RESEARCH REPORT

Individual Differences in Working Memory Capacity Predict Action Monitoring and the Error-Related Negativity

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Neuroscience suggests that the anterior cingulate cortex (ACC) is responsible for conflict monitoring and the detection of errors in cognitive tasks, thereby contributing to the implementation of attentional control. Though individual differences in frontally mediated goal maintenance have clearly been shown to influence outward behavior in interference-rich contexts, it is unclear whether corresponding differences exist in neural responses that arise out of the ACC. To investigate this possibility, we conducted an electrophysiological study using a variant of the Simon Task, recording event-related potentials (ERPs) in healthy normal individuals with varying working memory capacity (high vs. low spans; a behavioral proxy for variability in goal maintenance). Primary analyses focused on the magnitude of the error-related negativity (ERN), a response-locked ERP component associated with the commission of errors thought to arise because of action monitoring in the ACC. Our results revealed that frontally mediated working memory capacity may alter error monitoring by the ACC, with high spans showing a greater ERN than low spans. These individual differences were also observed in the posterior positivity, a response-locked ERP component associated with updating cognitive strategies, suggesting greater awareness of errors with increased working memory capacity. These results are interpreted within 2-process models of attentional control, suggesting individuals with greater working memory capacity may better maintain task goals by more strongly biasing neural activity in frontal–executive networks.

Keywords: error-related negativity, working memory capacity, individual differences, attentional control, anterior cingulate

Two-process models of attentional control suggest that prefrontal cortex (PFC) is responsible for the maintenance of task goals, whereas anterior cingulate cortex (ACC) is responsible for detecting errors that conflict with those goals (see Braver, Gray, & Burgess, 2007; Cohen, Botvinick, & Carter, 2000; Watson, Lambert, Miller, & Strayer, 2011). Consistent with these models, neuroimaging of cognition provides evidence for PFC activity that may be involved in goal maintenance. For instance, in the Stroop Task, incongruent trials elicit neural activity in PFC, and this activity may reflect maintenance of task goals (e.g., respond to ink colors while ignoring words; see Milham et al., 2001; Peterson et al., 2002). However, given that no task is process pure, lab standards like Stroop or a spatial variant like the Simon Task (Simon, 1969) often recruit other brain regions too. Notably, there is a prominent role for the ACC (Milham et al., 2001; Peterson et al., 2002) in selecting motor responses and/or in error monitoring. Indeed, current theories of human learning highlight the importance of the ACC in monitoring performance, providing negative feedback to errors, and strategically adjusting off-task behavior (see Simons, 2010). More generally, the ACC is thought to monitor the environment for possible sources of interference or conflict in information processing, including the commission of actual errors or other situations where response competition occurs, attempting to prevent such conflict going forward (Botvinick, Cohen, & Carter, 2004; Yeung, Botvinick, & Cohen, 2004). For example, ACC involvement increases when correct responses are accompanied by high conflict, underscoring a fundamental role for the ACC in conflict monitoring. Hence, the dual recruitment of PFC and ACC in neuroimaging studies of controlled cognition is consistent with two-process models wherein goal maintenance (supported primarily by PFC) and conflict monitoring (supported primarily by ACC) are separate but related mechanisms thought to underlie the successful implementation of attentional control.

The delineating of brain regions provides a more complete picture of the distinct role of major neural correlates involved in attentional control, but it leaves an important empirical question unresolved: How do the PFC and ACC interact to coordinate the dual mechanisms of goal maintenance and conflict monitoring, respectively? One possibility is that PFC actively maintains task goals, biasing corresponding activity in ACC in a top-down fashion (Miller & Cohen, 2001). Another complementary possibility is that conflict monitoring in ACC may influence corresponding activity in PFC, signaling the need for greater attentional control and the dynamic updating of task goals as needed, particularly if one is off task and has committed an error (Cohen, Botvinick, & Carter, 2000; Kerns et al., 2004). Indeed, both

