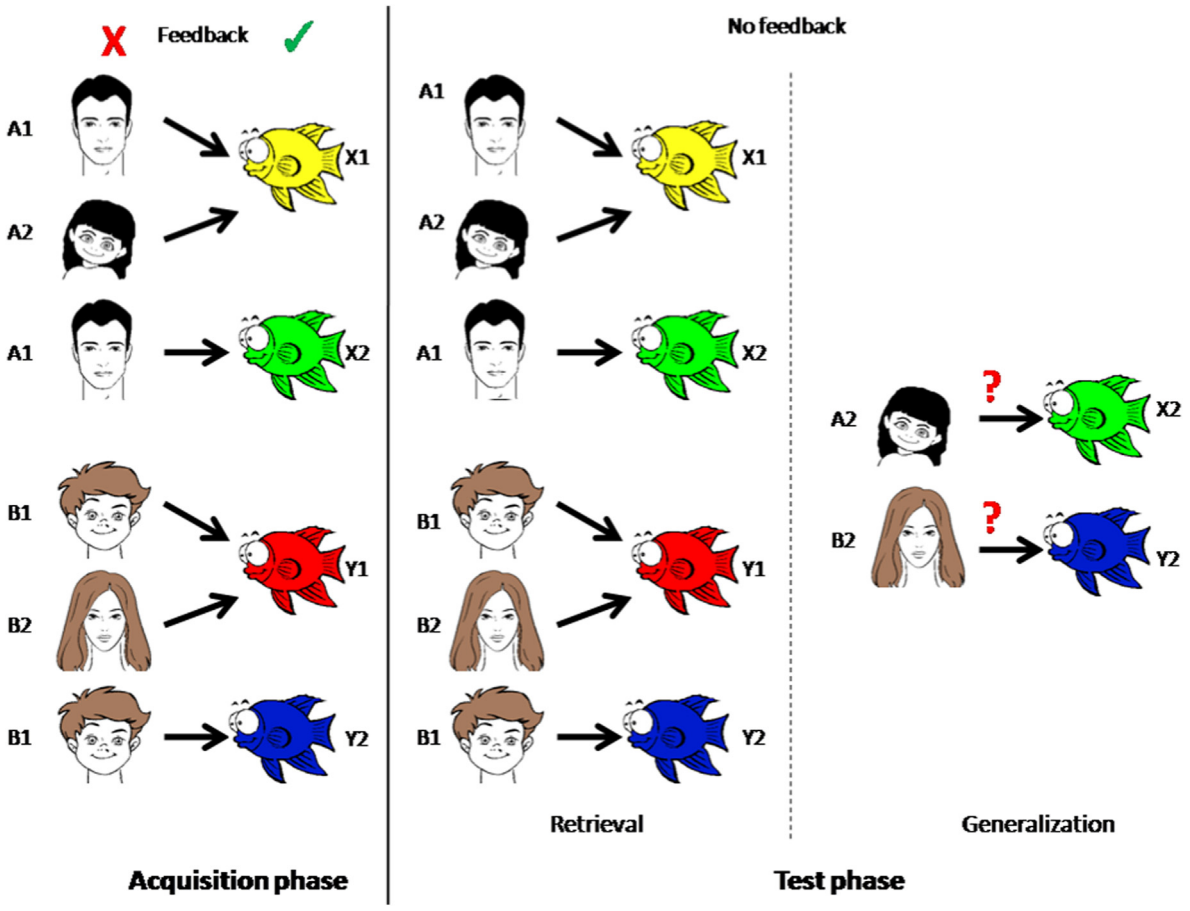

Figure 3. A schematic representation of the applied visually guided associative learning paradigm.

LEFT and RIGHT. The trials are organized into two main phases: acquisition and test. The test phase is further broken down to retrieval and generalization (see below). Depending on the phase the participant was in, the choice was (acquisition phase) or was not (test phase) followed by feedback on the correctness of the choice.

