

5

Decision making performance in Obsessive-Compulsive Disorder

Abstract

Several neuro-imaging studies in ocd have found the orbitofrontal cortex to be functionally abnormal. As these brain regions are presumed to be involved in complex decision making behaviour, studying this behaviour in ocd may provide further insight into the cognitive deficits accompanying the disorder. We therefore compared performance of 27 drug-free ocd patients and 26 healthy volunteers on the decision making task developed by Bechara and colleagues (1994).

Our results demonstrate comparable decision making behaviour in ocd patients and volunteers. Within ocd patients, risk taking was independently related to both level of anxiety and the severity of ocd symptoms. These data argue against a gross impairment of the ventromedial prefrontal cortex in ocd. However, the functions of these brain regions seem to be implicated in the disorder, possibly in a more subtle way than could be measured with this task.

Introduction

FUNDAMENTAL PROCESSING DEFICITS presumably underlie Obsessive-Compulsive Disorder (OCD). Although OCD patients are reported to be impaired on several neuropsychological tests, it is difficult to explain symptoms such as checking or ritualized hand washing from deficits on traditional tests.

OCD patients show increased levels of glucose uptake in the circuit linking the ventromedial prefrontal cortex (vmPFC) with subcortical structures (Baxter et al., 1988; Perani et al., 1995; Swedo et al., 1989) suggesting that the cognitive mechanisms associated with this circuit may be abnormal. According to the somatic marker hypothesis formulated by Damasio (1994), the vmPFC is essential for decision making. These brain regions are assumed to represent somatic states that are experienced while evaluating the emotional outcomes of actions. These representations bias decision making processes by signaling the aversive properties of response options. Indeed, patients with damage to the vmPFC show markedly impaired decision making behaviour (Bechara et al., 1994).

Clinically, OCD patients display difficulties in the regulation of emotional behaviour. In the current study, we tested the hypothesis that these deficits are accompanied by altered functioning of the vmPFC by studying performance of OCD patients on the decision making task of Bechara et al. (1994). Since cerebral metabolic activity in OCD reportedly normalizes with medication (e.g. Baxter et al., 1992), we decided to include only drug-free patients.

Methods

Subjects

Twenty-seven patients with Obsessive-Compulsive Disorder (DSM-IV) were included. Patients were drug-free for at least 4 weeks prior to testing. Comorbid depressive and anxiety symptoms were measured with the 17-item Hamilton Depression Rating Scale (HDRS) and the 14-item Hamilton Anxiety Rating Scale (HARS, Hamilton, 1960). Exclusion criteria were the presence of major medical illness, head injury, alcohol or substance abuse. All patients completed the Yale Brown Obsessive Compulsive Scale (Y-BOCS, Goodman et al., 1989) to assess severity of OCD symptoms. Twenty-six healthy volunteers were matched to the OCD group according to sex, age and intelligence as estimated with the Raven Standard Progressive Matrices (Raven, 1960) (see table 1).

Table 1 — Demographic and clinical data of subjects. Y-BOCS = Yale Brown Obsessive Compulsive Scale; HDRS = Hamilton Depression Rating Scale; HARS = Hamilton Anxiety Rating Scale.

	OCD mean (SD)	Volunteers mean (SD)
Age (<i>in years</i>)	34.9 (9.9)	31.2 (8.3)
IQ	112.5 (10.1)	113.5 (13.8)
Male : female	7 : 20	8 : 18
Length of illness (<i>in years</i>)	18.4 (12.3)	–
Y-BOCS total	25.1 (5.2)	0.0 (0.0)
HARS total	12.4 (3.7)	–
HDRS total	9.8 (3.4)	–

Decision making procedure

The procedure was similar to the one described by Bechara et al. (1994). Subjects were presented with four decks of identical cards and received *f* 2000,– (~ € 907,56) of play money. They were instructed that the task consisted of a long series of card selections and that the object was to maximize profits. For each drawn card they received a certain amount of money, however, some selections were followed by a penalty. The distribution of reward and punishment associated with each deck was unknown to the subjects. High rewards (*f* 100,– ~ € 45,38) were obtained on decks A and B. Since summated penalties outweighed them, these decks were risky. In contrast, although rewards on decks C and D were moderate (*f* 50,– ~ € 22,69), gains exceeded penalties so these decks were advantageous. Decks A/B and C/D differed from each other with respect to frequency and magnitude but not to the net amount of penalties.