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possibilities may be true, with dissociable roles for the dorsolateral PFC and ACC in implementing task goals and monitoring performance, respectively (see MacDonald, Cohen, Stenger, & Carter, 2000, for evidence from event-related neuroimaging of Stroop performance that clearly supports this particular theoretical alignment of these dual cognitive processes with their distinct neural bases in the PFC–ACC attentional control network). Additional insight into answering this PFC–ACC interaction question may come from considering research examining individual differences in working memory capacity (WMC; Engle, 2002), which is thought to reflect the ability to maintain task goals in interference-rich contexts. In an influential review, Kane and Engle (2002) synthesized a wealth of evidence from single-cell, brain imaging, and neuropsychological research to argue that PFC, as well as networked posterior and subcortical brain regions, is necessary for effective WMC.\footnote{Although the source of individual differences in WMC is still hotly debated, our conceptualization of the present study regarding PFC–ACC interaction was primarily motivated by the resistance-to-interference hypothesis of Randy Engle, Mike Kane, and colleagues (see Engle, 2002; Kane & Engle, 2002). However, we refer the interested reader to alternative theoretical frameworks that can be used to explain the behavioral effects associated with individual differences in WMC. These diverse but complementary frameworks include the attentional control hypothesis of Nelson Cowan (Cowan et al., 2005) or the retrieval hypothesis of Nash Unsworth (Unsworth & Engle, 2007), among others.}

Consistent with this argument, individual differences in WMC predict susceptibility to interference on traditional cognitive psychology lab tasks thought to require goal maintenance and top-down attentional control, including Stroop, antisaccade, and dichotic listening (Engle, 2002). For example, Kane and Engle (2003) found individuals with lower WMC were more likely to lose cognitive set and produced more naming errors (i.e., word reading) in a high-congruency Stroop Task. Finally, and perhaps most important for the purposes of the present study given our method, recent neuroimaging evidence with the Operation Span Task (OSPAN) revealed that individuals with greater WMC have elevated activity in the PFC–ACC attentional control network (see Faraco et al., 2011).

Though individual differences in WMC have been shown to influence outward behavioral responses in situations requiring attentional control and resistance to interference, it is less clear whether corresponding differences exist in underlying neural responses that arise out of the ACC, such as the error-related negativity (ERN). The ERN is a response-locked electrophysiological signature—an event-related potential (ERP)—associated with the commission of errors and thought to arise because of conflict or action monitoring in ACC (Gehring, Goss, Coles, Meyer, & Donchin, 1993; Holroyd & Coles, 2002). When recording neural activity from patients with lateral PFC damage, Gehring and Knight (2000) provided clear evidence of altered action monitoring and, most important for our purposes, the management of a source of interference tracked by ACC. That is, the ERN for the patients was equal for correct and error trials, an atypical pattern suggesting possible loss of task goals in PFC that could have appropriately influenced information processing and corresponding activity levels in ACC. In contrast, errors for age-matched controls generated greater ERN activity than correct trials, the typical pattern in this literature. Although these preliminary ERP results with neuropsychological patients support two-process models of attentional control where ERN activity and conflict monitoring in ACC can be modulated, top-down, through task goals maintained by PFC (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Cohen et al., 2000; Kane & Engle, 2002; Miller & Cohen, 2001), it is difficult to rule out the possibility that PFC–ACC interaction may be an emergent property restricted to those with brain damage.

Present Study

Therefore, rather than relying on neuropsychological patients to empirically investigate possible top-down bias in the attentional control network, the present study was the first to use an individual
differences approach, borrowed from the cognitive psychology tradition on WMC described earlier. Specifically, we recorded ERPs in select samples of healthy young adults who varied in WMC and who received a Simon Task in order to recruit both PFC and ACC (Peterson et al., 2002). We made two straightforward predictions. First, if Kane and Engle’s (2002) core supposition on attentional control is accurate and there is a necessary relationship between underlying PFC function and effective WMC, then individual differences in WMC should influence neural activity in ACC and, thus, the magnitude of the ERN. According to this prediction, individuals with greater WMC are better at maintaining task goals in interference-rich contexts; consequently, they may use enhanced PFC function to bias error processing and conflict signatures in ACC, showing greater ERN responses too (see Faraco et al., 2011, for additional neuroimaging evidence in support of this prediction). Furthermore, although we have emphasized PFC–ACC interactions thus far, particularly with respect to the magnitude of the ERN, individual differences in WMC may influence other posterior ERP signatures too. For example, individual differences in WMC may alter the magnitude of the posterior positivity (Pe), a response-locked ERP component following the ERN that is associated with awareness of errors and updating of cognitive strategies (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). Hence, a second prediction was that those with greater WMC would show a greater Pe response, reflecting a differential ability to consciously refresh task goals following the loss of cognitive set, a possibility revisited in the Discussion section.

