Individual differences in electrophysiological responses to performance feedback predict AB magnitude

Mary H. MacLean · Karen M. Arnell

Abstract The attentional blink (AB) is observed when report accuracy for a second target (T2) is reduced if T2 is presented within approximately 500 ms of a first target (T1), but accuracy is relatively unimpaired at longer T1–T2 separations. The AB is thought to represent a transient cost of attending to a target, and reliable individual differences have been observed in its magnitude. Some models of the AB have suggested that cognitive control contributes to production of the AB, such that greater cognitive control is associated with larger AB magnitudes. Performance-monitoring functions are thought to modulate the strength of cognitive control, and those functions are indexed by event-related potentials in response to both endogenous and exogenous performance evaluation. Here we examined whether individual differences in the amplitudes to internal and external response feedback predict individual AB magnitudes. We found that electrophysiological responses to externally provided performance feedback, measured in two different tasks, did predict individual differences in AB magnitude, such that greater feedback-related N2 amplitudes were associated with larger AB magnitudes, regardless of the valence of the feedback.

Keywords Attention · Cognitive control · Event-related potential · ERP · Attentional blink · Feedback

When two to-be-attended targets are presented in a rapid serial visual presentation (RSVP) stream, the accuracy for the second target (T2) is reduced when it is presented within 500 ms after the first target (T1), relative to longer T1–T2 separations (Raymond, Shapiro, & Arnell, 1992). This phenomenon has been named the *attentional blink* (AB; Raymond et al., 1992). The magnitude of the AB can be captured in the change in T2

accuracy as a function of the temporal separation, or *lag*, between T1 and T2 (MacLean & Arnell, 2012). The AB has traditionally been interpreted as reflecting attentional limitations according to which processing of T1 interferes with and/or delays the allocation of attention to T2, if T2 is presented before T1 processing has been completed (e.g., Chun & Potter, 1995; Jolicœur & Dell'Acqua, 1998; Shapiro, Arnell, & Raymond, 1997). However, several recent models of the AB have implicated cognitive control as influential to the production of the AB.

The attentional blink and cognitive control

Several models of the AB, such as the temporary-loss-of-control (TLC) model (Di Lollo, Kawahara, Ghorashi, & Enns, 2005), the boost-and-bounce model (Olivers & Meeter, 2008), the threaded-cognition model (Taatgen, Juvina, Schipper, Borst, & Martens, 2009), and the overinvestment hypothesis (Olivers & Nieuwenhuis, 2006), feature cognitive control as being central to the presence of the AB. In several of these models, strong cognitive control predicts an increased AB. For example, in the boost-and-bounce model (Olivers & Meeter, 2008), it is proposed that selection of the T1 item leads to an excitatory "boost" that carries over to the distractor immediately trailing T1. In response to the incorrect boosting of the distractor into working memory, cognitive control is used to initiate an inhibitory "bounce" that prevents subsequent items, including T2, from inclusion into working memory. Similarly, according to the threaded-cognition model (Taatgen et al., 2009), selection of T1 initiates an overexertion of cognitive control (the "memory function") that results in impaired T2 detection at short lags. Note that according to both the boost-and-bounce and threaded-cognition models, better goal-driven cognitive control leads to larger ABs because greater cognitive control exerted to protect T1 identification comes at the cost of T2 identification at short lags. While it may seem counterintuitive that better cognitive

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control may predict larger ABs, some data do suggest that somewhat distracted performance (due to the need to perform an additional task) may decrease the AB by reducing attentional investment (Olivers & Nieuwenhuis, 2005, 2006), and that the probability of failing to correctly identify T2 is predicted by investment in T1 (Shapiro, Schmitz, Martens, Hommel, & Schnitzler, 2006). The overinvestment hypothesis posits that the typical goal-dedicated, but inappropriate, overinvestment of attention (relative to what is needed) actually increases the AB by allowing distractors to gain enough attention to become effective competitors for limited working memory resources (Olivers & Nieuwenhuis, 2006).

In summary, various models of the AB have proposed that increased cognitive control, which may be advantageous for identifying single targets, is not advantageous in an AB task, in which two targets are presented closely in time, resulting in an AB.

Individual differences in the AB and investment

The AB is not observed in some individuals, so-called "nonblinkers" (Martens, Munneke, Smid, & Johnson, 2006), and individuals differ reliably in the magnitude of their ABs (Dale & Arnell, in press; McLaughlin, Shore, & Klein, 2001). The degree to which individuals rigidly try to perform the two tasks in order and try hard to perform well on both tasks could predict the magnitude of their ABs via the degree of attentional investment and cognitive control: Individuals who invest more in the task and in T1 may have larger AB magnitudes.

In general, studies of the AB have shown that individual-difference measures that reflect, or are associated with, a flexible, diffuse, and less-invested processing style (e.g., Fredrickson & Branigan, 2005; Rowe, Hirsh, & Anderson, 2007) predict smaller AB magnitudes, and measures that reflect, or are associated with, a rigid, focused, and more-invested processing style (e.g., Ashby, Isen, & Turken, 1999) predict larger AB magnitudes. Individual-difference measures that predict a small AB include the following: high state and trait positive affect and low state and trait negative affect (MacLean & Arnell, 2010; MacLean, Arnell, & Busseri, 2010), high scores on the personality factors of extraversion and openness to experience and low scores on the neuroticism factor (MacLean & Arnell, 2010), flexible and effective control of working memory (Arnell, Stokes, MacLean, & Gicante, 2010; Arnell & Stubitz, 2010; Colzato, Spapé, Pannebakker, & Hommel, 2007), and a tendency to see the global or big picture in a display (Dale & Arnell, 2010). The importance of attentional investment to the AB has also been shown more directly using pretrial alpha event-related desynchrony (alpha ERD), which is a measure of attentional investment at the start of the RSVP trial. Alpha ERD was higher when T1 was correct (vs. incorrect) and when T2 was correct (vs. incorrect) at longer lags. However, alpha ERD was lower when T2 was correct (vs. incorrect) at shorter lags, suggesting that greater pretrial investment of attention exacerbates the AB (MacLean & Arnell, 2011).

