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P3a and mismatch negativity in individuals with moderate Intermittent Explosive Disorder

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ABSTRACT

The mismatch negativity (MMN) and the P3a have so far only sparsely been used to investigate neural correlates of impulsivity. This study compares the MMN, P3a, and P3b between individuals with and without the propensity for impulsive, uncontrollable outbursts of temper (referred to here as moderate Intermittent Explosive Disorder, mIED). The MMN reflects automatic change detection, whereas the P3a reflects involuntary attentional mechanisms. Both MMN and P3a can be elicited in the absence of attention. Results showed that the P3a was significantly smaller in the mIED group compared to the control group, whereas neither MMN nor P3b differed between groups. These data provide the first ERP correlates of mIED, showing that mIED affects cognitive mechanisms of involuntary control of attention (as reflected in the P3a). Because the P3a receives main contributions from the frontal, and the MMN from the temporal lobe, results support the notion that increased impulsivity is related to frontal, rather than temporal lobe function.

The propensity for impulsive, uncontrollable outbursts of temper that are not in proportion to a precipitating event is more common than realized, and represents a source of important social and clinical problems [3,25,27,29,40]. In its extreme form, this propensity includes uncontrollable episodes of aggressive impulses that result in serious assaultive acts or destruction of property. If the degree of aggression expressed during such explosive episodes is grossly out of proportion to the precipitating psychosocial stressor, individuals showing such episodes fulfil the diagnostic criteria of Intermittent Explosive Disorder (IED) as defined in the DSM-IV-TR (unless the explosive episodes are better accounted for by another mental disorder or by direct physiologic effects of a substance or a general medical condition) [11,25,29,34]. However, many individuals with the propensity for impulsive, uncontrollable temper tantrums perform actions during impulsive episodes that are, for the most part, judicially not relevant (e.g., door slamming, loud shouting, binge eating or binge drinking, smashing dishes etc.). Therefore, these individuals do not fulfil the diagnostic criteria for IED, but they nevertheless often suffer from the disadvantageous impact of their temper tantrums on their family, their health, and their occupational career. This calls for more systematic research on this moderate form of IED (mIED) [27] with regards to its biological correlates, etiology, and possibilities for therapeutic intervention.

A previous study [27] investigated oscillatory brain activity (as measured with electroencephalography, EEG) in individuals with mIED. Individuals with mIED (compared to normal controls) showed increased power of oscillatory activity in the beta band, and decreased power of oscillatory activity in the theta band in response to both auditory and visual stimulation (compared to a rest condition without stimulus presentation). This was taken to reflect that individuals with mIED are more agitated by, and show greater stress responses to sensory stimuli, consistent with the heightened central nervous arousal reported for impulsive aggression [3], and with the restlessness and hyperactivity of some disorders characterized by substantial impulsivity (such as attention deficit/hyperactivity disorder) [10]. Moreover, based on the oscillatory EEG data of the participants, the mentioned previous study [27] also used linear discriminant analysis to develop a model of classification functions which discriminated both groups (with and without mIED) significantly. That model was used in the present study as a classifier for the analysis of EEGs of participants (i.e., based on the model of classification functions, EEGs of participants were assigned either to the group with or without mIED). The result of this classification complemented the assessment of mIED based on diagnostic interviews (see Supplementary Material for details).

To further examine the neural underpinnings of mIED, the present study compared event-related brain potentials (ERPs) between individuals with mIED and control subjects. So far, only little systematic ERP research on (pathologic) impulsivity has been carried out, except a few studies investigating the P3b in individuals with impulsive aggression. The P3b is a subcomponent of the P300

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potential which can be elicited by infrequent target stimuli that have to be detected among frequent non-target stimuli [35] (thus, the P3b requires directed, voluntary attentional focusing). The P3b has been found to be reduced in individuals with impulsive aggression who were also diagnosed with antisocial personality disorder [4,19], and in college students with impulsive aggression [28]. Further studies have shown associations between reduced P3b and impulsivity in cocaine-dependent individuals [30], as well as in individuals with DSM-IV diagnosis of alcohol dependence [9]. These studies suggest that some forms of pathologic impulsivity (related to aggression and substance abuse) are related to problems in the voluntary focusing of attention.

