

Clinical Usefulness of the Iowa Gambling Task in Severe Alcohol Use Disorders: Link with Relapse and Cognitive-Physiological Deficits

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Background: Decision-making impairments have been repeatedly evaluated in severe alcohol use disorders (SAUD) using the Iowa Gambling Task (IGT). The IGT, capitalizing on strong theoretical background and ecological significance, allowed identifying large-scale deficits in this population and is now a standard decision-making assessment in therapeutic settings. However, the clinical usefulness of the IGT, particularly regarding its ability to predict relapse and its link with key cognitive-physiological deficits, remains to be clarified.

Methods: Thirty-eight recently detoxified patients with SAUD and 38 matched healthy controls performed the IGT, a neuropsychological task using monetary rewards to assess decision making under uncertainty and under risk. Disease characteristics (e.g., duration and intensity), cognitive abilities, psychopathological comorbidities, and physiological damage were also measured, as well as relapse rates 6 months later.

Results: Compared to controls, patients with SAUD presented a dissociation between preserved decision making under uncertainty and impaired decision making under risk. In the SAUD group, while relapsers (55% of the sample) presented lower global cognitive functioning and stronger liver damage than nonrelapsers at detoxification time, no difference was found between these subgroups for the IGT. IGT results were not related to alcohol-consumption characteristics or cognitive-physiological deficits.

Conclusions: SAUD is not related to a global IGT deficit, as suggested earlier, but rather to a specific impairment for decision making under risk. This deficit is not associated with other disease-related variables and has no relapse prediction power. These results question the clinical usefulness of the IGT as a tool identifying key treatment levers and guiding (neuro)psychological rehabilitation.

Key Words: Alcohol Dependence, Decision Making, Executive Functions, Liver Dysfunction, Cognitive Impairments.

IT HAS LONG been established that alcohol-related neurotoxicity causes large-scale cerebral modifications in severe alcohol use disorders (SAUD) (Bühler and Mann, 2011). These brain changes are associated with intense cognitive impairments encompassing a wide range of functions such as perceptive, attentional, memory, and executive abilities (Stavro et al., 2013). Among these cognitive consequences, decision-making deficits are considered as a central feature of SAUD. Decision making, globally defined as the selection of a behavioral response among alternatives (based on a

cost–benefit evaluation of the estimated consequences), leading to various cognitive, psychological, and social outcomes (Paulus, 2007), is a key ability in human beings. Addictive states have thus been centrally conceptualized as “decision-making diseases”: The joint influence of increased substance-related reactivity (i.e., limbic overactivation, manifested by craving and attentional biases) and reduced executive control (i.e., prefrontal underactivation, leading to reduced inhibition abilities) drives patients to promote drug-related choices, leading to addiction persistence (Wiers and Stacy, 2006). While this classical dual-process view (Stacy and Wiers, 2010) is currently challenged (Hommel and Wiers, 2017; Melnikoff and Bargh, 2018), the most influential neurocognitive models of addictions still consider altered decision making as a key theoretical and clinical factor (e.g., Koob and Volkow, 2016; Volkow and Baler, 2015; Wise and Koob, 2014).

The most commonly used neurocognitive task to explore decision making in SAUD is the Iowa Gambling Task (IGT; Bechara et al., 1994, 1997), in which participants receive an amount of money and are asked to pick up cards from 4 decks to increase their gains (each card selection leading to a

