

Impaired filtering of irrelevant information in dysphoria: An ERP study

Max Owens¹, Ernst H. W. Koster², and Nazanin Derakshan¹

¹*Birkbeck University of London, UK*

²*Ghent University, Belgium*

Address for correspondence:

Nazanin Derakshan, PhD
Affective and Cognitive Control Lab
Department of Psychological Sciences
Birkbeck University of London
Malet Street
London, WC1E 7HX
UK.

Email: n.derakshan@bbk.ac.uk

Abstract

Behavioural findings have led to proposals that difficulties in attention and concentration in depression may have their roots in fundamental inhibitory impairments for irrelevant information. These impairments may be associated with reduced capacity to actively maintain relevant information to facilitate goal directed behaviour. In light of mixed data from behavioural studies, the current study using direct neural measurement, examines whether dysphoric individuals show poor filtering of irrelevant information and reduced working memory capacity for relevant information. Consistent with previous research, a sustained event related potential (ERP) asymmetry, termed contralateral delay activity (CDA), was observed to be sensitive to working memory capacity and the efficient filtering of irrelevant information from visual working memory. We found a strong positive correlation between the efficiency of filtering irrelevant items and visual working memory capacity. Specifically, dysphoric participants were poor at filtering irrelevant information, and showed reduced working memory capacity relative to high capacity non-dysphoric participants. Results support the hypothesis that impaired inhibition is a central feature of dysphoria and are discussed within the framework of cognitive and neurophysiological models of depression.

Keywords

attentional control, depression, inhibition, working memory capacity, contralateral delay activity, filtering efficiency

Introduction

Depression is recognised as a severe multifaceted disorder that includes affective, physiological, as well as cognitive symptoms. The cognitive symptoms observed in depression are typically deficits in attention and concentration (Mohanty & Heller, 2002). In a recent review (Levin, Heller, Mohanty, Herrington, & Miller, 2007) it was suggested that these cognitive symptoms may have their roots in fundamental executive function impairments. Generally, executive function describes a collection of top-down cognitive processes, mainly located in the prefrontal cortex, which control attention to produce goal directed behaviour (Miller & Cohen, 2001). Hertel (1994) has proposed that depressed individuals have difficulty exercising attentional control in order to allocate available resources to task demands. It is thought that this is mainly reflected in inhibitory deficits, where an important function of inhibitory processes is to limit the disruptive influence of distractors on relevant information. One way inhibition may do this is by filtering out task-irrelevant information (Friedman & Miyake, 2004).

Interestingly, a wealth of cognitive neuropsychological research has investigated cognitive inhibition in depression using, among others, the *n*-back task (Harvey et al., 2005), oddball task (Kaiser et al., 2003), anti-saccade task (Sweeney, Strojwas, Mann & Thase, 1998), stroop task (Gohier et al., 2009) and rapid serial visual presentation tasks (Rokke, Arnell, Koch & Andrews, 2002). Although attention and concentration problems are considered important symptoms of depression, these studies have provided mixed support for the idea that depression is characterized by inhibitory deficits (Joormann et al., 2007). Often inhibitory performance was quite similar in depressed versus non-depressed individuals with mainly severely depressed individuals being characterized by marked impairments.

As studies have mainly used tasks relying on behavioural outcomes (i.e., reaction times, errors) one important limitation is that the absence of impairments does not imply that inhibitory functioning is as efficient in depressed as in non-depressed individuals. These behavioural outcomes do not generally speak to the *mechanisms* underlying inhibition of task-irrelevant material. To this end, Dillon and Pizzagalli (2007) advocated a neuroscientific approach to study in a more direct fashion the brain mechanisms involved in inhibitory-related processes (see also Aron, 2007). For instance, in a recent fMRI study, activity within the prefrontal cortex and basal ganglia was found to precede filtering of irrelevant items in the posterior parietal cortex and this in turn predicted inter-individual differences in visual working memory capacity (McNab & Klingberg, 2008).

Inhibition has been proposed to facilitate efficient goal directed behaviour by reducing the access and maintenance of irrelevant information in working memory (Hasher, Zacks, & May, 1999). A crucial function of the working memory system is to keep relevant information readily retrievable when the task context provides interfering information that would lead to an inappropriate response. The amount of relevant information that can remain active is the result of an ability to use attention to avoid distraction (Engle, 2002). In this view inhibition modulates individual differences in working memory capacity (WM capacity; Kane, Bleckley, Conway, & Engle, 2001). As such, recent reviews and models of cognition in depression (Joormann et al. 2007; De Raedt & Koster, 2010) have highlighted inhibitory impairments as an important cognitive risk factor for depression. That is, within the context of emotion regulation, efficient inhibition of task-irrelevant and/or negative information is crucial in regulating negative affect. Clear demonstration of the effects of inhibitory dysfunction on working memory capacity in depression may then provide valuable insight into understanding why some people are more prone to cognitive risk factors which increase the severity of depressive episodes such as depressive rumination (Nolen-Hoeksema, Wisco

& Lyubomirsky, 2008), and help provide an index for cognitive therapies which target working memory (De Raedt, Koster, & Joormann, 2010).

While cognitive symptoms of depression have mainly been described in terms of their verbal products such as the process of uncontrolled and persistent negative thoughts which characterizes depressive rumination (Nolen-Hoeksema, Wisco & Lyubomirsky, 2008), executive dysfunction may be expected to disrupt attentional control for processes of both the visual and verbal subsystems of working memory due to their integrated structure (Repovs & Baddeley, 2006). Thus, if dysphoria is associated with impaired inhibition of irrelevant information a reduction in visual working memory capacity should be expected.

