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Effects of mindfulness-based cognitive therapy on neurophysiological correlates of performance monitoring in adult attention-deficit/hyperactivity disorder

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HIGHLIGHTS

- Mindfulness-based cognitive therapy (MBCT) suggests enacting comparable neurophysiological effects related to attention and self-regulation as pharmacological treatments for ADHD.
- Enhanced error-positivity (Pe) amplitudes indexing error saliency/awareness were associated with ameliorated inattention symptoms.
- Increased NoGo-P3 amplitude reflecting greater inhibitory control correlated with attenuated hyperactivity/impulsivity symptomatology.

ABSTRACT

Objective: To examine whether mindfulness-based cognitive therapy (MBCT) would enhance attenuated amplitudes of event-related potentials (ERPs) indexing performance monitoring biomarkers of attention-deficit/hyperactivity disorder (ADHD).
Methods: Fifty adult ADHD patients took part in a randomised controlled study investigating ERP and clinical measures pre-to-post MBCT. Twenty-six patients were randomly allocated to MBCT, 24 to a wait-list control. Main outcome measures included error processing (ERN, Pe), conflict monitoring (NoGo-N2), and inhibitory control (NoGo-P3) ERPs concomitant to a continuous performance task (CPT-X). Inattention and hyperactivity–impulsivity ADHD symptoms, psychological distress and social functioning, and mindfulness skills were also assessed.
Results: MBCT was associated with increased Pe and NoGo-P3 amplitudes, coinciding with reduced 'hyperactivity/impulsivity' and 'inattention' symptomatology. Specific to the MBCT; enhanced Pe amplitudes correlated with a decrease in hyperactivity/impulsivity symptoms and increased 'act-with-awareness' mindfulness skill, whereas, enhanced P3 correlated with amelioration in inattention symptoms.
Conclusions: MBCT enhanced ERP amplitudes associated with motivational saliency and error awareness, leading to improved inhibitory regulation.
Significance: MBCT suggests having comparable modulation on performance monitoring ERP amplitudes as pharmacological treatments. Further study and development of MBCT as a treatment for ADHD is warranted, in addition to its potential scope for clinical applicability to broader defined externalising disorders and clinical problems associated with impairments of the prefrontal cortex.

1. Introduction

The human performance monitoring system synchronises flexible and adaptive goal-directed cognition and behaviour, encompassing error processing and conflict monitoring subsystems which update and modify subsequent response. Neural substrates

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of this complex network are multi-levelled and interdependent, although predominantly postulated within the region of the prefrontal cortex (PFC), specifically the dorsal anterior cingulate cortex (ACC) (Botvinick et al., 2004), and purported to be heavily involved in self-monitoring and self-regulation.

Increased dopaminergic system activity in the ACC correlates with electro-cortical amplitude increases in the early (50–150 ms) error-related negativity (ERN) and later (200–400 ms) positive voltage, error-positivity (Pe) event-related potentials (ERPs) (Holroyd and Coles, 2002; Biehl et al., 2011), evoked in response to conscious detection of error-making. Controversy exists regarding the functional significance of the ERN; whether it reflects activation of the error detection system to mismatch (Gehring et al., 1993) and corresponding correct-related negativity (CRN) (Vidal et al., 2000); a global conflict monitoring system activated by error vs. correct response choice (Yeung et al., 2004a); emotional response to making a known error (Luu et al., 2000); or more likely the interplay between cognitive and affective dynamics in error processing (Yeung, 2004b). The Pe generally reflects awareness of a committed error (Nieuwenhuis et al., 2001; Overbeek, 2005; Shalgi et al., 2009), implying a greater degree of affective evaluation to error significance compared to the ERN (Falkenstein, 2004). Impairments in performance monitoring are proposed to underlie symptoms of attention-deficit/hyperactivity disorder (ADHD) (Shiels and Hawk, 2010), supported by the clinical efficacy of methylphenidate-based pharmacology (Sunohara et al., 1999) which increase catecholamine release, such as dopamine (Missale et al., 1998).

Furthermore, depleted prefrontal cerebral dopamine, via branched chain amino acids (BCAAs) ingestion, has shown to attenuate N2 and P3 ERP component amplitudes (Neuhaus et al., 2009). The N2 (150–400 ms) reflects global performance monitoring processes associated with attention and motor response preparation (Eimer et al., 1996; Donkers and van Boxtel, 2004). Specifically, increased amplitude of the NoGo-N2 (evoked when a response is made for 'NoGo' stimuli) has been associated with increased conflict monitoring (Donkers and van Boxtel, 2004), and response inhibition (Falkenstein et al., 1999; Nieuwenhuis et al., 2004). The NoGo-P3 (300–500 ms) reflects a 'closure' potential of this inhibitory gating response circuit, exclusive to response inhibition execution (Donkers and van Boxtel, 2004). Attenuated ERN, Pe, NoGo-N2 and NoGo-P3 amplitudes evoked during tasks examining performance monitoring have been found in children (Albrecht et al., 2008; Senderecka et al., 2011) and adults (Prox et al., 2007; McLoughlin et al., 2009) with ADHD and other externalising problems (Sokhadze et al., 2008; Ruchsow et al., 2005; Franken et al., 2007; Brazil et al., 2009), representing biomarkers which subsequently 'normalise' when treated with pharmacology, targeting neurotransmission (Sunohara et al., 1999). Pharmacological treatments have limitations however, as average response rates are often lower in adults compared to children alongside related safety issues of medication abuse/addiction, and an overall lack of evidence for long-term treatment effects.

Mindfulness-based cognitive therapy (MBCT) is the coalescence of cognitive behavioural therapy (CBT) and mindfulness; a form of sustained attention training. Proposals subsume MBCT's therapeutic working pathways into: (a) attention regulation, (b) emotion regulation, (c) somatic awareness, (d) distancing from a self-focused perspective (Hölzel et al., 2011). Gaining a more sophisticated conscious relational understanding and active control of such internal domains during MBCT enhances cognitive flexibility, acute present-moment attention and bio-regulation, enabling insight and adaptation of maladaptive cognitions and behaviours underlying psychiatric symptoms.

Extant clinical applications of MBCT point to its versatility, possibly due to its potential for multi-faceted channels of efficacy. Disorders whose aetiology and maintenance are associated with dysfunctional fronto-limbic PFC-amygdala cortical networks and consequent emotion regulation, such as depression (Kenny and Williams, 2007; van Alderen et al., 2012), suicide vulnerability (Williams et al., 2006), bipolar disorder (Williams et al., 2008), generalised anxiety disorders (Evans et al., 2008), and borderline personality disorder (Sachse et al., 2010), have shown preliminary (or with depression, more extensive) promising clinical response to MBCT. Furthermore, MBCT has shown to be on par with antidepressant medication for relapse prevention (40–50% MBCT vs. 60% treatment-as-usual) (Teasdale et al., 2000; Ma and Teasdale, 2004; Kuyken et al., 2008; Segal et al., 2010). Attention regulation during MBCT provides a fitting rationale for its therapeutic application to ADHD. A modified protocol already indicates its feasibility for managing ADHD symptoms (Zylowska et al., 2008), warranting larger scale controlled trials. In line, non-clinical applications of intensive mindfulness training have shown to optimise response inhibition (Sahdra et al., 2011).