Results

Gambling task performance per block of 20 card selections was compared between groups. For each block, the number of cards taken from the disadvantageous decks (A & B) and the advantageous decks (C & D) were counted. Repeated measures analysis of variance on the total number of disadvantageous selections revealed only a main effect of time ($F_{4,48} = 10.51$; $p < 0.000$). This

demonstrates that patients and controls displayed a comparable decrease in risk taking over time.

Pearson's product-moment correlation coefficients were calculated between risk taking and symptom severity of OCD patients, with risk taking defined as the total number of cards taken from advantageous decks versus that from disadvantageous decks. Risk taking was significantly correlated with total Y-BOCS ($r = 0.416$, $p = 0.031$) and HARS ($r = 0.546$, $p = 0.003$) but not with HDRS scores ($r = 0.343$, *ns*). HARS and Y-BOCS scores appeared virtually orthogonal to each other ($r = -0.039$, $p = 0.845$). In order to explore the unique risk adjustment patterns associated with OCD and anxiety symptoms, we divided OCD patients into two subgroups using mean Y-BOCS and HARS scores as cut-off points. HARS scores were used as a covariate in the Y-BOCS groups and vice versa. Subgroups did not differ on age, IQ and HDRS scores. Analysis of covariance demonstrated a main effect of group in the Y-BOCS groups ($F_{1,24} = 11.90$; $p = 0.002$). In the HARS-groups, a trend towards a group x time interaction was found ($F_{4,21} = 2.66$; $p = 0.061$) (see figure 1ab).

Discussion

Our main finding was that decision making performance of OCD patients as a group did not differ from that of healthy volunteers. One explanation for this negative finding is that the hyperactivity of the vmPFC in OCD is accompanied by a specific impairment that is not expressed in decision making as it is in patients with damage to the vmPFC.

Our data also show that the ability to adjust on the gambling task was independently associated with both anxiety and OCD-severity. Patients with low to moderate OCD took less risk than patients with moderate to severe OCD, while risk taking of volunteers was intermediate.

Although these results need replication because of the limited number of subjects, they suggest an inverse relationship between symptoms and risk adjustment. To our knowledge, such a relationship has not been reported in OCD before. Its explanation therefore remains somewhat speculative. It is suggested, however, that individuals with high trait anxiety are more reactive to punishment, leading to increased expectancies of punishment (Zinbarg et al., 1998). Thus, the unpredictable occurrence of punishment in this task may have elicited powerful somatic signals in OCD patients. Apparently, moderately affected patients were able to adapt to these strong signals. In contrast, patients with high

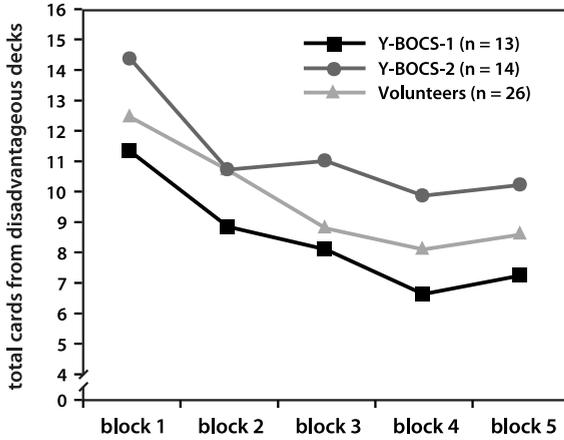


Figure 1a — Decision making performance on the Bechara gambling task. Total number of disadvantageous cards selected in each block of 20 cards: ocd patients with low to moderate ocd symptoms (Y-BOCS-1), ocd patients with moderate to severe ocd symptoms (Y-BOCS-2) and healthy volunteers.