There is recent ERP evidence to demonstrate that allocation of memory capacity to irrelevant information is significantly correlated with individual differences in overall WM capacity (Vogel, McCollough & Machizawa, 2005). High WM capacity individuals tend to filter out irrelevant information and focus attention on the most relevant items within a cognitive task, whereas low WM capacity individuals tend to be less efficient and allocate attentional resources to irrelevant information. Vogel et al (2005) measured WM capacity in a paradigm where on some trials participants were required to selectively remember a set of items (red rectangles) in the presence of task irrelevant distractors (blue rectangles). WM capacity was then estimated from performance on the task with participants typically grouped based on a median split of their accuracy scores. To observe the association between the ability to efficiently filter irrelevant information and WM capacity each participant's brain activity was recorded during the task using EEG. Vogel and Machizawa (2004) previously observed a large negative voltage over posterior regions contralateral to the position of the to-be-remember items on the display (Contralateral Delay Activity, CDA). CDA amplitudes have been found to be sensitive to the number of items remembered during each trial,

increasing significantly between arrays of up to 4 items (McCollough, Machizawa, & Vogel, 2007).

In Vogel et al. (2005) the CDA was used as a direct neurophysiological measure of filtering efficiency, “*we used the CDA as a direct neurophysiological measure of whether or not the irrelevant distractor items unnecessarily consumed memory capacity. For example on the trials in which two red items were presented simultaneously with two blue items, if an individual was perfectly efficient at remembering only the red items and excluding the blue items from memory, then the CDA amplitude should be equivalent to that observed when two red items were presented alone. By contrast, if an individual was perfectly inefficient at excluding the blue items, all four of the items in the array (two red and two blue) would be stored in memory, resulting in an amplitude equal to that when four red items alone were presented*” (Vogel et al., 2005, p. 500). High WM capacity individuals were found to selectively filter out the irrelevant items. Low WM capacity individuals were found to have CDA amplitudes in the presence of distractors which were more similar to that of the 4 item array, indicating they tended to inefficiently allocate attentional resources to irrelevant information.

The main aim of the current study was to examine the nature of impaired inhibition in depression, in relation to WM capacity using the visual working memory task used by Vogel et al. (2005). We predicted that, relative to high capacity non-dysphoric individuals, dysphoric individuals will have reduced working memory capacity for relevant information, and will have similar CDA amplitudes in the distractor condition and the 4 item condition compared to the 2 item condition, reflecting inefficient filtering of irrelevant information.

Methods

Participants

The study was advertised online through the Birkbeck College and University College of London automated experiment management systems. Participants were not allowed to take part in the study if they suffered from migraine. 51 right handed participants (27 male and 24 female) were selected for the study based on their initial scores on the Beck Depression Inventory, BDI-II (Beck, Steer & Brown, 1996). The inventory consists of 21 items assessing the severity of symptoms of depression. Each item has a four point scale ranging from 0 to 3. The cut-off for presence of mild depressive symptoms is a score of 14 on the BDI (Beck et al., 1996). However, for the current study participants were allocated to the dysphoric group only if their score was greater than or equal to 20 as attention deficits have been shown to appear at a moderate level for the BDI II within non-clinical university based samples (Rokke et al., 2002). Participants were allocated to the non-dysphoric group if their score was 5 or below. Accordingly, the dysphoric group had a mean score of $M = 25.88$ ($SD = 7.04$) and the non-dysphoric group a mean score of $M = 2.18$ ($SD = 1.75$). All participants were tested within two weeks of their first assessment. At testing, each participant provided demographic information by self report (see Table 1) and was reassessed on the BDI-II. In the dysphoric group ($N = 17$) all scored above the cut-off for the presence of mild depressive symptoms ($M = 24.94$, $SD = 7.08$) and the non-dysphoric group ($N = 34$) scored below their cut-off ($M = 2.03$, $SD = 2.12$).

Insert Table 1

Stimuli and Procedure

Stimuli were presented on a 17inch LCD with a refresh rate of 16.6ms. The experimental task was programmed and run using DMDX programming software (Forster & Forster, 2003) on a Dell Optiplex GX520. Stimulus design and procedure were adapted from those of Vogel et al. (2005). In the task participants were presented with trials consisting of two stimulus arrays, a memory array, and a test array. Participants were instructed to remember target items (red rectangles) from the memory array across a short retention period; accuracy for the target items was then accessed in the test array. The two stimulus arrays were each presented within $4^\circ \times 7.2^\circ$ rectangular regions that were centred 3° from a white central fixation cross on a black background viewed at a distance of 60 cm. Within a trial each array (see Figure 1) was presented either on the left or right side of the cross and consisted of two or four rectangles ($0.64^\circ \times 1.21^\circ$). Participants were instructed which array to attend by a white arrow above the central fixation cross.

Insert Figure 1 here

The colour of each rectangle could be either red (target rectangle) or blue (distractor rectangle) depending on trial condition. Each rectangle was oriented randomly along one of four positions (vertical, horizontal, left 45° , right 45°). Rectangle positions were also random with the constraint that the distance between rectangles was at least 2° .

Each trial began with a central fixation (together with the white arrow) which remained on screen for 700ms. After presentation of the cross and arrow, on both sides of the fixation, arrays of either 2 red rectangles (2 item condition), 4 red rectangles (4 item condition) or 2 red rectangles and 2 blue rectangles (distractor condition) were presented for 100ms (memory array). All rectangles were then removed from the display for 900ms

(retention period). All rectangles were then redisplayed for 2000ms (test array). Participants were instructed to maintain fixation during each trial and remember the orientations of the red rectangles on the side indicated by the arrow. During the test array participants responded with one of two button presses to indicate whether the direction of one of the red rectangles changed or did not change. The inter-trial interval was varied randomly between 1500 and 2000ms. Array size (conditions: 2 item, 4 item & distractor condition), arrow direction (left & right), change and no change trials were randomized and presented equally often across the experiment. Participants completed a short practice phase consisting of 24 trials (8 per condition) before the experimental blocks. The experiment was split into 7 blocks of 84 trials (196 trials per condition), totalling 588 total trials across the experiment. Within each block participants were given a short break after half of the trials were completed. Experimental session's lasted approximately 120 minutes. After the experiment participants were debriefed and paid £15 for their time.