Another ERP component that has been compared between impulsive and non-impulsive individuals is the mismatch negativity (MMN). The MMN is an ERP component which can be elicited by deviant auditory stimuli that are presented occasionally among a series of auditory standard stimuli [32]. A previous study [16] reported that the amplitude of the mismatch negativity (MMN) is increased in individuals with increased "dysfunctional impulsivity" [13]. Dysfunctional impulsivity is a "tendency to act with less forethought than most people of equal ability when this tendency is a source of difficulty" [16, p. 164], that is, a tendency that is also characteristic for mIED (although mIED also comprises other characteristics). Because the neural mechanisms underlying the generation of the MMN operate in the absence of attention, the findings of that study [16] suggest that dysfunctional impulsivity influences cognitive mechanisms even on a pre-attentive (i.e., involuntary) processing level.

In the present study, we presented participants with an auditory oddball paradigm suited to elicit an MMN, a P3a (elicited by distracter stimuli), and a P3b (elicited by target sounds). The P3a is another subcomponent of the P300 ERP, and taken to reflect the (involuntary) allocation, or switch, of attention [14,22,35]. Like the MMN, the P3a can be elicited even when subjects do not have to attend to a stimulus or a stimulus dimension [14,35]. Thus, the MMN was used here to compare processes of automatic auditory change detection, the P3a to compare involuntary, and the P3b to compare voluntary, processes of attention between individuals with and without mIED.

The main neural generators of the MMN have been localized in the temporal lobe (although the MMN is also modulated by frontal lobe sources) [1,2,20,31,36]. The neural generators of the P3a are not precisely clear [35], but so far the most consistent finding across studies is involvement of (dorso)lateral prefrontal cortex in the generation of the P3a, perhaps partly due to its role for the control of sensory-limbic integration [26] (other structures reported to be involved in involuntary attention switching include superior temporal, temporo-parietal, and parietal cortical areas, the parahippocampal and anterior cingulate gyri, and the hippocampus) [2,35]. Therefore, measuring MMN and P3a also aimed at exploring whether individuals with and without mIED differ with regards to neural processes mainly located in the temporal lobe (reflected in the MMN), or in the frontal lobe (reflected in the P3a), or in both the frontal and the temporal lobe. This was motivated by previous studies showing relations between frontal lobe (dys)function and IED [7], impulsive aggression [5,12,33,39], and impulsivity [6,21].

We hypothesized that P3a, P3b, and MMN would differ between individuals with and without mIED. Based on the mentioned studies [4,9,19,28,30], directed hypotheses were only made for the P3b (which was expected to be smaller in individuals with mIED), and the MMN (which was expected to be larger in individuals with mIED). In addition to ERPs, we also obtained impulsivity scores of the I_7 inventory (as an index of the impulsivity facet *impulsive action*) [15,27] with the directed hypothesis that I_7 scores would be higher in the mIED than in the control group.

78 right-handed (lateralization quotient > 80), normal hearing university students participated in the study (age range 18–34 years, mean 25.9 years). None of the participants was a professional musician (none of the subjects had ever participated more than 3 years in extracurricular music lessons or performances), and no participant was taking any medication. Exclusion criteria were past or current diagnosis of a personality disorder, of an obsessive-compulsive or a major depressive disorder, past neurological illness or traumatic brain injury with hospital stay or coma. None of the subjects met DSM-IV criteria for borderline or antisocial personality disorder. Participants were recruited via the participant database of the Max Planck Institute for Cognitive Neuroscience, and via flyers and posters inviting participants to take part in a study on brain correlates of increased impulsivity and temper tantrums. Written informed consent was obtained, the study was approved by the local ethics committee, and conducted according to the declaration of Helsinki.