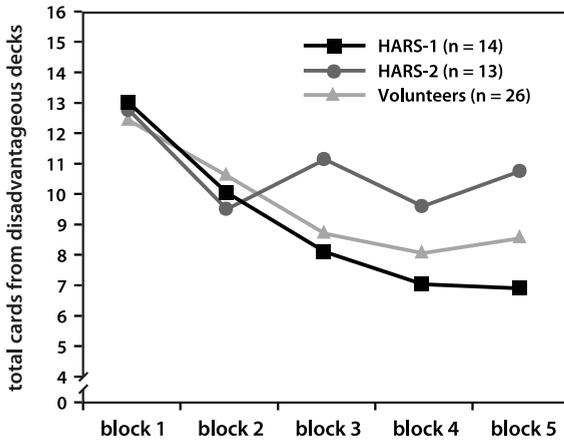


Figure 1b — Decision making performance on the Bechara gambling task. Total number of disadvantageous cards selected in each block of 20 cards: ocd patients with low to moderate anxiety (hars-1), ocd patients with moderate to high anxiety (hars-2) and healthy volunteers.

OCD-severity tended to choose for high immediate reward. With respect to this, Mischel et al. (1989) found that children's ability to postpone immediate reward was related to their ability to cope with stress or frustration. Thus, risk taking of these OCD patients could reflect a psychological inability to handle negative emotions, not only in this task but in daily life as well.

Independently from OCD severity, moderate anxiety seems to support risk avoidance; this effect apparently breaks down with high levels of anxiety. High anxiety could have interfered with the inhibition of dominant responses (Arnstén, 1997) resulting in risk taking on this task.

In conclusion, we found no evidence for a clear-cut impairment of vmPFC function in OCD. However, its function varies with symptom severity, suggesting that its role in OCD is more complex than previously thought.

References

- American Psychiatric Association (1994) *Diagnostic and statistical manual of the mental disorders* (4th edition) Washington DC: American Psychiatric Association.
- Arnsten AFT (1997) Catecholamine regulation of the prefrontal cortex. *Journal of Psychopharmacology* 11, 151–162.
- Baxter LR, Schwartz JM, Mazziotta JC, Phelps ME, Pahl JJ, Guze BH & Fairbanks L (1988) Cerebral glucose metabolic rates in nondepressed patients with obsessive-compulsive disorder. *American Journal of Psychiatry* 145, 1560–1563.
- Baxter LR, Schwartz JM, Bergman KS, Szuba MP, Guze BH, Mazziotta JC, Alzraki A, Selin CE, Fern HK & Phelps M (1992) Caudate glucose metabolic rate changes with both drug and behavior therapy for obsessive-compulsive disorder. *Archives of General Psychiatry* 49, 681–689.
- Bechara A, Damasio AR, Damasio H & Anderson SW (1994) Insensitivity to future consequences following damage to the human prefrontal cortex. *Cognition* 50, 7–15.
- Damasio AR (1994) *Descartes' error: emotion, reason and the human brain*. New York: Putnam.
- Goodman WK, Price LH, Rasmussen SH, Mazure C, Fleischmann RL, Hill CL, Heninger GR & Charney DS (1989) The Yale-Brown obsessive-compulsive scale: 1. Development, use, and reliability. *Archives of General Psychiatry* 46, 1006–1011.
- Hamilton M (1960) Diagnosis and rating scale for depression. *British Journal of Psychiatry* 3, 76–78.
- Mischel W, Shoda Y & Rodriguez M (1989) Delay of gratification in children. *Science* 244, 933–938.
- Otto MW (1992) Normal and abnormal information processing. A neuropsychological perspective on obsessive compulsive disorder. *Psychiatric Clinics of North America* 15, 825–848.
- Raven J (1960) *Guide to the Standard Progressive Matrices, sets A, B, C, D and E*. Lewis: London.
- Swedo SE, Shapiro MB, Grady CL, Cheslow DL, Leonard HL, Kumar A, Friedland R, Rapoport SI & Rapoport JL (1989) Cerebral glucose metabolism in childhood onset obsessive compulsive disorder. *Archives of General Psychiatry* 46, 518–523.
- Zinbarg RE & Mohlman J (1998) Individual differences in the acquisition of affectively valenced associations. *Journal of personality and social psychology* 74, 1024–1040.

