RISK PREFERENCE IN PATIENTS WITH OBSTRUCTIVE SLEEP APNOEA SYNDROME IS MODULATED BY THE GAIN OR LOSS CONTEXT

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Background: The aim of the present study was to assess the propensity for risk taking among patients with obstructive sleep apnoea syndrome by means of a single-outcome gambling task that involved actual monetary losses and gains.

Material/Methods: We recruited 23 patients and 17 controls matched for sex, age and education. To explore the influence of previous outcomes on risky behaviour, we calculated the proportion of risky choices following sequences of one, two or three consecutive gains or losses.

Results: Patients with OSAS made significantly more risky choices than the controls. However, like the controls, they made more risky choices after two and three losses than after one, and fewer risky choices after two and three gains than after one. Their level of impulsivity did not differ from that of the controls.

Conclusions: Our results show that OSAS induces a shift towards risk preference, but the ability to fully monitor and control ongoing behaviour remains intact.

Keywords: OSAS; gambling task; risk preferences; gains; losses
Obstructive sleep apnoea syndrome (OSAS) is associated with a number of adverse health consequences and cognitive difficulties. The overall pattern of cognitive impairment in OSAS is complex, and research in this field is mixed. On balance, OSAS has been found to have negative effects on cognition (particularly attention/vigilance), declarative memory, and executive functions (Daurat, Sarhane, & Tiberge, 2016; Kielb, Ancoli-Israel, Rebok, & Spira, 2012). Different factors may contribute to cognitive impairment, including nocturnal hypoxemia, sleep fragmentation, excessive daytime sleepiness, and functional and structural brain alterations (for a review, see Daurat et al., 2016).

The present study investigated risk taking in untreated patients with OSAS. This population is known to present a higher rate of road traffic accidents - commonly attributed to sleepiness, but which may actually stem from increased risk taking, as has been suggested by several previous studies (Daurat, Ricarrere, & Tiberge, 2013; Delazer, Zamarian, Frauscher, Mitterling, Stefani, Heidbreder, & Högl, 2016). These studies, which looked at making decisions under uncertainty, found that patients with OSAS tended to favour riskier options. Using the Iowa Gambling Task (IGT), the authors showed that patients selected cards that brought a higher immediate reward despite the more severe delayed punishment. Altered performance on the IGT was interpreted as the impaired use of feedback from previous trials for current decisions, mediated by orbitofrontal/ventromedial prefrontal cortex (OFC/vmPFC) dysfunction (Bechara, Damasio, & Damasio, 2000). Damage in these regions may disrupt the ability to utilize emotional markers to guide decision making, and to learn from past experience to make advantageous decisions (Bechara et al., 2000). However, the IGT is a complex instrument involving multiple emotional but also cognitive processes. Thus, it is unclear which of these processes is behind the changes associated with OSAS. For example, the medial temporal lobe declarative memory system that is known to be disturbed in patients (Sarhane & Daurat, 2016; Sarhane, Etcheverry, Tiberge, & Daurat, 2014) plays a critical role in successful performance (Maia & McClelland, 2004). Thus, patients’ impaired IGT performance may be linked to difficulty in learning the task.

To our knowledge, none of the published studies utilized a simple measure to directly study risk preferences in patients with OSAS. The aim of the present study was thus to assess potential changes in risk taking by means of a single-outcome gambling task that involved actual monetary losses and gains. Using this type of task, researchers have shown that preferences for either taking or avoiding risk depend on the gain/loss context (Gehring & Willoughby, 2002; Goyer, Woldorff, & Huettel, 2008; Karajan, Porjesz, Rangaswamy, Tang, Chorlian, Padmanabhapillai et al., 2009), as predicted by prospect theory (Kahneman & Tversky, 1979; Tversky & Kahneman, 1981, 1992; De Martino, Kumaran, Seymour, & Dolan, 2006). When deciding between relatively risky and relatively safe options, individuals typically have higher preferences for riskier options when the choice is...
made after experiencing a financial loss, while they generally prefer safer options when the choice takes place after experiencing a financial gain (Masaki, Takeuchi, Gehring, Takasawa, & Yamazaki, 2006; Losecaat Vermeer, Boksem, & Sanfey, 2014). Based on prior findings showing increased risk taking in patients with OSAS (Daurat et al., 2013; Delazer et al., 2016), we expected to observe a shift towards risk preference in this population. Moreover, since one possible explanation for this preference is the impaired use of feedback, we did not expect previous outcomes (i.e., successive financial gains or losses) to affect patients’ subsequent risk preferences.