Parallel to treatment trials, advancing scientific knowledge of the working mechanisms of MBCT for psychiatric disorders is equally justified, contributing to improved clinical efficacy. To this regard, we were interested whether the attention training component of MBCT would have comparable therapeutic pathways as pharmacological treatments facilitating better attention regulation in ADHD (Volkow et al., 2007), via enhanced neurotransmission pathways. We hypothesised the mindfulness process would improve attention and self-regulation, examining this hypothesis using ERPs related to sustained attention and inhibitory control, further associated with ACC activity. To this end, ERPs provide reliable measures of brain function, regulated by neurotransmission (Cassidy et al., 2012). Specifically, we postulated amplitudes of ERPs pertaining to error-processing (ERN and Pe), and inhibitory processes (NoGo-N2 and NoGo-P3) would increase following exposure to MBCT, reflecting optimised performance monitoring. Furthermore, we anticipated ERP changes associated with the MBCT would improve clinical symptoms. To examine this hypothesis, correlational analyses using increment change measures of ERP, clinical, and mindfulness indexes were also conducted.

2. Methods

2.1. Sample

Sixty-one adult ADHD patients were recruited via Radboud University Nijmegen Medical Centre outpatient unit, 32 randomly allocated to the treatment condition (MBCT), and 29 to a wait-list (WL) control group. Subsequently, 11 patients (6 MBCT; 5 WL) did not attend the T2/post testing session. For two cases we dropped their participation because one did not attend the full 12-week MBCT intervention, the second started extra mindfulness training outside the intervention; and the further nine dropped out of the study due to time/scheduling/organisation limitations. Leaving 50 participating patients for the present study; 26 randomly allocated to the MBCT, and 24 to the WL.

Inclusion criteria were primary diagnosis of ADHD, *DSM-IV-TR* confirmed by three psychiatrists, in patients aged 18–65 years. Exclusion criteria were substance abuse/dependence within the last 6 months, co-morbid psychotic-, borderline-, antisocial-, and behavioural disorders, and learning difficulties. Of the 50 patients, 31 [15 (48.3%) MBCT; 16 (61.6%) WL] received pharmacological medication: 19 methylphenidate-based, 8 dextroamphetamine-based, and 4 anti-depressant medications (paroxetine, shown to have no mediating effects on the ERPs collected (de Bruijn et al., 2006)), leaving 19 non-medicated patients. Stimulant medication dosage was stabilized two weeks, non-stimulants 4 weeks, before

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participation and no changes were made to medication during the study.

2.2. MBCT intervention

The MBCT course was adapted from the protocol for depressive disorders (Segal et al., 2002), consisting of 12 weekly sessions for 3 h (for details of exercises, see Zylowska et al., 2009). Workbooks incorporating psycho-educative modules specific to ADHD were utilised, alongside assignments guided by compact disks (CDs) requiring on average 30–45 min self-practise per day. Maintenance of self-practise was monitored by the trainer. The course was administered by a psychiatrist specialising in ADHD, with 9 years experience as an MBCT trainer.

2.3. Procedure

Informed written consent to participate in a controlled randomised study (ethically approved by CMO, Arnhem-Nijmegen) was obtained from each patient before undergoing 2 × ±2-h sessions; pre-and-post MBCT for the MF group, or two sessions spaced 12-weeks apart preceding the onset of their MBCT course for the WL group. Randomisation (random number tables) was conducted prior to pre/T1 data collection. Each session comprised the completion of clinical scales, followed by an EEG recording concomitant to a standard visual continuous performance task (CPT-X).

Patients were instructed to keep muscular activity relaxed (e.g., shoulders, forehead) and refrain from eye movement/blinking, as much as possible during EEG recording periods.

The CPT-X task involved sequential presentation of 5 letters (A, F, H, Y, X): h = 2 cm, w = 1.5 cm, white on a black background. Patients were instructed to press a button as quickly and accurately as possible whenever they saw letters 'A', 'F', 'H', or 'Y' (396 Go stimuli), and not to press whenever they saw an 'X' (99 NoGo stimuli). Overall, 495 stimtuli (20% inhibition rate) were presented in 3× 165 stimuli blocks, with rest intervals between each block. Stimulus duration was 500 ms, random interstimulus interval (ISI) between 750–2200 ms. Before recording, a 30 stimuli (24 Go) practise block ensured task comprehension.

2.4. Clinical measures

Clinical scales were administered pre-and-post: (a) Conners' Adult ADHD Self-rating Scale (CAARS-S:SV) (Conners et al., 2008), measuring global *DSM-IV* ADHD symptoms, and 'hyperactivity–impulsivity' and 'inattention' subdomains; (b) outcome questionnaire (OQ 45.2) (Lambert and Finch, 1999), assessing: 'symptom distress', 'interpersonal relations', and 'social role'; (c) Kentucky Inventory of Mindfulness (KIMS) (Baer et al., 2004), measuring core mindfulness skills: 'observe', 'describe', 'act-with-awareness', and 'accept-without-judgement', with valid psychometric applicability to clinical populations (Baum et al., 2010).

2.5. Electrophysiological recording (online)

EEG data were acquired using Brain Vision Recorder 1.03 software and QuikAmps 72 hardware (http://BrainProducts.com), recorded from 30 Ag/AgCl active electrode sensors with integrated noise subtraction circuits (actiCAP: Brain Products) located in accordance with the 10–10 electrode system (sites: Fp1, Fp2, AFz, F7, F3, Fz, F4, F8, FC5, FC1, FCz, FC2, FC6, T7, C3, Cz, C4, T8, CP5, CP1, CP2, CP6, P7, P3, Pz, P4, P8, O1, Oz, O2). Average online reference was used, and referenced to the right mastoid offline. Ground electrode on the forehead. Vertical and horizontal ocular activity were calculated by bipolar derivations of electro-oculogram signals recorded using Ag/AgCl cup electrodes above and below the left eye, and 1 cm to the outer canthi of each eye, respectively. Impedance was maintained <10 KΩ. Electrical signal was continuously sampled at a digitization rate of 500 Hz, with a band-pass filter of 0.1–100 Hz.