If individual differences in AB magnitude result, at least in part, from individual differences in cognitive control and attentional investment, then measures of an individual's investment in tasks and task-relevant information and their concern with their performance may predict the magnitude of their ABs.

Event-related potential (ERP) indices of investment in task performance

The error-related negativity (ERN) is a negative ERP observed shortly following the commission of a response, and it is observed to be largest (i.e., most negative) following errors in performance (Gehring, Goss, Coles, Meyer, & Donchin, 1993). In Gehring et al.'s study, they observed that indices of error detection (presumably the result of monitoring) and adjustment were related to the size of the ERN. In particular, the size of the ERN increased as the force used to make the erroneous response decreased, as the likelihood that performance on the following trial would be correct, and as the degree of response slowing on the following trial (when correct) increased. Therefore, the ERN appears to be a reliable index of performance monitoring and appraisal. ERN amplitude can be modulated by motivational factors, such that a larger ERN is observed when accuracy is emphasized (Gehring et al., 1993). ERN amplitude has also been correlated with personality factors related to reward and punishment sensitivity (BIS/BAS), such that individuals high in the BIS factor tended to have larger ERN amplitudes (Boksem, Tops, Wester, Meijman, & Lorist, 2006). Neuroticism and conscientiousness have also been found to predict the magnitude of the motivational effect on ERN amplitude (Pailing & Segalowitz, 2004). Greater neuroticism was associated with a greater increase in ERN amplitudes as the incentive for accuracy increased, while conscientiousness showed the opposite pattern. Thus, the size of the ERN appears to be related to investment in tasks and task performance.

The ERN is often measured as the difference in amplitude of the negative ERP (which we will refer to as the *medial-frontal negativity* or *MFN*) that shortly follows the commission of incorrect responses relative to the ERP following correct responses. This difference measure reflects the accuracy-related modulation of the MFN. Should AB magnitude correlate positively with this difference measure, that would indicate that individuals who are more

responsive to incorrect performance *relative* to correct performance have larger AB magnitudes. Given that we do not hypothesize that the relationship between the strength of response to performance and AB magnitude should be confined to accuracy-related modulation of the MFN, we hypothesize that greater AB magnitude will be related to a stronger response to performance in general (i.e., larger, more negative MFNs), regardless of accuracy.

ERN amplitude has good test–retest reliability, indicating that ERN amplitude is a good candidate for a biological correlate of cognitive traits (Segalowitz et al., 2010). The evidence that ERN amplitude correlates with personality factors also suggests that ERN amplitude reflects a trait (Boksem et al., 2006; Pailing & Segalowitz, 2004). That ERN amplitude differs according to motivational effects (Gehring et al., 1993) and can differ according to subsequent behavioral adjustments (Rodriguez-Fornells, Kurzbuch, & Münte, 2002) suggests that the ERN amplitude could reflect trait-like differences in degrees of investment in tasks and in concern about performance (i.e., motivation).

In addition to examining responses to actual performance, as reflected in the ERN, it is also possible to examine responses to information that is relevant to performance—that is, feedback. The feedback-related negativity (FRN) is the second negative-going deflection (N2) observed following feedback and is typically observed to be larger following feedback that indicates that an error has occurred (Hajcak, Moser, Holroyd, & Simons, 2006; Yeung & Sanfey, 2004). Some evidence has suggested that the FRN is the same component as the ERP referred to as the N2, which can be observed following rare or "oddball" stimuli (Holroyd, Pakzad-Vaezi, & Krigolson, 2008). As with the ERN, it has been suggested that the FRN reflects the evaluation of outcomes in the context of goals and motivations (Simons, 2010).

The amplitude of the FRN has been shown to mirror the magnitude of the prediction error: As the likelihood of a negative outcome decreases (i.e., negative outcome not expected), the amplitude of the FRN to the negative outcome increases (Bellebaum & Daum, 2008). However, this modulation of FRN amplitude with the magnitude of prediction error has only been observed in individuals who learned strategies for maximizing reward in a learning task. Furthermore, the FRN amplitude is smaller following mistakes when, on a subsequent trial, participants make the same erroneous response than when they make a different later response (Cohen & Ranganath, 2007; van der Helden, Boksem, & Blom, 2010). Neuroticism also predicts FRN amplitude. Specifically, for individuals high in neuroticism, the FRN following uninformative feedback is larger than that observed following negative feedback (Hirsh & Inzlicht, 2008).

Most often the FRN is measured as a difference measure obtained by subtracting the components evoked by correct/positive feedback from those evoked by incorrect/negative feedback. This difference measure reflects valence-related modulation of the N2—that is, the amplitude of the N2 following feedback is more negative (larger) following incorrect/negative feedback than following correct/positive feedback. Should AB magnitude correlate positively with this difference measure, that would indicate that individuals who are more responsive to "incorrect" feedback *relative* to "correct" feedback have larger AB magnitudes. Other individual differences have been related to differences in the valence-related modulation of responses to feedback (e.g., Foti & Hajcak, 2009; Hirsh & Inzlicht, 2008).

However, the N2 following feedback stimuli is not modulated only by valence; for example, it is also modulated by the magnitude or value of reward stimuli (Santesso et al., 2008) and by the degree to which negative feedback is expected (Bellebaum & Daum, 2008; Hajcak, Moser, Holroyd, & Simons, 2007). So, it appears that the N2 is a component sensitive *in general* to performance-relevant information contained in the feedback. The purpose of the difference measure is to eliminate the components commonly evoked by "correct" and "incorrect" feedback—for example, those related to the frequency, expectedness, and value of the feedback—leaving only those related to the valence of the feedback (Holroyd & Krigolson, 2007). Given that we do not hypothesize that the relationship between the strength of response to feedback and AB magnitude should be confined to the valence-related modulation of the feedback-related N2, we hypothesize that greater AB magnitude will be related to a stronger response to feedback in general (i.e., larger, more negative N2s), regardless of valence. Other individual differences have been related to the general response to feedback (i.e., a main effect, regardless of feedback valence; see, e.g., Santesso, Dzyundzyak, & Segalowitz, 2011; Tucker, Luu, Frishkoff, Quiring, & Poulson, 2003).