MATERIAL AND METHODS

Participants
The participants were 23 patients with OSAS and 17 healthy non-obese and non-snoring controls. Patients were recruited through a university hospital’s sleep disorders laboratory. They had all been diagnosed as having mild-to-severe OSAS, with an apnoea-hypopnoea index (the number of apnoea/hypopnoea episodes per hour of total sleep time) > 10 ($M = 44, SD = 20$). None were being treated for OSAS.

The control group was recruited from among university and hospital employees, and matched with the patients for age, sex and education level. As the controls’ sleep was not recorded, sleep disturbance was excluded on the basis of the self-report Epworth Sleepiness Scale (ESS; Johns, 1991), the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), and the Berlin questionnaire (Netzer, Stoohs, Netzer, Clark, & Strohl, 1999). Although, we cannot fully exclude the presence of any undiagnosed sleep disorders without a sleep EEG in controls, none of these well-established questionnaires indicated disturbed sleep. All control participants had ESS and PSQI scores ≤ 5, and were classified as being at a low risk of OSAS (Berlin score < 2). The demographic and clinical characteristics of the patients and healthy controls are set out in Table 1. The questionnaires’ results were confirmed by responses to a clinical interview, indicating the absence of snoring, sleepiness, nocturnal urination, apnoeas, and choking arousals, as well as by the reported absence of cardiovascular disease and other diseases classically associated with OSAS (e.g., diabetes).

The participants did not suffer from any neurological (e.g., stroke, seizure disorder, neurodegenerative disease) or psychiatric disease (e.g., schizophrenia, psychosis), chronic lung disease, drug or alcohol abuse; they did not use medications that could impair memory (e.g., hypnotics, benzodiazepines). Finally, the participants did not suffer from cognitive impairment and mental retardation, as reflected by a score < 28 on the Mini Mental State Examination (Folstein, Folstein, & McHugh, 1975), depression as reflected by a score < 7 on the Beck Depression Inventory (Beck & Beamesderfer, 1974) or anxiety as reflected by a score < 50 on the State-Trait Anxiety Inventory (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) (Table 1).
Participants were instructed to maintain a regular sleep-wake schedule (i.e., to sleep from 11 p.m. to 7 a.m.) for the 3 days before the morning testing session. Actimeters (Actiwatch®; CamNtech, Cambridge, UK) were used to check compliance with the protocol.

All participants provided their written consent after being fully informed of the nature and characteristics of the study, which was conducted in line with the Declaration of Helsinki.

### Procedure

**Single-outcome gambling task**

We administered an adapted version of the single-outcome gambling task devised by Kamarajan et al. (2009). The experimental task is illustrated in Figure 1. The participants were placed in front of a computer screen on which the task was displayed. Responses were made by the left- or right-clicking of a computer mouse using the index or middle finger of the right hand. The task included a practice block, followed by four experimental blocks of 88 trials each. Each trial began with a fixation stimulus, consisting of a fixation cross flanked by two grey boxes. After 300 milliseconds (ms), the numbers 10 and 50 (corresponding to monetary sums in Euro cents) were displayed inside each box (choice stimulus; CS). Participants were instructed to select one of the boxes by either left- or right-clicking the mouse. The location of these amounts (left or right) was counterbalanced across trials. Once an amount had been selected, the fixation cross disappeared to indicate that a response had been made (post-choice stimulus; PCS). The total duration of the CS and PCS steps (i.e., the time window for participants to make their choice) was 2000 ms. In the absence of a response, the