Data preparation

Working memory capacity

Each participant's WM capacity was estimated from their performance on the task using a standard formula (Cowan, 2001). The formula is $K = S(H - F)$, where K is WM capacity, S is the size of the array (i.e., 4 or 2), H is the hit rate or proportion of correct responses when a change is present, and F is the false alarm rate or the proportion of incorrect responses when no change is present. Memory capacity varies considerably within a population; as a result there are low and high WM capacity individuals. To account for variation in memory capacity, non-dysphoric participants were divided into high and low capacity groups using a median split on their K scores (cf. Vogel et al., 2005). The split led to

the loss of one participant (who had a median score) resulting in 16 high capacity non-dysphoric participants and 17 low capacity non-dysphoric participants.

ERP recording

Participants were seated in an electrically isolated, sound proof room with dimmed lighting. Before recording each participant was given the instruction to avoid large movements during the task, to focus on the cross in the centre of the screen in order to avoid saccades during trials, and to try to time their blinks after button responses to stimuli within the inter-trial interval. The EEG was recorded using 32 Ag/AgCl sintered ring electrodes mounted on a fitted cap (EASYCAP) according to the international 10/20 system. The horizontal electrooculogram (EOG) was recorded from two electrodes placed 1 cm to the left and right of the external canthi to measure horizontal eye movements. Vertical EOG was recorded from a single electrode placed below the left eye to measure eye blinks. Electrode impedance was below 5 k Ω . EEG data were recorded referenced to the left mastoid, and re-referenced offline to the mean of the left and right mastoids (linked mastoids). EEG recordings were filtered with bandpass at 0.01–80 Hz and sampled at 250 Hz.

EEG processing

EEG data were processed in two stages using the MATLAB extension EEGLAB (Delorme & Makeig, 2004) and the EEGLAB plugin ERPLAB (Lopez-Calderon & Luck, 2010). EEG data were processed using both artifact correction and rejection. First Independent Component Analysis (ICA) was conducted to identify ocular, muscle, and noise components (Jung et al., 2001). Artifactual ICA components were then identified and removed from the data using standard methods (Jung, Makeig, Humphries et al., 2000; Jung, Makeig, Westerfield et al., 2000; Onton, Westerfield, Townsend, & Makeig, 2006).

Specifically, ICA was first applied to continuous EEG data to create time courses of temporally independent signals spatially filtered from the EEG data of each channel. Stereotypical artifactual wave shapes (e.g. blink activity: brief, large, deflections at frontal electrode sites and deflections of opposite polarity at the vertical EOG) were matched with that of simultaneous ICA time courses. Potential artifactual ICA components were then verified by plotting their scalp topography and removed if maps provided further evidence that the component was predominately artifactual activity (e.g., blink activity projects most strongly to far frontal sites). Artifact detection and rejection was then conducted on epoched uncorrected data files to identify and remove trials containing blinks and large eye movements at the time of stimulus presentation. Trials with ocular artifacts at stimulus presentation were removed from both behavioural and ICA corrected continuous data. The percentage of trials remaining after artifact rejection for each group was: 86% for the Dysphorics, 85% for the NDLC group and 86% for the NDHC group. Across each group ERPs were based on an average of $M = 169.23$ ($SD = 4.32$) trials for the two item condition, $M = 168.63$ ($SD = 5.86$) trials for the four item condition, and $M = 171.28$ ($SD = 8.33$) trials for the distractor condition. The groups did not differ on the number of artifact free trials per condition. Participants with rejection rates over 25% were removed from the analysis which resulted in the removal of one participant from the low capacity non-dysphoric group leaving 16 participants in this group.

Contralateral Delay Activity (CDA)

CDA is computed as the difference in mean amplitude between activity in hemispheres contralateral and ipsilateral to the memory array during the retention period. Activity from posterior electrode sites (P3/4, P7/8, PO3/4, PO7/8, O1/2) within the time period of 300–900 ms after onset of the memory array was used in the calculation of CDA

(see figure 2 for display of contralateral and ipsilateral activity for each group by electrode sites). Contralateral waveforms were calculated by averaging activity recorded at right hemisphere electrode sites when participants were cued to remember items on the left side of the central fixation with activity recorded from the left hemisphere electrode sites when participants were cued to remember items on the right side of the central fixation.

Conversely, ipsilateral waveforms were calculated by averaging the activity recorded at right hemisphere electrode sites when participants were cued to remember items on the right side of the central fixation with activity recorded from the left hemisphere electrode sites when participants were cued to remember items on the left side of the central fixation, see figure 3 for contralateral and ipsilateral activity collapsed across posterior electrode sites.

Insert Figure 2 here

Insert Figure 3 here

ERP analysis – filtering efficiency

CDA waveforms provide within group representations of the number of items held in working memory. The sensitivity of CDA makes it possible to use this measure to accurately determine the efficiency of inhibitory processes during the task. Analysis of CDA used the method of Vogel et al. (2005). This method uses a formula to determine each participant's ability to efficiently filter irrelevant information. The formula provides a quantitative measure of whether CDA amplitudes in the distractor condition are more similar to that in the four items or the two items condition. Scores range from 1 (efficient: identical to 2 item) to 0

(inefficient: identical to 4 item)¹. The formula is, $FE = (F-D)/(F-T)$, where FE is filtering efficiency, F is the amplitude for 4 items, D is the amplitude in the distractor present condition and T is the amplitude in the 2 items condition.

Results

Previous research has shown that WM capacity and filtering efficiency scores are strongly correlated (Vogel et al., 2005). Figure 3 below shows the correlation between each participant's filtering efficiency and working memory capacity in the present study. In line with previous research we found that these measures were strongly correlated across all participants, ($r = .63, n = 49, p < .001$). This finding shows that low capacity individuals, in the current sample, have low filtering efficiency scores and high capacity individuals have high filtering efficiency scores².