The amplitude of the N2 components to positive and negative feedback (which are used to measure the FRN) have good test–retest reliability, indicating that N2/FRN amplitude is also a good candidate for a biological correlate of cognitive traits (Segalowitz et al., 2010). The evidence that FRN amplitude correlates with a personality factor also suggests that it could reflect a trait (Hirsh & Inzlicht, 2008). That FRN amplitude differs according to the magnitude of the prediction error (Bellebaum & Daum, 2008) and of subsequent behavioral adjustments (Bellebaum & Daum, 2008; Cohen & Ranganath, 2007; van der Helden et al., 2010) suggests that FRN amplitude could, like ERN amplitude, reflect trait-like differences in the degrees of investment in task and performance.

The present study

It has been suggested that cognitive control and the overinvestment of attention contributes to the production of the

AB (Di Lollo et al., 2005; Olivers & Meeter, 2008; Olivers & Nieuwenhuis, 2006; Taatgen et al., 2009). Thus, the degrees to which individuals invest in tasks and their performance may predict individual differences in AB magnitude. The ERN (and its MFN components) and the FRN (and its N2 components) are thought to reflect the responses to performance outcomes in the context of expectations and goals. ERN amplitude has been shown to reflect the strength of the incongruence between expected/correct behavior and the context of erroneous outcomes (e.g., Danielmeier, Wessel, Steinhauser, & Ullsperger, 2009; Rodriguez-Fornells et al., 2002). FRN amplitude has been shown to reflect the degrees to which individuals adopt and enforce strategy (i.e., invest in tasks) to achieve goals (Bellebaum & Daum, 2008; van der Helden et al., 2010). Thus, we hypothesize that both the MFN and feedback-N2 amplitudes will predict AB magnitude, such that greater MFN/N2 amplitude will be associated with larger AB magnitudes.

In this study, we measured the response-locked ERN and its component MFNs generated by a flanker task, as well as the stimulus-locked FRN and its component N2s generated by feedback, in two separate tasks: an AB task and a time production task. We correlated AB magnitude with the amplitudes of the MFNs and the feedback-N2s to look for relationships between the AB and feedback responsivity. We also correlated AB magnitude with the ERN and FRN using incorrect-minus-correct trial difference measures as predictors, to see whether any of the relationships between feedback responsivity and the AB are modulated by the accuracy of performance or the valence of the feedback.

Method

Participants

The participants were 71 Brock University undergraduate students (45 females, 26 male; mean age = 20 years), recruited through the Brock Psychology Department's online system for participant recruitment. Due to an error in the recording procedures, electroencephalography (EEG) data for the AB task were unavailable for four of the participants. In addition, 26 participants did not complete the flanker task, which was the final task in the study.

Procedure

Participants completed the AB task first, then the time production task, and finally the flanker task. The stimuli were displayed and responses were logged using E-Prime (Psychology Software Tools Inc., Sharpsburg, PA), running on a desktop PC and presented on a CRT monitor (refresh rate of 60 Hz).

AB task The AB task consisted of four blocks of 60 RSVP trials; 120 of these were T2-absent trials (T1 only), and 120 contained both T1 and T2. When present, T2 was shown three items, or 252 ms, after T1 (lag 3) on half of the trials, or eight items, 672 ms, after T1 (lag 8) on the other half. All trial types were presented randomly within each block. T1 was always presented in white font as the sixth item in the stream. T1 was a single uppercase letter randomly selected from among the letters M, N, P, R, S, T, U, V, W, Y, and Z (11-alternative forced choice). T2 was the uppercase letter X, always presented in black font. The distractor items consisted of single uppercase letters always presented in black font and randomly selected from the alphabet, with the exclusion of the letters I, O, Q, and X. No letter was ever repeated sequentially. Each trial began with a fixation cross for 417 ms, followed immediately by the RSVP stream. The RSVP stream consisted of 15 stimuli with an SOA of 84 ms/ item (no blank interstimulus interval) presented on a gray background. At the end of each stream, participants indicated the identity of the white letter (T1 response) and then reported whether an X was present or absent (T2 response). Participants made unspeeded T1 and T2 responses using a keyboard in response to onscreen prompts presented after each RSVP stream. The participants were instructed that the X was not always present, but that when present it could appear at any point after the white letter. In an attempt to control the false alarm rate, we instructed participants to indicate that an X was present only if they were "pretty sure" that they had seen one. Following each participant's T2 response was an interstimulus interval of 1,000 ms and then the presentation of feedback for 167 ms. The feedback accurately indicated whether the participant's T2 response was correct ("Correct!" was presented following a hit or correct rejection) or incorrect ("Incorrect!" was presented in following a miss or false alarm). For 20 participants, the feedback on both correct and incorrect trials was presented in black font. For the other 51 participants, "Correct" was presented in blue font and "Incorrect" was presented in red font. There were no differences in the groups' AB performance as a function of black versus colored feedback, and feedback color did not influence any of the relationships reported below; therefore, the data were collapsed across this variable. An intertrial interval (ITI) of 1,000 ms followed the feedback.