<table>
<thead>
<tr>
<th></th>
<th>OSAS group</th>
<th>Control group</th>
<th>t test</th>
<th>p value</th>
<th>Cohen’s d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>53.30 (8.59)</td>
<td>50.53 (8.26)</td>
<td>1.06</td>
<td>0.31</td>
<td>0.33</td>
</tr>
<tr>
<td>Education (years)</td>
<td>13.43 (2.95)</td>
<td>13.88 (2.73)</td>
<td>-0.49</td>
<td>0.63</td>
<td>0.16</td>
</tr>
<tr>
<td>Subjective sleep quality (PSQI)</td>
<td>7.62 (2.01)</td>
<td>3.94 (1.33)</td>
<td>4.28</td>
<td>0.0001</td>
<td>2.15</td>
</tr>
<tr>
<td>Subjective sleepiness (ESS)</td>
<td>11.54 (4.10)</td>
<td>5.41 (2.89)</td>
<td>5.23</td>
<td>0.0001</td>
<td>1.73</td>
</tr>
<tr>
<td>Depression (BDI)</td>
<td>5.08 (2.06)</td>
<td>2.11 (3.03)</td>
<td>2.38</td>
<td>0.05</td>
<td>1.14</td>
</tr>
<tr>
<td>Anxiety-Trait (STAI-A)</td>
<td>30.95 (8.16)</td>
<td>27.94 (8.42)</td>
<td>1.14</td>
<td>0.26</td>
<td>0.36</td>
</tr>
<tr>
<td>Anxiety-State (STAI-B)</td>
<td>39.57 (10.16)</td>
<td>32.82 (7.28)</td>
<td>2.27</td>
<td>0.05</td>
<td>0.76</td>
</tr>
<tr>
<td>MMSE</td>
<td>29.69 (.70)</td>
<td>29.53 (1.06)</td>
<td>0.59</td>
<td>0.56</td>
<td>0.18</td>
</tr>
<tr>
<td>Impulsivity (Barratt)</td>
<td>61.65 (12.04)</td>
<td>59.06 (9.24)</td>
<td>0.74</td>
<td>0.46</td>
<td>0.24</td>
</tr>
</tbody>
</table>

*Note. PSQI = Pittsburgh Sleep Quality Index; ESS = Epworth Sleepiness Scale; BDI = Beck Depression Inventory; STAI = State-Trait Anxiety Inventory; MMSE = Mini Mental State Examination*
trial was considered null, and a new one was initiated. After a 700-ms interstimulus interval (ISI), the choice appeared in a box in the centre of the screen: green if the gambled sum had been won, red if it had been lost (feedback stimulus; FS). As each trial could lead to the gain or loss of 10 or 50 cents, there were four possible outcomes. The feedback screen was displayed for 800 ms, followed by a black screen for an intertrial interval of 1000 ms. The probability of a new trial being a gain or loss condition was 50%, in randomized order across the task. The participants were unaware of this parameter.

At the beginning of the task, a real 10-Euro note was placed on the desk within view of the participant. Real currency was used because gambling with fictional amounts does not involve the same risk-taking behaviour. The remaining amount was shown on the screen at the end of each block, and the amount of money on the desk was updated accordingly. At the end of the experiment, the final amount was awarded to the participant. We used the proportion of risky (i.e., 50-cent) choices as the dependent variable. To explore the influence of previous outcomes on risky behaviour, we calculated the proportions of risky choices following sequences of one, two or three consecutive gains or losses. To ensure that there was a sufficient number of valid trials to assess each condition, we only considered the last three trials at most, as not all participants had a run of four consecutive wins/losses.

Fig. 1. Sequence and length of the various events making up a trial of the gambling task. The numbers 10 and 50 (corresponding to monetary sums in euro cents) were displayed inside each box (choice stimulus; CS = 800 ms). Once an amount had been selected by the participant, the fixation cross disappeared to indicate that a response had been made (post-choice stimulus; PCS). The total duration of the CS and PCS steps corresponded to the time window for participant to make their choice (= 2000 ms). The selected amount appeared as the feedback stimulus (FS; 800 ms), either in red (to indicate a loss) or in green (to indicate a gain). A) a typical trial showing a loss of 10 cents in the red box, B) another trial featuring a gain of 50 in the green box, and C) the duration of the task events.
Barratt Impulsiveness Scale (BIS-11)

The BIS-11 is a 30-item, self-report measure of impulsivity across attentional (inability to concentrate), motor (acting without thinking), and planning (lack of future orientation) dimensions (Patton, Stanford, & Barratt, 1995). Each of the 30 items is rated on a 4-point scale ranging from 1 (Rarely/never) to 4 (Almost always).