2.6. Signal analysis (offline)

ERP analysis was conducted using Brain Vision Analyzer 2.0 Data were filtered between 0.1–30 Hz (24-dB/octave slope), via zero-phase shift band-pass (IIR Butterworth) and 50 Hz notch filters. Ocular artefacts were corrected using the regression method (Gratton et al., 1983). Data were segmented into: (1) response-locked false alarms to NoGo stimuli (FA), (2) response-locked correct hits to Go stimuli (CH), (3) stimulus-locked NoGo trials (NoGo-T), (4) stimulus-locked Go trials (Go-T); epochs from −200 to 600 ms relative to response or stimulus onset. Artefact rejection removed trials where voltages exceeded ±50 μV. Data were baseline corrected from −200 to −50 ms for response-locked, and −200 to 0 ms for stimulus-locked epochs, before computation of averages for each condition. Only correct responses to Go or correctly rejected NoGo trials were used for stimulus-locked averages, for subsequent grand average calculation. The minimum number of trials used for response-locked ERPs was <5.

Visual inspection of individual participant averages determined maximal peak windows, measured from baseline to peak, for the following ERP components of interest: (1) response-locked evoked potentials: ERN (30–150 ms), Pe (200–450 ms) for false alarms to NoGo stimuli, and CRN (30–150 ms), Pc (200–450 ms) for correctly rejected NoGo. Stimuli-locked components included: N2 (220–400 ms), and P3 (300–550 ms) extracted on Go and NoGo trials.

Difference waveforms were computed for the ERN/Pe [FA–CH], and NoGo-N2, NoGo-P3 [NoGo-T–Go-T]. The same peak temporal windows (above) identified maximal amplitudes. For illustrative purposes, topographical maps were calculated from grand averaged difference waveforms. Despite debate concerning normalisation procedures for ERP amplitudes prior to spatial distribution mapping, vector scaling normalisation was not employed following critique and recommendation of the unreliability of such methods to spatial data distributions (Urbach and Kutas, 2002). Current source density maps were calculated via spherical spline interpolation, where order of splines = 4, maximum degree of Legendre polynomial = 10, smoothing constant λ = 1e-5.

2.7. Statistical analysis

2.7.1. Behavioural measures

Time (pre, post) × Condition (FA, CH/correct Go-T, correct NoGo-T, omitted Go-T) × Group (MBCT, WL) repeated-measures ANCOVA (r-ANCOVA) compared accuracy score data. Time (pre, post) × Condition (FA, CH) × Group (MF, WL) r-ANCOVA was applied to response time data.

2.7.2. Evoked potentials

Response-locked components were maximal at fronto-central electrode sites: ERN = FCz; CRN = Fz; Pe = Cz; Pc = FCz. Stimuli-locked component maximal amplitudes = FCz for Go-N2/NoGo-N2; and Cz for Go-P3/NoGo-P3.

Time (pre, post) × Condition (Go, NoGo) × Site (Fz, FCz, Cz) × Group (MF, WL) r-ANCOVAs examined response-locked components. Time (pre, post) × Condition (Go, NoGo) × Site (Fz, FCz, Cz, Pz) × Group (MF, WL) r-ANCOVAs examined stimulus-locked components. Amplitude and latency measures were analysed separately.

All analyses (clinical, behavioural, ERP) were co-varied for medication status, age and sex. Where assumption of sphericity was

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violated, Greenhouse-Geisser corrections were applied. Follow-up analyses applied conservative Bonferroni correction.

Bivariate Pearson *r* correlation were applied to increment change indexes subtracting pre from post means for each measure, to examine correlations between ERP and clinical/mindfulness data.

3. Results

Due to the various findings analysed vs. reporting length constraints, non-significant results are not explicitly reported in the following sections.

3.1. Missing data

Six datasets (2 MBCT, 4 WL) could not be included in the analyses; two were dropped due to too few trials (<5) available for the response-locked ERPs, and for a further four datasets a technical issue meant no response markers were logged in the case of two pre/T1 and two post/T2 recording sessions. Of the remaining $N = 44$, complete clinical datasets were not available for two patients (1 MBCT, 1 WL); in one case the pre/T1, the other the post/T2, questionnaires were not completed at the time of testing due to practical/time constraints.

3.2. Demographics and baseline comparisons

The statistically viable $N = 44$ sample (24 MBCT vs. 20 WL), was matched between groups for Age ($p = .15$: MBCT = 39.5 (9.5) years, range = 19–53; WL = 33.9 (9.8) years, range = 22–50), Sex ($p = .14$: MBCT = 15F, 9 M; WL = 8F, 12 M), and Medication Status ($p = .42$: MBCT = 14 med, 10 non-med; WL = 14 med, 6 non-med). Although medication status did not differ between groups, it was still factored as a statistical co-variate. Clinical, behavioural and ERP measures did not significantly differ between groups at T1/baseline.

3.3. Behavioural data

As expected, a main effect of Condition was evident for task accuracy scores ($F(3, 120) = 92.828$, $p < .0001$), and also for RTs ($F(1, 40) = 5.724$, $p = .022$). Despite no significant Group effect/Time × Group interaction, number of FAs significantly decreased pre-to-post in the MBCT group alongside a significant slowing in RTs, not present in the WL (see Table 1).

3.4. ERN/CRN

As expected, main effects of Condition ($F(1, 41), = 19.059$, $p < .0001$) and Site ($F(2, 82) = 5.555$, $p = .01$) were evident, reflecting higher ERN amplitudes compared to CRN. There was no main effect of Group ($p = .85$), although a Time × Condition × Site × Group ($F(2, 82) = 3.357$, $p = .05$) interaction indicated overall ERN amplitude attenuation pre-to-post MBCT, contrary to amplitude increase at Fz and Cz in the WL. However, follow-up post hoc *t*-tests revealed such amplitude changes were not significant. Despite no main effect of Medication status ($p = .18$), a trend Condition × Site × Medication ($F(2, 82) = 3.035$, $p = .07$) interaction was found. Post-hoc tests showed overall medicated patients had higher ERN amplitudes compared to non-medicated, significantly so at Cz only ($F(1, 42) = 7.370$, $p = .01$). A Group × Medication post hoc data-split indicated there were no significant pre-to-post differences in either medicated or non-medicated patients for either group, aside for medicated patients exposed to MBCT showed a significant decrease in ERN amplitude at Cz ($t(13) = -2.323$, $p = .04$) [−9.84(8.4) μV to −7.27(6.7) μV).

Taking latency, main effects/interaction of Site ($F(2, 82) = 6.490$, $p = .002$), Group ($F(1, 41) = 5.452$, $p = .03$), and Condition × Site ($F(2, 82) = 3.145$, $p = .05$) were found. Although no main effect of Medication ($p = .85$), a Site × Medication ($F(2, 82) = 3.695$, $p = .03$) interaction revealed faster ERN/CRN latencies from pre-to-post in both MBCT and WL groups, regardless of medication status. Post-hoc Group × Medication tests were not significant, except for medicated patients undergoing MBCT showed significantly reduced ERN latency at Cz ($t(13) = 3.821$, $p = .002$) [71.6(19.6)–59.9(23.9) ms].