Time production task The time production task was adapted from Hirsh and Inzlicht (2008) and consisted of four blocks of 42 trials apiece. Each trial began with a fixation cross (167 ms) followed by a 2,000-ms blank response period. Participants were instructed to press the spacebar when they thought that 1 s had elapsed since the fixation cross disappeared (i.e., that the screen had been blank). Following the 2,000-ms blank response period, feedback was presented for

917 ms, followed by a blank ITI of either 1,000, 1,500, or 2,000 ms. On 112 (or 2/3) of the trials, the feedback was informative, indicating whether the participant's estimate was correct ("+") or incorrect ("–"). On the remaining 56 (1/3) of the trials, the feedback was uninformative regarding performance ("?"). This feedback—correct, incorrect, or uninformative—was presented in white font for 20 participants, and in green ("+"), red ("–"), or white ("?") for the other 51 participants. As with the AB task, feedback color did not influence time estimation performance or any of the relationships reported below, so the data were collapsed across this variable. Participants were instructed before the task began regarding the meanings of the different feedback symbols. Initially an estimate was considered correct if it was presented within 100 ms of 1 s (i.e., 900–1,100 ms into the blank response period). Following a correct estimate, this window decreased by 10 ms, and following an incorrect estimate, the window increased by 10 ms. This procedure ensured that all of the participants received "incorrect" feedback on approximately the same number of trials (~1/2 of the informative-feedback trials, or 56 trials).

Flanker task The flanker task consisted of four blocks of 160 trials. Each trial consisted of a flanker stimulus presented for 117 ms, followed by a 1,250-ms blank ITI before the flanker stimulus for the next trial. The flanker stimuli consisted of a string of five uppercase letters (Ss or Hs) in black font presented on a gray background. On half of the trials, the flanker stimulus was congruent, such that all five letters were the same. On the other half of the trials, the flanker stimulus was incongruent, such that the letter in the middle of the string differed from the four flanking letters. Participants were asked to indicate what the middle letter of the letter string was by pressing either the "s" or the "h" button on a keyboard. Participants were instructed to respond as quickly as possible while still being accurate. In order to increase errors, if a participant had less than 10 % errors in a given block, a message was presented during the following break telling the participant that he or she was responding too slowly.

EEG acquisition EEG was recorded continuously using tin electrodes embedded in an Electro-Cap (Electro-Cap International Inc., Eaton, Ohio) from 29 sites distributed according to the 10–20 system, with an electrode placed anterior to Fz as the ground. EEG was recorded using the linked left and right earlobes as references, and was re-referenced to a common average of the EEG. EEG data were acquired with Neuroscan acquisition software (Compumedics USA, Charlotte, NC), using a 32-channel NeuroScan SynAmp. The data were sampled at a rate of 500 Hz. An electrooculogram (EOG) recorded horizontal eye movements using electrodes placed on the outer canthus of each eye, and vertical eye

movement and blinks using electrodes placed on the infra- and supraorbital regions of each eye. Impedances for both the EEG and EOG were maintained below 10 kΩ. The EEG was low-pass filtered offline at 30 Hz using a roll-off of 48 dB/octave.

Data analysis

FRN analysis For the EEG data from both the AB and time production tasks, epochs were created beginning 200 ms before and 1,000 ms after the presentation of the feedback. Epochs were baseline corrected using the 200-ms prefeedback interval. Epochs with amplitudes exceeding $\pm 50\,\mu$V were rejected. Epochs were then visually examined, and remaining epochs that contained artifacts were also rejected. The epochs that remained after all exclusions were then averaged. In the AB task, averages were created for each combination of T2 condition (absent, lag 3, and lag 8) and T2 response (correct or incorrect). Only epochs in which T1 was correct were included in these averages. In the time production task, averages were created according to the type of feedback presented: uninformative, correct, or incorrect.

Group averages were then created and examined for each task to isolate the site where the feedback-related N2 was maximal and the time windows for defining two ERP peaks: the positivity preceding the N2 (the P2) and the N2, used to isolate the FRN. For both tasks, the ERPs were scored at FCz, where the N2 was largest. From visual inspection of the average waves for each task, for purposes of scoring the P2 was defined as the most positive value between 160 and 230 ms in the AB task, and between 150 and 250 ms in the time production task. The N2(AB) was defined as the most negative value between 230 and 300 ms in the AB task, and the N2(time) as the most negative value between 200 and 300 ms in the time production task.

ERN analysis Epochs were created from 500 ms before to 250 ms after response. Epochs with amplitudes exceeding $\pm 50\,\mu$V were rejected. The epochs were then visually examined, and remaining epochs with artifacts were also rejected. Of the trials still remaining, only those with RTs >200 ms and <1,000 ms were then averaged. An average was created for each combination of congruency (congruent and incongruent) and performance (correct and incorrect). Group averages were then created and examined to identify the site where the performance-related MFN was maximal and the time windows for defining two ERP peaks: the positivity preceding the MFN (the PP) and the MFN. ERPs were scored at Cz, where the MFN was largest. The PP was defined as the most positive value between −100 and −20 ms, and the MFN was defined as the most negative value between −50 and 50 ms.

Results

Behavioral results

AB task performance The AB data from 13 participants were excluded due to either poor T1 performance (10 participants with <40 %) or poor T2 sensitivity at lag 8 (three participants with <10 % hits minus false alarms) on the AB task. The mean T1 accuracy for the remaining participants was 65.95 % (*SD* = 14.13). Note that the unusually low T1 accuracy, which is still well above chance, is possibly due to the unintentional emphasis on T2 performance that the presence of T2 performance feedback introduced. T2 performance was conditionalized on T1 accuracy. The mean number of hits for T2 at lag 3 was 47.81 (*SD* = 28.23), the mean number of T2 hits at lag 8 was 81.74 (*SD* = 14.50), and the mean rate of false alarms on T2-absent trials was 16.93 (*SD* = 12.10). The mean T2 performance (hits – false alarms) was 21.09 % at lag 3 (*SD* = 18.74) and 65.76 % at lag 8 (*SD* = 18.14). A paired-samples *t* test showed significantly lower T2 performance at lag 3 than at lag 8, indicating the presence of an AB [$t(41)$ = 18.71, $p <$ .001]. The individual AB magnitude was represented by the standardized residual of lag 3 T2 performance when lag 8 T2 performance was controlled (i.e., the residual variability in lag 3 T2 performance after removing the shared variance with lag 8 T2 performance). This standardized residual is computed by using linear regression to predict lag 3 T2 performance with lag 8 T2 performance and saving the standardized residual (for a more detailed account of this method, see MacLean & Arnell, 2012). This measure of AB magnitude (henceforth referred to henceforth as *lag 3 T2 performance*) removes individual differences in T2 performance while reflecting the degree to which T2 performance is relatively impaired when T2 is presented within the AB period. Note that higher values indicate less impairment and a smaller AB magnitude. The residual measure of lag 3 T2 performance was reliable, as determined using split-half correlations (r = .58). A similar residual variable (*lag 8 T2 performance*) was created for lag 8 T2 performance when lag 3 T2 performance was controlled. The residual measure of lag 8 T2 performance was also reliable, as determined using split-half correlations (r = .73).