Method

Participants

ERPs were recorded from 24 undergraduates (age range = 18–37 years old; \(M = 23.1\) years) at the University of Utah, who received course credit in exchange for their participation. All participants were right-handed with normal or corrected-to-normal vision and had no self-reported history of neurological or psychiatric disorders (e.g., epilepsy, depression) or serious brain trauma.

Materials and Procedures

The experiment was conducted in two sessions. In Session 1, to assess individual differences in WMC, participants (\(N = 371\); age range = 18–37; \(M = 21.4\)) were given a computerized OSPAN Task (Unsworth, Heitz, Schrock, & Engle, 2005). In this task, participants simultaneously memorized letters while solving math problems. These dual-task memory/math instructions challenged participants’
goal maintenance, revealing individual differences in OSPAN and, presumably, variability in frontally mediated WMC or attentional control, reflecting a differential ability to manage interference. In Session 2, a subset of participants, sampled from the top (i.e., high WMC, $N = 12$, average absolute span score = 58.8) and bottom (i.e., low WMC, $N = 12$, average absolute span score = 16.4) of the distribution of OSPAN scores from Session 1, returned for the follow-up ERP study (see Conway et al., 2005, for further discussion of the utility of extreme-group designs in attentional control research). Individuals with high WMC ($M = 23.2$ years old; three male, nine female) and low WMC ($M = 22.9$ years old; four male, eight female) were comparable in age and gender, respectively.

Session 2 took place one day to several months after Session 1. In Session 2, participants had electrodes applied and were tested in a variant of the Simon Task. In this task, participants were instructed to respond as quickly and accurately as possible, pushing one of two buttons corresponding to the direction of arrows presented visually on a computer screen, ignoring the spatial location of the arrows (cf. Castel, Balota, Hutchison, Logan, & Yap, 2007). Arrows were presented in one of three conditions: neutral, congruent, and incongruent. Each arrow was preceded by a fixation cross presented for 400 ms in the center of the display followed by a blank screen for 100 ms. The arrow was then presented until the participant responded or 1,500 ms had elapsed. No feedback was provided. In the neutral condition, arrows were presented centrally. In the congruent condition, arrows appeared on the same side of the space as the direction they were facing (e.g., a left-facing arrow on the left side of space, situated 5 degrees of visual angle in the horizontal plane from the central fixation point that appeared at the onset of every trial). In the incongruent condition, arrows were presented on the opposite side of the space as the direction they were facing (e.g., a left-facing arrow on the right side of space, situated 5 degrees of visual angle in the horizontal plane from the fixation point). To maximize the likelihood that individual differences in WMC would influence Simon Task performance, we used a high proportion of congruent trials (75% congruent; 900 trials) and few incongruent or neutral trials (12.5%; 150 trials/condition). It is noteworthy that Kane and Engle (2003) used similar trial proportions to investigate the influence of individual differences in WMC on Stroop performance.

To enable collection of ERP data while participants performed the Simon Task, MED 10-mm diameter Ag/AgCl biopotential electrodes were used for all recordings, and impedance did not exceed 10 kohm. Electroencephalographic (EEG) and electrooculographic (EOG) signals were amplified by a Grass Model 12 Neurodata Acquisition System. All signals were sampled every 2 ms for 1,500 ms, beginning 100 ms prior to stimulus onset. EEG activity was recorded with a 0.1-Hz high-pass and 100-Hz low-pass analog filter at five electrode sites (i.e., Fz, Cz, Pz, C3, C4) based on the International 10–20 System (Jasper, 1958) and referenced to the software-averaged linked mastoids. ERP data were subsequently digitally filtered with a Boxcar filter with 4.2 Hz/3 dB and 13.5 Hz/0 dB cutoffs. EOG activity was simultaneously recorded at bipolar vertical and horizontal locations, and artifacts were corrected offline (Gratton, Coles, & Donchin, 1983). Returning to our earlier companion predictions, we expected that greater WMC would increase the magnitude of two distinct ERP signatures: the ERN and the Pe, both of which may be associated with greater attentional control.

