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Electrophysiological correlates of the disrupted processing of anger in alcoholism

Pierre Maurage [a,*], Salvatore Campanella [b], Pierre Philippot [c], Nicolas Vermeulen [c], Eric Constant [d], Olivier Luminet [c], Philippe de Timary [d]

[a] Cognitive Neuroscience unit, Faculty of Psychology, catholic University of Louvain, 10 Place C. Mercier, 1348 Louvain-la-Neuve, Belgium
[b] Department of Psychiatry (EEG), Brugmann Hospital, Free University of Brussels, 4 Place Vangehuchten, 1020 Brussels, Belgium
[c] Clinical Psychology Research unit, Faculty of Psychology, catholic University of Louvain, 10 Place C. Mercier, 1348 Louvain-la-Neuve, Belgium
[d] Department of Psychiatry, St Luc Hospital, catholic University of Louvain, 10 Avenue Hippocrate, 1200 Brussels, Belgium

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ABSTRACT

Objective: Recent studies have shown that alcoholism is characterized by a deficit in the processing of emotional facial expressions (EFE), and that this deficit could be "emotion specific". The present study explored the hypothesis that there is a specific deficit for the EFE of anger compared to another negative emotion (disgust). Moreover, on the basis of event-related potentials (ERPs), this study aimed at determining the locus of this deficit in the information-processing stream.

Methods: Fifteen patients suffering from alcoholism and fifteen matched healthy controls took part in the study, which used a "modified emotional" oddball paradigm. ERPs were recorded in response to repetitions of a particular facial expression (i.e. anger) and in response to two deviant (rare) stimuli obtained by a morphing procedure, one depicting the same emotion as the frequent stimulus, the other depicting a different emotion (i.e. disgust). The participants' task was to press a key as soon as they spotted the deviant stimulus.

Results: Behavioural data showed an absence of categorical perception effect for anger (but not for disgust) stimuli among alcoholic patients. Moreover, electrophysiological data revealed that alcoholism is associated with an impaired processing of anger at the attentional level (N2b/P3a complex), extending to the decisional level (P3b).

Conclusion: This study demonstrated disturbed processing of anger in alcoholism, at behavioural and electrophysiological levels. These preliminary results strengthen the proposition of a specific deficit for anger, and localize its possible origin to the attentional level (N2b/P3a complex) of the information processing stream. The clinical implications of these results are discussed.

1. Introduction

The decoding of emotional facial expressions (EFE) has been extensively investigated in normal individuals over the last decades, leading to a huge amount of data (Camras et al., 1993; Ekman, 1984). The appropriate processing of EFE is clearly a major skill for the development and maintenance of adapted interpersonal relations (Ekman, 1989; Feldman et al., 1991). In this perspective, studies have explored EFE decoding deficits in different psychopathologies (Power and Dalgleish, 1997), such as schizophrenia (Archer et al., 1992), social phobia (Winton et al., 1995) and depression (Hale, 1998).

More specifically, recent electrophysiological studies demonstrated the usefulness of an "emotional oddball paradigm" (based on the detection of an infrequent deviant stimulus among a succession of frequent standard stimuli) in the exploration of EFE deficits: One possible use of this paradigm is to define, for each clinical population showing an EFE deficit, where the disturbance originates in the information processing stream (Campanella and Philippot, 2006). Psychopathy (Campanella et al., 2005), anxiety (Rossignol et al., 2005), drug addiction (Mejias et al., 2005) and schizophrenia (Campanella et al., 2006) have been investigated using this technique, with results suggesting that the initial level of impairment leading to disturbed EFE processing is specific for each population (i.e. perceptual level for schizophrenia, attention level for depression, decisional level for anxiety and psychopathy). Nevertheless, all the stimuli used in these experiments were faces depicting a neutral expression for the frequent stimulus and an EFE for the rare stimuli, as illustrated in Fig. 1 (part A). The key limitation of this method is that it leads to major physical differences between the frequent (neutral) and rare (emotional) stimuli, as the physical distance between a face displaying a neutral or an emotional state is not controlled for. This bias weakens the conclusions that can be drawn from this paradigm, because one cannot exclude the possibility that the differences observed between

* Corresponding author. Université catholique de Louvain, Faculté de Psychologie, NESC, Place du Cardinal Mercier, 10, B-1348 Louvain-la-Neuve, Belgium. Tel.: +32 10479245; fax: +32 10473774.
E-mail address: pierre.maurage@uclouvain.be (P. Maurage).

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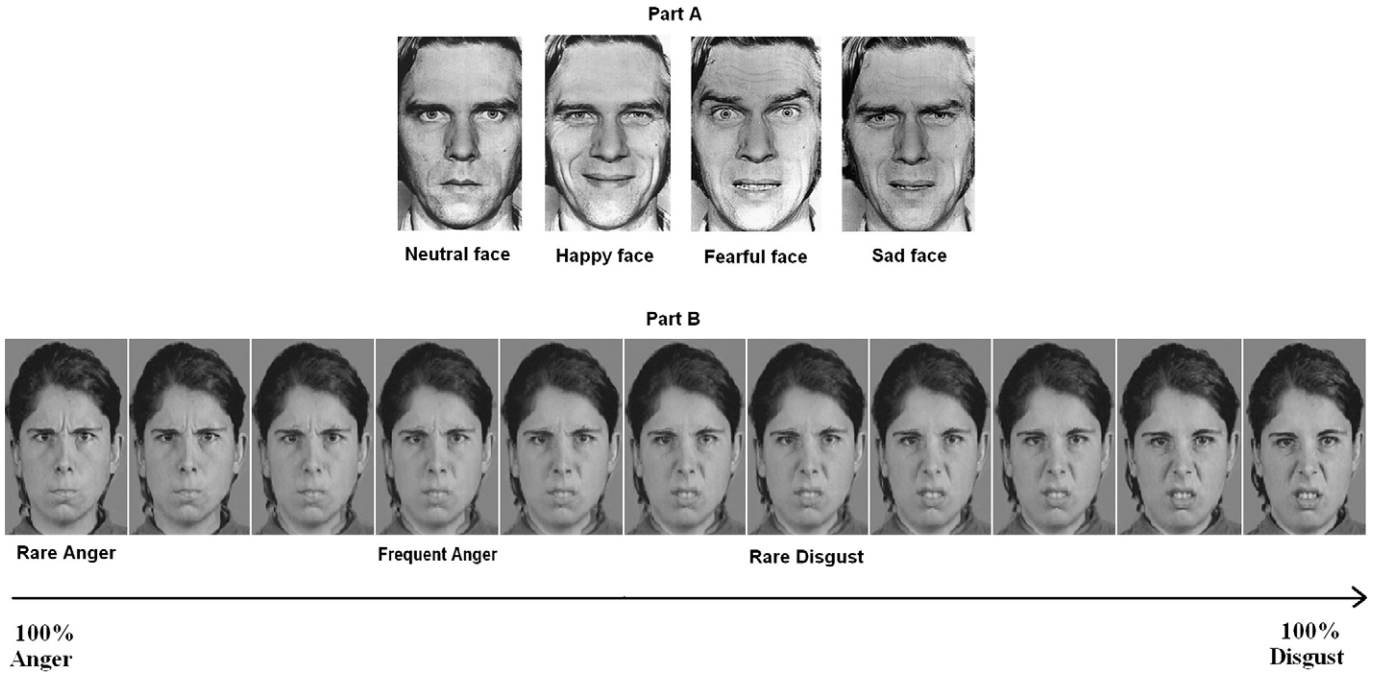

Fig. 1. Illustration showing the stimuli used in previous studies based on an emotional oddball paradigm (Part A) and the stimuli (stemming from the morphing procedure) used in our modified emotional oddball paradigm (Part B).

frequent and rare stimuli could at least partly be explained by uncontrolled physical variations (and not by the explored emotional dimension).

The present study used an adaptation of this emotional oddball paradigm to control for the physical differences and ensure that the results observed were linked to the emotional factor. Indeed, a morphing technique allowed the generation of a continuum of different morphed faces moving linearly from one EFE to another, with an identical physical distance between the frequent and every rare stimulus. For example, as seen in Fig. 1 (part B), a morphed continuum moving from an angry face to a disgusted one enabled the creation of one frequent face (i.e. containing 65% anger and 35% disgust) and two rare faces (i.e. respectively containing 95% anger–5% disgust and 35% anger–65% disgust). Hence the physical distance between the frequent stimulus and the two rare ones was identical (30%), but the first rare stimulus displayed the same emotion as the frequent one (here, anger), while the second mainly showed the other emotion (here, disgust). This technique thus led to a "purer" oddball paradigm, where the non-emotional (physical) differences are controlled for: The results are then uniquely linked to the emotional variations (see "Task and procedure" for further methodological details).