Time production task performance The average time at which participants indicated that 1 s had passed was 980 ms (*SD* = 146), ranging from 348 to 1,294 ms; on average, participants tended to prematurely indicate that 1 s had passed by 20 ms. The mean number of trials on which participants received negative feedback was approximately 1/3 of the 168 trials (*M* = 58, *SD* = 4),

with very little variability (individual rates varied from 55 to 71).

Flanker task performance A paired-samples *t* test indicated that accuracy on congruent trials (*M* = 85.53 %, *SD* = 10.15) was significantly greater [$t(35)$ = 5.75, $p <$ .001] than accuracy on incongruent trials (*M* = 79.83 %, *SD* = 12.15). Error rates (both congruent and incongruent trials) varied from 3 % to 46 % (*M* = 17.58, *SD* = 10.81). Reaction times on correct congruent trials (*M* = 472, *SD* = 53) were significantly faster [$t(35)$ = 8.92, $p <$ .001] than reaction times on correct incongruent trials (*M* = 493, *SD* = 53). Thus, our results display the typical flanker effects.

Electrophysiological results

ERP results For the purposes of examining mean effects, the N2 and MFN were estimated using a peak-to-peak measure of amplitude. The N2 (for both the AB and time production tasks) and MFN (for the flanker task) amplitudes were both calculated as the difference in peak amplitude between the N2 or MFN and the P2 or PP that preceded it (i.e., P2 – N2, PP – MFN). Larger values on this peak-to-peak measure indicate larger N2/MFN amplitudes.

FRN (AB task) N2s were observed in the group averages and at the individual level following the feedback in the AB task (see Fig. 1). A paired-samples *t* test comparing the N2 (AB) peak-to-peak measure following "correct" and "incorrect" feedback was significant [$t(53)$ = 2.95, $p =$ .005], indicating that the N2(AB) was larger following "incorrect" feedback (*M* = 7.22, *SD* = 3.67) than following "correct" feedback (*M* = 6.37, *SD* = 3.14). Note, however, the sizeable N2(AB) following "correct" feedback.
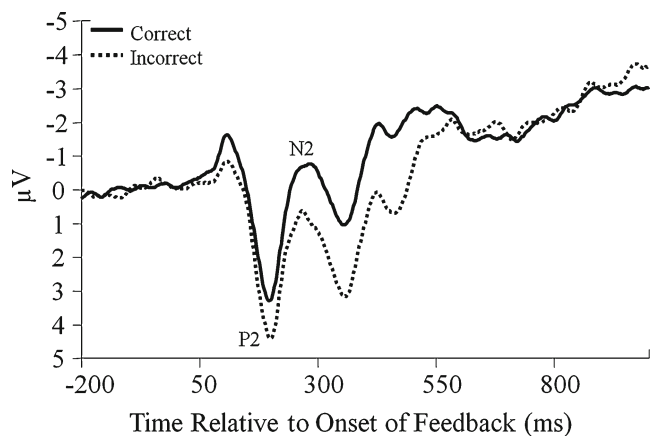

Fig. 1 Group averaged waveform for the feedback-related negativity (N2) and its preceding positivity (P2), time-locked to the feedback in the attentional blink (AB) task at electrode FCz. Both average waveforms are averaged across lags and only include those trials on which T1 performance was correct

FRN (time production task) We observed N2s in the group averages and at the individual level following the feedback in the time production task (see Fig. 2). A repeated measures analysis of variance (ANOVA) was performed with the factor Feedback Type (uninformative, correct, and incorrect) using the N2(time) peak-to-peak measure. A significant main effect of feedback type emerged [$F(2, 106) = 19.61$, $p < .001$], such that the N2(time) was largest following "incorrect" feedback ($M = 5.76$, $SD = 3.36$), second largest for uninformative feedback ($M = 4.87$, $SD = 3.06$), and smallest for "correct" feedback ($M = 3.69$, $SD = 2.81$). Note again the sizeable N2(time) following "correct" feedback. Post-hoc paired *t* tests revealed that all N2(time) differences were significant (incorrect – uninformative, uninformative – correct, incorrect – correct; all ps < .01).

ERN (flanker task) We observed apparent MFNs in the group averages and at the individual level following both correct and incorrect responses in the flanker task (see Fig. 3). A 2 × 2 repeated measures ANOVA was performed with the factors Flanker Type (congruent and incongruent) and Performance (correct and incorrect) using the MFN peak-to-peak measure. The only significant effect was a main effect of performance [$F(1, 35) = 48.73$, $p < .001$], such that the MFN was larger following incorrect ($M = 5.15$, $SE = .57$) than following correct ($M = 1.67$, $SE = .25$) responses. However, a sizeable MFN was still observed following correct responses.