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specific win and/or loss). Two decks (A and B, unfavorable decks) lead to high gains but also high losses, with a negative total outcome in the long run, while the 2 others (C and D, favorable decks) lead to lower gains but very reduced losses, with a positive total outcome. Participants are not aware of this gain/loss ponderation between decks and have to progressively infer it and sharpen their decision making to maximize gains. The IGT, initially developed to quantify decision-making abilities in neurological patients presenting ventromedial prefrontal cortex lesions, has since been extensively used in psychiatric and addictive disorders (Gowin et al., 2018). This computerized task presents a double strength. First, it is anchored in a strong theoretical model, the “somatic marker hypothesis” (Damasio, 1994), arguing that, when confronted with uncertainty, human beings consider the expected positive or negative outcomes of their future actions by using emotional or somatic markers (i.e., anticipatory affective states based on the reactivation of previous experiences). Second, it presents a strong ecological value. Indeed, the IGT is based on real monetary rewards, thus mimicking real-life decision making (i.e., being confronted with uncertain or risky situations with potential rewarding or punishing outcomes). The IGT thus efficiently models real-life decision making, notably regarding alcohol consumption, as it implies obtaining deferred positive outcomes by avoiding immediate gratification.

Reduced IGT performance has been repeatedly found in alcohol use disorders. Initial studies have identified a globally reduced net score (i.e., subtraction between the number of cards selected from advantageous minus disadvantageous decks) in recently detoxified alcohol-dependent patients (Goudriaan et al., 2005). This result has been confirmed in more recent explorations including patients at early (Cordovil De Sousa Uva et al., 2010) or midterm (Tomassini et al., 2012) alcohol detoxification stages, but also people presenting prolonged abstinence (Fein et al., 2004). Reduced IGT performance has also been documented among SAUD patients presenting psychopathological comorbidities (e.g., borderline personality disorder [Dom et al., 2006; George-miller et al., 2013]; antisocial traits or conducts [Kim et al., 2006; Miranda et al., 2009]). The brain correlates of decision-making deficits have also been identified (Le Berre et al., 2014), suggesting that IGT-reduced performance is strongly correlated with gray matter loss in ventromedial prefrontal cortex, dorsal anterior cingulate cortex, and right hippocampus in SAUD. Two recent meta-analyses (Kovács et al., 2017; Stephan et al., 2017), reviewing IGT results in alcohol use disorders, confirmed this general trend by concluding that, while some contradictory results have been reported (Zorlu et al., 2013), SAUD is associated with significantly reduced IGT net score. However, several concerns still remain about the empirical and clinical significance of such a deficit.

A first issue relates to the usefulness of IGT to predict relapse. While the measure of decision-making impairment is worthwhile per se at the fundamental level, the inclusion of

IGT measure (and the associated neuropsychological rehabilitation proposals) in clinical settings should only be recommended if this tool has a proven relation with clinical outcomes, and centrally with relapse risk. Quite surprisingly, the link between IGT performance and midterm relapse (i.e., after 6 months, the classical threshold used in relapse prediction studies) has not been measured in SAUD. Preliminary data (Bowden-Jones et al., 2005; De Wilde et al., 2013) have suggested that IGT might constitute a reliable prediction tool to distinguish relapsers from nonrelapsers, but follow-up measures have only been performed at 3 months postdetoxification, offering limited insights on the midterm impact of IGT scores on relapse. Moreover, these data were obtained on very small and unmatched groups (i.e., only 6 relapsers; Bowden-Jones et al., 2005) or on polysubstance abusers (i.e., De Wilde et al., 2013). Further explorations are thus needed, which is even reinforced by studies showing that (i) IGT deficits are stable across the consecutive detoxification stages (Cordovil De Sousa Uva et al., 2010); (ii) IGT deficit persists after long-term abstinence, suggesting that SAUD patients can achieve persistent abstinence despite remaining decision-making deficits (Fein et al., 2004). These results suggest a stability of decision-making deficits across the successive stages of the disease, which is in contradiction with the proposal that IGT results might constitute an efficient relapse predictor.

A second issue is related to the link between IGT performance and other alcohol-related deficits. It has been shown that decision-making deficits could be at least partly underlain by memory or executive function impairments (Barry and Petry, 2008; Noël et al., 2007), but their relations with general cognitive measures usually performed in therapeutic settings (e.g., Montreal Cognitive Assessment [MoCA]), as well as with clinical (e.g., disease duration and intensity) and physiological (e.g., liver impairments) alcohol-related factors are unknown. As the IGT deficit presented by SAUD patients is actually identical (Bottesi et al., 2015) or even lower than the one reported in gambling disorder (Kovács et al., 2017), impaired decision making may not be linked to substance consumption itself but rather to stable personality factors, thus questioning its position as a core deficit in SAUD. A better characterization of these links appears as a strong prerequisite to evaluate how IGT measures could be implemented in clinical settings to complement the current routine examinations proposed.