In addition, this study focused on alcoholism. Alcoholism leads to social and interpersonal dysfunctions, and notably to a degradation in the alcoholic's social life (e.g. Buu et al., 2007; Kornreich et al., 2002). These negative social consequences of alcoholism can partly be attributed to a direct effect of alcohol on the subject's interpersonal behaviour. Moreover, some alcoholic subjects also present (independently of alcohol consumption) emotional deficits that directly alter their social competences. Various defects in emotional processing have indeed been described in alcoholism: Alexithymia (Taieb et al., 2002; Uzun et al., 2003), emotional intelligence (Riley and Schutte, 2003; Szczepanska et al., 2004), and EFE decoding (Oscar-Berman et al., 1990; Townshend and Duka, 2003). Globally, alcoholic individuals overestimate the intensity of the EFE, misinterpret these EFE, and are not aware of this impairment (Kornreich et al., 2001). The link between emotional and social deficits (Nixon et al., 1992) could lead to a vicious circle: A deficit in EFE decoding, induced by the neurotoxicity of alcohol, worsens interpersonal problems, which in turn may increase alcohol consumption used as a coping strategy (Kornreich et al., 2002). Nevertheless, other studies only showed limited impairments (Frigerio et al., 2002) and even no deficit for the EFE decoding in alcoholism (Uekermann et al., 2005). It thus seems crucial that the presence of this deficit is confirmed. The present study explored this deficit by means of event-related potentials (ERPs). ERPs have been used for decades to explore cerebral functioning in alcoholism, mainly showing reduced amplitude and delayed latency of the P300 component (Hansenne, 2006; Polich, 2004; Porjesz and Begleiter, 2003). Nevertheless, most of the previous studies used basic visual or auditory stimuli (see, for example, Porjesz and Begleiter, 1981; Rodriguez Holguin et al., 1999) and alcoholism has not yet been investigated with an emotional oddball paradigm using EFE.

It furthermore appears that the specificity of this deficit varies across emotions: While some emotions seem to be correctly evaluated (particularly fear and disgust), alcoholism is mainly linked with impairment for anger (Frigerio et al., 2002; Marlatt, 1979; Philippot et al., 1999). Moreover, this impairment has been found to be correlated with the frequency of aggressive and violent behaviour among alcoholics (Bushman and Cooper, 1990; Wall and Wekerle, 2002). It thus seems highly relevant to explore the differential impairment among emotions, particularly regarding anger deficit, in alcoholism. Anger decoding, which seems to be impaired in alcoholism, was compared to disgust decoding, used as a control emotion (as disgust decoding appears to be preserved in alcoholism, e.g. Philippot et al., 1999).

Finally, earlier studies on psychopathology (see Campanella and Philippot, 2006 for a review) showed that ERPs allow the successive stages of cognitive processing to be separated, and therefore the initial level of impairment in a specific population to be defined. Indeed, different ERP components, reflecting different functional processes, are produced in an oddball paradigm, mainly:

- Perceptual processing: First, the P100, a positive deflection maximal at occipital sites around 100 milliseconds (ms) after appearance of the stimulus. This wave is generated in response to every visual stimulus and reflects the exogenous cortical activity associated with the early primary visual processing of the stimulation (Heinze and Mangun, 1995). It has been shown more recently (e.g. Debruille et al., 1998; Seeck et al., 1997) that the P100 may also be modulated by the

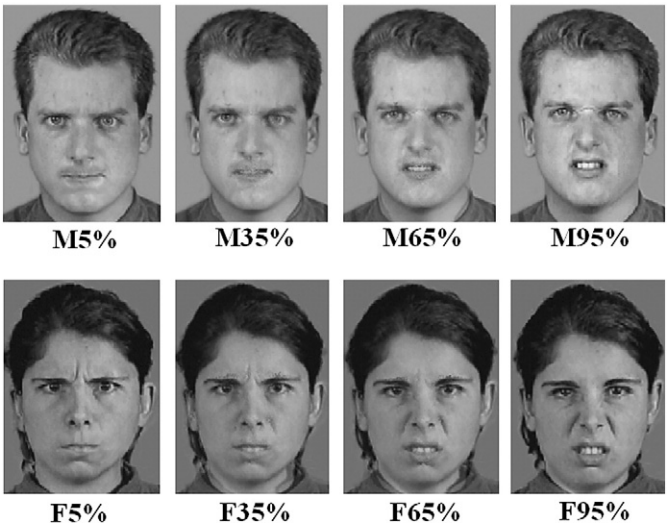

Fig. 2. Illustration of the eight morphed faces used in this study. The percentages represent the proportion of disgust displayed in the face (M = Male; F = Female).

processing of more complex aspects of the stimulus, like familiarity or attention. Second, the *N170*, a negative waveform maximally recorded at occipito-temporal sites around 170 ms after the stimulus onset. This wave, generated by the face fusiform area (e.g. Kanwisher and Yovel, 2006) is associated with a high-level perceptual stage specifically dedicated to the encoding of human faces and leading to the generation of a complete facial representation (Bentin et al., 1996). Indeed, the N170 presents a higher amplitude for faces than for other categories of objects, even if only part of the face is presented (particularly the eyes, Bentin et al., 1996). Moreover, this component is modulated by emotion and familiarity (e.g. Campanella et al., 2000, 2002a).

- Attention processing: The *N2b*/*P3a* complex, called "attention orienting complex" (Halgren and Marinkovic, 1995) is usually observed in an oddball paradigm via the subtraction of the ERPs obtained for rare and frequent stimuli. This complex is generated by the anterior cingulate cortex (e.g. Crottaz-Herbette and Menon, 2006), and is composed of two components (1) the N2b, a negativity maximal at occipital sites around 250 ms, which refers to the attention switch needed to take new information into account (Naatanen and Picton, 1986; Suwazono et al., 2000) or to pay attention to biologically significant events in order to cope with them (Campanella et al., 2002a, 2002b), and (2) the P3a, a positive wave maximal at frontal sites around 300 ms, which is linked to stimulus novelty and indexes the orientation of attention towards salient events (Knight, 1991).

- Decisional processing: The *P3b*, initially observed by Sutton et al. (1965). This long-lasting positive potential, peaking at parietal sites between 300 and 700 ms after stimulus arrival, is linked to the closure of cognitive processing before starting the motor response (Hansenne, 2006; Polich, 2004).

Table 1
Categorization of morphed faces by 20 independent judges

Proportion of disgust (%)	Anger response (%)	Disgust response (%)
5	97	3
35	93	7
65	15	85
95	7	93

Note: Judges had to decide whether randomly-presented blended images showed an angry or disgusted expression. Mean percentage of anger and disgust responses are presented for each morphed level (mean of M and F). The percentage in the first column represents the proportion of disgust in the blended image (e.g., 5% is a morphed image composed of 5% disgust and 95% anger).

On this basis, as each of these waves is associated with a cognitive stage and could be specifically impaired, an effect appearing on the P100 or N170 could reflect a deficit linked to the early visuo-spatial processing of EFE, a deficit of the N2b/P3a complex could be interpreted as a deficit in the allocation of attentional resources to "emotional" task processing, and a P3b alteration could reveal a "response-related" decisional impairment (Campanella and Philippot, 2006 for a review). Moreover, as the successive stages are not independent (Maurage et al., 2007), a deficit at the early stages could hamper the subsequent stages: For example, a perceptual deficit (indexed by a delayed/reduced N170) could prevent attention being directed to new incoming information (indexed by a delayed/reduced N2b/P3a complex) and lead to an inadequate decision (indexed by deficient P3b). The ERPs will thus allow the initial stage of the emotional processing bias in alcoholism and its influence on the subsequent processing to be identified during the cognitive task.

To summarize, this study used an emotional oddball paradigm based on morphed EFE of anger and disgust to answer the following questions:

(1) Is the deficit in EFE decoding classically observed in alcoholism identical across different emotions (anger and disgust), or is this deficit specific to certain emotions? On the basis of previous studies, it could be hypothesized that the deficit will be specific to anger, with marked impairment for this emotion at the behavioural and electrophysiological levels, and preserved performance for disgust.

