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Short communication

Impaired affective prosody decoding in severe alcohol use disorder and Korsakoff syndrome

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ABSTRACT

Recognizing others' emotions is a fundamental social skill, widely impaired in psychiatric populations. These emotional dysfunctions are involved in the development and maintenance of alcohol-related disorders, but their differential intensity across emotions and their modifications during disease evolution remain underexplored. Affective prosody decoding was assessed through a vocalization task using six emotions, among 17 patients with severe alcohol use disorder, 16 Korsakoff syndrome patients (diagnosed following DSM-V criteria) and 19 controls. Significant disturbances in emotional decoding, particularly for negative emotions, were found in alcohol-related disorders. These impairments, identical for both experimental groups, constitute a core deficit in excessive alcohol use.

1. Introduction

The effective decoding of affective stimuli from our social environment is essential for adapted communication and interpersonal behaviors. This ability is impaired in a wide range of psychopathological states, and notably in alcohol use disorders: severe alcohol use disorder is particularly characterized by disturbances in emotional facial expression decoding (Donadon and Osório, 2014), and these deficits are involved in disease development and relapse (Thoma et al., 2013). However, beyond emotional facial expressions, human beings frequently express their emotional states through voices, which constitute key means of social communication (Belin et al., 2011). Emotional prosody processing is thus another crucial skill for successful social interactions (Frühholz et al., 2016), and some studies have recently suggested that individuals with severe alcohol use disorder present reduced abilities to decode emotional prosody (Maurage et al., 2009; Monnot et al., 2001; Sorocco et al., 2010; Uekermann et al., 2005).

This deficit of high clinical relevance has however been little explored in other alcohol use disorders, and particularly in Korsakoff syndrome. This frequent neurological complication of severe alcohol use disorder is centrally characterized by retrograde and anterograde memory deficits (Butters and Brandt, 1985), and most frequently results from a combination of chronic alcohol consumption and thiamine deficiency (Oscar-Berman, 2012). Cognitive and brain damages related to Korsakoff syndrome appear far more intense than those usually reported in patients with severe alcohol use disorder (Sullivan and Pfefferbaum, 2009). This observation led to the proposal of a continuity (Ryback, 1971) between uncomplicated individuals with severe alcohol use disorder and Korsakoff syndrome patients, manifested by a linear worsening of cognitive and cerebral deficits, Korsakoff syndrome patients presenting much more serious behavioral and cerebral impairments (Oscar-Berman, 2012). While the continuity theory has received empirical support from studies showing a continuum between severe alcohol use disorder and Korsakoff syndrome for memory abilities (Pitel et al., 2008), the validity of this proposal has not been experimentally tested for other impairments observed in alcohol-related disorders, among which emotional decoding.

Our main aim was thus to test prosody decoding abilities in patients presenting severe alcohol use disorder or Korsakoff syndrome, using a validated battery, to offer a direct comparison between these populations and explore the continuity hypothesis, but also to underline the fundamental and clinical relevance of emotional prosody deficits in these populations.

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2. Materials and methods

2.1. Participants

Sixteen individuals with Korsakoff syndrome (6 women, mean age: 51.4 years old), 17 individuals with severe alcohol use disorder (9 women, mean age: 50.7 years old) and 19 matched controls (10 women, mean age: 51.8 years old) took part in the study. Korsakoff syndrome individuals were diagnosed with "alcohol-induced major neurocognitive disorder (amnestic confabulatory type)" (DSM-V criteria), according to the clinical interview conducted by a psychiatrist and a neurologist. All Korsakoff syndrome participants had a history of severe alcohol use disorder (consumption before treatment: 1.92–50.97 alcohol units/day) with persisting memory loss after long-term abstinence (abstinence duration: 76–812 days). Individuals with alcohol-related disorder were diagnosed with severe alcohol use disorder (DSM-V criteria, consumption before treatment: 4.80–48.00 alcohol units/day), and were recruited after detoxification treatment (abstinence duration: 14–606 days). Patients were recruited at the Neuropsychiatric Hospitals of Saint-Martin and Beau-Vallon (Belgium). Control participants had low alcohol consumption (0.14–2.69 alcohol units/day). All participants had no history of serious psychiatric illness, medical/neurological condition and polysubstance abuse (except tobacco). Subclinical psychopathological factors were controlled with self-reported questionnaires assessing anxiety [State/Trait Anxiety Inventory (Spielberger et al., 1983)] and depression [Beck Depression Inventory (Beck et al., 1996)], and the Montreal Cognitive Assessment test (MoCA, Nasreddine et al., 2005). These data are part of a larger project on cognitive and affective functions in alcohol-related disorders (see Brion et al., 2017). The Ethics Committee of the Medical School (Université catholique de Louvain) approved the study conducted according to the principles of the Declaration of Helsinki. Participants' characteristics are provided in Table 1.