Relationships between AB performance and ERPs

For the purpose of examining individual differences in the N2 and MFN, new variables were created by saving the standardized residuals of the FRN or ERN peak scores, controlling for the P2 or PP peak scores for each separate task (i.e., the residual variability in the N2 or MFN peak
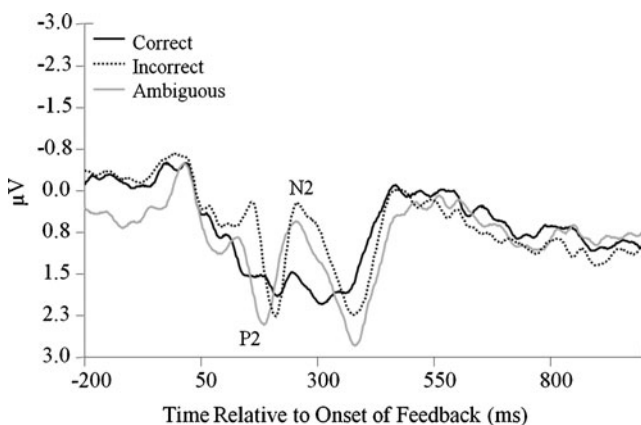

Fig. 2 Group averaged waveforms for the feedback-related negativity (N2) and its preceding positivity (P2), time-locked to the feedback in the time production task at FCz
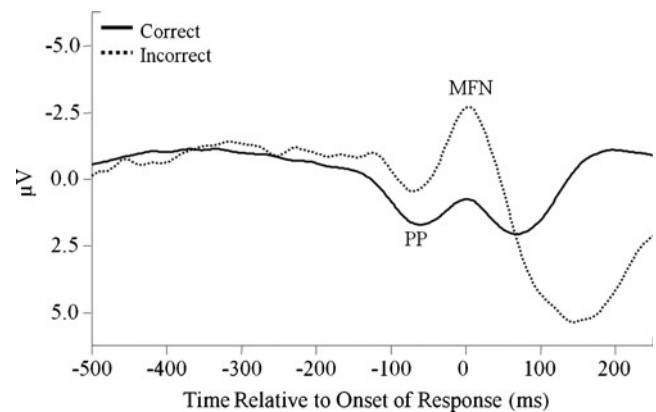

Fig. 3 Group averaged waveforms for the medial-frontal negativity (MFN) and its preceding positivity (PP), time-locked to response in the flanker task at Cz

score after removing shared variance with the P2 or PP peak score). All standardized residuals are the product of a linear regression with the ERP of interest (N2 or MFN) as the dependant variable and the preceding ERP (P2 or PP) as the predictor variable. These new residual variables were used for all correlations and regressions, given that (1) they are more reliable than the peak-to-peak difference measures used to examine the mean effects, and (2) this method controls for individual differences in the peak used as the reference peak (e.g., the P2 or PP for the N2 or MFN). The reliability (internal consistency) for all residual ERP measures was extremely high, as determined using split-half correlations: N2(AB), $r = .91$; N2(time), $r = .87$; MFN, $r = .80$.

As we discussed above, it was predicted that the N2 following feedback of all kinds—in other words, the general strength of the response to feedback—would relate to AB task performance. In order to test this possibility, the N2 (AB) residual amplitude scores were averaged across all T2 types, creating N2(AB) residual amplitude scores for both "correct" and "incorrect" feedback. The correct and incorrect N2s were also averaged across tasks (AB and time production) to create overall N2 residual amplitude scores for "correct" and "incorrect" feedback. It was also possible that the relationship between individual differences in feedback responsivity and the AB would be modulated by the valence of the feedback. In order to examine this, we also created difference measures for the FRN in both the AB and time production tasks and the ERN in the flanker task. The difference measures were calculated by subtracting the standardized residual measures of the FRN/ERN following "correct" feedback from that following "incorrect" feedback.[1]

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[1] This method for creating a difference measure eliminates the possibility of confounding latency effects with amplitude effects that the other method—subtracting entire waveforms prior to scoring the peak amplitude—can create.

N2s and MFNs The N2s were highly positively correlated with each other across feedback types and within, between, and averaged across (overall) tasks (all *p*s < .001), and they were somewhat positively related to the MFNs, but not significantly. T1 accuracy was not found to correlate with any of the ERPs (see Tables 1 and 2).[2] Lag 8 T2 performance correlated negatively with correct MFN amplitude (see Table 1), such that greater lag 8 T2 performance was associated with a more negative (i.e., larger) MFN to correct performance. Lag 3 T2 performance correlated positively with all N2 amplitudes, such that greater lag 3 T2 performance (i.e., smaller AB magnitude) was associated with more positive (i.e., smaller) N2s in both the AB and time production tasks (see Table 2). Although these relationships were only significant with the N2s to "correct" feedback (see Fig. 4a and b) from each task (incorrect *p*s < .10), lag 3 T2 performance was significantly positively correlated with overall N2s to both "correct" and "incorrect" feedback (averaged across tasks; see Fig. 4c and d). As Pearson's *r* is sensitive to extreme scores, and it appears from the scatterplots in Fig. 4 that our sample did contain at least one extreme score, we also calculated Spearman rank-order correlations (rho). These analyses did not change the pattern or significance of any of the relationships concerning the N2: lag 3 T2 performance and overall correct N2, *r* = .39 (*p* = .002); overall incorrect N2, *r* = .28 (*p* = .034). However, the relationship between lag 8 T2 performance and the correct MFN was not significant according to the Spearman rank-order correlation (*r* = −.29, *p* = .068). Similarly, when the one extreme standardized score (>3) was removed, all previously significant relationships remained significant (all *p*s < .05).

The results of the correlations were also replicated when we examined mean differences in lag 3 T2 performance on the basis of a median split of participants according to N2 amplitudes to "correct" feedback on each task (time production and AB). In both cases, individuals with more negative (i.e., larger) N2s to "correct" feedback had lower lag 3 T2 performance [*M*(AB) = −.34, *M*(time) = −.33] than did individuals with more positive (i.e., smaller) N2s to "correct" feedback [*M*(AB) = .31, *M*(time) = .27]: AB, *t*(52) = 2.57, *p* = .013; time, *t*(52) = 2.31, *p* = .025.