A third issue concerns the dissociation between decision making under uncertainty and decision making under risk. Although most earlier studies only reported the global net score, several works (Bechara and Martin, 2004; Brand et al., 2006) have suggested that the IGT might not constitute a unitary task, as it could actually be divided into 2 subparts, each related to a specific psychological process: During the IGT, participants progressively learn the respective reinforcement contingencies associated with each deck, leading them to switch from decision making under uncertainty (early part of the task, where the win/loss ratio of each

deck is not identified yet) to decision making under risk (late part of the task, where the participant can consciously decide to opt more frequently for the identified risky decks). The IGT impairment shown by SAUD patients might be mostly related to less efficient decision making under risk (Brevers et al., 2014; Noël et al., 2007), but the differential links between these 2 phases and alcohol-related variables or relapse remain totally unexplored.

Clarifying these concerns would thus help to establish the clinical usefulness of the IGT, as pointed out by the above-mentioned meta-analyses (Kovács et al., 2017; Stephan et al., 2017). These works have indeed underlined that the actual link between IGT score and disease outcomes like relapse has never been experimentally explored, and that studies specifically examining decision-making deficits in SAUD are needed to clarify the contribution of impaired decision making to therapeutic outcomes. The present study will thus explore the clinical usefulness of the decision-making deficit observed in SAUD through the IGT, to determine (i) whether performance at this task constitutes a reliable predictor of relapse 6 months later and is related to other key deficits in SAUD, and (ii) whether SAUD is related to a dissociation between uncertainty-related and risk-related subscores, and whether these subscores are differentially related to clinical and physiological factors.

MATERIALS AND METHODS

Participants

Thirty-eight patients diagnosed with SAUD according to DSM-5 criteria (American Psychiatric Association, 2013), and 38 matched healthy controls took part in the study. SAUD diagnosis was established during an exhaustive interview performed by a trained psychiatrist. The mean number of DSM-5 criteria met among patients was 8.21 (SD = 1.91). The mean Alcohol Use Disorders Identification Test (AUDIT; classically used as a global screening of the dangerousness of alcohol consumption; Saunders et al., 1993) score was 27.66 (SD = 7.40), and the mean Michigan Alcoholism Screening Test (MAST; Selzer, 1971) score was 31.92 (SD = 9.54), clearly confirming the presence of SAUD. The semistructured interview was also used to determine medical history, alcohol-consumption characteristics, and psychopathological comorbidities. SAUD participants were recruited during their detoxification stay (Emile Roux Hospital, Limeil-Brévannes, France) and had all abstained from alcohol for at least 14 days (mean abstinence duration: 31.66 days, SD = 16.33). They were free of any current other psychiatric diagnosis except nicotine dependence, as 29 patients were occasional or regular smokers (mean number of cigarettes per day: 3.73, SD = 7.47). Their mean daily alcohol consumption before detoxification was 16.45 standard alcohol units (SD = 8.66), an alcohol unit corresponding to 10 grams of pure ethanol. The mean number of previous detoxification treatments was 1.89 (SD = 2.17), and the mean duration of SAUD was 24.68 years (SD = 12.79). The Hospital Anxiety and Depression scale (Zigmond and Snaith, 1983) showed that the mean depressive (5.92, SD = 2.07) and anxious (7.87, SD = 3.03) scores presented by patients were below the cutoff scores for mild depression/anxiety (Stern, 2014), only 1 patient presenting severe anxious symptomatology. Healthy participants were free of any past or current psychiatric disorder, drug/substance abuse, or (sub)clinical psychopathological states. The mean alcohol