 Insert Figure 4 here

To determine if dysphoria was associated with reduced working capacity (WM capacity) and impaired filtering efficiency (FE), performance of the dysphoric group was compared to that of each of the non-dysphoric sub-groups³. A multivariate ANOVA was conducted with Group (dysphoric, NDHC, and NDLC) as between subject factor and WM Capacity (K scores) and filtering efficiency (FE) as dependent variables.

Working memory capacity

For working memory there was a main effect of Group, $F(2,46) = 24.941, p < .001$ ⁴. The NDHC group showed higher K scores ($M = 2.62, SD = 0.24$), than the dysphoric, ($M = 1.53, SD = 0.63$), and the NDLC group ($M = 1.65, SD = 0.45$). Using a Bonferroni adjustment

these differences were found to be significant in post hoc comparisons between the NDHC and NDLC groups, $p < .001$, and for the NDHC group and dysphoric individuals, $p < .001$. There were no group differences between the dysphoric and the NDLC groups, $p = 1$.

CDA analysis

Figure 5 shows grand mean CDA waveforms as a function of condition for the dysphoric group and non-dysphoric sub-groups averaged across posterior electrode sites. For all groups it appears that within the 300-900ms time window CDA amplitudes were highest for the 4 item array followed by the distractor condition and 2 item conditions. A mixed ANOVA with Group (dysphoric, NDLC, NDHC) as the between subject factor and Condition (2 item, distractor, 4 item) as the within subject factor yielded a main effect of Condition, Wilks Lambda = .30, $F(2,45) = 51.11$, $p < .001$ showing that CDA amplitudes were significantly different between all conditions ($ps < .001$, Bonferroni corrected) (see Table 2 for descriptive statistics). A significant difference between the 4 item and 2 item conditions shows CDA amplitudes are sensitive to the number of representations held in memory (McCollough et. al, 2007). CDA amplitudes were also significantly different for these two conditions in comparison to the distractor condition, indicating all participants did not completely filter the distractors and stored at least some irrelevant information in visual working memory.

Analysis also revealed a significant Group X Condition interaction, Wilks Lambda = .73, $F(4,90) = 3.76$, $p < .008$. This interaction showed that for dysphorics and the NDLC group the differences in CDA amplitudes between the distractor and 4 item conditions was lower than that of the NDHC group (Mean differences of .11, .16, and .31, respectively, $F(2,46) = 6.02$, $p < .006$). This suggested that the NDHC group held fewer irrelevant items in working memory relative to the NDLC and the D groups. In contrast the differences in CDA amplitudes between the 4 item and the 2 item condition were not different across groups,

$F < 1$. To investigate the relationship between CDA amplitudes reflecting the ability to filter irrelevant items from working memory and the number of items held in working memory we conducted the filtering efficiency analyses below.

 Insert Figure 5 here

 Insert Table 2 here

Filtering efficiency (FE)

FE was calculated using the formula: $FE = (F-D)/(F-T)$, where F is the amplitude for 4 items, D is the amplitude in the distractor present condition and T is the amplitude in the 2 items condition. Scores range from 1 (efficient: identical to 2 item) to 0 (inefficient: identical to 4 item). FE scores ranged from .05 to .78. The dysphorics had a mean FE score of .27 ($SD = .18$). The NDLC group had a mean FE score of .29 ($SD = .12$), and for the NDHC group the mean FE score was .51 ($SD = .18$). Analysis revealed a main effect of Group, $F(2,46) = 9.674$, $p < .001^5$. The NDHC group were significantly more efficient at filtering irrelevant information from storage in visual working memory than dysphoric and NDLC individuals. Using Bonferroni adjustments these differences were found to be significant in post hoc comparisons between NDHC and NDLC groups, $p = .002$, and NDHC and dysphoric groups, $p = .001$. There were no group differences between dysphoric and NDLC groups, $p = 1$.

Additional analysis

We conducted additional analyses to rule out the possibility that the poor levels of performance in the NDLC and dysphoric groups was simply due to an inability to voluntarily allocate visual attention to the task (the relevant side of the memory array as indicated by the cue). To assess voluntary attention to the task, the difference in mean amplitude was measured between contralateral activity and ipsilateral activity from 75 – 175ms after onset of the memory array at posterior electrode sites (P3/4, P7/8, PO3/4, PO7/8, O1/2) (cf. Fukuda & Vogel, 2009). This time range encompasses early visual sensory responses (P1/N1) reflecting spatial attention to the task. Mean amplitudes were compared between groups (NDHC, NDLC, dysphoric) and within condition (2 item, 4 item, distractor) using a mixed ANOVA. We found that P1/N1 amplitudes did not differ by group, $F(2, 46) < 1$ ruling out the possibility that dysphorics and the NDLC differed from the NDHC in their ability to voluntarily orient spatial attention to the task.

Discussion

In the present study we examined the nature of impaired inhibition of irrelevant information in depression, in relation to working memory capacity. For this purpose we administered a well-investigated task (Vogel, Woodman & Luck, 2001; Vogel et al., 2005) which provides a specific neural marker of filtering of irrelevant information and working memory capacity. The results of the study are that (1) filtering efficiency and working memory capacity are positively related; and (2) inhibitory functioning in dysphoric individuals is significantly lower than high capacity non-dysphoric individuals and similar to functioning of non-dysphoric individuals low in WM capacity. These findings are discussed below.