(2) Is this deficit associated with a dysfunction in the perceptual stages (P100 and N170 components), in the allocation of attentional resources (N2b/P3a complex) and/or in the decisional stage (P3b component) of cognitive processing? Basic visuo-spatial functions, which are indexed by P100 and N170 components, seem to be globally spared in alcoholism (Kornreich et al., 2002; Parsons and Nixon, 1993). On the other hand, deficits have been found at a behavioural level among alcoholics for tasks involving attention abilities (e.g. Noel et al., 2001; Tedstone and Coyle, 2004). The electrophysiological components associated with these attention abilities, namely the N2b/P3a complex (Halgren and Marinkovic, 1995), could thus be impaired in alcoholism. As suggested above, this attention deficit could then hamper the subsequent stages, namely the decisional stage indexed by P3b. This possible cumulative deficit was explored using correlations and regression analyses.

2. Materials and methods

2.1. Participants

Fifteen inpatients (eight women), diagnosed with alcohol dependence according to DSM-IV criteria, were recruited during the third

Table 2
Illustration of the four triads of stimuli

Continue A–D	Face	Rare within[a]	Frequent	Rare between[b]
Triad 1	Male (M)	95% A[c]–5% D	65% A–35% D	35% A–65% D
Triad 2	Female (F)	95% A–5% D	65% A–35% D	35% A–65% D
Triad 3	Male (M)	5% A–95% D	35% A–65% D	65% A–35% D
Triad 4	Female (F)	5% A–95% D	35% A–65% D	65% A–35% D

Note that the physical difference between frequent and rare (within or between) stimuli was kept constant (30%).

[a] Rare within: stimulus depicting the same emotion as the frequent one (e.g., an anger stimulus in a sequence of anger stimuli).

[b] Rare between: stimulus depicting a different emotion to the frequent one (e.g., a disgust stimulus in a sequence of anger stimuli).

[c] A = Anger, D = Disgust.

week of their treatment in a detoxification centre (St Luc Hospital, Brussels, Belgium). They had all abstained from alcohol for at least two weeks, were free of any other psychiatric disorder (as assessed by an exhaustive psychiatric examination), and were all right-handed. The mean age of alcoholism onset was 28.7 years (S.D. 9.71), the mean disease duration was 234.7 months (S.D. 208.4), the mean alcohol consumption just before detoxification was 16.7 drinks per day (S.D. 4.87) and the mean number of previous detoxification treatments was 2.8 (S.D. 1.2). Patients were matched for age, gender and level of education with a control group composed of 15 volunteers who were free of any history of psychiatric disorder or drug/substance abuse (as assessed by an exhaustive psychiatric examination). The mean alcohol consumption in the control group was 3.4 drinks per week (S.D. 2.57). Exclusion criteria for both groups included major medical problems, neurological disease (including epilepsy), visual impairment and poly-substance abuse. Education level was assessed according to the number of years of education completed since starting primary school. Patients and control participants were assessed for several psychological measures: State and trait anxiety (STAI A and B, Spielberger et al., 1983), depression (BDI, Beck and Steer, 1987), interpersonal problems (Horowitz et al., 1988) and alexithymia (Bagby et al., 1994). Although control subjects were not receiving any medication, eight alcoholic individuals were receiving low doses of benzodiazepines (mean dose: 13.33 mg/day, S.D. 14.47). The testing took place at least 12 h after the last dose had been taken. Participants were provided with full details regarding the aims of the study and the procedure to be followed. After receiving this information, all participants gave their informed consent. The study was approved by the ethical committee of the "University of Louvain medical school".

2.2. Task and procedure

We used a visual and emotional oddball paradigm based on a change-detection task, in which participants were presented, in each experimental block, with one regularly repeated standard stimulus and two deviant stimuli, which had to be detected as quickly as possible (Campanella et al., 2002a, 2002b, 2004, 2005, 2006). Two human faces, one male (M) and one female (F), displaying expressions of anger and disgust were selected from a standardized set (Beaupré et al., 1999), and a continuum from anger to disgust was constructed for each face ("M anger" to "M disgust" and "F anger" to "F disgust") using morphing software, "Morph 5.2.1." (see Campanella et al., 2000; Young et al., 1997 for more technical details about the morphing procedure). Four morphed faces were created for each continuum. The morphed faces were based on the blending of the two initial faces in the proportions 5:95 (i.e. 5% "M disgust" and 95% "M anger"), 35:65, 65:35, and 95:5. We will refer to these morphed faces using (1) the gender of the stimulus (M or F) and (2) the proportion of disgust contained in the stimulus (5, 35, 65 or 95%). For example, M5% refers to the male face with 5% disgust (and 95% anger). On the basis of these eight stimuli (four morphing levels × two faces, as illustrated in Fig. 2), a pilot study conducted on 20 healthy controls (see Table 1), showed that M5% and F5% were significantly identified as showing an anger expression, while M95% and F95% were perceived as disgust. More importantly, M35% and F35% were predominantly identified as anger, whereas M65% and F65% were identified as disgust.

Four triads of stimuli were then generated to be used in our oddball paradigm (as shown in Table 2 and Fig. 3). For example, the triad M5%–35%–65%, where M35% (perceived as anger) constituted the frequent stimulus, and M5%–M65%, respectively perceived as displaying the same emotion as the frequent stimulus (M5% — rare within) or the other emotion (M65% — rare between), were the two deviant stimuli. The three other triads were: (1) F5% (anger, rare within) -F35% (anger, freq) -F65% (disgust, rare between); (2) M35% (anger, rare between) -M65% (disgust, freq) -M95% (disgust, rare within); and (3) F35% (anger, rare between) -F65% (disgust, freq) -F95% (disgust, rare within). The advantage of this method is twofold. First, it makes sure that the physical distance (namely the percentage on the morphing continuum) between the frequent stimulus and the rare stimulus is constant across all trials (30%). Second, it allows each stimulus to be the frequent one in one triad and the rare one in another triad. This ensures that potential ERP differences observed between frequent and rare stimuli cannot be explained by the physical difference between the frequent and rare patterns *per se* (Schröger et al., 1994).

During the ERP recordings, participants sat in a dark room on a chair placed at 1 m from the screen with their head restrained in a chin rest. Before starting the task, subjects had to fix their gaze at a small white cross in the centre of the screen. Then, a stimulus (6 cm horizontal and 8 cm vertical in size and subtending a visual angle of 3×4°), was presented for 500 ms on a black background. A black screen was then displayed as an inter-stimulus interval, lasting for a random period of between 1300 and 1600 ms. This inter-stimulus interval was followed by another stimulus, presented for 500 ms, etc. From the stimulus onset, participants had 1500 ms to respond. Participants were presented with a total of 16 blocks, each consisting of 50 stimuli: 42 frequent ones (e.g. M65%, disgust), 4 rare within (e.g. M95%, disgust) and 4 rare between (e.g. M35%, anger). The order of the 16 blocks was counterbalanced across subjects. The participants had to signal as quickly as possible the occurrence of a rare stimulus by pressing a response button with their right forefinger. Response time and error rate were recorded. There were two categories of error: Omission (i.e., subject did not press the response key when a deviant stimulus appeared) and false recognition (i.e., subject pressed the response key when a standard stimulus appeared). Participants were told that speed was important but not at the cost of accuracy. Only correct responses (i.e., deviant stimuli for which the subject pressed the response key) were considered for analysis of reaction times and ERP.

2.3. EEG recording and data analysis

The electroencephalogram (EEG) was recorded by 32 electrodes mounted in an electrode Quick-Cap. Electrode positions included the standard 10–20 system locations and intermediate positions. Recordings were taken with a linked mastoid physical reference. The EEG was amplified by battery-operated A.N.T.® amplifiers with a gain of 30,000 and a band-pass of 0.01–100 Hz. The impedance of all electrodes was kept below 10 kΩ. The EEG was recorded continuously (sampling rate 500 Hz, A.N.T. Eeprobe software) and the vertical electrooculogram (VEOG) was recorded bipolarly from electrodes placed on the supraorbital and infraorbital ridges of the left and right eyes. Trials contaminated by EOG artifacts (mean of 10%) were eliminated off-line. The recording was then re-referenced using a common average (Bertrand et al., 1985). A baseline correction was computed using a 200 ms interval (namely the interval between 200 ms prior to stimulus and stimulus onset) as baseline. Epochs were created starting 200 ms prior to stimulus onset and lasting for 850 ms. Codes synchronized with stimulus delivery were used to selectively average the epochs associated with different stimulus types. In order to compute different averages of ERP target stimuli for each subject individually, two parameters were coded for each stimulus: (1) the stimulus type: rare WITHIN, rare BETWEEN, and in order to have the same number of averaged frequent and rare stimuli, only the frequent stimuli just preceding the rare ones were averaged as FREQUENT (Horn et al., 2003); (2) the response type: response key press for rare stimuli, no response key press for frequent stimuli). Data were finally filtered using a 30 Hz low-pass filter.