During the acquisition phase, the participants learned a series of antecedent-consequent pairs in a trial-and-error manner. When antecedents A1 or A2 were shown, the correct consequent was X1. On the other hand, when antecedent B1 or B2 were presented, the correct consequent was Y1. Visual feedback on the correctness of the subject's choice was provided immediately in the form of the words CORRECT (in green) and INCORRECT (in red) displayed on the screen under the antecedent-consequent pair. This way, besides the face-fish associations, the participants also learned that the antecedent A1 (B1) is equivalent to antecedent A2 (B2) in terms of their relation to the same consequent. Next, the participants had to learn new stimulus pairs. In case of antecedents A1 and B1, the correct consequents were X2 and Y2, respectively. Of the eight possible stimulus combinations, six were presented in the acquisition phase of the equivalence learning task. New association were presented mixed with the previously learned ones. The subjects had to accomplish a certain number of correct decisions, 4 when the first association was presented, and it was increased by 2 upon the presentation of each new association that followed up to a maximum of 12. Thus, the number of trials in the acquisition phase was not constant, it depended on the effectiveness of the learning of the participants.

In the test phase, the task remained the same, but visual feedback was no longer given. During the retrieval part of the test phase the already learnt six stimulus pairs were tested. In the generalization (or transfer) part of the test phase, hitherto not presented, new stimulus pairs were also tested. These were predictable if the participant had managed to acquire the equivalence rule (antecedent A1 and A2 are equivalent upon the connected consequences, similarly B1 and B2, too). Here the participants had to choose that antecedent A2 and B2 were coupled to consequences X2 and Y2, respectively. Participants were not informed that new associations would have to be formed, too. These new stimulus pairs were mixed with the earlier ones. The number of trials in the test phase was constant for each participant. Altogether 48 trials (36 previously learned and 12 new, predictable associations) had to be completed in the test phase.

More detailed description of the paradigm can be found in our previous studies [29, 30].

4.3. Data analysis

The performance of the participants was characterized with four main parameters: the number of trials necessary for the completion of the acquisition phase (NAT), association learning error ratio (ALER), retrieval error ratio (RER), and generalization error ratio (GER). Error ratios were calculated by dividing the number of incorrect trials by the total number of trials. Reaction times were recorded for the acquisition phase, the retrieval part of the test phase and the generalization part of the test phase.

After having determined that the data were non-normally distributed (Shapiro-Wilk $p < 0.05$), comparisons between BPD patients and controls were performed with the Mann-Whitney U test. The level of significance was set at $p = 0.05$. For the descriptive characterization of the data, medians and ranges were used.

Declarations

Author contribution statement

Anett Rosu; Kálmán Tót: Performed the experiments; Analyzed and interpreted the data; Wrote the paper.

György Godó: Performed the experiments; Analyzed and interpreted the data.

Szabolcs Kéri: Conceived and designed the experiments; Contributed reagents, materials, analysis tools or data.

Attila Nagy: Conceived and designed the experiments; Contributed reagents, materials, analysis tools or data; Wrote the paper.

Gabriella Eördegh: Conceived and designed the experiments; Performed the experiments; Analyzed and interpreted the data; Wrote the paper.

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Data availability statement

Data will be made available on request.

Declaration of interests statement

The authors declare no conflict of interest.

Additional information

No additional information is available for this paper.