We calculated working memory and filtering efficiency according to methods developed by Vogel and colleagues. The present findings replicated the results of Vogel et

al. (2005); specifically, across all participants, filtering efficiency and working memory capacity were positively correlated with high capacity individuals showing high filtering efficiency and low capacity individuals showing low filtering efficiency scores. Thus, the current study provided further evidence that misallocation of attentional resources to irrelevant information may drive individual differences in overall working memory capacity (Vogel et al., 2005). In this regard, the results of the present study are also in line with theoretical proposals and recent neural evidence which have linked inhibition and working memory (Hasher, Zacks & May, 1999; McNab & Klingberg, 2008).

Based on previous work we hypothesized that due to impaired inhibition (Joormann et al., 2007), dysphoric individuals would have lower WM capacity relative to high capacity non-dysphoric individuals. Our hypothesis was supported by the results: dysphoric individuals were characterized by reduced filtering of irrelevant information and working memory capacity relative to high capacity non-dysphoric individuals. No differences were observed for WM capacity or FE when the dysphoric group were compared to low capacity non-dysphoric group. These findings emerged at the neural and behavioural level respectively. Importantly, it was found that the performance of the dysphoric group was not associated with poor voluntary attention to task and was related specifically to inefficient filtering of irrelevant information so these results provide an important validation for the use of this methodology in cognitive research in depression.

Previous behavioural paradigms so far have provided mixed results for the existence of inhibitory impairments in depression. Specifically, our findings showed that it is important to separate the (control) non-dysphoric group by their working memory capacity scores as it was through this division that the similarity between non-dysphoric (low working memory capacity) and dysphoric individuals could be observed. In the absence of such a division the differences between dysphoric and non-dysphoric groups may be masked by their differences

in working memory capacity. Our findings further highlight the benefit of combining behavioural and electrophysiological measures in examining the inhibitory processes linked with depression. The current approach also highlights that with a more specific measurement, it can be observed that dysphoric individuals are less efficient in filtering irrelevant information.

The results of the present study imply that impaired inhibition is a central feature of dysphoria, and are thus important for cognitive theory and research in depression. It has been proposed that impaired inhibition may act as a cognitive vulnerability factor for depression as it could explain some of the typical cognitive symptoms of depression (e.g., lack of concentration and memory deficits) as well as being associated with a reduced capacity to engage in emotion regulation (cf. Joormann et al., 2007). The further elucidation of the nature of inhibitory deficits in depression is also of importance from a translation research perspective. That is, a more precise understanding of the cognitive impairments in depression will allow examination of its direct role in the pathophysiology of depression and can illuminate potential ways to remediate such problems (De Raedt et al, 2010). There are several interventions, such as repetitive transcranial magnetic stimulation (Leyman, De Raedt, Vanderhasselt, & Baeken, in press) or cognitive training regimes (MacLeod, Koster, & Fox, 2009; Siegle, Ghinassi, & Thase, 2007) that could strengthen inhibitory control and may alleviate depressive symptoms.

There are some restrictions to the present study. The use of a dysphoric sample hampers generalisation to clinically depressed populations. However, if anything, our findings provide an overestimation of inhibitory functioning in depression as a largely high-functioning student population was tested who had slightly lower depression scores than observed in clinical samples. Also, as a result of the use of neutral stimuli, the current study cannot inform whether inhibition is impaired and working memory capacity is reduced in the

context of emotional material. There is growing evidence of inhibitory impairment for emotional material in depression (Joormann et al., 2007; Derakshan, Salt, & Koster, 2009). However, it is interesting that in the current study basic impairments were observed. As these impairments may be even more pronounced for emotional material, future research will need to be conducted to extend findings of the current study to emotional processing in depression. Finally, the current study did not consider the influence of anxiety and given the comorbidity between anxiety and depression, it is important for future research to examine the independent and interactive effects of anxiety and depression in relation to filtering efficiency. Still, it should be noted that impaired inhibitory functions have been more strongly associated with depression (Joormann et al., 2007) than anxiety.

In conclusion, the present study provides clear evidence of fundamental attentional control impairments in depression. Results indicate that dysphoric individuals have trouble filtering distracting irrelevant information from the focus of attention while engaged in goal directed behaviour. This disruption of information processing may have severe cognitive and emotional consequences. The results of the present study collectively show utilizing direct neural and behavioural measures offers a promising way forward for exploring attentional control impairment in depression.

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Footnotes

1. Calculation of filtering efficiency (FE) can produce outliers if mean CDA amplitudes for the 2 item condition are, for example, greater than the 4 item condition (i.e. negative FE). However, all participants in the current study had FE scores within the range of 0 to 1 so were included in the analysis.
2. A significant positive correlation was found for both the full non-dysphoric group $r = .44$, $n = 32$, $p = .011$, and the dysphoric group $r = .83$, $n = 17$, $p < .001$.
3. It must be noted that firstly a two-group analysis comparing the dysphoric and the full non-dysphoric group was conducted to examine if the two groups differed overall in terms of Overall K and FE. The analysis showed that the dysphoric group had significantly lower levels of K and FE than the non-dysphoric group.
4. The full non-dysphoric group had significantly higher WM capacity ($M = 2.13$, $SD = .60$) than the dysphoric group ($M = 1.53$, $SD = .63$), $F(1,47) = 10.279$, $p = .002$.
- 5 The full non-dysphoric group had significantly greater FE ($M = .40$, $SD = .19$) than the dysphoric group ($M = .27$, $SD = .18$), $F(1,47) = 4.98$, $p < .05$.

Table captions

Table 1. Demographic information for dysphoric and non-dysphoric groups.

Table 2. Mean CDA amplitudes (standard deviations in brackets) for dysphorics, non-dysphoric low capacity (NDLC), non-dysphoric high capacity (NDHC), and Condition; 2 item, 4 item, and distractor.

Figure captions

Figure 1. Example of a distractor condition in a change trial. Participants are instructed to remember the orientations of the red rectangles (light grey), and respond during the test array with one of two buttons to indicate whether a change was present or not.