Statistical analysis

The normality of the data was verified by measuring skewness and kurtosis. We used either a repeated-measures analysis of variance (ANOVA) or a t test to look for differences between the patient and the control groups. Post hoc tests were conducted using the Bonferroni method for multiple comparisons. Correlations between variables were investigated using Pearson’s correlation coefficient.

RESULTS

Clinical measures and impulsivity measure

As expected, the patients reported significantly worse sleep quality (PSQI) and more daytime sleepiness (ESS) than the controls (see: Table 1). Depression and anxiety-state scores were also significantly higher in patients than in the controls, but well below pathological thresholds. The level of impulsivity did not differ between the groups.

Gambling task

Risky choices

The proportion of risky choices ranged from 0.42 to 0.99 in the OSAS group and from 0.10 to 0.74 in the control group, in accordance with previous findings using this sort of task with a small reward (e.g., Goyer et al., 2008; Karamajan et al., 2009).

We carried out a 2 (group: OSAS vs. control) x 4 (block: 1, 2, 3, 4) x 2 (valence: gain vs. loss) repeated-measures ANOVA on the proportion of risky choices. There was a significant main effect of the group, \( F(1, 38) = 4.18, p < 0.05, \eta_p^2 = 0.1 \), indicating that the patients (\( M = 0.63, SD = 0.14 \)) made more risky choices than the controls (\( M = 0.52, SD = 0.17 \)). The proportion of risky choices also varied as a function of preceding outcome valence: participants chose the risky option more often when the previous outcome was a loss than when it was a gain, \( F(1, 38) = 11.94, p < 0.001, \eta_p^2 = .24 \). Neither the main effect of the block nor the interaction effects between the other factors were significant (\( ps > .29 \)).

To examine the influence of previous outcomes on risky behaviour, we carried out a 2 (group: OSAS vs. control) x 2 (valence: gain vs. loss) x 3 (preceding consecutive trials: 1 vs. 2 vs. 3) repeated-measures ANOVA on the proportion of risky choices. The results confirmed the significant main effects of the group, \( F(1, 38) = \)
4.05, \( p < 0.05, \eta^2_p = 0.1 \), and valence, \( F(1, 38) = 16.75, p < 0.0001, \eta^2_p = .31 \).

Moreover, both the trial effect, \( F(2, 76) = 4.4, p < 0.05, \eta^2_p = 0.1 \), and the Valence \( \times \) Trial interaction effect were significant, \( F(2, 76) = 9.23, p < 0.0001, \eta^2_p = 0.20 \). The proportion of risky choices increased with the number of previous successive losses, and decreased with the number of previous successive gains (Fig. 2). The proportion of risky choices was significantly higher after two \((M = 0.63, SD = 0.19)\) \((p < 0.05, d = 0.28)\) or three \((M = 0.64, SD = 0.20)\) \((p < 0.05, d = 0.32)\) losses than after one loss \((M = 0.58, SD = 0.17)\). By the same token, participants made fewer risky choices after two \((M = 0.52, SD = 0.18)\) \((p < 0.0001, d = 0.47)\) or three gains \((M = 0.49, SD = 0.20)\) \((p < 0.0001, d = 0.61)\) than after one gain \((M = 0.60, SD = 0.16)\).

### Reaction times

A 2 (group: OSAS vs. control) \( \times 2 \) (valence: gain vs. loss) \( \times 3 \) (trials: 1, 2, 3) repeated-measures ANOVA on reaction times only revealed a significant effect of the Valence \( \times \) Group interaction, \( F(1, 38) = 6.22, p < 0.05, \eta^2_p = 0.144 \). The patients responded faster after a loss \((M = 664.15, SD = 120.71)\) than after a gain \((M = 627.42, SD = 86.54)\) \((p < 0.01, d = 1.36)\), while no difference was found in the control group (gains: \( M = 674.58, SD = 170.31 \); losses: \( M = 689.42, SD = 161.84 \)) \((p > 0.05)\).