consumption in the control group was 1.13 standard alcohol units per day (SD = 1.08), and their mean AUDIT score was 3.79 (SD = 1.97), all control participants being in a low-risk alcohol consumption (i.e., AUDIT score lower than 8). Education level was assessed according to the number of years of education completed since starting primary school. Exclusion criteria for both groups included major medical problems, neurological disease, and polysubstance abuse. Participants were informed with full details regarding the aims of the study, provided written informed consent to take part in the study, and were tested individually in a quiet room. Participants were not paid for their participation. SAUD patients were individually contacted by phone 6 months after starting the detoxification process to determine their alcohol-consumption status, namely the presence of relapse (i.e., reappearance of SAUD as evaluated by DSM-5 criteria): Twenty-one patients were classified as relapsers and 17 as nonrelapsers. The study protocol was approved by the ethical committee of the University of Créteil and was conducted in accordance with the Declaration of Helsinki, as revised in 2008.

Measures

Cognitive and Physiological Measures. Global cognitive abilities were evaluated through the MoCA (Nasreddine et al., 2005), assessing visuospatial abilities, short-term memory, executive functions, attention, working memory, language, and orientation. MoCA scores range from 0 to 30, a score below 23 indicating mild cognitive impairments (Carson et al., 2018). Physiological measures related to liver functions were also performed, as liver lesions were estimated using liver stiffness measurement performed with a FibroScan® device (Echosens™ Paris, France). The measurement was performed after at least 2 hours fasting during the first week after alcohol withdrawal. Results were considered as reliable if at least 10 valid measurements were obtained and if the interquartile range was below one-third of the median.

Experimental Measure. The IGT consists of a computerized neuropsychological task in which participants were facing 4 virtual card decks (A, B, C, and D). Participants were instructed to choose, for each trial, a card from 1 deck by clicking on it with the mouse. Immediately after each choice, the resulting gain (upper part of the screen, e.g., 150 points) and loss (lower part of the screen, e.g., -200 points) were indicated, as well as participants' total gain/loss for the whole task. Instructions underlined the possibility for the participant to switch between decks at each trial, the aim of the task being to maximize their net income (gain/loss ratio). It was explicitly stated that the gain/loss ratio was not random and varied across decks, some decks being more advantageous. Participants started with 2,000 points and performed 100 trials, without feedback regarding their strategy. The A and B decks were disadvantageous, leading to higher gains (150 points) but also higher losses (-100 to -1,600), leading to a negative net result (250 points lost for each 10 cards chosen in A and B). The C and D decks were advantageous, leading to lower gains (100 points) but also lower losses (-50 to -650), leading to a positive net result (250 points won for each 10 cards chosen in C and D). Healthy participants learn the distinction between advantageous and disadvantageous decks, leading to a progressive switch toward C-D decks. The IGT net score is calculated by subtracting the total number of selections from disadvantageous decks (A + B) from the total number of selections from advantageous decks (C + D), and subscores can be calculated using the same formula on subparts of the task (i.e., items 1 to 20, 21 to 40, 41 to 60, 61 to 80, and 81 to 100). A comparison of first (decision making under uncertainty) and second (decision making under risk) parts of the task is also classically performed by comparing the first 40 items to the 40 last ones (Brevers et al., 2014; Noël et al., 2007).

Statistical Analyses

Statistical analyses were performed using IBM SPSS Statistics (Version 25.0; IBM Corp., Armonk, NY), and the following strategy was used. First, between-group comparisons were performed on demographic characteristics (age, gender, education level). Second, a repeated-measures analysis of variance was performed, with groups (SAUD patients, controls) as between-subject factor and IGT parts (early [trials 1 to 40] vs. late [trials 61 to 100]) as within-subject variable, to explore the distinction between decision making under uncertainty and decision making under risk. Third, 2 SAUD groups were determined, namely patients who had relapsed after 6 months and patients who had remained abstinent, and group comparisons were performed on demographic, psychopathological, cognitive, and physiological measures (through independent-samples *t*-tests), as well as repeated-measures analysis of variance, with groups (relapsers, nonrelapsers) as between-subjects factor and IGT parts as within-subject variable. Significant main effects and interactions (corrected through Greenhouse-Geisser correction when needed) were followed by univariate contrasts (post hoc independent-samples *t*-tests). Alpha level was set at 0.05. Finally, correlational analyses were performed using Pearson's correlations to explore the links between IGT scores and psychological, cognitive, and physiological factors in the SAUD group.