The classification of subjects into those with and without mIED was based on a structured interview, and on an EEG measurement designed to assess mIED [27] (the EEG classification is described in the Supplementary Material). Participants were assigned to one of the two groups (with or without mIED) only when the classification based on the interview, and the classification based on the EEG, were congruent. This was the case for 60 subjects (age range 18–33 years, mean 25.1 years). The remaining 18 subjects were excluded from further data analysis. From the 60 individuals included in the data analysis, 39 participants (27 females) were classified to the group without mIED, and 21 (15 females) to the group with mIED (mean age did not differ between groups, $p > .9$). The relatively high rate of mIED subjects in the study population is presumably due to the recruitment procedure, which partly used flyers and posters inviting to take part in a study on brain correlates of increased impulsivity and temper tantrums.

Interviews were conducted in a fashion reminiscent of a previous study [27]. The interview started with the explanation that individuals differ with regard to how they experience and express anger, and that the purpose of the interview was to assess which kind of anger-type they are, followed by five questions: subjects were asked (1) whether they had over the course of the last months outbursts of temper during which they performed actions that they could not control, and – if so – (2) whether these actions were in excess of what they consider as appropriate with respect to the precipitating event. Furthermore, participants were asked (3) whether their temper outbursts first seemed to be switched on and then switched off, (4) whether they had regretted violent acts performed during an outburst immediately after the episode was over, and (5) whether such regret had not been helpful in averting such behaviour during subsequent episodes. Participants who answered all of these five questions with "yes" were assigned to the mIED group, those who answered all questions with "no" to the group without mIED (except when they met criteria for the implosive type of mIED, see next paragraph; individuals with mixed answers were not included in the data analysis).

Because some individuals with mIED may show auto-aggressive episodes (e.g., in extreme cases, self-injurious behaviour), rather than aggressive outbursts, subjects were also asked whether, over the course of the last months, they had experiences of uncontrollable inner rage during which they directed their aggression inwards, rather than outwards. With this regard, two subjects answered all five questions mentioned in the previous paragraph with "yes" and were, therefore, included in the mIED group.

Interviews were carried out blinded, i.e., without knowledge about the classification of subjects based on the EEG data. Interviews were conducted in a separate session that either preceded or followed the EEG session.

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The EEG-based classification of subjects into those with and without mIED is described in the Supplementary Material. In brief, 23 statistical values were calculated from the spectral EEG data (such as power values in specific frequency ranges), and then entered into a model of standardized canonical discriminant function coefficients obtained in a previous study [27]. The model returns a classification of each data set, that is, for each data set which was entered into the analysis, the model returns a probability for the group membership (with, without mIED) of this data set. If the classification based on this procedure was identical with the classification based on the structured interview, participants were included in the study.

In the MMN experiment, standard stimuli were piano tones (General Midi sound #2) with a presentation time of 600 ms and an f0 pitch of 396 Hz (corresponding to g′). There were three types of deviants: frequency deviants (f0 pitch = 440 Hz), timbre deviants (tones played by guitar, marimba, trumpet, strings, or flute with the standard f0 pitch of 396 Hz), and omission deviants. Deviants were pseudorandomly intermixed between standards in a fashion that deviants were preceded by at least two, and not more than four, standards. 70% of stimuli were standards, and each deviant occurred with a probability of 10%. There were 540 stimuli in total (378 standards, and 54 deviants of each type). Stimuli were played with ~60 dB SPL under computerized control on a synthesizer (ROLAND JV 8010; Roland Corporation, Hamamatsu, Japan). Standards directly following deviants were not evaluated. All piano tones had the same decay of loudness, like the normal decay of piano tones played at a rate of 0.6 Hz, with no further silent interval (inter-onset-interval = 0 ms). Stimuli were generated on a synthesizer (ROLAND JV 8010; Roland Corporation, Hamamatsu, Japan), and presented via headphones under computerized control.