3.5. Pe/Pc

Main effects of Time ($F(1, 41) = 5.573$, $p = .02$), Condition ($F(1, 41) = 36.276$, $p < .0001$), and Condition × Site ($F(2, 82) = 3.552$, $p = .033$) indicated amplitudes increased pre-to-post in both groups, and significantly so at FCz for Pe ($t(23) = -2.613$, $p = .02$) [9.75(5.7)–13.99(7.3) μV] in the MBCT group (Fig. 1 and Fig. 2), contrary to Fz ($t(19) = -2.809$, $p = .01$) [8.5(3.5)–12.4(5.3) μV], and Cz ($t(19) = -2.139$, $p = .05$) [10.1(3.7)–12.09(5.1) μV] in the WL (Fig. 1 and Fig. 2). There were no significant findings for Pe/Pc latency measures.

3.6. NoGo-N2

Time ($F(1, 41) = 14.241$, $p = .001$), Site ($F(1, 41) = 8.071$, $p < .0001$) main effects, and Site × Group ($F(3, 123) = 3.011$, $p = .033$) interaction were evident. As there was a trend Time × Condition × Group ($F(1, 41) = 3.204$, $p = .081$) interaction, posthoc tests were conducted, indicating general increase in Go and NoGo-N2 amplitudes across sites in the WL, significantly so for Go-N2 at Fz ($t(19) = 3.902$, $p = .001$) [−2.79(2.6) to −4.20(2.9) μV], and Pz ($t(19) = 2.164$, $p = .04$) [.436(2.3) to −.115(2.0) μV]. Conversely, amplitude attenuation was evident for NoGo-N2 pre-to-post MBCT at Fz ($p = .86$) [−2.22(3.6) to

Table 1
Accuracy and reaction times for the continuous performance task in patients with ADHD: MBCT vs. WL control group.

	MBCT ($N = 24$) $\bar{X}$ (σ)		Comparison	WL ($N = 20$) $\bar{X}$ (σ)		Comparison	Effects/interaction
	Pre	Post		Pre	Post		
Behavioural variable							
FA (N)	25.3 (17)	19.8 (15)	$p = .001^{***}$	26.2 (17)	25.6 (15)	$p = .83$	Condition: $p < .0001^{**}$
FA (%)	25.5 (17)	20.0 (15)	$p = .001^{***}$	26.5 (17)	25.9 (15)	$p = .83$	
CH (N)	382.5 (54)	388.6 (27)	$p = 28$	393.5 (5)	393.8 (4)	$p = .82$	
CH (%)	96.6 (54)	98.1 (27)	$p = .28$	99.4 (5)	99.4 (4)	$p = .82$	
C-NoGo (N)	73.6 (18)	79.2 (15)	$p = .001^{***}$	72.8 (17)	73.5 (15)	$p = .82$	
C-NoGo (%)	74.3 (18)	80.0 (15)	$p = .001^{***}$	73.5 (17)	74.2 (15)	$p = .82$	
Reaction time							
FA (ms)	287.6 (63)	314.8 (41)	$p = .044^{*}$	290.7 (40)	300.7 (41)	$p = .25$	Condition: $p = .022^{*}$
CH (ms)	371.8 (50)	380.7 (42)	$p = .17$	359.1 (58)	357.9 (48)	$p = .89$	

FA, false alarms to NoGo stimuli; CH, correct hits to Go stimuli; C-NoGo, correctly rejected NoGo stimuli. * $p < .05$; ** $p < .01$; *** $p < .001$.

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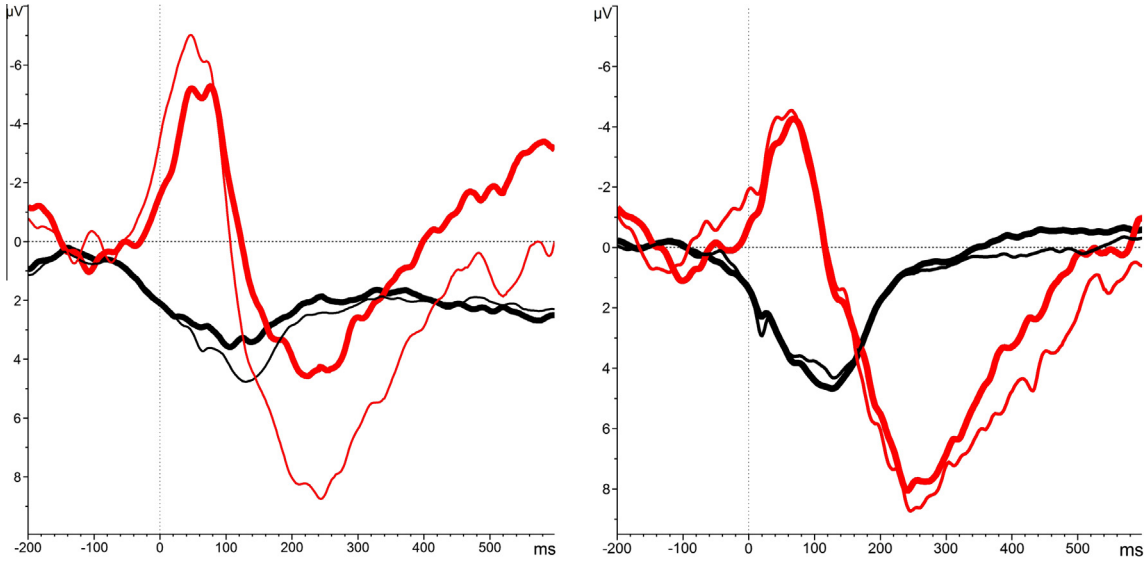

Fig. 1. ERN/Pe (red) and CRN/Pc (black) pre (thick) and post (thin) for the MBCT group at FCz (left) and WL group (right) at Cz [0 ms = response onset]. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

−2.11(3.1) μV], Cz, (p = .47) [−2.30(4.3) to −2.02(3.8) μV], and Pz (p = .51) [−1.64(2.8) to −1.34(2.9) μV], compared to overall increase for Go-N2, significantly so at FCz ($t(23)$ = 2.095, p = .05) [−1.43(2.8) to −2.18(2.7) μV]. No main effects of Group (p = .64), or Medication (p = .29).

Examining N2 latency, Site ($F(3,57)$ = 3.406, p = .05) and Time × Site × Group ($F(3,57)$ = 3.50, p = .04) effects showed decreased latency (faster peaking) for NoGo-N2 pre-to-post MBCT, compared to an overall slowing in WL, and slowing of Go-T for both groups. Although, latency change in both groups were marginal, not significant.