### Correlations between the proportion of risky choices or reaction times and clinical measures

No significant correlations were found between the proportion of risky choices or reaction times and clinical measures (i.e., subjective sleepiness, subjective sleep quality, anxiety and depression scores) in either the patients or the controls \((ps > 0.05)\).
DISCUSSION

OSAS made the patients more likely to make risky choices. However, they remained sensitive to the outcome of their previous gambles.

More specifically, the fact that the patients more often chose the risky option and were faster to respond after a loss than after a gain suggests that OSAS induced a shift towards risk preference. However, although they generally made more risky choices than the controls, the patients modulated their preferences according to their history of gains and losses. They were more likely to select the risky option if they had lost money on the previous trials, and less likely to make a risky choice if they had won money on the previous trials. Thus, our results show that patients are sensitive to previous outcomes and are able to adjust their ongoing behaviour. The finding that risk-taking behaviour in patients is modulated by the gain or loss context of the decision is consistent with previous studies where similar tasks were administered to healthy participants (Gehring & Willoughby, 2002; Goyer et al., 2008; Kamarajan et al., 2009; Masaki et al., 2006), and more generally with the large body of literature on risk preference (Tversky & Kahneman, 1981, 1992).

Previous studies using the IGT showed that patients with OSAS (Daurat et al., 2013) or a subgroup of patients (Delazer et al., 2016) chose risky card decks throughout the task, whereas controls gradually shifted from risky to advantageous decks. The pattern of the patients’ performances resembled that of patients with OFC/vmPFC dysfunction (Bechara, Damasio, Damasio, & Anderson, 1994), selecting cards that brought higher immediate rewards despite more severe penalties. VmPFC/OFC signals are causally important for guiding decision making (Bechara et al., 2000). The medial region of the PFC, which includes the anterior cingulate cortex (ACC), has also been associated with cognitive control and monitoring. For example, a recent neuroimaging study showed that VmPFC and ACC activation supports safe behaviour after a monetary gain, and risky behaviour following a monetary loss (Losecaat Vermeer et al., 2014). Using gambling tasks similar to that of the present study, event-related potential studies have identified the ACC as the source of a medial-frontal negativity (MFN) component that is typically elicited by the processing of previous outcomes (Goyer et al., 2008; Masaki et al., 2006). Thus, the ACC appears to be critical for guiding choice behaviour based on the consequences of previous actions. Because hypoxia is correlated with IGT performance, it has been suggested that these brain regions are affected by hypoxemia, thus explaining the inability to use affective information to guide choice selection (Daurat et al., 2013). Our data did not support this hypothesis, suggesting instead that the vmPFC-ACC is not affected by OSAS, or only to a very limited extent. This is consistent with a prior study (Daurat, Huet, & Tiberge, 2014) that showed that while episodic memory is disturbed, the processes of memory monitoring and memory control are preserved.

Evaluating the neural substrate of impaired decision taking is complex. In addition to the OFC/vmPFC and ACC regions, choosing between options associ-
ated with different rewards or punishments depends on transcortical networks encompassing numerous brain areas, such as the mesencephalic dopaminergic system, dorsolateral prefrontal cortex (Labudda et al., 2008), amygdala (De Martino et al., 2006) and ventral striatum (Allain, 2013; Floresco, St Onge, Ghods-Sharifi, & Winstanley, 2008). Dopamine is known to modulate risk-based decision making and is associated with risk seeking or avoidance, depending on which receptor subtypes are involved and which population is considered (Linnet, Moller, Peterson, Gjedde, & Doudet, 2011; St Onge & Floresco, 2009; Stopper & Floresco, 2011; Stopper, Khayambashi, & Floresco, 2013; Zuckerman & Kuhlman, 2000). A decrease in D2/D3 receptor availability in the ventral striatum, associated with reduced alertness and increased sleepiness, has been found after one night of sleep deprivation (SD; Volkow et al., 2012). As D2/D3 receptors in the ventral striatum modulate risk-taking propensity (Linnet et al., 2011), it has been suggested that the reduced availability of D2/D3 receptors due to SD facilitates engagement in risky behaviour (Volkow et al., 2012). A similar mechanism may account for the increased risk tendency found in patients with OSAS. Moreover, it has been showed that 49 h of SD or 7 consecutive nights of sleep restriction in healthy participants increased risk-taking (Killgore, Balkin, & Wesensten, 2006; Maric, Montvai, Werth, Storz, Leemann, Weissengruber et al., 2017).