The experimental session started with filling out the I_7 questionnaire [15]. The first part of the EEG recording session was conducted for the electrophysiological assessment of mIED (this part was identical to a previous study [27] and is described in the Supplementary Material). Subsequently, after a short break, the MMN experiment followed, for which participants were informed about the different types of deviants, asked to press a response button for the timbre (but not for the frequency or the omission) deviants, and instructed to look at a fixation-cross during the presentation of the stimuli.

The EEG was recorded from 39 electrodes of the 10-20 system (FPZ, FP1, FP2, AFZ, AF3, AF4, AF7, AF8, FZ, F3, F4, F5, F6, F7, F8, FCZ, FC5, FC6, FC3, FC4, FT7, FT8, CZ, C3, C4, T7, T8, CP5, CP6, PZ, P3, P4, P7, P8, POZ, O1, O2), as well as from the nose-tip and the right mastoid (M2), using an electrode placed on the left mastoid (M1) as reference. Sampling rate was 500 Hz. After the measurements, EEG data were re-referenced to the algebraic mean of both mastoid electrodes (M1 and M2) to obtain a symmetric reference.

For the ERP analysis, EEG data were filtered using a 0.25–25 Hz band-pass filter (2309 points, Blackman window, finite impulse response) to reduce artefacts. For further rejection of movement artefacts, each sampling point was centred in a gliding window, and rejected if the standard deviation within a 200 or 800 ms gliding window exceeded 30 μV at any EEG-electrode. Eye-artefacts were rejected whenever the standard deviation of a 200 ms gliding window exceeded 25 μV at the vertical, or the horizontal EOG (rejections were controlled by the author). ERPs were calculated using a 100 ms pre-stimulus baseline.

For the statistical analysis of ERPs, mean amplitude values were computed for six regions of interest (ROIs): anterior left (AF7, AF3, F7, F3, FT7, FC3), anterior midline (AFZ, FZ, FCZ), anterior right (AF8, AF4, F8, F4, FT8, FC4), posterior left (P3, C3, P7, T7, CP5), posterior midline (CZ, PZ, POZ), and posterior right (P4, C4, P8, T8, CP6). Frontal ROIs were used to analyze MMN, and P3a potentials (as well as potentials of the re-orienting negativity, RON). Parietal ROIs were used to analyze P3b potentials. To test whether ERPs

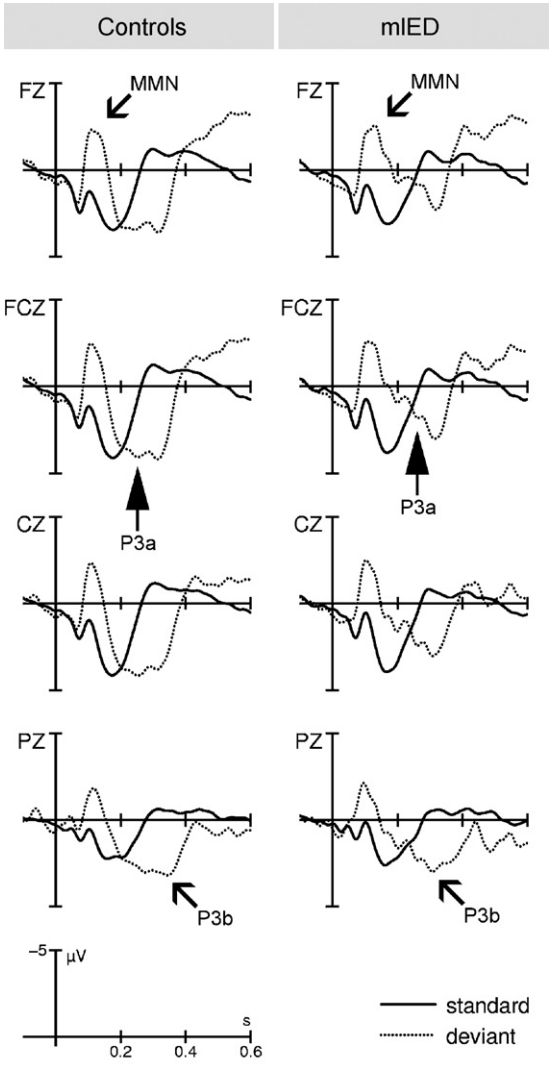

Fig. 1. Grand-average ERP waveforms of standards (solid line) and frequency deviants (dotted line), separately for the control group (left panel), and the mIED group (right panel). The P3a was significantly smaller in the mIED group compared to the control group.