RESULTS

Group Comparisons on Demographic Measures

As shown in Table 1, there were no significant group differences for age, $t(74) = 0.10, p = 0.92$, gender, $\chi^2(1, n = 74) = 0.00, p = 1$, and education level, $t(74) = 0.09, p = 0.93$.

Group Comparisons on Experimental Measures

As illustrated in Fig. 1, a main group effect was found for IGT performance, SAUD patients presenting globally lower scores than controls, $F(1, 74) = 6.42, p = 0.013$. A main subpart effect was also identified, which indexes an increasing

performance during task as lower scores were observed for trials 1 to 40 compared to trials 61 to 100, $F(1, 74) = 15.21, p < 0.001$. These main effects were qualified by a group \times subpart interaction, $F(1, 74) = 10.81, p = 0.002$: SAUD patients were impaired for the second part of the task, that is, decision making under risk, $t(74) = 3.30, p = 0.001$, but not for the first part, that is, decision making under uncertainty, $t(74) = 0.15, p = 0.88$.

Relapsers Versus Nonrelapsers Comparison on Experimental Measures

As shown in Table 2, relapsers and nonrelapsers did not significantly differ on demographic, age, $t(36) = 1.92, p = 0.062$, gender, $\chi^2(1, n = 36) = 0.23, p = 0.52$, education level, $t(36) = 0.33, p = 0.74$, and psychopathological, depression $t(36) = 0.52, p = 0.60$, anxiety, $t(36) = 0.67, p = 0.51$, measures, but relapsers presented lower MoCA scores than nonrelapsers, $t(36) = 2.24, p = 0.032$, as well as increased liver damage, that is, liver stiffness score, $t(36) = 2.48, p = 0.021$. Nevertheless, regarding the comparison of IGT subparts between relapsers and nonrelapsers, no significant main group effect, $F(1, 36) = 0.75, p = 0.394$, subpart effect, $F(1, 36) = 0.17, p = 0.679$, or group \times subpart interaction, $F(1, 36) = 0.95, p = 0.337$, were found.

Complementary Correlational Analyses

In the SAUD group, IGT results (total score and sub-scores) were not correlated with demographic ($r < 0.262, p > 0.112$), alcohol-consumption ($r < 0.156, p > 0.305$), psychopathological ($r < 0.235, p > 0.122$), cognitive ($r < 0.244, p > 0.140$), or physiological ($r < 0.334, p > 0.102$) measures.

Table 1. Demographic Characteristics and Experimental Results of the Severe Alcohol Use Disorders (SAUD) and Healthy Control (CTRL) Groups: Mean (SD)

	SAUD (N = 38)	CTRL (N = 38)	Group comparison (<i>p</i> -value)
<i>Demographic measures</i>			
Gender ratio (M/F)	29/9	29/9	1
Age (in years)	46.95 (11.30)	46.66 (13.42)	0.92
Education level (in years)	12.50 (2.42)	12.45 (2.88)	0.93
<i>Experimental measures</i>			
IGT total score	4.47 (23.33)	24.74 (28.76)	0.001
IGT—trials 1 to 20	1.37 (5.86)	-1.21 (6.65)	0.077
IGT—trials 21 to 40	0.27 (7.53)	3.21 (7.48)	0.091
IGT—trials 41 to 60	0.37 (8.34)	6.53 (8.54)	0.002
IGT—trials 61 to 80	2.42 (5.35)	7.37 (10.76)	0.013
IGT—trials 81 to 100	0.05 (7.39)	8.84 (10.14)	<0.001

IGT, Iowa Gambling Task.