Figure 2: a,b,c. Contralateral and ipsilateral activity time locked to the memory array by condition (2 item, 4 item, distractor) at posterior electrode sites: P3/P4, P7/P8, PO7/PO8, O1/O2 for dysphoric(2a), non-dysphoric high capacity (2b) and non-dysphoric low capacity (2c) groups.

Figure 3. Grand averaged waveforms for activity contralateral and ipsilateral time locked to the memory array for dysphoric, non-dysphoric high capacity (NDHC) and non-dysphoric low capacity (NDLC) groups across posterior electrode sites.

Figure 4. Correlation between memory capacity and the efficiency of excluding distractors from storage in visual working memory for dysphoric, non-dysphoric high capacity (NDHC) and non-dysphoric low capacity (NDLC) groups.

Figure 5. Grand averaged CDA waveforms (contralateral – ipsilateral activity) for Dysphoric, non-dysphoric high capacity (NDHC) and non-dysphoric low capacity (NDLC). Each graph shows waveforms by trial condition; 2 item (CDA_2 item condition), distractor (CDA_distractor condition) and 4 item (CDA_4 item condition). Highlighted region shows analysis window (300ms-900ms).

Table 1

Demographic Information Across Groups

Group	Time 1 Mean BDI	Time 2 Mean BDI	Gender		Mean Age
			Female	Male	
Dysphoric	25.88 (7.04)	24.94 (7.08)	12	5	25.06 (10.13)
Non-dysphoric	2.18 (1.75)	2.03 (2.12)	12	22	29.00 (10.09)
NDHC	2.59 (1.82)	2.17 (2.05)	5	12	29.47 (12.70)
NDLC	1.76 (1.64)	1.88 (2.20)	7	10	28.53 (6.92)

Table 2

Mean CDA Amplitudes for Group and Condition

	Dysphoric	NDLC	NDHC	Condition Mean
2 Item	-0.48 (.42)	-.74 (.54)	-.92 (.49)	-.71 (.59)
4 Item	-.93 (.49)	-1.26 (.76)	-1.55 (.67)	-1.24 (.70)
Distractor	-.82 (.48)	-1.09 (.66)	-1.24 (.58)	-1.04 (.51)