to standard and deviant tones differed from each other, amplitude values of ERPs were analyzed statistically by repeated measures MANOVAs. MANOVAs were conducted separately for ERPs elicited by frequency, omission, and by timbre deviants, with factors condition (standard, deviant), hemispheric distribution (left, midline, right ROIs), and group (without, with mIED). Time windows for statistical analyses were 100–180 ms (MMN), 200–300 ms (P3a), 350–400 ms (P3b), and 400–600 ms (RON).

As expected, I_7 scores were higher in the group with mIED ($M = 45.3$, $SEM = 4.1$) compared to the group without mIED ($M = 38.9$, $SEM = 3.1$), the difference between groups being significant ($p = .05$; one-sided two-samples t-test).

Fig. 1 shows the ERP waveforms elicited by standards and (task-irrelevant) *frequency deviants* (see Fig. 2 for iso-potential maps). In each of the groups the deviants evoked a significant MMN. The MMN did not differ between groups with regard to its amplitude (see Table 1 for mean amplitudes and statistical tests). Similarly, peak latencies were almost identical for both groups (control group: 140 ms; mIED group: 138 ms). In the control group, the MMN was followed by a clear P3a (with a peak latency of 238 ms), whereas the P3a was considerably smaller in the mIED group (with a peak at around 260 ms), the amplitude difference between groups being

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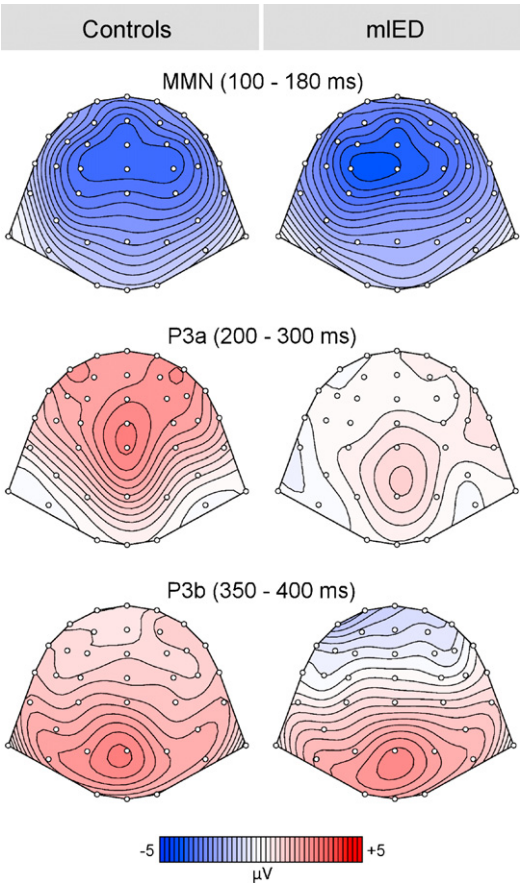

Fig. 2. Iso-potential maps of grand-average ERP effects (difference ERPs: standards subtracted from frequency deviants), interpolated over the time windows used for statistical analysis (see also legend of Table 1). View from top.

significant ($p < .0005$; see Table 1 for detailed statistics). Notably, in the same time interval, ERPs were similar for both groups at parietal leads (a MANOVA in the time interval from 200 to 300 ms for parietal ROIs did not show an interaction between factors condition and group, $p > .11$). This indicates that the difference between groups at frontal electrodes was due to a difference in P3a, not P3b potentials. When examining the ERPs on a single subject level, the P3a was absent in six subjects of the mIED group (i.e., subsequent to the MMN, there was no sampling point in which the ERPs of deviants were more positive than those of standards), whereas a P3a was elicited in all subjects of the control group. Even when excluding these six subjects from the analysis, the difference in P3a potentials between groups was significant (as indicated by an interaction between factors condition and group, $F(1,52) = 4.86$; $p < .05$). This indicates that the amplitude difference between groups was not only due to a subgroup of individuals without P3a.