Given that both overall N2 amplitudes (correct and incorrect) correlated significantly with lag 3 T2 performance and also correlated significantly with each other, a simultaneous multiple regression was performed predicting lag 3 T2 performance with both overall N2s to determine whether the two overall N2 amplitudes explain the same variability in lag 3 performance. The model was significant, R^2 = .38, *p* = .013, and the overall correct N2 was nearly a

Table 1 Correlations between attentional blink (AB) task performance and medial-frontal negativities to "correct" and "incorrect" feedback in the flanker task

	Correct	Incorrect
T1 accuracy	.09	−.29
Lag 8 T2 performance	−.35[*]	−.17
Lag 3 T2 performance	.19	−.09

n = 39, [*] *p* < .05 (two-tailed)

significant unique predictor (semipartial *r* = .25, *p* = .053), over and above the overall incorrect N2, while the overall incorrect N2 was not (semipartial *r* = −.05, *p* = .694). The results of the multiple regression indicate that the correlation between overall incorrect N2 amplitude and lag 3 T2 performance is entirely due to shared variance between the overall correct and incorrect N2 amplitudes, but that the relationship between overall correct N2 amplitude and lag 3 T2 performance can also be attributed to variance unique to the overall correct N2 amplitude.[3]

FRNs and ERNs (difference measures) Correlations between AB performance measures and the FRN and ERN difference measures were performed to see whether the relationship between individual differences in feedback responsivity and the AB was modulated by feedback valence. T1 accuracy was significantly correlated with the ERN difference measure, such that greater T1 accuracy was associated with a smaller accuracy-related ERP modulation (see Table 3). Lag 8 T2 performance was not found to correlate significantly with any of the FRN or ERN measures. Lag 3 T2 performance was also unrelated to the FRN or ERN measures, but showed a trend toward negative correlations. This suggests that, if anything, smaller ABs (reflected in higher lag 3 T2 performance) were somewhat associated with smaller accuracy- and valence-related modulations of the ERPs (given that higher FRN/ERN values reflect larger FRN/ERNs), which would be expected, given the findings above that larger N2s to "correct" feedback were especially strong predictors of greater ABs.

Trial-to-trial FRN(AB)

It is possible that the relationship between lag 3 T2 performance and overall N2(AB) magnitude could result from a large N2 on trial *n* leading to a short-term increase in investment that might be expected to increase long-lag T2

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[2] The pattern of relationships remained when the rate of negative feedback was controlled for in a multiple regression of lag 3 T2 performance.

[3] When the same simultaneous regression was performed separately for each task, the same pattern was observed, except that in the case of the N2s from the AB task, the N2 to "correct" feedback reached significance as a unique predictor of lag 3 T2 performance (semipartial *r*=.33, *p*=.007).

Table 2 Correlations between attentional blink (AB) task performance and feedback N2s in the AB and time production tasks

	AB		Time			Overall	
	Correct	Incorrect	Uniform.	Correct	Incorrect	Correct	Incorrect
T1 accuracy	–.10	–.14	–.09	.00	.08	–.07	–.06
Lag 8 T2 perform.	–.24	–.13	.07	–.06	–.07	–.16	–.12
Lag 3 T2 perform.	.39[**]	.26	.17	.31[*]	.25	.38[**]	.29[*]

$n = 54$, [*] $p < .05$, [**] $p < .01$
(two-tailed)

accuracy but to impair short-lag T2 accuracy on trial $n + 1$. To test this possibility, N2s in the AB task were averaged according to T2 performance on the subsequent trial (correct or incorrect), as well as the T2 condition on the subsequent trial (lag 3, lag 8, and absent)—for instance, N2 (on trial n) when T2 performance was incorrect on the subsequent ($n + 1$) trial, in which T2 was presented at lag 3. This yielded six different averages for each individual. From visual inspection of the group average waveforms at site FCz, the P2 was defined as the most positive peak between 160 and 200 ms, and the N2 was defined as the most negative peak between 200 and 260 ms.

A 3 × 2 repeated measures ANOVA was performed with the factors T2 Type on the Subsequent Trial (absent, lag 3, and lag 8) and T2 Performance on the Subsequent Trial (correct and incorrect), using the N2(AB) peak-to-peak measure as the dependent variable.[4] Note that two individuals were missing data for their lag 8 incorrect averages (i.e., they had no artifact-free epochs for lag 8 incorrect). We found a significant main effect of T2 performance [$F(1, 48) = 22.88$, $p < .001$], such that the N2 was larger when it preceded trials on which T2 performance was incorrect ($M = 7.88$, $SE = 0.52$) rather than trials on which T2 performance was correct ($M = 6.14$, $SE = 0.39$). A significant interaction between T2 performance and T2 type also emerged [$F(2, 96) = 12.01$, $p < .001$; see Fig. 5]. In contrast to the possibility described above, the nature of this interaction was such that the N2 was larger when it preceded trials on which T2 performance was incorrect rather than trials on which T2 performance was correct, but only for trials on which T2 was presented at lag 8 [$t(48) = 5.09$, $p < .001$] or T2 was absent [$t(50) = 5.33$, $p < .001$]. We found no difference in the preceding trial's N2 for correct and incorrect T2 trials when T2 was presented at lag 3 [$t(51) = −0.36$, $p = .721$].

General discussion

The purpose of this investigation was to determine whether the response to performance and to feedback on performance, as measured by the MFN and N2, would predict AB magnitude,

and whether this relationship was modulated by whether the feedback/performance was correct or incorrect. It has been proposed in several models that cognitive control and overinvestment contribute to the production of the AB (Olivers & Meeter, 2008; Olivers & Nieuwenhuis, 2006; Taatgen et al., 2009). On the basis of these models and of findings showing that individual differences that reflect, or are associated with, a flexible, diffuse, and less-invested processing style predict smaller AB magnitudes (e.g., Dale & Arnell, 2010; MacLean & Arnell, 2010; MacLean et al., 2010), we hypothesized that greater AB magnitude should be associated with larger performance-related MFN and feedback-related N2 amplitudes. Specifically, individuals who demonstrate strong responses to performance and feedback may be more invested in the task and concerned with their performance, and thus have been hypothesized to have larger AB magnitudes than do individuals with weaker responses and weaker cognitive control/less investment.