Figure 1

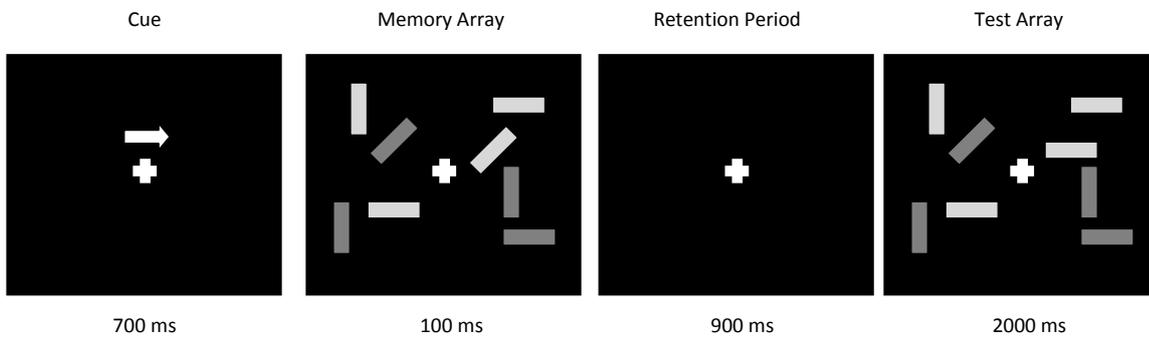


Figure 2a

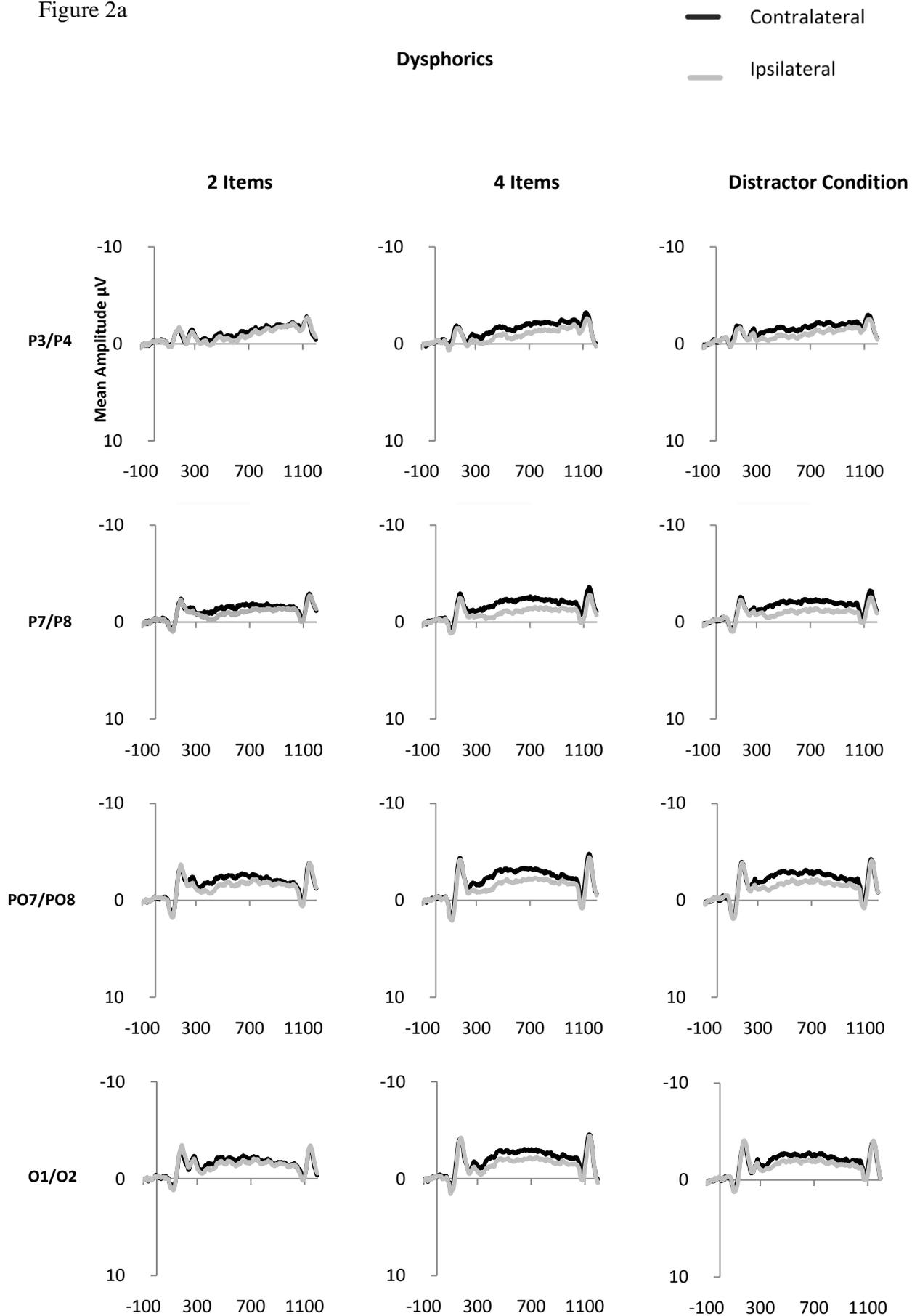


Figure 2c