Table 1
Mean amplitudes of ERPs (separately for the control group and the mIED group) and results of MANOVAs. MANOVAs were computed with factors condition (standard, deviant), hemispheric distribution (left, midline, right ROIs), and group (without, with mIED).

ERP	Mean (SEM) amplitudes		Condition	Condition × group
	Control group	mIED group	F_{58} (p-value)	F_{58} (p-value)
Frequency deviants				
MMN	−3.78 (.28)	−3.94 (.37)	**142.13 (.0001)**	.37 (.55)
P3a[a]	3.31 (.45)	.35 (.68)	**21.5 (.0001)**	**14.01 (.0005)**
P3b	2.91 (.42)	2.26 (.80)	**39.72 (.0001)**	.63 (.43)
RON	−2.65 (.40)	−3.08 (.73)	**56.46 (.0001)**	.31 (.58)
Omission deviants				
MMN	−4.78 (.46)	−5.50 (.69)	**163.36 (.0001)**	.81 (.37)
P3b	2.87 (.42)	3.64 (1.01)	**48.64 (.0001)**	.69 (.41)
Timbre deviants				
MMN/N2b	−7.05 (.70)	−6.08 (.72)	**144.45 (.0001)**	.78 (.38)
P3b	10.87 (1.16)	12.29 (1.20)	**163.97 (.0001)**	.62 (.44)

Time windows for statistical analyses were 100–180 ms (MMN), 200–300 ms (P3a), 350–400 ms (P3b), and 400–600 ms (RON). Bold font indicates statistical significance.

[a] The MANOVA on the P3a also indicated a main effect of group ($p < .02$).

The P3a was followed by a P3b that was maximal over parietal leads (best to be seen in the iso-potential maps of Fig. 2), and a frontal re-orienting negativity (RON, taken to reflect a re-orientation of attention after a distracting stimulus) [37]. Neither P3b, nor RON amplitudes differed between groups (see Table 1 for statistics).

For all effects (MMN, P3a, P3b, and RON) MANOVAs indicated interactions between factors condition and hemispheric distribution ($p < .001$ in all tests), reflecting that mean amplitude values of these effects were larger over midline electrodes compared to lateral leads (see also Fig. 2). The slight left-hemispheric distribution of the MMN in the mIED group was statistically not significant ($p > .24$).

The (task-irrelevant) *omission deviants* elicited an MMN-like negativity (but no clear P3a) with similar amplitudes in both groups, followed by a parietal positivity that was maximal at around 350 ms (presumably a P3b), and whose amplitude did also not differ between groups (see Table 1 and Supplementary Figure 1). Likewise, the (task-relevant) *timbre deviants* elicited an MMN/N2b followed by a P3b and a RON with amplitudes that did not differ between groups (see Table 1 and Supplementary Figure 2).

The present study provides the first ERP comparison between individuals with and without mIED. The P3a elicited by distracter stimuli (frequency deviants) was decreased in the mIED group, indicating that the neural resources underlying involuntary attentional processes differ between individuals with and without mIED (the P3a is taken to reflect switches of attention that do not require volitional effort). Notably, more than 25% of the subjects in the mIED group did not show any P3a potentials, whereas the P3a was elicited in all subjects of the control group (and the difference of P3a amplitudes between groups remained significant even when excluding the mIED subjects with absent P3a). This indicates that the difference in P3a potentials between groups was not due to a greater temporal jittering of P3a latencies, but due to diminished, or absent, P3a potentials in the subjects of the mIED group.