Results

Reaction time (RT) and accuracy data were analyzed with a 2 (WMC: high vs. low) $\times$ 3 (condition: neutral, congruent, incongruent) repeated-measures analysis of variance (ANOVA) with Greenhouse–Geisser correction. Trials with RTs $< 50$ ms or $> 1,500$ ms were excluded from all analyses reported here, removing $< 1\%$ of data. Mean RTs and accuracy are presented in Tables 1 and 2, respectively, as a function of WMC and Simon Task condition. The RT analysis on correct trials revealed a main effect of condition, $F(1.2, 26.9) = 431.8$, $MSE = 389$, $p < .001$, $\eta^2_p = .95$. Planned comparisons using a Bonferroni least squares difference ($p < .05$) indicated a Simon effect: congruent trials (334 ms) had a faster mean RT than neutral trials (366 ms), which were faster than incongruent trials (460 ms). Congruent trials were also faster than incongruent trials. Neither the main effect of WMC nor its interaction with condition was significant (both $ps > .30$). Turning to the analysis of accuracy, once again there was a main effect of condition, $F(1.03, 22.7) = 72.3$, $MSE = 109$, $p < .001$, $\eta^2_p = .77$. Planned comparisons using a Bonferroni least squares difference ($p < .05$) indicated a Simon effect: congruent trials (100%) were more accurate than neutral trials (97%), which were more accurate than incongruent trials (76%). Congruent trials were also more accurate than incongruent trials. Neither the main effect of WMC nor its interaction with condition was significant (both $ps > .20$), though the accuracy data trended in the expected direction. Specifically, those with low WMC appeared more likely to lose cognitive set in the Simon Task, responding to spatial location instead of arrow direction on incongruent trials, diminishing their accuracy (see Table 2).

The response-locked ERP data presented in Figure 1 were analyzed with a 2 (trial accuracy: correct vs. error) $\times$ 2 (WMC: high vs. low) repeated-measures ANOVA. Trials with EEG or EOG artifacts were not included in the analyses reported here, removing $< 4\%$ of data. In Figure 1, the time at which a response was made is indicated by the vertical dotted line. Two distinct ERP components are evident in Figure 1: the ERN and the Pe. The ERN is depicted as a negative deflection, peaking approximately 150 ms after the response and was analyzed using a base-to-peak measure at electrode Cz with a peak window between 120 and 180 ms. A significant effect of accuracy revealed that the ERN was larger following errors than following correct trials, $F(1, 22) = 29.3$, $MSE = 711$, $p < .001$, $\eta^2_p = .57$. Most important, trial accuracy and WMC interacted, $F(1, 22) = 7.6$, $MSE = 711$, $p < .05$, $\eta^2_p = .26$, and planned follow-up comparisons with a Bonferroni least squares difference ($p < .05$) revealed the pattern we had predicted: a larger ERN on error trials for those with greater WMC. However, the ERN on correct trials did not differ with WMC.\(^2\)

\(^2\) Consistent with the extreme-group approach advocated by Conway et al. (2005), the range of absolute span scores for our 12 high-WMC (i.e., 50–68) and 12 low-WMC (i.e., 7–25) participants compared favorably to the upper (i.e., .51) and lower quartiles (i.e., .28) for absolute span as reported by Unsworth et al. (2005); see their Table 2).

Owing to the large number of trials in the present study, including the additional compatible filler trials used to achieve the 75% congruency proportion in our Simon task, the average number of errors, collapsed across condition, in the computation of ERN/Pe analyses was 37.4 per subject.
Using a similar repeated-measures ANOVA, the error-related positivity, Pe, was analyzed at electrode Pz, using a base-to-peak measure with a peak window between 300 and 500 ms, and is depicted in Figure 1 as the positive inflection following the commission of an error and the ERN. The analysis revealed an effect of trial accuracy, $F(1, 22) = 33.7, MSE = 3,518, p < .001, \eta^2 = .61$; an effect of WMC, $F(1, 22) = 5.3, MSE = 4,489, p < .05, \eta^2 = .19$; and a Trial Accuracy × WMC interaction, $F(1, 22) = 5.3, MSE = 3,518, p < .05, \eta^2 = .19$. As with the ERN, planned follow-up comparisons revealed the expected pattern: Pe did not differ with WMC on correct trials but was larger for those with high WMC on error trials.