References

[1] M. Swartz, et al., Estimating the prevalence of borderline personality disorder in the community, J. Pers. Disord. 4 (3) (1990) 257–272.
[2] D.E. Battle, Diagnostic and statistical manual of mental disorders (DSM), Codas 25 (2) (2013) 191–192.
[3] M.M. Perez-Rodriguez, et al., Striatal activity in borderline personality disorder with comorbid intermittent explosive disorder: sex differences, J. Psychiatr. Res. 46 (6) (2012) 797–804.
[4] A. O'Neill, T. Frodl, Brain structure and function in borderline personality disorder, Brain Struct. Funct. 217 (4) (2012) 767–782.
[5] E. Lis, et al., Neuroimaging and genetics of borderline personality disorder: a review, J. Psychiatry Neurosci. 32 (3) (2007) 162–173.
[6] A. Lamers, et al., Nonacceptance of negative emotions in women with borderline personality disorder: association with neuroactivity of the dorsal striatum, J. Psychiatry Neurosci. 44 (5) (2019) 303–312.
[7] G.Y. Wang, et al., ACC GABA levels are associated with functional activation and connectivity in the fronto-striatal network during interference inhibition in patients with borderline personality disorder, Neuroimage 147 (2017) 164–174.
[8] J.G. Stewart, et al., Neurophysiological activity following rewards and losses among female adolescents and young adults with borderline personality disorder, J. Abnorm. Psychol. 128 (6) (2019) 610–621.
[9] S.K. Fineberg, et al., Differential valuation and learning from social and nonsocial cues in borderline personality disorder, Biol. Psychiatr. 84 (11) (2018) 838–845.
[10] Z. Unoka, J.R. M, Neuropsychological deficits in BPD patients and the moderator effects of co-occurring mental disorders: a meta-analysis, Clin. Psychol. Rev. 44 (2016) 1–12.
[11] C.E. Myers, et al., Dissociating hippocampal versus basal ganglia contributions to learning and transfer, J. Cognit. Neurosci. 15 (2) (2003) 185–193.
[12] N. Bodi, et al., Associative learning, acquired equivalence, and flexible generalization of knowledge in mild Alzheimer disease, Cognit. Behav. Neurol. 22 (2) (2009) 89–94.
[13] S. Keri, et al., Dissociation between medial temporal lobe and basal ganglia memory systems in schizophrenia, Schizophr. Res. 77 (2-3) (2005) 321–328.
[14] A. Oze, et al., Acquired equivalence and related memory processes in migraine without aura, Cephalalgia 37 (6) (2017) 532–540.
[15] Á. Pertich, et al., Maintained visual-, auditory-, and multisensory-guided associative learning functions in children with obsessive–compulsive disorder, Front. Psychiatr. 11 (1287) (2020).
[16] Z. Giricz, et al., Visually guided associative learning in pediatric and adult migraine without aura, Cephalalgia 41 (2020) 176–184, 0333102420958388.
[17] Z. Unoka, et al., Intact implicit statistical learning in borderline personality disorder, Psychiatr. Res. 255 (2017) 373–381.
[18] O.P. Hornung, et al., Declarative and procedural memory consolidation during sleep in patients with borderline personality disorder, J. Psychiatr. Res. 42 (8) (2008) 653–658.
[19] T. Beblo, et al., Deficits in visual functions and neuropsychological inconsistency in Borderline Personality Disorder, Psychiatr. Res. 145 (2-3) (2006) 127–135.
[20] C. Mensebach, et al., Neural correlates of episodic and semantic memory retrieval in borderline personality disorder: an fMRI study, Psychiatr. Res. 171 (2) (2009) 94–105.

[21] A.C. Ruocco, N. Bahl, Material-specific discrepancies in verbal and visual episodic memory in borderline personality disorder, Psychiatr. Res. 220 (1-2) (2014) 694–697.
[22] M. Rentrop, et al., Response inhibition in borderline personality disorder: performance in a Go/Nogo task, Psychopathology 41 (1) (2008) 50–57.
[23] C.E. Myers, et al., Dissociating medial temporal and basal ganglia memory systems with a latent learning task, Neuropsychologia 41 (14) (2003) 1919–1928.
[24] G. Eordegh, et al., Impairment of visually guided associative learning in children with Tourette syndrome, PLoS One 15 (6) (2020) e0234724.
[25] A.A. Moustafa, C.E. Myers, M.A. Gluck, A neurocomputational model of classical conditioning phenomena: a putative role for the hippocampal region in associative learning, Brain Res. 1276 (2009) 180–195.
[26] C.E. Myers, et al., Learning and generalization deficits in patients with memory impairments due to anterior communicating artery aneurysm rupture or hypoxic brain injury, Neuropsychology 22 (5) (2008) 681–686.
[27] M.T. Ullman, M.Y. Pullman, A compensatory role for declarative memory in neurodevelopmental disorders, Neurosci. Biobehav. Rev. 51 (2015) 205–222.
[28] R. Marsh, T.V. Maia, B.S. Peterson, Functional disturbances within frontostriatal circuits across multiple childhood psychopathologies, Am. J. Psychiatr. 166 (6) (2009) 664–674.
[29] G. Eordegh, et al., Multisensory guided associative learning in healthy humans, PLoS One 14 (3) (2019) e0213094.
[30] G. Braunitzer, et al., The development of acquired equivalence from childhood to adulthood-A cross-sectional study of 265 subjects, PLoS One 12 (6) (2017) e0179525.