3.7. NoGo-P3

Higher amplitudes were yielded for NoGo-P3 compared to Go-P3 in both groups (Condition: $F(1, 41)$ = 60.080, p < .0001). Site ($F(3, 123)$ = 3.289, p = .02), Condition × Site ($F(3,123)$ = 11.631, p < .0001), Condition × Site × Group ($F(3, 123)$ = 2.696, p = .05), and Time × Site × Group ($F(3, 123)$ = 2.514, p = .06) and Time × Condition × Group ($F(1, 41)$ = 3.220, p = .08) trends, showed significant increase in Go-P3 ($t(23)$ = −2.986, p = .007) [5.04(2.7) to 5.96(2.8) μV], and NoGo-P3 ($t(23)$ = −2.502, p = .02) [8.82(4.4) to 10.10(4.1) μV] amplitudes at Pz pre-to-post MBCT (Fig. 3 and Fig. 4), contrary to parietal (Pz) decrease in the WL for Go-P3 (p = .42) [6.14(2.2) to 5.76(2.5) μV], and NoGo-P3 (p = .40) [9.69(2.8) to 9.13(3.7) μV] (Fig. 3 and Fig. 4).

Taking latency, Time ($F(1, 41)$ = 4.048, p = .05), Condition ($F(1, 41)$ = 4.594, p = .04), Site ($F(3, 123)$ = 7.673, p < .0001), and Condition x Site ($F(3, 123)$ = 12.073, p < .0001) interaction show overall increase in Go/NoGo-P3 latency in both groups.

3.8. Clinical effects

Examining the CAARS-SV, main effects of Group ($F(1, 38)$ = 4.713, p = .04), Domain ($F(2, 76)$ = 28.884, p < .0001), further to Time × Group ($F(1, 38)$ = 9.248, p = .004), and Time × Domain × Group ($F(2, 76)$ = 5.227, p = .01) interactions, showed reduced 'inattention' ($t(22)$ = 4.891, p < .0001), 'hyperactivity/impulsivity' ($t(22)$ = 3.161, p < .0001), and global ADHD index ($t(22)$ = 4.239, p < .0001) symptoms pre-to-post MBCT exclusively. (see Table 2).

Examining the outcome questionnaire (OQ-45.2), main effect of Domain ($F(2, 76)$ = 28.885, p < .0001), and Time × Group ($F(1, 38)$ = 4.924, p = .033) interaction indicated amelioration in 'symptom distress' ($t(22)$ = 2.392, p = .03), 'social role' ($t(22)$ 2.265, p = .03), and global score ($t(22)$ = 2.964, p = .007), in the MBCT group only (Table 2).

3.9. Mindfulness skills

Main effect of Domain ($F(3, 114)$ = 3.338, p = .03), and Time × Group ($F(1, 38)$ = 22.845, p < .0001) interaction, reflected improved mindfulness skills for all domains in the MBCT group pre-to-post; 'observe' ($t(22)$ = −3.301, p = .003), 'describe' ($t(22)$ = −2.459, p = .022), 'act-with-awareness' ($t(22)$ = −4.350, p < .0001), and 'act-without-judgement' ($t(22)$ = −2.681, p = .01). As expected, no significant changes were evident in the WL group. (Table 2).

3.10. Correlational analyses

Increases in act-with-awareness on the KIMS correlated with decreases in CAARS global scores ($r(23)$ = −.832, p < .001), 'inattention' ($r(23)$ = −.618, p = .002), and 'hyperactivity/impulsivity' ($r(23)$ = −.893, p < .001) subdomains in the MBCT group. Likewise, increases in KIMS act-without-judgement correlated with decreases in global CAARS ($r(23)$ = −.632, p = .001), 'inattention' ($r(23)$ = −.632, p = .001), and 'hyperactivity/impulsivity' ($r(23)$ = −.533, p = .009) exclusive to MBCT. Conversely, CAARS 'inattention' and KIMS 'observe' were positively correlated in the WL ($r(19)$ = .537, p = .02).

Examining mindfulness/CAARS and ERP measures; no significant correlations pertained to the ERN in either group, nor for the Pe in the WL. However, reduction in CAARS 'hyperactivity/impulsivity' correlated to increased Pe amplitudes at Fz ($r(23)$ = −.456, p = .03) and Cz ($r(23)$ = −.453, p = .03) pre-to-post MBCT only, further to increased KIMS act-with-awareness associated with increased Pe amplitude at Fz ($r(23)$ = .491, p = .02).

Increased P3 amplitudes correlated to increased mindfulness skills pre-to-post MBCT only. KIMS 'describe' with the Go-P3 at Cz ($r(23)$ = .483, p = .02), and FCz ($r(23)$ = .416, p = .05). act-without-judgement and the Go-P3 at Cz ($r(23)$ = .513, p = .01), and Pz ($r(23)$ = .545, p = .007), further to the NoGo-P3 at Cz ($r(23)$ = .466, p = .03), and Pz ($r(23)$ = .484, p = .02). Furthermore, reduced scores on the CAARS-inattention subdomain and increased amplitudes at

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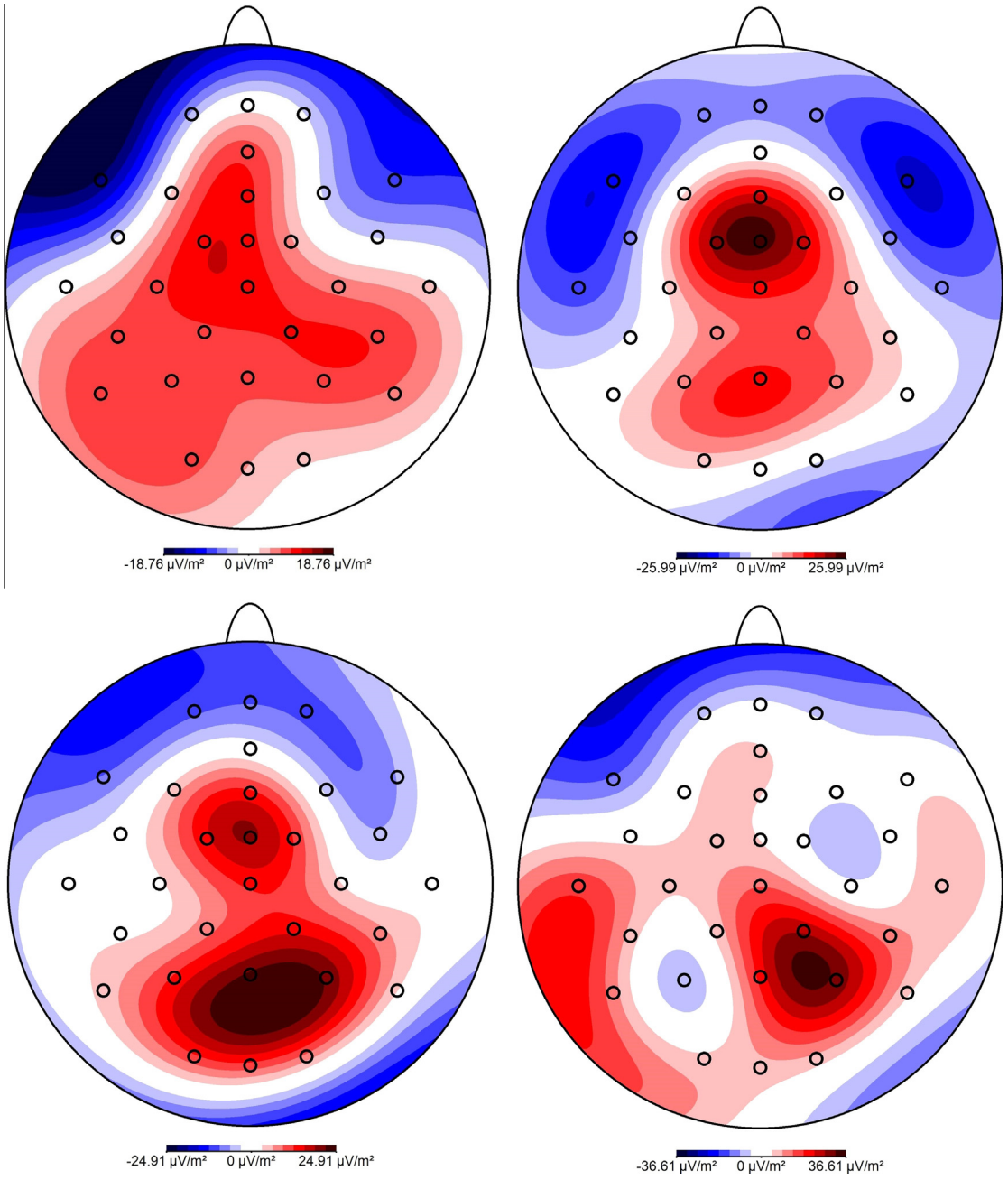