The present results bear an interesting resemblance to results from studies investigating the P3a in individuals with attention deficit/hyperactivity disorder (AD/HD), which is also often characterized by uncontrollable, impulsive temper tantrums (in addition to hyperactivity, inattention and lack of concentration) [10]. Smaller amplitudes of the P3a (elicited using a go/nogo-like paradigm) have been reported in children with AD/HD compared to control children [38], and an early subcomponent of the novelty-P3 (a P3a-like ERP component associated with the orienting response and the detection and evaluation of novelty) was reported to have smaller amplitudes over fronto-central left-hemisphere recording sites in a group of AD/HD children (compared to controls) [18], although this finding was not replicated in a subsequent study with a similar experimental design [41]. Consistent with these findings, smaller P3a amplitudes were also shown in different groups of AD/HD children, namely a group with predominantly inattentive children, and a group with the combined inattentive and hyperactive type [24]. In addition to studies on AD/HD, reduced P3a (or frontal P300) amplitudes have also been reported for impulsive individuals with chronic cocaine dependence [8], for male subjects with both increased impulsivity and alcohol problems [23], and for college students with impulsive aggression [28]. In the lat-

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ter study, clear P300 potentials were elicited at frontal electrodes by task-irrelevant rare deviants in normal controls, whereas this effect was almost absent in the impulsive-aggressive group. The results of these studies corroborate the present findings of an association between increased impulsivity and lack of P3a potentials and thus suggest a close link between increased impulsivity and cognitive mechanisms of involuntary control of attention (for P3a effects in patients groups not suffering from increased impulsivity such as patients with obstructive sleep apnea syndrome see Ref. [17]).

The P3b did not differ between groups, suggesting that mIED is not related to voluntary processes of attention. This stays in contrast to results of studies reporting reduced P3b potentials in individuals with impulsive aggression – however, these studies investigated clinical populations with co-morbidities such as antisocial personality disorder [4,19,28], and drug abuse [9,30]. In the present study, mIED did not co-occur with such disorders, perhaps accounting for the lack of a group difference in the P3b amplitudes; this issue remains to be specified.

Similarly, the MMN did not differ between individuals with and without mIED (note that deviants may also have elicited N1 differences due to refractoriness effects). Because the MMN is mainly generated in the temporal lobe, and the P3a mainly in the frontal lobe (see Introduction), this result suggests that mIED affects frontal, rather than temporal lobe activity, consistent with other studies showing relations between frontal lobe (dys)function and Intermittent Explosive Disorder or impulsive aggression [5,7,12,33,39].

The present MMN results contrast results of a previous study from Franken et al. [16] which reported that the MMN amplitude correlates with dysfunctional impulsivity (being larger in individuals with higher impulsivity). This difference between studies is perhaps due to the investigation of different impulsivity facets: dysfunctional impulsivity (as investigated by Franken et al. [16]) primarily refers to poor response inhibition and rapid, unplanned reactions, whereas mIED refers to uncontrollable, impulsive temper tantrums. Future studies should clarify which impulsivity facets exert influences on the neural mechanisms underlying the generation of the MMN (and the P3a), and how reliable such influences are. For this purpose, it would also be useful if more studies that primarily investigate the P3a (and not impulsivity) would obtain measures of impulsivity from the participants. With regards to the P3a, further studies could also examine the possible use of the P3a as a diagnostic index of pathologic impulsivity.

In conclusion, the present results reveal ERP correlates of mIED, showing that the P3a (but not the P3b) is reduced in individuals with mIED. This indicates that cognitive mechanisms of involuntary (but not voluntary) control of attention are affected by the propensity for impulsive, uncontrollable, excessive outbursts of temper. The MMN did not differ between groups, suggesting that processes of automatic auditory change detection are not affected by mIED, and that mIED affects processes mediated by frontal, rather than temporal lobe structures. Future studies could examine whether the P3a can be used as a diagnostic index for pathologic impulsivity.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.neulet.2009.05.047.

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