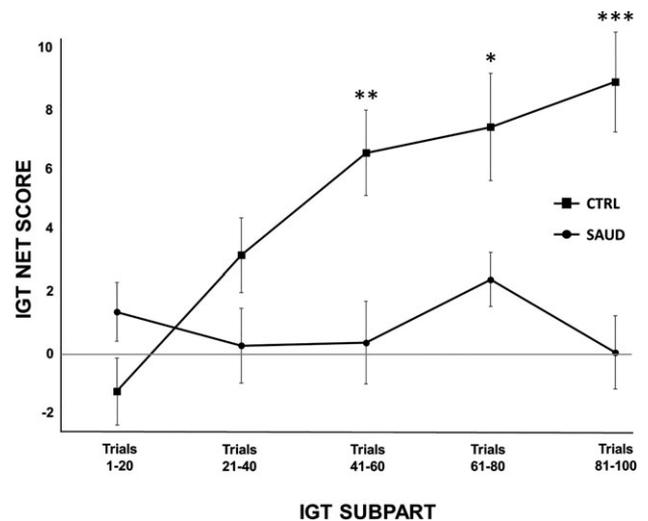


Fig. 1. Iowa Gambling Task (IGT) net scores (i.e., number of cards taken from advantageous minus disadvantageous decks) for each subpart of the task (i.e., trials 1 to 20, 21 to 40, 41 to 60, 61 to 80, and 81 to 100) among patients with severe alcohol use disorders (SAUD) and matched controls (CTRL). Error bars represent the standard error. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Table 2. Demographic, Psychopathological, and Experimental Characteristics at Detoxification Time of Patients with SAUD Who Relapsed or Remained Abstinent 6 Months Later: Mean (SD)

	Relapsers (N = 21)	Nonrelapsers (N = 17)	Group comparison (p-value)
<i>Demographic measures</i>			
Gender ratio (M/F)	17/4	12/5	0.52
Age (in years)	50.00 (12.17)	43.18 (9.12)	0.062
Education level (in years)	12.62 (2.71)	12.35 (2.09)	0.74
<i>Alcohol-consumption characteristics</i>			
Number of DSM-5 criteria	8.33 (2.01)	8.06 (1.82)	0.66
AUDIT score	27.95 (8.17)	27.29 (6.56)	0.78
MAST score	30.95 (9.54)	33.12 (9.71)	0.50
SAUD duration	26.95 (14.66)	21.88 (9.72)	0.21
Abstinence duration (in days)	34.57 (17.94)	28.06 (13.76)	0.21
Consumption before detoxification	17.24 (8.85)	15.47 (8.59)	0.54
Number of previous detoxifications	2.38 (2.57)	1.29 (1.36)	0.11
<i>Psychopathological measures</i>			
HAD anxiety	7.57 (2.89)	8.24 (3.25)	0.51
HAD depression	5.76 (1.81)	6.12 (2.39)	0.60
<i>Cognitive and physiological measures</i>			
MoCA	23.52 (5.04)	26.65 (3.06)	0.032
Liver stiffness score	8.52 (4.68)	5.26 (1.86)	0.021
<i>Experimental measures</i>			
IGT total score	1.52 (23.71)	8.12 (23.02)	0.39
IGT—trials 1 to 20	0.29 (6.01)	2.71 (5.56)	0.21
IGT—trials 21 to 40	-1.14 (8.01)	2.00 (6.71)	0.20
IGT—trials 41 to 60	0.29 (9.28)	0.47 (7.29)	0.95
IGT—trials 61 to 80	3.23 (5.60)	1.41 (4.99)	0.30
IGT—trials 81 to 100	-1.14 (7.44)	1.53 (7.26)	0.27

AUDIT, Alcohol Use Disorders Identification Test; HAD, Hospital Anxiety and Depression scale; IGT, Iowa Gambling Task; MAST, Michigan Alcoholism Screening Test; MoCA, Montreal Cognitive Assessment; SAUD, severe alcohol use disorders.