Discussion

Utilizing an individual differences approach, our ERP results demonstrated that variability in WMC altered the magnitude of electrophysiological signatures that underlie attentional control. As shown in the left side of Figure 1, individual differences in WMC modulated the magnitude of the ERN, a brain potential generated by ACC that has been linked to action or conflict monitoring (Gehring et al., 1993, Gehring, Liu, Orr, & Carp, in press). Presumably, individuals with greater WMC were better able to maintain task goals and, therefore, had a robust ERN on error trials when they were off task, whereas those with lower capacity had a relatively muted ERN following an error. At a methodological level, our study moved beyond outward behavioral measures of individual differences in attentional control. Rather, we examined physiological patterns of brain activity in underlying frontal-executive neural networks that were unique to groups with varying levels of WMC. In doing so, our study provided novel electrophysiological evidence in support of two-process models of attentional control, where PFC maintains task goals in interference-rich contexts and ACC detects possible sources of conflict or interference. Specifically, differential neural resources in PFC, indexed by variability in WMC, appeared to bias the processing of conflict in the attentional control network including the ACC (Cohen et al., 2000; Kane & Engle, 2002). As shown in the right side of Figure 1,
individual differences in WMC also modulated the magnitude of the Pe, a brain potential thought to originate in posterior cingulate cortex (PCC; see Vocat, Pourtois, & Vuilleumier, 2008). The Pe is associated with error awareness or salience and the controlled adjustment of response strategies (Falkenstein et al., 2000). The ERN and Pe are thought to reflect independent aspects of posterror processing, with the former linked to error or conflict monitoring and the latter associated with conscious error recognition and remedial action (Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001).

Taken together, the combination of these ERN/Pe signatures suggests individuals with greater WMC have a more finely tuned attentional control network and, therefore, were more likely to monitor potential sources of interference in their actions and to consciously refresh task goals following the loss of cognitive set. Indeed, PFC is thought to play a critical role in controlled action-monitoring and refreshing of the task goal, respectively. We suggest this is clear evidence of networked PFC activity, where the top-down bias in information processing and the resolution of interference occurs to a greater extent in individuals with high WMC (see Faraco et al., 2011, for similar arguments). As shown in Figure 1, on trials where actions were consistent with task goals maintained in PFC, representing correct performance, there was no evidence of postresponse activity in either ACC or PCC for either high- or low-WMC participants (as reflected by the ERN and Pe, respectively). However, when outward behavior was inconsistent with task goals maintained in PFC, representing an error, there was neural activity in ACC and in PCC associated with conflict monitoring and refreshing of the task goal, respectively. We suggest this clear evidence of networked PFC activity, where the top-down bias in information processing and the resolution of interference occurs to a greater extent in individuals with high WMC.

In this way, our ERP findings support Kane and Engle’s (2002) core supposition regarding attentional control and the necessary relation between underlying PFC function and effective WMC.

Caveats and Future Directions

In contrast to the WMC differences we reported for both ERP signatures, we did not observe significant group differences in either accuracy or RT. At the very least, one might have expected the enhanced conflict monitoring in ACC and/or the error management in PCC to promote better avoidance of errors for those with greater WMC. What might explain this discrepancy between more traditional behavioral measures and the electrophysiological data? With respect to accuracy, the most likely explanation is that reduced sensitivity due to ceiling effects prevented us from detecting the differential implementation of attentional control (where experiment-wide accuracy, collapsed across all 1,200 trials, was >96%). Indeed, as shown in Table 2, akin to the significant group differences in Stroop accuracy on incongruent trials reported by Kane and Engle (2003), in our Simon Task, individuals with reduced WMC trended toward greater susceptibility to errors in the incongruent condition. In this light, the superior conflict monitoring and enhanced PFC–ACC interaction of individuals with greater WMC may have also had a self-limiting influence on outward behavior, reducing these individuals’ likelihood of an error. Turning to RTs, one might have expected a reduced Simon effect with greater WMC, reflecting better management of interference on incongruent trials where an arrow’s orientation and spatial location are incompatible. However, once again, using a high-congruency version of the Stroop Task, Kane and Engle found individuals with greater WMC were no faster on incongruent trials than other conditions. Although Kane and Engle’s behavioral work with the Stroop Task inspired our use of a high-congruency Simon Task in the present study, the relative magnitude of online ERP signatures on error versus correct trials was more sensitive to individual differences in attentional control than more traditional measures of accuracy or RT.