Nevertheless, while SD has been shown to increase the propensity to take risks (Harrison & Horne, 2000), this is a complex effect that is modulated by different factors. Changes in risk preference induced by SD depend on the type of task and whether the decision is framed in terms of gains or losses (Killgore et al., 2006, 2008; McKenna, Dickinson, Orff, & Drummond, 2007). Furthermore, monetary rewards have been shown to attenuate the effects of SD (Horne & Pettitt, 1985; Hsieh, Li, & Tsai, 2010). SD participants have been found to be less willing to engage in risky behaviour when they have the opportunity to win and keep real money (Killgore et al., 2007). Using a flanker task, Hsieh et al. (2010) showed that monetary incentives reduce the effects of SD on error monitoring, as reflected by the reduced amplitude of the event-related negativity (ERN) component generated by the ACC. Thus, the fact that our participants had the opportunity to earn real money may account for the differences between previous results based on the IGT and those of the present study. In previous studies, patients may have preferred options that generated the most arousal or interest rather than the most profit, especially since they did not gamble with actual money. This may explain why they continued to choose the risky decks throughout the task. By contrast, our task involved real monetary gains or losses. Thus, real monetary stakes possibly (or probably) make patients more motivated and more engaged in the task, but also more cautious, explaining why they modify their choices according to previous outcomes.

Another possible explanation for the discrepancy between previous studies and the present one is the difficulty of learning the IGT task. Contrary to the single-outcome gambling task used in our study, the IGT involves numerous cognitive processes. The medial temporal lobe memory system plays a critical role
in IGT performance. Participants must maintain and update a representation of the contingencies associated with multiple decks of cards over time, in order to make advantageous decisions. Difficulty integrating more temporally distant outcomes has been observed in participants who report daytime sleepiness (Olson, Weber, Rauch, & Killgore, 2016). Moreover, source memory for the outcomes (i.e., recalling which deck produced which result) is critical for successful IGT performance (Whitney & Hinson, 2012), and source memory is known to be disturbed in patients with OSAS (Sarhane & Daurat, 2016; Sarhane et al., 2014).

Finally, a simple possible explanation for the increase in risky choices found in patients is the effort required to increase stimulation. Sleepiness is a major symptom of OSAS. Patients may select the risky choice more often to counteract their level of sleepiness by creating an element of stimulation/excitement, all the while preserving their ability to fully monitor and control ongoing behaviour. Furthermore, their level of impulsivity, as assessed by the BIS-11, was similar to that of control participants, suggesting that they did not display any risk-taking behaviour in their daily lives, although a recent study suggests that risk-taking behavior in healthy sleep-deprived subjects may depend on certain personality traits, such as disinhibition (Rusnac, Spitzenstetter, & Tassi, 2018). Thus, taken together, the results of the present study suggest that in the real world patients may (be able to) transcend their higher risk tendency – at least in some circumstances/situations.

**CONCLUSIONS**

In conclusion, this experimental investigation proved useful for understanding the shift towards risk preference induced by OSAS, although it did not allow us to draw any firm conclusions as to the mechanisms behind this shift. Further studies are therefore needed to explore this issue. Functional brain imaging could prove very helpful in this respect, as could ERPs, with the measure of the ERN-MFN components. Future studies could also explore patients’ ability to integrate gamble outcomes in greater depth, by manipulating the proportion of gain trials or by the use of neurofeedback (Choi, 2013; Mirski et al., 2014). Finally, despite the absence of differences in the degree of patients’ impulsivity, it may be interesting to explore other dimensions of sensation seeking or more broadly other personality traits involved in risk taking.

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