DISCUSSION

Using the IGT, a theoretically anchored and ecological task, we investigated decision-making abilities in SAUD, to explore their predictive role in relapse, their links with cognitive-physiological deficits, and the possible dissociation between decision making under uncertainty and risk. Among the numerous neurocognitive consequences of SAUD, decision-making impairments are considered as a cornerstone symptom, addictions being centrally characterized by a reduced ability to make efficient behavioral choices in everyday life. However, while reduced IGT performance has been recurrently observed in SAUD, and while the IGT is now often integrated as a clinical tool in therapeutic settings, the processes underlying this deficit and its role in disease maintenance remained to be determined. Here, we centrally show that (i) SAUD is not related to a global decision-making deficit, but rather to a dissociation between preserved decision making under uncertainty and impaired decision making under risk; (ii) this deficit, while clearly related to a core ability for adapted behaviors, does not appear associated with increased relapse risk in SAUD, nor with other cognitive-physiological deficits. These results cast doubts on the usefulness of the IGT as a tool to estimate SAUD relapse risk through decision-making evaluation in clinical settings.

First, the observed dissociation between decision making under uncertainty and decision making under risk,

confirming earlier results (Brevers et al., 2014; Noël et al., 2007), is crucial as these 2 parts of the IGT are related to distinct underlying processes. During the first part, participants have to learn the reinforcement contingencies related to each card deck, using an exploratory strategy. This initial phase, based on effective learning through negative and positive feedbacks (i.e., somatic markers), is thought to rely on unconscious processes. Conversely, the second part of the task implies to switch to decision making under risk, when the contingencies are learned and conscious, leading to the emergence of rationale and strategy-based choices (Persaud et al., 2009). Our results suggest that SAUD patients, as they have a preserved performance in the first part, do not present an impaired ability to evaluate the contingencies associated with each deck (e.g., the absence of perseverative deck choice or systematic bias in the exploratory phase) and to perform decision making under uncertainty. Conversely, the large-scale deficit presented in the second part shows that SAUD is related to a reduced ability to benefit from this preserved exploration phase to extract efficient decision strategies, resulting in suboptimal deck choices. However, it should be noted that this deficit in decision making under risk, usually interpreted as reflecting an increased sensitivity to short-term rewards (Cordovil De Sousa Uva et al., 2010; Tomassini et al., 2012), can result from different sources of impairments. As suggested earlier (Dunn et al., 2006), the mechanisms involved in the second phase are heterogeneous, reduced performance being potentially related to intensified appetite toward immediate wins (underlain by sensation-

seeking traits or impulsive personality), but also to impaired executive control abilities (Brevers et al., 2013), or even to difficulties in working memory, updating or cognitive flexibility, hampering the identification, encoding, and/or implementation of efficient decision strategies. It can even be proposed that the reduced performance observed in the second part is merely resulting from the absence of learning during the first one, SAUD patients being unable to infer the win/loss ratio associated with each deck, thus remaining under a decision making under uncertainty context during the whole task (Kim et al., 2011). These various possible interpretations of the psychological processes responsible for impaired performance point out a main limitation of the IGT, namely its multidetermined nature, which hampers to spot the impaired cognitive subcomponents and reduces its clinical usefulness, as underlined below.