In the future, researchers may wish to conduct follow-up studies where task instructions explicitly encourage participants to stress speed over accuracy or vice versa, rather than stressing both dependent measures equally. By emphasizing accuracy at the expense of speed, one may be able to simultaneously decrease errors and to increase the magnitude of the ERN in attentional control paradigms, bringing outward behavioral measures and underlying ERP signatures into better accord (e.g., see Gehring et al., 1993). Regardless, it is theoretically important for two-process models of attentional control that we observed group differences in performance monitoring in the absence of either explicit experimenter feedback on errors or speed–accuracy trade-off instructions. Specifically, our ERP results in Figure 1 suggest that those with greater WMC are more likely to spontaneously utilize greater PFC resources, monitoring their actions and independently updating task goals when confronted with errors (where, as shown in Table 2, those mistakes were primarily driven by goal lapses on interference-rich incongruent trials for both groups). This pattern held even though our two WMC extreme groups were reasonably equivalent on two outward measures of behavior, RT and accuracy, rather than overly confounded by them, with perhaps a slightly greater probability of errors on incongruent trials for those with low WMC.

Throughout this article, we have argued that the ERP data reflect PFC–ACC interactions and the top-down bias of possible sources of interference in the attentional control network. More specifically, the PFC appears to bias conflict monitoring in ACC by influencing the magnitude of the ERN. This further alters downstream processing of errors by PCC, including Pe magnitude. The Pe is, in turn, associated with updating task goals, most likely cycling back to goal maintenance and an important role for PFC. Of course, in a recurrent network like this one, it can be difficult to rule out alternative explanations. Nevertheless, our results most clearly support the notion that PFC directly biases action monitoring in ACC, converging with interpretations of ERP results obtained in frontal lobe patients with compromised attentional control (Alain, McNeely, Yu, Christensen, & West, 2002; Gehring, Himle, & Nisenson, 2000; Gehring & Knight, 2000). Our study

4 For example, rather than directly influencing ACC activity per se, PFC may indirectly bias the magnitude of the ERN instead. Here, PFC may influence information processing in posterior visual pathways feeding into response systems, thereby enhancing the amount of response conflict on error trials detected by ACC (Yeung et al., 2004). We thank an anonymous reviewer for pointing out this alternative possibility.
extends this ERP literature by establishing a novel, theoretically
derived approach to the study of cognitive control. It advances our
understanding of the cognitive control system, particularly in the
context of working memory and executive control. This is an
important advance because of potential interpretive complications
associated with the use of neuropsychological patients, including variability
in the cause, extent, or location of lesions (see Banich, 2004).

Therefore, our study convincingly demonstrated that ERN modu-
lation is most likely due to true PFC–ACC interaction and top-
down bias of compensatory systems monitoring performance
rather than a restricted, emergent property associated with brain
damage in PFC. However, in contrast to patients with lateral PFC
damage where the magnitude of the ERN was influenced only
on correct trials (Gehring & Knight, 2000), individual differences in
WMc influenced the magnitude of the ERN only on error trials.
Consequently, while PFC–ACC interaction is not limited to neu-
ropsychological patients per se, the nature of the interaction dis-
ociates when comparing those with PFC damage with those with
reduced WMC.

Summary and Conclusions

To summarize, our ERP results indicated that individuals with
greater WMC have a more strongly biased attentional control
network governed by PFC and, therefore, may be more likely to
spontaneously monitor their actions, detect errors as reflected by a
larger ERN, and refresh task goals as reflected by a larger Pe
following the loss of cognitive set. Our results are more broadly
consistent with the literature on individual differences in cognitive
control, where greater WMC permits more flexible attentional
control over sources of interference (Engle, 2002). Ultimately,
PFC–ACC interactions may play a role in detecting-off-task be-
havior, updating task goals, and reducing errors in everyday life
(e.g., see Kane et al., 2007, on WMc and mind wandering).

Clearly, our study suggests that combining cognitive psychological
and electrophysiological approaches can be theoretically prof-
itable. This is particularly true when utilizing individual differ-
ences in WMc to predict the magnitude of brain potentials like the
ERN, a neural signature we leveraged as a biomarker of interfer-
ence in testing two-process models of attentional control.

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