Fig. 2. Current source density (CSD) maps for the peak in the Pe difference waveform in the MBCT group (above) and WL (below) group at pre/T1 (left) and post/T2 (right).

Cz for Go-P3 ($r(23) = -.429$, $p = .046$), and NoGo-P3 ($r(23) = -.476$, $p = .02$), were evident.

4. Discussion

To our knowledge this is the first study to explore the effects of MBCT on ERP markers and related clinical amelioration in adult ADHD. Primarily, we investigated whether amplitudes of ERPs indexing performance monitoring, related to inattention and hyperactivity–impulsivity symptoms, would increase following MBCT. In accord, MBCT enhanced electro-cortical amplitudes of later evoked ERPs associated with error awareness, motivational saliency, and inhibitory control, alongside amelioration in inattention and hyperactivity/impulsivity symptoms.

4.1. Error processing

Neurophysiological change pertained to later error processing; as the ERN component was mitigated by medication confounds regardless of group, suggesting MBCT did not have direct regulatory effects upon 'automatic' visual error detection via mismatch template upgrading, or improved conflict monitoring, further supported by the absence of modulation upon the N2 component. Rather, a significant increase in the fronto-central Pe implies an increase in conscious error processing (Overbeek, 2005) and subjective significance towards error-making, engaging also an increased affective component (Falkenstein, 2004). To this regard, poor error processing in externalising disorders such as ADHD and psychopathy have been associated with impaired affective processing

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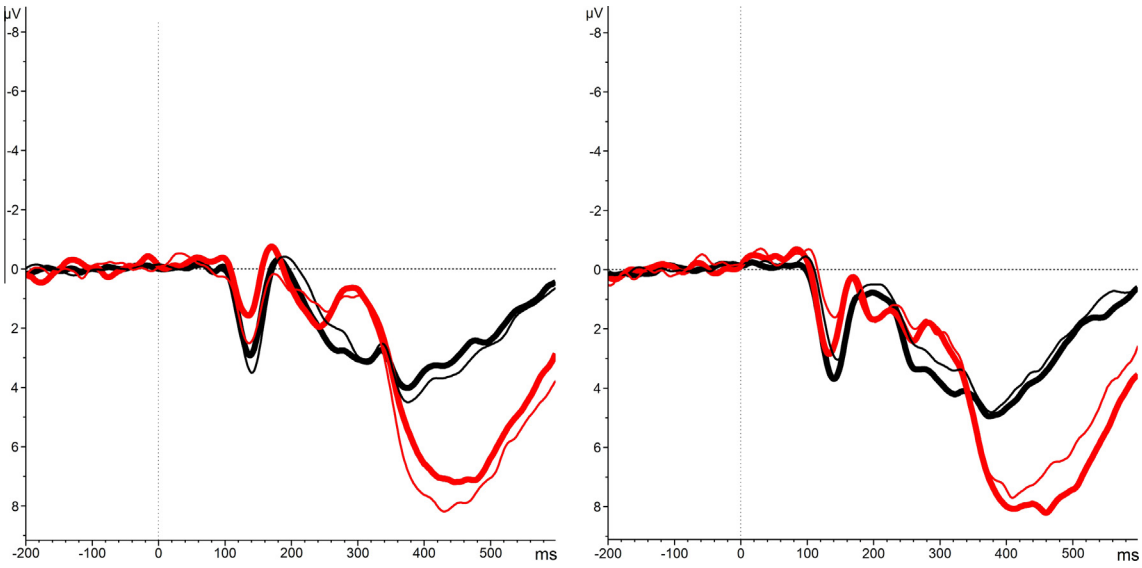

Fig. 3. NoGo (red) and Go (black) pre (thick) and post (thin) for N2 and P3 components for MBCT group at Pz (left) and WL group (right) at Pz [0 ms = stimulus onset]. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

towards the conscious appraisal of error-making, also reflected in post-error slowing times which fail to adjust responses accordingly (Brazil et al., 2009; Barkley, 1997a). Although increased Pe amplitude was also yielded in the WL group, the mindfulness-skills data sheds further light on these findings, as it is plausible to hypothesise the increase in Pe amplitude, indexing error awareness, was driven by an overarching increase in global cognitive and affective self-awareness, reflected by improvement in act-with-awareness on the KIMS following MBCT which also correlated with increased Pe amplitudes. In this vein, a prior study examining degrees of trait mindfulness using the KIMS found act-with-awareness to be significantly lower in adult ADHD patients (Smalley et al., 2009).

Conversely, a recent study in healthy undergraduates exposed to mindfulness training via CDs totalling approximately 30 min, showed training was associated with a reduction in Pe amplitude (Larson et al., 2013). However, participants underwent a considerably briefer application of a differing mindfulness training known as mindfulness-based stress reduction (MBSR). MBSR focuses on 'ethical living' and regulation of the autonomic nervous system, supported by the additional finding of decreased systolic blood pressure in the mindfulness group (Larson et al., 2013). This contrast in findings supports the discreteness of the two mindfulness systems (i.e., MBCT vs. MBSR), whereby the attention training component of MBCT appears apt for ADHD and related psychiatric disorders.