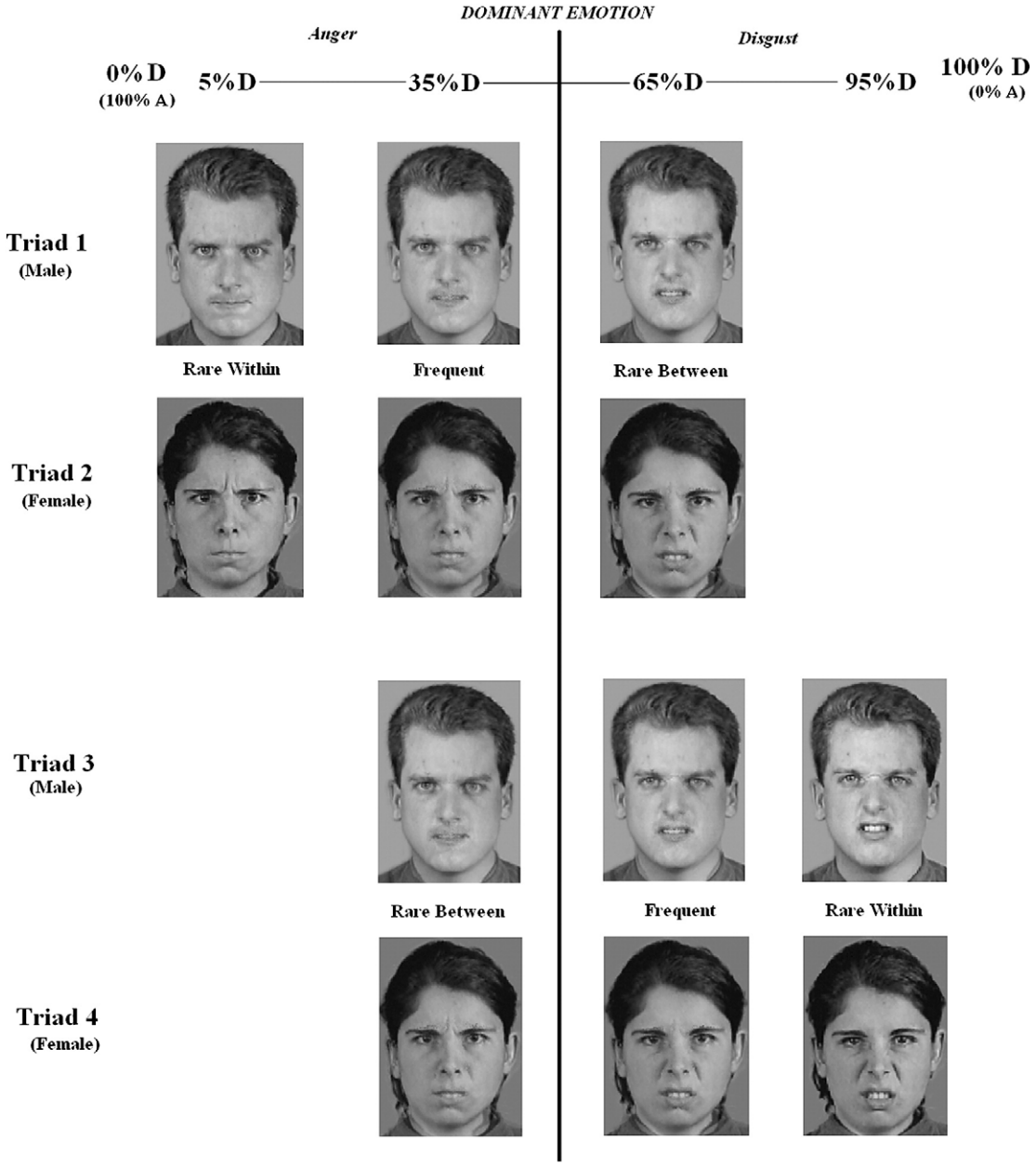

Fig. 3. Illustration of the four triads used in the study (A = Anger; D = Disgust; F = Female; M = Male).

A general time window was first determined globally for the identification of each ERP component on the basis of the ERP literature (90–160 ms for P100, 160–210 ms for N170, 300–450 ms for N2b and P3a, 450–650 ms for P3b). Peak selection was conducted as follows: For each subject individually and each component of interest, individual peak amplitudes and maximum peak latencies were obtained from several electrodes separately for the ERPs resulting either from the waveforms evoked by the rare stimuli (P100, N170 and P3b) or from the subtraction of waveforms evoked by frequent and rare deviant stimuli (N2b and P3a): Oz, O1, O2, T5, T6 for P100 and T5, T6 for N170 (Bentin et al., 1996), Oz, O1, O2 for N2b (Halgren and Marinkovic, 1995), Fz, F3, F4 for P3a (Knight, 1991) and Pz, P3, P4 for P3b (Polich, 2004). These values were tested using repeated measures of analysis of variance (ANOVA — Greenhouse–Geisser corrections were applied when appropriate) and paired sample *t*-tests. ANCOVAs (i.e., analyses of covariance, testing the potential influence of psychopathological measures on the results), Pearson's correlations and multiple linear regression were also computed when needed (see below in the results section for details). The results section will only present the significant results.

3. Results

3.1. Psychopathological measures

As shown in Table 3, there were no group differences for age [$F(1,28)=0.04$, N.S.], or education [$F(1,28)=0.33$, N.S.], but the two groups differed for depression [$F(1,28)=10.80$, $p<0.05$], anxiety state [$F(1,28)=6.42$, $p<0.05$], anxiety trait [$F(1,28)=25.08$, $p<0.01$], interpersonal problems [$F(1,28)=7.40$, $p<0.05$] and alexithymia [$F(1,28)=5.55$, $p<0.05$]. In order to test the potential effect of gender, psychopathological measures (depression, anxiety, interpersonal problems and alexithymia) and medication (in the alcoholic group), ANCOVAs were performed, including these variables as covariables in our ANOVA statistical analyses. There was no significant influence of

Table 3
Patient and control characteristics: mean (S.D.)

	Controls ($N=15$)	Alcoholics ($N=15$)	Group effect
Age (in years)	48.1 (7.24)	49.13 (8)	N.S.
EL[1]	13.5 (2.3)	13 (2.1)	N.S.
BDI[2]	3.18 (3.6)	7.33 (2.9)	$p<0.05$
STAI A[3]	28.8 (8.4)	39.5 (11.3)	$p<0.05$
STAI B[3]	31.2 (5.8)	50.2 (10.9)	$p<0.01$
IIP[4]	0.98 (0.6)	1.7 (0.6)	$p<0.05$
TAS 20[5]	43.4 (6)	51.3 (9.3)	$p<0.05$

NS = Non-significant.
[1]EL = Education Level.
[2] BDI = Beck Depression Inventory (Beck and Steer, 1987).
[3] STAI = State and Trait Anxiety Inventory (Spielberger et al., 1983).
[4] IIP = Inventory of Interpersonal Problems (Horowitz et al., 1988).
[5] TAS-20 = Twenty-item Toronto Alexithymia Scale-II (Bagby et al., 1994).

gender, psychopathological measures or medication level on any behavioural or electrophysiological results ($p>0.05$ for every test).

3.2. Behavioural data

A 2×2×2 ANOVA with emotion (anger, disgust) and condition (within, between) as within-factors and group (alcoholic individuals, controls) as between-factor was carried out separately for accuracy and reaction times. These results are illustrated in Table 4.

3.2.1. Accuracy

An Emotion × Condition interaction [$F(1,28)=18.47$, $p<0.001$] showed that while in both conditions "within" stimuli were more difficult to detect than "between" ones, the "disgust within" category led to more errors, suggesting that variations in emotional intensity in faces are easier to detect for anger than for disgust.