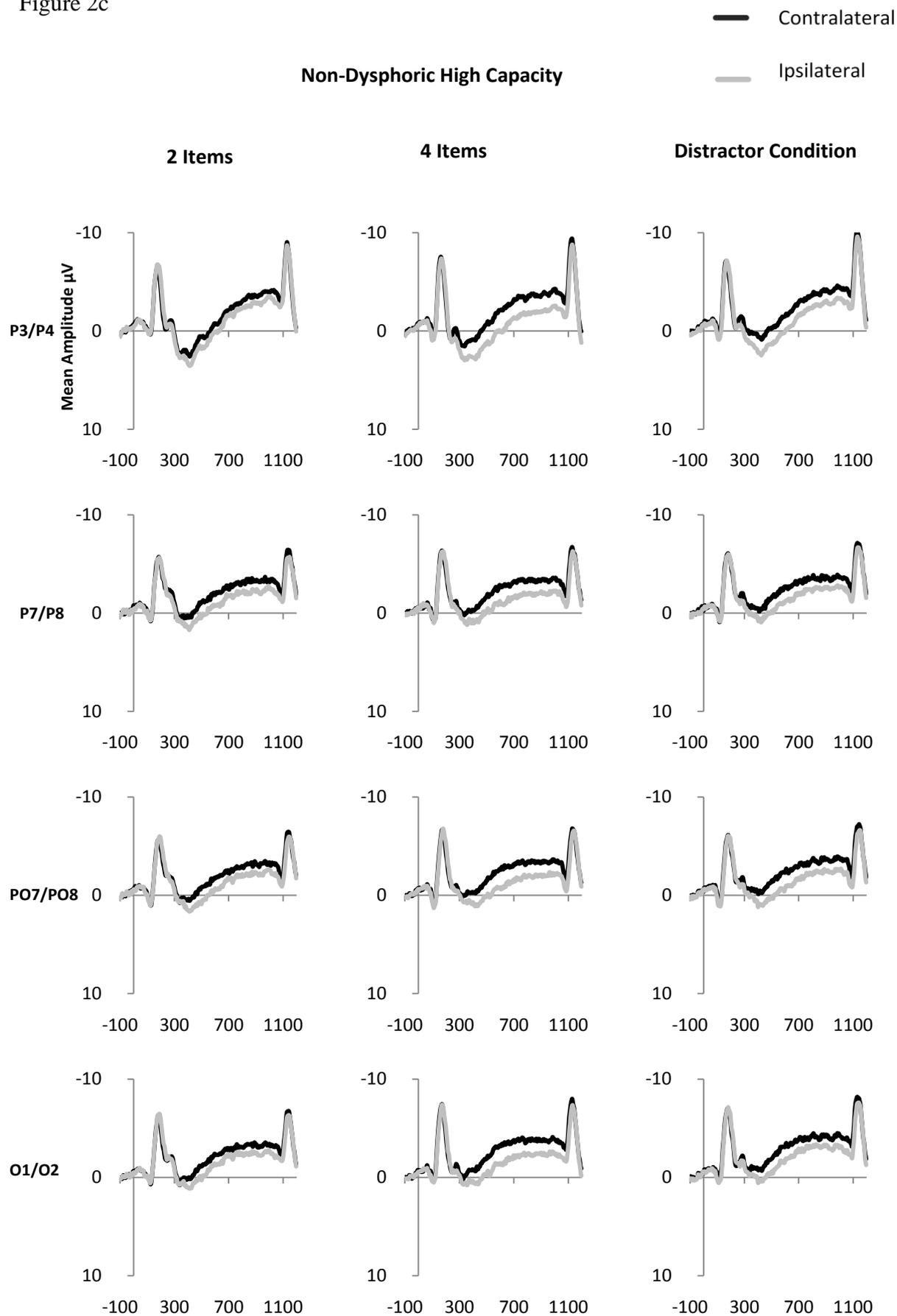


Figure 3

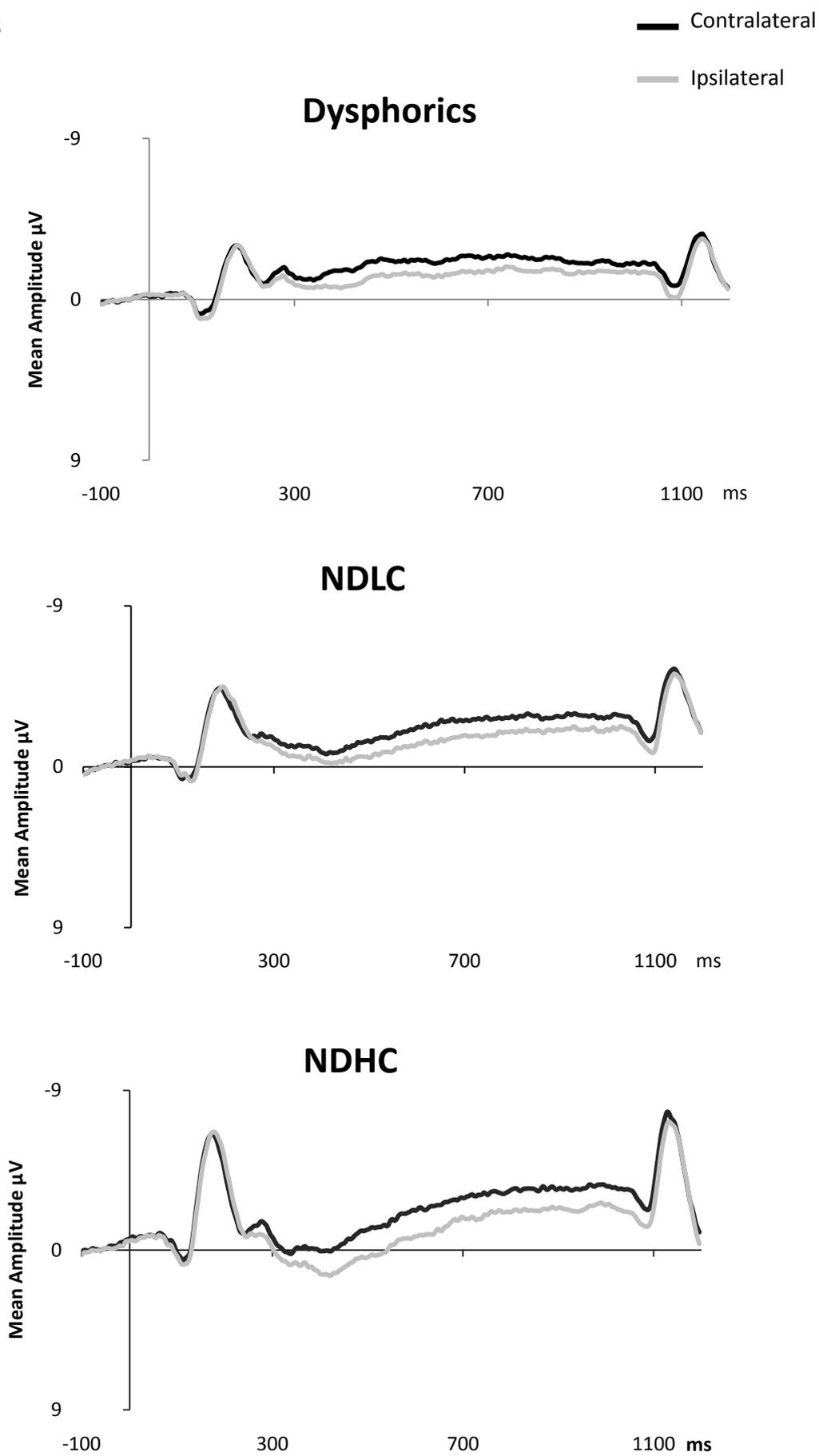


Figure 4

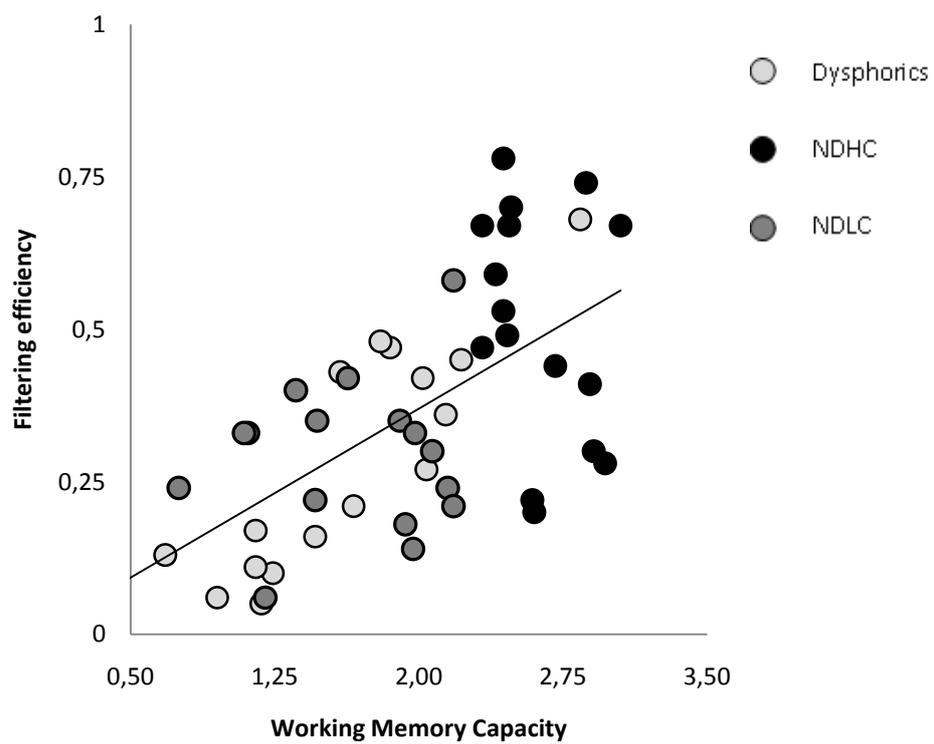


Figure 5