2.2. Task and procedure

The experimental task was based on the Montreal Affective Voices battery (Belin et al., 2008), a validated set of nonverbal vocalizations expressing neutral, anger, disgust, fear, happiness or sadness emotions. Participants had to decide, for each of the 120 sequentially and randomly displayed stimuli (20 stimuli per emotion), which emotion was presented by clicking on the correspondent label. Each trial followed the same procedure: warning beep (250 ms), blank screen (500 ms), target vocalization (600–1800 ms), response screen (1800 ms). As response was already allowed during vocalization, participants had a total of 2400–3600 ms to answer. Task duration was approximatively 15 min. Accuracy was computed for the global score and each emotion's subscore (no RT was recorded).

2.3. Statistical analyses

Statistical analyses were performed using SPSS 21. One-way ANOVAs were conducted with group (severe alcohol use disorder, Korsakoff syndrome, control) as between-subject factor and demographical/psychopathological/cognitive measures as within-subjects factors. A 3 × 6 ANCOVA was then conducted with group as between-subjects factor, emotional category (neutral, anger, disgust, fear, happiness, sadness) as within-subjects factor, and education level, trait anxiety, depression and MoCA as covariates. Violations of sphericity were corrected by the Greenhouse–Geisser correction. Post-hoc analyses were calculated for significant main effects and interactions.

3. Results

As shown in Table 1, no significant effect was found for Group [$F(2,45) = 2.37$, $p = 0.104$] or Emotion [$F(5,225) = 0.77$, $p = 0.507$]. A significant Group × Emotion interaction was found [$F(10,225) = 2.35$, $p = 0.036$]. Post-hoc t-tests showed that for anger, control participants presented less detection errors than Korsakoff syndrome patients [$t(33) = 2.12$, $p = 0.039$] and patients with severe alcohol use disorder [$t(34) = 2.40$, $p = 0.020$], who did not differ [$t(31) = 0.68$, $p = 0.495$]. For fear, control participants presented less detection errors than Korsakoff syndrome patients [$t(33) = 2.28$, $p = 0.027$] and patients with severe alcohol use disorder [$t(34) = 2.85$, $p = 0.007$], who did not

Table 1
Raw scores for control participants, patients with severe alcohol use disorder, and Korsakoff syndrome participants [mean (S.D.)] and group comparisons for demographic, psychopathological/cognitive and experimental measures.

	Controls (n = 19)	Alcohol use disorder (n = 17)	Korsakoff syndrome (n = 16)	Group effect (p -value)	Post-hoc comparisons* (p -value)		
					Control vs. alcohol use disorder	Control vs. Korsakoff	Alcohol use disorder vs. Korsakoff
Demographic measures							
Age (years)	51.8 (7.2)	50.7 (8.2)	51.4 (8.5)	0.912	0.661	0881	0.805
Gender ratio (F/M)	10/9	9/8	6/10	0.596 (χ^2)			
Education level (years)	16.9 (4.3)	13.6 (2.7)	11.1 (2.2)	**<0.001**	**0.003**	**<0.001**	**0.036**
Alcohol consumption (units/day)	0.9 (0.8)	23.45 (11.2)	14.8 (12.4)	**<0.001**	**<0.001**	**<0.001**	**0.045**
Abstinence duration (days)	3.0	92.7 (146.4)	115.9 (215.9)	0.051	/	/	0.719
Psychopathological and cognitive measures							
Beck Depression Inventory	2.9 (6.8)	11.3 (6.2)	6.2 (5.1)	**0.002**	**<0.001**	0.120	**0.030**
State Anxiety Inventory	29.5 (7.8)	35.0 (23.3)	38.9 (9.2)	0.185			
Trait Anxiety Inventory	33.3 (8.5)	51.0 (12.4)	44.4 (10.5)	**<0.001**	**<0.001**	**0.003**	0.105
MoCA	28.5 (1.1)	26.9 (1.1)	22.4 (2.1)	**<0.001**	**<0.001**	**<0.001**	**<0.001**
Prosody identification accuracy*				Group × Emotion interaction (p -value)			
Anger	82.6 (8.7)	60.6 (25.1)	57.8 (22.7)	**0.036**	**0.020**	**0.039**	0.495
Disgust	96.8 (5.6)	95.9 (7.9)	98.4 (3.5)		0.739	0.350	0.328
Fear	88.9 (8.4)	76.4(16.8)	71.9 (16.7)		**0.007**	**0.027**	0.622
Happiness	98.9 (2.7)	96.8 (6.1)	97.2 (5.5)		0.688	0.987	0.693
Sadness	92.6 (12.1)	93.2 (11.0)	93.7 (7.4)		0.606	0.393	0.486
Neutral	88.1 (18.0)	92.3 (9.7)	87.5 (14.5)		0.683	0.725	0.389