4.2. Inhibitory gating regulation

Enhanced NoGo-P3 amplitudes were exclusive to the MBCT intervention. Although both the NoGo-N2 and P3 index a neural inhibitory gating circuit (Donkers and van Boxtel, 2004), evidence purports the NoGo-N2 reflects the early regulatory stage of conflict monitoring, whereas the NoGo-P3 has been related to cognitive and motor control involved in response inhibition (Bekker et al., 2004; Dimoska et al., 2006; Jonkman, 2006; Smith et al., 2008), suggesting MBCT targeted the latter mechanistic stage. Pertinently, the Pe and P3 ERPs are hypothesised to index overlapping higher-order processing of consciously salient events (Ridderinkhof et al., 2009). Ergo, we can infer MBCT targeted this shared processing system of these intrinsically related but discrete components.

These results are in line with research suggesting ADHD may not necessarily represent an inhibition-specific dysfunction, but impaired response execution (Banaschewski et al., 2004). Improved bio-regulation skills via body-scanning and present-moment embodiment exercises undergone during MBCT may have contributed to increased motor control, and regulation of the NoGo-P3 component of the inhibitory gating 'circuit'. This connects well with the idea that ADHD is characterised by dysregulation in inhibitory control, as opposed to an absolute inhibitory gating deficit (Yong-Liang et al., 2000), whereby MBCT improved inhibitory regulation subsystems. Amelioration in hyperactivity/impulsivity symptoms, additional to improvement in inattention on the CAARS, provides further support.

4.3. Attention training

The findings so far indicate improvement in performance monitoring related to attentional processes of MBCT, enhancing error awareness and appropriate inhibition regulation towards better impulsivity regulation. Injecting the behavioural data into our analysis; the patient group as a whole responded accurately to Go-T (mean range 96.6–99.4%), suggesting our ADHD sample did not have marked sustained attention impairment, rendering it unlikely that MBCT elicited task improvement via enhanced focused attention. Thus, redefining our perspective of attention to precisely delineate how the symptom of 'inattention' was ameliorated, a distinction can be drawn between focused attention and awareness/vigilance. The latter more open, reflexive, and enabling attentional 'switching' capacity, epicentral to 'open monitoring' attention practised in MBCT.

Modulation of focused linear attention vs. parallel levels of awareness can be explained by molecular models of attention; the former predominately regulated by dopamine homeostasis, and the latter by adrenergic activity and associated norepinephrine neurotransmission (Deth, 2003). The NoGo-P3 is associated with monoamine neurotransmitters such as norepinephrine (Nieuwenhuis et al., 2005). Although the neurotransmission network of the brain is integral and highly complex, norepinephrine has not been closely linked to error monitoring ERPs, such as the ERN, mediated by dopamine neurotransmission (Meyer et al., 2012). Whilst ADHD is commonly associated with dopamine imbalance, research

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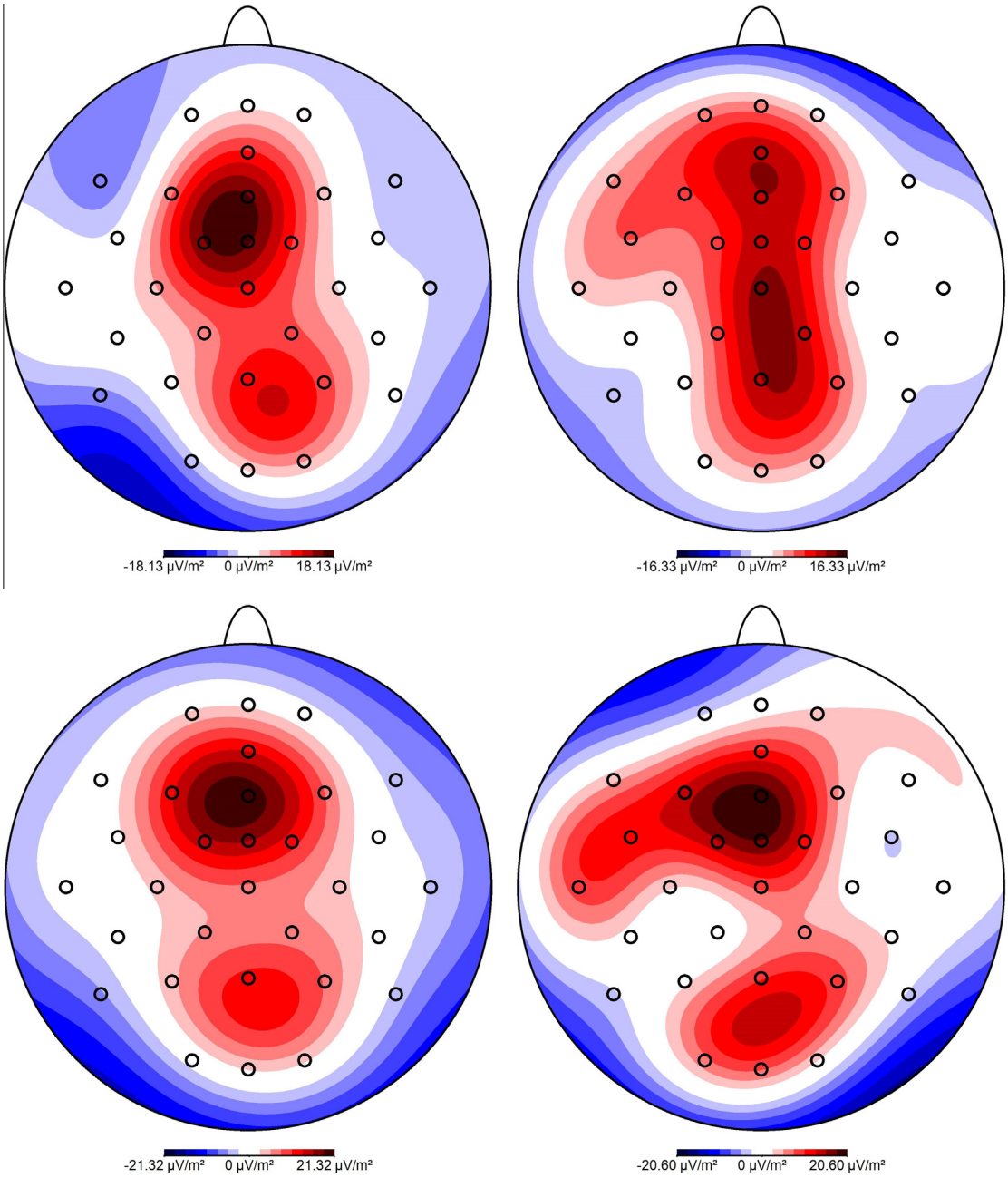