3.2.2. Reaction times (RTs)

There was a Group × Condition × Emotion [$F(1,28)=6.61$, $p<0.05$] interaction: Alcoholic subjects had globally higher RTs, but in the control group, "between" stimuli led to faster RTs than "within" stimuli for anger ($t(14)=3.14$, $p<0.01$) and disgust ($t(14)=3.75$, $p<0.01$), while in the alcoholic group, this effect was present for disgust ($t(14)=5.35$, $p<0.001$) but not for anger ($t(14)=-0.83$, N.S.).

3.3. Event-related potentials

For each component of interest, 2×2×2×3 (5 for P100, 2 for N170) ANOVAs were computed separately for latencies and amplitudes, with group (alcoholic individuals, controls) as between-factor, and emotion (anger, disgust), condition (within, between) and location (Oz, O1, O2, T5, T6 for P100; T5, T6 for N170; Oz, O1, O2 for N2b; Fz, F3, F4 for P3a; Pz, P3, P4 for P3b) as within-factors. The electrophysiological results are shown in Table 5, and the Figs. 4 and 5 illustrate the frequent, rare and subtraction waveforms for alcoholic and control groups, respectively. Fig. 6 presents the N2b, P3a and P3b amplitudes for each group, condition and emotion.

Table 4
Behavioural results: accuracy (mean number of errors) (S.D.) and Reaction times (ms) (S.D.)

Group		Anger between	Disgust between	Anger within	Disgust within
Controls ($N=15$)	Accuracy	0.8 (1.5)	1 (2.2)	1.8 (2.9)	4.53 (5.4)
	RTs	599 (111)	586 (107)	653 (122)	662 (122)
Alcoholics ($N=15$)	Accuracy	1.53 (2.4)	2.4 (3.4)	0.67 (1.9)	4.4 (3.4)
	RTs	716 (92)	682 (81)	711 (62)	756 (83)

3.4. P100–N170

There was no significant main effect or interaction effect for P100 and N170, either for amplitudes or for latencies.

3.5. N2b

3.5.1. Latencies

There was a main effect for Group [$F(1,28)=19.68$, $p<0.01$]: The N2b latency was shorter for the control groups.

3.5.2. Amplitudes

There was a Group × Emotion × Condition interaction [$F(1,28)=5.89$, $p<0.05$]: In the control group, higher N2b amplitudes were observed for "anger within" as compared to "anger between" ($t(14)=4.63$, $p<0.001$) and for "disgust between" as compared to "anger between" ($t(14)=6.88$, $p<0.001$).

3.6. P3a

3.6.1. Latencies

Two interactions were observed: (1) Condition × Group [$F(1,28)=5.64$, $p<0.05$]: P3a latency was shorter for controls, and the deficit in the

Table 5
Electrophysiological results: (a) mean latencies (ms (S.D.)) and (b) mean amplitudes (μV (S.D.)) for P100 (0z), N170 (T6), N2b (Oz), P3a (Fz) and P3b (Pz) components for each condition, among controls and alcoholics

(a)			Alcoholics	Controls	Group effect
Between trials	Anger	P100	133 (10)	137 (13)	NS
		N170	189 (14)	187 (16)	NS
		N2b	386 (22)	351 (73)	**
		P3a	373 (29)	320 (50)	**
		P3b	607 (59)	510 (50)	***
	Disgust	P100	138 (10)	135 (10)	NS
		N170	187 (12)	185 (16)	NS
		N2b	400 (45)	330 (59)	**
		P3a	359 (31)	336 (50)	*
		P3b	578 (41)	509 (60)	***
Within trials	Anger	P100	136 (12)	137 (14)	NS
		N170	188 (17)	188 (16)	NS
		N2b	387 (43)	326 (57)	**
		P3a	415 (25)	316 (49)	***
		P3b	556 (43)	516 (42)	***
	Disgust	P100	137 (13)	135 (13)	NS
		N170	190 (13)	185 (16)	NS
		N2b	379 (44)	323 (52)	**
		P3a	400 (33)	346 (39)	**
		P3b	584 (33)	493 (41)	***
(b)			Alcoholics	Controls	Group effect
Between trials	Anger	P100	4.7 (1.8)	6.1 (2.3)	NS
		N170	−4.7 (2.3)	−3.1 (1.5)	NS
		N2b	−4.5 (3.1)	−4.4 (2)	NS
		P3a	5.5 (3.7)	3 (1.5)	NS
		P3b	4.5 (2.9)	6.4 (2.8)	*
	Disgust	P100	5 (1)	6 (2.2)	NS
		N170	−4.6 (2.8)	−2.9 (2.1)	NS
		N2b	−4.6 (3)	−4.5 (2.3)	NS
		P3a	5 (4.2)	4.8 (2.7)	NS
		P3b	3.7 (2.9)	5.2 (2.2)	*
Within trials	Anger	P100	5.7 (2.5)	6.2 (2.3)	NS
		N170	−3.9 (2.3)	−3.4 (1.4)	NS
		N2b	−4.8 (2.9)	−4.6 (1.7)	NS
		P3a	4.4 (1.6)	4.2 (2.4)	NS
		P3b	3.1 (2.2)	6 (2.5)	*
	Disgust	P100	4.8 (2.8)	5.8 (2.2)	NS
		N170	−4.6 (2)	−4 (1.6)	NS
		N2b	−4.6 (2.2)	−4.8 (2.6)	NS
		P3a	5.2 (2.4)	4.1 (2.1)	NS
		P3b	5 (3.7)	6.1 (2.4)	*

NS = Non-significant; *$p<0.05$; **$p<0.01$; ***$p<0.001$.

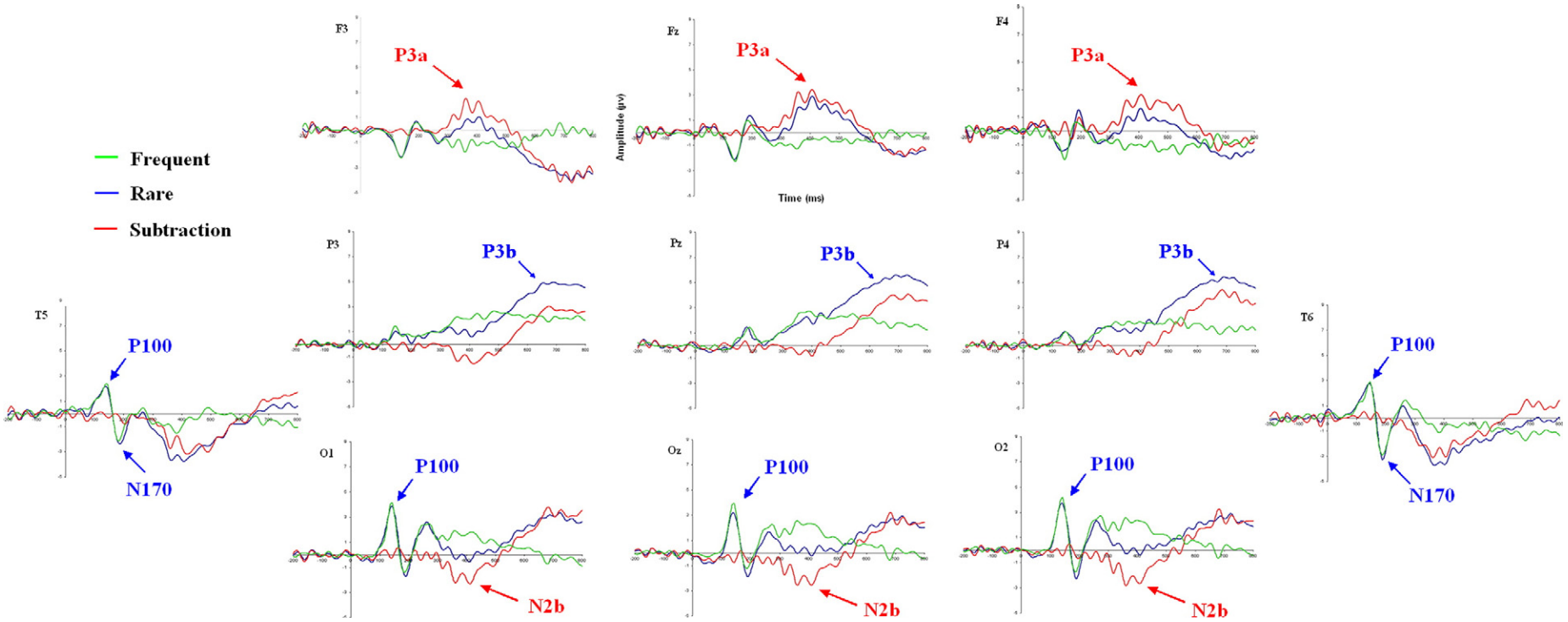

Fig. 4. Mean electroencephalographic results (for all conditions) in the alcoholic group on Fz, F3, F4 (P3a), Pz, P3, P4 (P3b), Oz, O1, O2, T5 and T6 (P100, N170 and N2b) for frequent, rare and subtraction waveforms.