* Covariates appearing in the model are evaluated at the following values: Education = 14.06, Trait Anxiety Inventory = 42.53, Beck Depression Inventory = 6.65, MOCA = 26.01. Significant results are in bold text.

differ [$t(31) = 0.49$, $p = 0.622$]. No significant group comparisons were found for other emotions.

4. Discussion

This study explored the generalization of the continuity hypothesis between patients with severe alcohol use disorder and Korsakoff syndrome (which has only been explored in cognitive abilities) to emotional prosody decoding. Regarding patients with severe alcohol use disorder, results are in line with previous studies showing impaired emotional prosody decoding (Maurage et al., 2009; Monnot et al., 2001; Sorocco et al., 2010; Uekermann et al., 2005). Only one study had investigated prosody processing in Korsakoff syndrome, showing impaired interpretation of speech when prosodic and semantic contents were incongruent (Snitz et al., 2002). The present paradigm however offered a purer evaluation of emotional prosody by using vocalizations, thus avoiding any semantic content interference. Korsakoff syndrome is thus related to a general impairment in non-verbal prosody decoding, with particularly impaired anger and fear identification. This increased impairment for negative emotional states is in line with earlier results in severe alcohol use disorder (Maurage et al., 2009). Importantly, this deficit cannot be explained by psychopathological comorbidities among patients with severe alcohol use disorder and Korsakoff syndrome, anxiety and depression being included as covariates in the analyses. Similarly, impaired cognitive abilities cannot explain the experimental results as education level and MoCa were also included as covariates. These results efficiently complement earlier works (Brion et al., 2017), which used a binary categorization task (i.e. anger–happiness) to explore crossmodal processing when facing congruent or incongruent emotions in these populations. The present paradigm, by presenting increased complexity (i.e. six emotions to be identified through multiple-choice) provides deeper insight regarding the differential decoding of basic emotions, particularly by showing that impaired prosody processing is centrally found for specific negative emotions in alcohol-related disorders.

Importantly, individuals presenting severe alcohol use disorder or Korsakoff syndrome do not differ regarding prosody decoding, which shows that the continuity theory, based on a linear worsening of cognitive abilities between these two stages of alcohol-related disorders, does not extend to emotional processing. Emotional abilities thus appear to constitute a stable impairment during the evolution of alcohol-related disorders, without major deterioration related to disease evolution and neurological complications. While future studies will have to test the continuity theory across other abilities, this result suggests that differential evolution might occur across cognitive-affective deficits during the transition between severe alcohol use disorder and Korsakoff syndrome. Several limits have however to be acknowledged, centrally regarding abstinence duration and alcohol consumption. Hence, the severe alcohol use disorder group included patients with various abstinence periods, and alcohol consumption was gathered only by self-reported measures (leading to potential over or underestimation). Moreover, as these criteria significantly varied between experimental groups and as the sample size was relatively small, conclusions should be interpreted with caution.

Despite these limits, our study reinforces the proposal that affective impairments constitute a core process in alcohol-related disorders, strongly disrupting social life and thus favoring relapse. As no causal link can be drawn from cross-sectional designs, it cannot be determined whether these affective impairments are a consequence of excessive alcohol consumption and/or a premorbid risk factor, already present before the emergence of alcohol use disorder. A better understanding of these affective processing deficits in severe alcohol use disorder and Korsakoff syndrome, especially to determine their interactions with excessive alcohol consumption, is thus urgently needed, notably to promote the development of new rehabilitation tools reducing interpersonal difficulties encountered by these patients.

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