Fig. 4. CSD maps for the peak in the P3 difference waveform in the MBCT group (above) and WL group (below) for pre/T1 (left) and post/T2 (right).

concedes dysfunction in the noradrenergic system also has adverse effects on attentional symptomatology (Pilszka et al., 1996). This would implicate that the main regulatory role of MBCT in our ADHD sample was likely by top-down control of PFC areas such as the ACC via norepinephrine activity, reflected in later peaking ERPs indexing higher-order processing. However, noradrenergic networks regulating norepinephrine are postulated to originate in the locus coeruleus, a structure of the brain stem (Nieuwenhuis et al., 2005). This opens the possibilities regarding mechanisms of action in mindfulness-based treatments due to an encompassing scope to engage attentional 'top-down' neural pathways that interplay with emotional and bio-regulatory 'bottom-up' pathways. Pertinent to ADHD applications, interestingly, the reticular activating system, a collection of nuclei at the base of the brainstem heavily synchronised with noradrenergic neurons of the locus coeruleus, is theorised to function sub-optimally within the disorder, causally linked to deficiencies in executive and attentional functioning (Garcia-Rill, 1997).

4.4. Affective self-regulation

Aside the predicted attentional working pathway of MBCT, increased emotion regulation in our ADHD sample may also be relevant. Enhanced Pe amplitude implicates increased affective evaluation to errors (Overbeek, 2005; Falkenstein, 2004). This may have been functionally 'counterbalanced' by an emotion regulatory mechanism, as maladaptively high levels of error awareness and evaluative significance theoretically pose to impede error processing due to error 'fixation', hypothetically interfering with optimal task performance. Furthermore, improvements in inhibitory control may also be considered within a framework of enhanced self-regulation of affect-motivation-arousal

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Table 2
Clinical scale measures.

Psychometric variable	MBCT $\bar{X}$ (σ)		Comparison	WL $\bar{X}$ (σ)		Comparison	Effects/interaction
	Pre	Post		Pre	Post		
CAARS-SV							
InA raw score	15.8 (4.2)	12.2 (4.8)	$p < .0001^{***}$	17.4 (3.6)	17.4 (3.3)	$p = 1.0$	Domain: $p < .0001^{***}$
InA T score#	71.9 (10)	62.2 (12)	$p < .0001^{***}$	77.4 (9.8)	77.8 (9.3)	$p = .79$	Time × Group: $p = .004^{**}$
H/I raw score	13.7 (4.8)	10.8 (4.6)	$p = .005^{**}$	14.3 (4.4)	14.0 (5.6)	$p = .62$	Time × Domain × Group: $p = .005^{**}$
H/I T score#	62.5 (11)	55.5 (11)	$p = .004^{**}$	64.7 (11)	63.7 (13)	$p = .56$	
G raw score	29.5 (7.5)	23.0 (8.5)	$p < .0001^{***}$	31.7 (5.8)	31.3 (6.7)	$p = .74$	Group: $p = .04^{*}$
G T score#	71.2 (11)	61.8 (12)	$p < .0001^{***}$	72.2 (7.2)	71.8 (8.3)	$p = .83$	
OQ-42.5							
Symptom distress	43.2 (13)	38.2 (11)	$p = .03^{*}$	42.8 (8.0)	43.1 (10)	$p = .87$	Domain: $p < .0001^{***}$
Interpersonal relations	21.3 (2.5)	20.3 (2.8)	$p = .07$	21.1 (3.8)	22.2 (2.9)	$p = .07$	Time × Group: $p = .033^{*}$
Social roles	14.9 (3.1)	13.5 (2.8)	$p = .03^{*}$	15.3 (3.5)	14.9 (2.4)	$p = .56$	
Global score	79.3 (14)	72.0 (14)	$p = .007^{**}$	79.1 (12)	80.2 (13)	$p = .66$	
KIMS							
Observe	22.8 (8.3)	27.6 (8.2)	$p = .003^{**}$	23.3 (6.1)	23.1 (7.5)	$p = .90$	
Describe	17.2 (5.8)	19.6 (5.1)	$p = .022^{*}$	17.3 (6.0)	17.8 (7.4)	$p = .53$	Time × Group: $p < .0001^{***}$
Act-with- awareness	12.9 (4.7)	18.0 (5.5)	$p < .0001^{***}$	12.6 (4.2)	11.6 (4.1)	$p = .18$	
Act-without-judgement	19.8 (7.3)	24.4 (6.7)	$p = .01^{**}$	22.7 (4.7)	20.2 (7.4)	$p = .07$	Domain: $p = .03^{*}$

CAARS-SV abbreviations: InA, inattention DSM-IV symptoms; H/I, hyperactivity/impulsivity DSM-IV symptoms; G, global DSM-IV ADHD symptoms; #T scores, comparison of raw scores to age and sex matched normative samples. T scores above 65 represent clinically significant symptoms. * $p < .05$; ** $p < .01$; *** $p < .001$.

(Barkley, 1997b), suggesting the MBCT increased motivational saliency, in turn, self-regulation and engagement with the attention task. Relevantly connected to the previous section, increased prefrontal norepinephrine outflow has been associated with high motivational salience towards stimuli (Ventura et al., 2008).

4.5. Limitations

This study presents various limitations. Firstly, the lack of an active control group is a significant methodological constraint. MacCoon et al. (2012) highlight the validity of using comparisons other than wait-list controls to increase the rigorousness of such research. The inclusion of an additional medication control group in the present study would be methodologically advantageous to examine the inferred hypothesis that MBCT has similar effects on pertinent neurotransmission systems in ADHD as pharmacology. Secondly, over half the patients were on psychotrophic medication. Albeit, medicated patients were equally dispersed within each group, medication was kept stable pre-to-post, and it was only found as a trend statistical confound for the ERN, which was not significantly affected by the MBCT. Thirdly, self-report CAARS is not ideal to gauge ADHD symptoms. Ergo, lack of an objective assessment is limiting, although, the self-reported surveys were aimed to supplement the primary ERP and behavioural measures.

5. Summary

MBCT increased Pe and NoGo-P3 ERP amplitudes, collectively improving inhibitory gating regulation, akin to a reflexive 'switching' ability, associated with underlying noradrenergic-regulated global attention processes related to enhanced awareness/vigilance. In alignment, dopaminergic-regulated focused attention may not have been the principal facet by which the MBCT worked, supported by the lack of modulation on the ERN. Furthermore, improved motor and emotion regulation, reflected in associated P3 and Pe amplitudes, plausibly had 'bottom-up' homeostatic effects on neural systems related to performance monitoring, so that functional ability was maintained and suitably adaptive. These findings advocate the development and further study of MBCT for ADHD interventions, in addition to its potential scope for clinical applicability to broader defined externalising disorders and problems associated with impairments of the prefrontal cortex.

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Conflict of Interest

None.

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