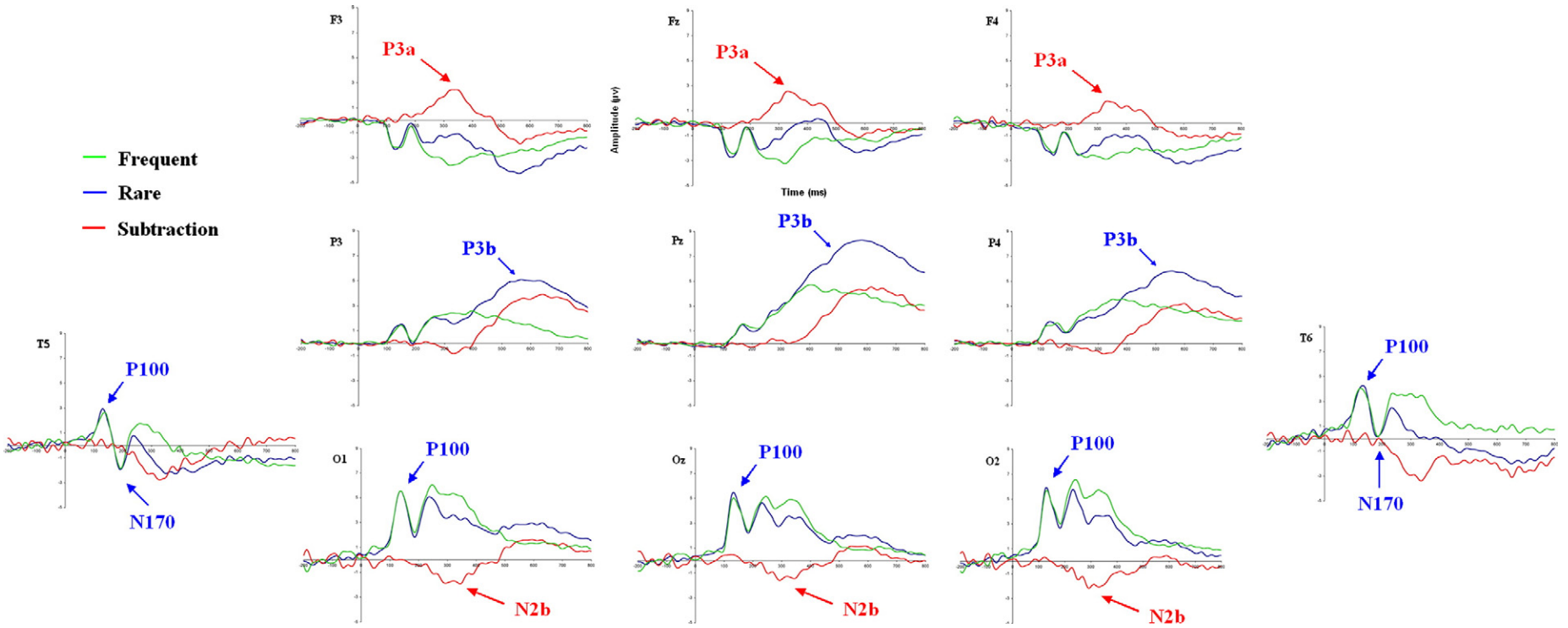

Fig. 5. Mean electroencephalographic results (for all conditions) in the control group on Fz, F3, F4 (P3a), Pz, P3, P4 (P3b), Oz, O1, O2, T5 and T6 (P100, N170 and N2b) for frequent, rare and subtraction waveforms.

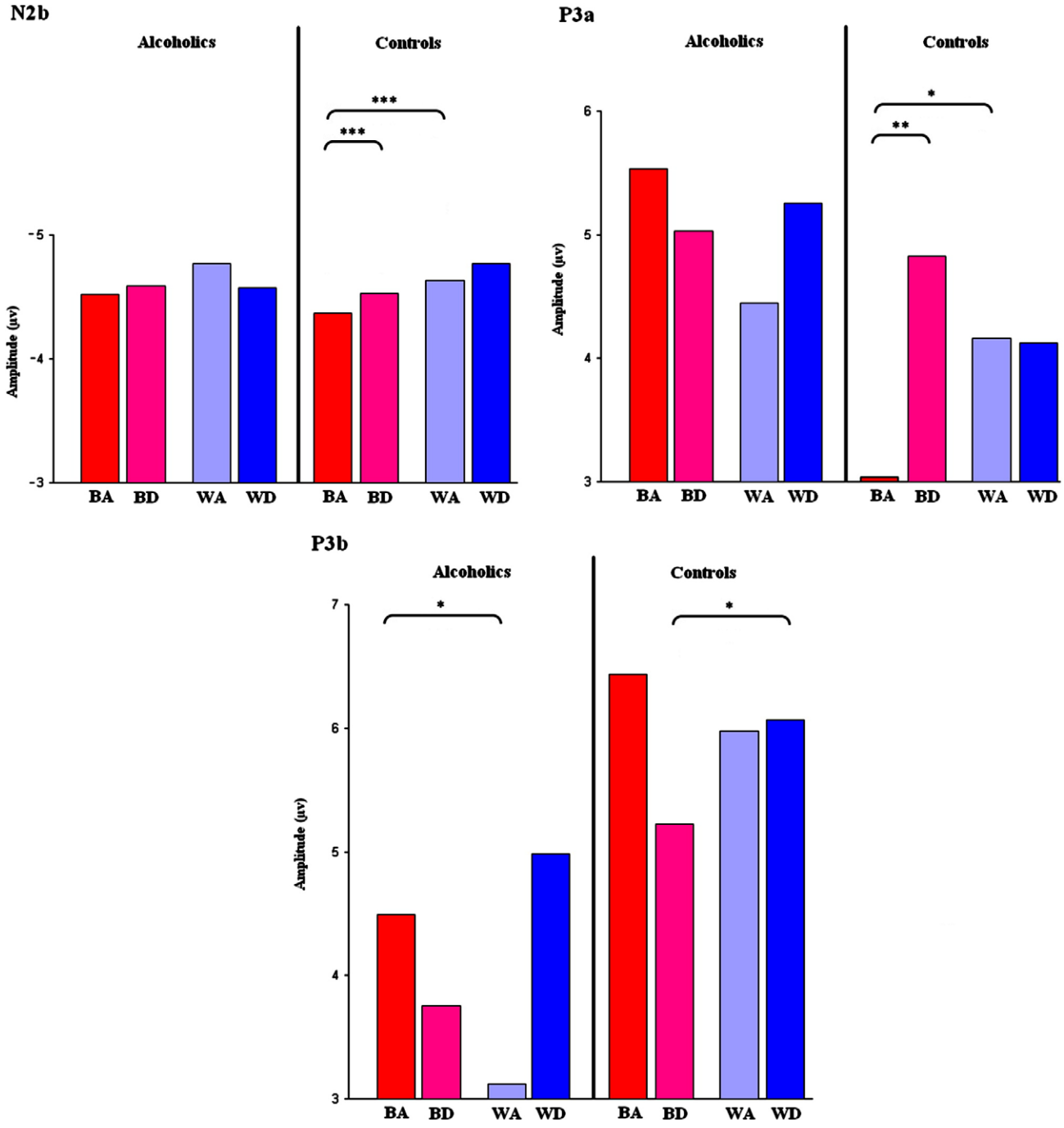

Fig. 6. Electrophysiological results: Mean amplitudes (µV) for N2b (Oz), P3a (Fz) and P3b (Pz) components for each condition and emotion, among controls and alcoholics (A = Anger, B = Between, D = Disgust, W = Within). The main significant differences are underlined: *$p<0.05$; ** $p<0.01$; *** $p<0.001$.

alcoholic subjects was mainly observed in the "within" condition: the difference "within"–"between" was only significant among alcoholics ($t(14)=7.25$, $p<0.01$); (2) Group × Emotion [$F(1,28)=15.11$, $p<0.01$]: the deficit in the alcoholic subjects was mainly observed for anger stimuli as, in the control group, the disgust stimuli led to longer latencies than the anger stimuli ($t(14)=2.70$, $p<0.05$), and the opposite result was observed among alcoholics ($t(14)=-2.40$, $p<0.05$).