Indeed, the second key result is that the IGT performance has not been identified here as a reliable relapse predicting factor in SAUD. While a clear impairment for decision making under risk was observed among SAUD patients compared to healthy controls, this deficit was not significantly different between patients who maintained abstinence and those who had relapsed after 6 months. Earlier results (Bowden-Jones et al., 2005; De Wilde et al., 2013) had suggested that IGT might predict relapse, but these preliminary data suffered from important limitations, including short-term relapse measures (6 weeks to 3 months after IGT testing), limited unmatched samples, and uncontrolled comorbidities. Our study, measuring relapse after the gold-standard period (6 months) and on larger as well as more controlled samples, casts doubt on this proposal: Neither the IGT global net score nor the subscores have been identified as relapse predictors. This result is in line with studies showing persisting IGT impairments even among SAUD patients achieving long-term abstinence (Körner et al., 2015). Actually, we even suggest that unspecific cognitive measures like the MoCA or general physiological damage measures (i.e., liver stiffness) might constitute more efficient relapse predictors than the IGT (excluding the alternative proposal that the lack of link between IGT and relapse would be explained by limited statistical power). Again, this lack of observed predictive value of the IGT might be related to its multidetermined nature: The IGT only offers, through the net score and subscores, composite outputs summing up various and unrelated deficits (Billieux et al., 2010; Noël et al., 2007) which might be differentially involved in relapse. For some patients, the IGT deficit observed here might result from modifications of processes strongly determinant for relapse (e.g., reward sensitivity and inhibitory control impairments). However, for other patients, IGT score might mainly reflect impairments in processes not directly pertinent for disease maintenance (e.g., logic inference abilities, limited understanding of task instructions, and reduced interest for monetary rewards). While future studies are needed to clarify the underlying processes

involved in reduced IGT score among SAUD patients, the large variety of deficits that can possibly end up in a similarly reduced performance might explain the globally weak predictive power of the task. The observed inability of the IGT to detect future relapsers strongly questions its clinical usefulness. This task has been the focus of many experimental works in SAUD and might still be useful for research purposes, as it reliably indexes high-level decision-making impairments in this population. However, a compulsory condition for a task to be implemented in therapeutic settings relates to its clinical meaning, that is, its ability to measure a specific factor involved in disease maintenance, which could then be targeted by remediation programs. While the present results should be confirmed and extended in future studies, they suggest that the IGT might doubly fail in this regard: On the one hand, its multidimensional nature might hamper to spot the processes involved in decision-making deficit, thus offering no information regarding the cognitive factors to be addressed. On the other hand, reduced IGT performance does not appear to give key information regarding the expected course of SAUD and thus appears of reduced interest for clinicians. This limited clinical validity of the IGT is further reinforced by the complementary analyses showing a total absence of correlation between IGT scores and other disease-related factors: No link was observed with global cognitive abilities, alcohol-consumption characteristics, or physiological impairments. The dissociation between IGT performance and alcohol-severity indexes (i.e., SAUD duration, alcohol-consumption intensity, and abstinence duration) had already been reported earlier (Le Berre et al., 2014), suggesting that the decision-making impairments might actually constitute a premorbid risk factor for addictive disorders rather than a consequence of excessive alcohol consumption. The IGT might thus constitute an efficient tool to identify the risk for developing SAUD in preclinical populations, as reported earlier (Goudriaan et al., 2011), rather than a recommendable tool for assessing relapse risk once SAUD is installed.

These results should be replicated on larger samples, with a stronger control of psychiatric comorbidities (e.g., personality disorders, which were not evaluated here) and of patients' postdetoxification treatment (which might have varied across individuals in the present sample). Moreover, the relapse assessment chosen here (i.e., patient's self-report obtained through phone call) might have been influenced by uncontrolled factors (e.g., social desirability). More reliable methods (e.g., face-to-face clinical interviews) should be favored in future studies. Despite these limits, the absence of relapse predicting power shown here by the IGT, as well as its lack of links with disease-related variables, suggest that this task presents limited usefulness for clinical guidance, despite its apparent ecological value. In line with recent proposals (Kwako et al., 2016), we thus urge clinical practitioners to replace multidetermined tasks like the IGT by process-specific tasks, focusing on the evaluation of the key

psychological variables involved in relapse (e.g., executive subcomponents, attentional biases, and emotional processing) and offering clear guidelines for accurate neuropsychological rehabilitation programs in SAUD.

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CONFLICT OF INTEREST

All authors report no competing financial interests or potential conflict of interests and no connection with tobacco, alcohol, pharmaceutical, or gaming industries.

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