3.6.2. Amplitudes

There was a Group × Emotion × Condition interaction [$F(1,28)=8.127$, $p<0.01$]: In the control group, P3a had a larger amplitude for "anger within" than for "anger between" ($t(14)=2.91$, $p<0.05$) and "disgust between" led to larger amplitudes than "anger between" ($t(14)=3.54$, $p<0.01$).

3.7. P3b

3.7.1. Latencies

There was a main effect of Group [$F(1,28)=40.55$, $p<0.001$]: P3b latency was significantly longer for alcoholics than for controls.

3.7.2. Amplitudes

There was a Group × Emotion × Condition interaction [$F(1,28)=7.99$, $p<0.01$]: P3b was globally larger among controls, but in the control group P3b had a larger amplitude for "disgust between" than for "disgust within" ($t(14)=2.43$, $p<0.05$), with no differences for anger. The opposite pattern was observed in the alcoholic group: Amplitude was larger for "anger between" than for "anger within" ($t(14)=2.42$, $p<0.05$), with no differences for disgust.

Table 6
Pearson's correlations (r (*p*-value)) in amplitude and latency between N2b, P3a and P3b for each experimental condition in the alcoholic group

		N2b–P3a	N2b–P3b	P3a–P3b
Between anger	Latency	$r=0.524$ ($p<0.05$)	$r=0.591$ ($p<0.05$)	$r=0.622$ ($p<0.05$)
	Amplitude	$r=-0.911$ ($p<0.001$)	$r=-0.876$ ($p<0.001$)	$r=0.72$ ($p<0.01$)
Between disgust	Latency	$r=0.521$ ($p<0.05$)	$r=0.694$ ($p<0.01$)	$r=0.527$ ($p<0.05$)
	Amplitude	$r=-0.765$ ($p<0.001$)	$r=-0.577$ ($p<0.05$)	$r=0.656$ ($p<0.01$)
Within anger	Latency	$r=0.911$ ($p<0.001$)	$r=0.731$ ($p<0.01$)	$r=0.559$ ($p<0.01$)
	Amplitude	$r=-0.832$ ($p<0.001$)	$r=-0.891$ ($p<0.001$)	$r=0.584$ ($p<0.05$)
Within disgust	Latency	$r=0.533$($p<0.05$)	$r=0.575$ ($p<0.05$)	$r=0.517$ ($p<0.05$)
	Amplitude	$r=-0.585$ ($p<0.05$)	$r=-0.666$ ($p<0.01$)	$r=0.574$ ($p<0.05$)

3.8. Additional analyses

1° The results presented above focus on the rare stimuli, but group differences could also occur for frequent stimuli, which reflect the "purest" measure of perceptual and decisional processing (i.e., not contaminated by a motor response as the subject does not have to react to frequent stimuli). In order to test these potential group differences, ANOVAs (2×5 for P100, 2×2 for N170, 2×3 for P3b) were computed separately for latencies and amplitudes (with group as between-factor and location as within-factor) on the P100, N170 and

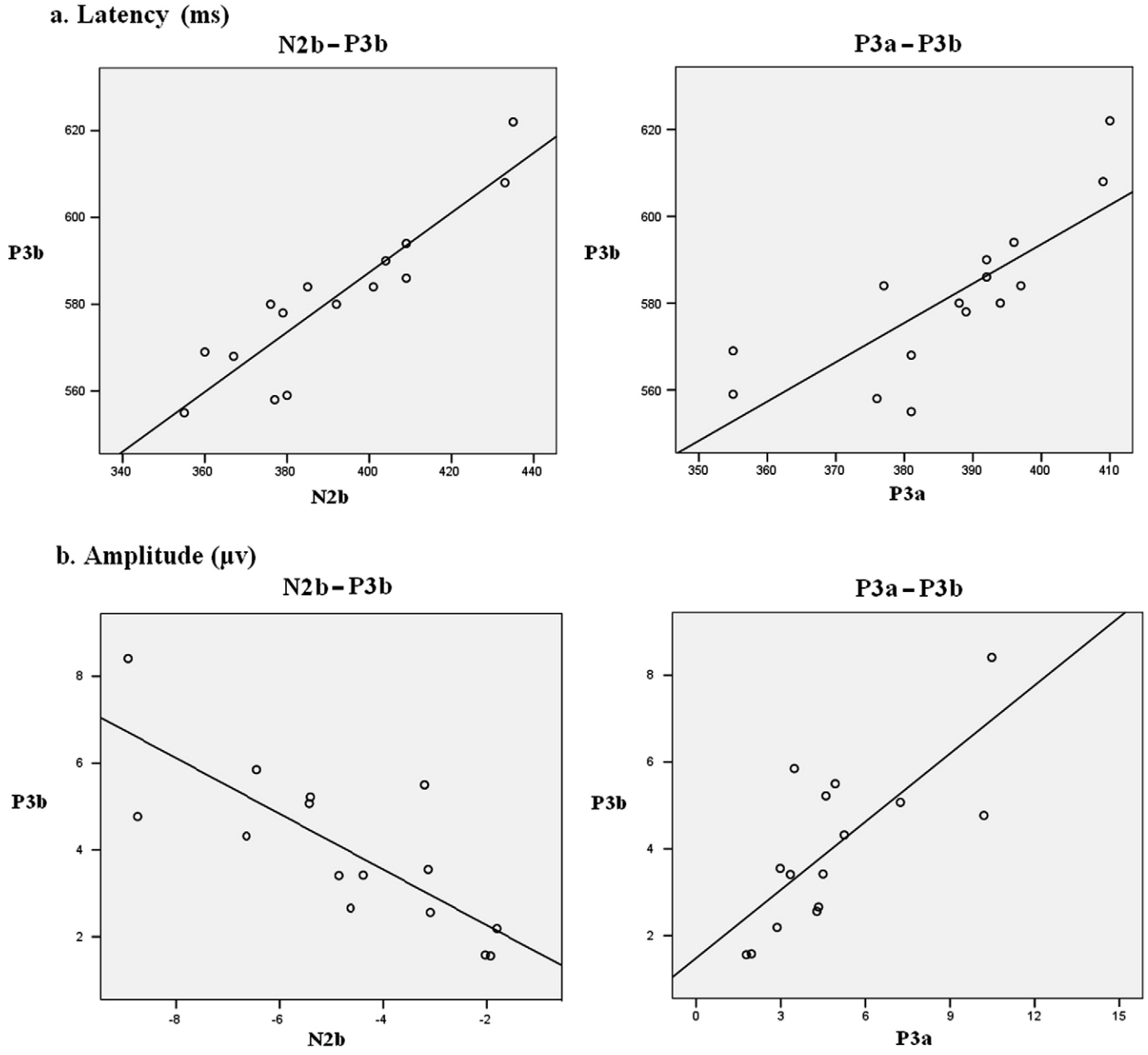

Fig. 7. Regression analysis results for latencies (part a) and amplitudes (part b) illustrating the predicting strength of N2b and P3a mean values on P3b results in the alcoholic group (ms = milliseconds; µV = microvolts).

P3b values for frequent stimuli (i.e., mean of all the frequent stimuli just preceding the rare ones). For P100 and N170, there was no significant main effect or interaction. For P3b, main group effects showed that controls had shorter latencies [$F(1,28)=21.03$, $p<0.001$] and higher amplitudes [$F(1,28)=5.57$, $p<0.05$] than alcoholics, thus showing that alcoholism was associated with an impaired P3b even when the subjects did not have to produce any motor response.

2° To test whether N2b, P3a and P3b are the successive stages of a continuum in cognitive processing (and thus whether the deficit in P3b could be considered as a consequence of impairments in earlier ERP components), two analyses were performed in the alcoholic group: (1) Pearson's correlations were computed between these three components in each experimental condition, for amplitude and latency (the results are shown in Table 6); (2) Multiple linear regression analyses were also computed to explore to what extent the mean N2b and P3a values (in latency and amplitude) could explain the results observed for P3b (the results are illustrated in Fig. 7): These analyses showed that the N2b-P3a results significantly predicted the P3b results, in latency [*N2b-P3b*: $F(1,14)=59.61$, $p<0.001$, $R^2=.82$; *P3a-P3b*: $F=22.21$, $p<0.001$, $R^2=.63$] and amplitude [*N2b-P3b*: $F=21.20$, $p<0.001$, $R^2=.62$; *P3a-P3b*: $F=15.68$, $p<0.01$, $R^2=.55$].

These two additional analyses clearly confirm the link between N2b, P3a and P3b, for latencies as well as amplitudes: The intensity of the deficit (delayed latency and reduced amplitude) at the attention stage (i.e., N2b and P3a) is significantly correlated with (and predicts) the deficit observed at the decision stage (P3b).

4. Discussion

It should first be noted that our original methodology was proved valid, as it led to a replication of the classical "categorical perception effect" among control subjects, i.e., faster RTs when the perceived emotion differs between rare and frequent stimuli (Between condition) than when the two stimuli are perceived as displaying the same emotion (Within condition) (Etcoff and Magee, 1992; Calder et al., 1996; Young et al., 1997). Indeed, control subjects detected "rare Between" trials faster than "rare Within" ones for both emotions. Nevertheless, the main implications of this study concern the behavioural and electrophysiological deficits observed in alcoholism.

On the one hand, we confirmed and extended earlier results concerning the deficits observed in alcoholism: (1) Our results reinforced the proposition of a general slowing in motor and cognitive processing among alcoholic subjects (Beatty et al., 1995; Fein et al., 1990; Sullivan et al., 2000, 2002). Indeed, longer RTs in detecting rare faces and longer latencies for N2b, P3a and P3b components were observed in the alcoholic group compared to controls; (2) The reduced P3b amplitude in the alcoholic group confirms the P3b amplitude reductions repeatedly described in alcoholism (see, e.g., Hansenne, 2006 for a review). These reduced amplitudes are usually considered as indicating a deficit in central nervous system inhibition (Porjesz and Begleiter, 2003) or in memory (Polich, 2004). As our task did not involve high demand on memory or inhibition processes, the P3b amplitude deficit in alcoholism (usually observed on the basis of tasks involving inhibition or memory processes) seems to index a general high-level decisional stages deficit, rather than only reflecting a memory or inhibition impairment. This proposition should be confirmed by future explorations, as it did not constitute the central focus of this study.

On the other hand, alcoholic subjects are particularly impaired for the processing of anger stimuli. At the behavioural level, we showed an absence of "categorical perception effect" for anger in alcoholism (i.e. no RTs difference between "anger within" and "anger between"), which suggests a specific deficit for the processing of anger (as compared to disgust). Nevertheless, due to a ceiling effect, no group differences were found for accuracy, which prevents any firm conclusion concerning the behavioural deficit in alcoholism for the categorical perception of anger. Future studies, based on more sensitive paradigms (avoiding ceiling effects) are thus needed to confirm this accuracy deficit.

This proposition of a specific impairment for anger stimuli in alcoholism was confirmed at the electrophysiological level. Indeed, while no differences between conditions were observed at the attention stage in the alcoholic group, control subjects clearly showed an enhanced processing of anger for N2b/P3a, as shown by: (1) *Higher N2b-P3a amplitudes for "anger within" than for "anger between"*. As the N2b/P3a amplitude reflects the amount of attention resources allocated to stimulus processing (Halgren and Marinkovic, 1995), this result suggests that controls need less attention to focus on an anger stimulus (as "anger between" condition requires detection of an anger stimulus among a succession of disgust stimuli), which could constitute an adaptive advantage in speeding up the motor reaction to anger (Marsh et al., 2005; Ohman et al., 2001); (2) *Higher N2b-P3a amplitudes for "disgust between" than for "anger between"*, showing that controls need more attention resources to disengage from anger (i.e. to detect a disgust stimulus among a succession of anger stimuli); (3) *Higher N2b-P3a amplitudes for "Anger within" than for the three other experimental conditions*, showing that the attention resources of control subjects are particularly engaged when they have to perceive intensity differences between different anger stimuli. These three results suggest that control subjects have a more intense processing of anger than disgust. Conversely, in the alcoholic group, there were no differences for N2b/P3a amplitudes between conditions, suggesting that alcoholics do not present the preferential processing for anger observed among control subjects. This observation supports the hypothesis of a deficit in the processing of anger in alcoholism and proposes that this deficit begins at the attention level (namely for N2b/P3a components), which confirms previous results (Noel et al., 2001) showing attention deficits in alcoholism.

Finally, we showed an extension of this specific deficit for anger to the decisional level. Indeed, concerning P3b amplitudes, results indicated that: (1) Among controls, "disgust between" led to the largest P3b amplitudes, which confirms their difficulty to disengage from anger: As P3b amplitude depends on the amount of information extracted from the event to make a decision (Rugg and Coles, 1995), a higher P3b amplitude reflects a more elaborate processing. The higher amplitude for "disgust between" thus confirms that controls need a deeper processing to detect a disgust stimulus among a succession of anger stimuli; (2) The opposite pattern was observed among alcoholics, thus confirming their deficient processing for anger: "Between anger" led to larger amplitudes than "within anger". In line with the N2b–P3a results, this suggests a difficulty for alcoholics to shift to anger; (3) Correlation and regression analyses showed a strong link between N2b–P3a and P3b components (in amplitude and latency) and thus the cumulative effect of the anger processing deficit: The greater the deficit for N2b–P3a, the greater the deficit for P3b. This finding confirms the linearity of the cognitive continuum and suggests an influence of early attention stages on later decisional ones. Nevertheless, correlation and regression analyses do not allow to conclude that there is a strict causal link between N2b/P3a and P3b deficits, as a mediating variable could be responsible for both deficits. Further explorations are thus needed to confirm the causal link between that N2b/P3a impairment and the deficit observed at later decisional stages (P3b).

4.1. Clinical implications and strengths of the study

The main proposition of our study is that the processing of anger is disturbed in alcoholism, and that this deficit begins at the attention level. This assumption has clinical implications, as it confirms earlier clinical studies (Bartek et al., 1999; Karno and Longabaugh, 2004) which stressed that alcoholic subjects have impaired perception and expression of anger, and that this specific deficit for anger increases

interpersonal problems. The difficulty identifying anger observed in our results, and thus the inability to correctly react by adapting one's behaviour, could increase interpersonal troubles and lead to the appearance and maintenance of a vicious circle (Kornreich et al., 2002): EFE decoding deficit (specifically for anger) would lead to interpersonal problems and social isolation. These increased interpersonal difficulties may lead to an enhanced alcohol consumption, used as a coping strategy, and the increased alcohol consumption may finally, because of alcohol neurotoxicity, increasingly deteriorate EFE decoding (Philippot et al., 2003). Our results thus clearly confirm the clinical observation of a specific deficit for the anger EFE decoding in alcoholism. Moreover, by showing that this deficit originates at the attention level, these results constitute a first step in the development of therapeutic programs focusing on EFE decoding abilities, by showing that these programs should first concentrate on the restoration of the ability to correctly manage the attention resources.

The main advantage of the present study is the use of a modified oddball paradigm based on a morphing technique to standardize the physical distance between the frequent and every rare stimulus, ensuring that the differences observed are linked to the emotional factor. Moreover, the strict pairing between groups and the additional analyses minimized potential confounding factors and alternative explanations for the results. It thus seems unlikely that our data were biased by confounding factors like gender, medication, depression, anxiety, interpersonal problems or alexithymia.

4.2. Limitations and conclusion

This study has some methodological limitations. Firstly, only two different faces were used and we cannot exclude the possibility that this limited variation in the stimuli influenced the results. A second limitation was the use of only two emotions, namely anger and disgust. The choice of these emotions was justified by earlier studies: The deficit for anger has been extensively described and, conversely, the decoding of disgust appears preserved in alcoholism (Frigerio et al., 2002; Kornreich et al., 2002, 2003; Philippot et al., 1999). However, it would be useful to further explore this hypothesis of a specific deficit for anger, and particularly to confirm that the processing of other emotions is preserved.

In conclusion, this study shows disturbed processing of anger in alcoholism, at a behavioural and electrophysiological level. These preliminary results need to be confirmed in further studies using a wider range of emotions and a larger sample of subjects. Nevertheless our results clearly confirm the proposition of a specific deficit for anger and localize the possible origin of this deficit at the level of "attention resource allocation" (N2b/P3a complex).

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