Our hypothesis regarding feedback was supported. The feedback-related N2 amplitude (correct or incorrect), but not the valence-related modulation (difference measure), was found to predict AB magnitude significantly, such that overall, larger N2 amplitudes to feedback were associated with larger AB magnitudes. This relationship was observed using N2 amplitudes that followed both "correct" and "incorrect" feedback in two different tasks, an AB task and a time production task. Thus, individual differences in AB magnitude were predicted by individual differences in overall N2 amplitude from the same AB task and in a completely unrelated time production task. N2 amplitudes to either "correct" or "incorrect" feedback did not predict T1 accuracy or long-lag T2 performance, so the relationship between N2 amplitude and AB task performance was confined to T2 performance at short lags (i.e., during the AB interval). The FRN difference measure did not predict AB magnitude, suggesting that the differential response to negative feedback was not what drove the relationship, but rather the response to feedback, regardless of valence. However, response to feedback on correct trials was the most reliable predictor of AB magnitude, and the results of a simultaneous regression suggested that the relationship between AB magnitude and the N2 to "correct" feedback was due in part to unique variance unrelated to the N2 to "incorrect" feedback.

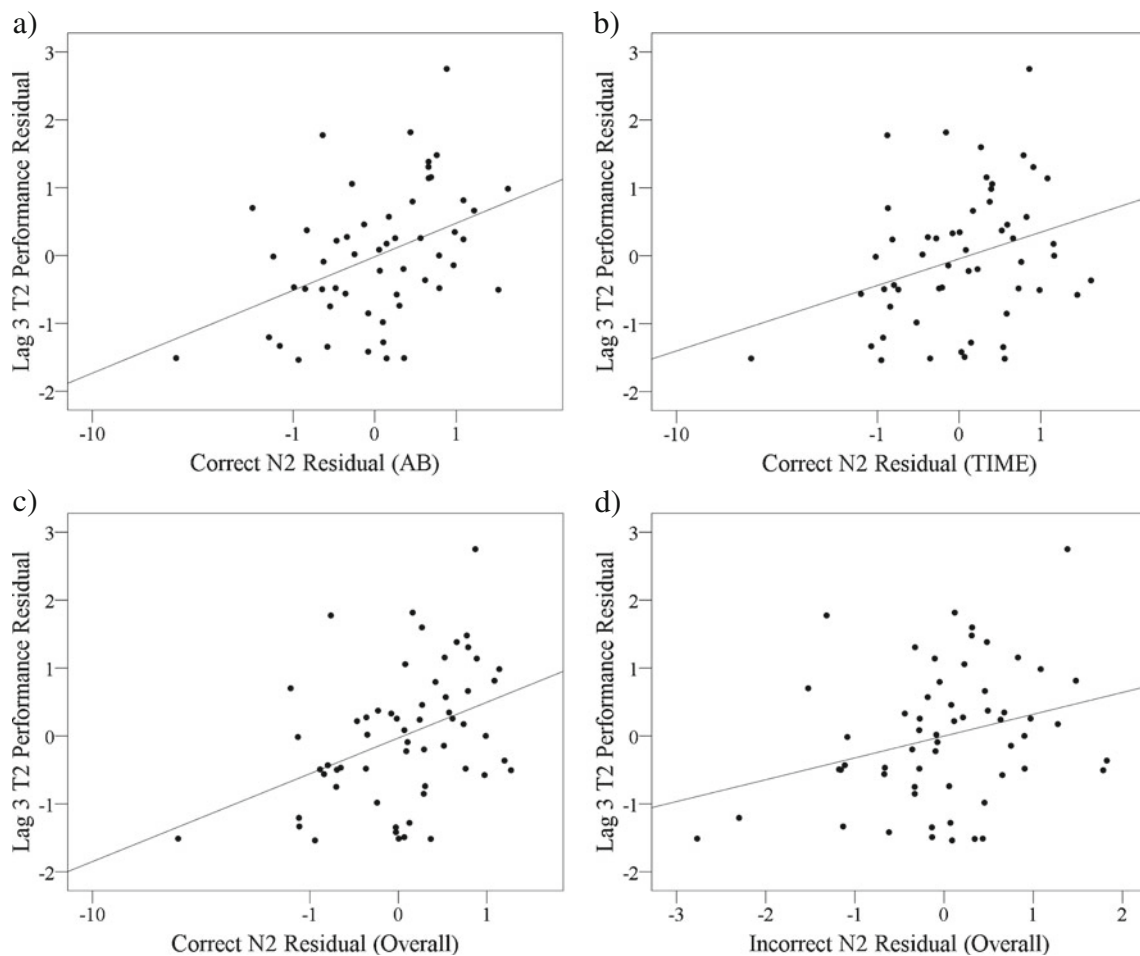[4] The type of feedback that produced the N2 did not interact with any of these effects.

Fig. 4 (a–d) Scatterplots depicting correlations between lag 3 T2 performance and the (**a**) correct N2(AB), (**b**) the correct N2(time), (**c**) the overall correct N2 (averaged across tasks), and (**d**) the overall incorrect N2 (averaged across tasks)

Evidence has also emerged that the valence modulation of the N2—that is, the FRN—is in fact due to a reduction in the negativity of the N2 caused by a positive-going component related to the processing of positive feedback (Holroyd et al., 2008). In other words, the valence effect of the N2 that is typically treated as the FRN is due to variability in the N2 to positive feedback, and not to variability in the N2 to negative feedback, which Holroyd et al. classified as a "common" N2. In this case, it is possible that the more reliable and unique relationship that we observed between the N2 to "correct" feedback and AB magnitude is due to individual differences in the positive-going component that reduces the size of the N2 to "correct" feedback, as compared to the N2 to "incorrect" feedback. Other individual differences have also been found to be uniquely related to the processing of positive feedback; for example, problem gamblers were found to have a smaller N2 to positive feedback than did controls, but not to differ from controls in response to negative feedback (Hewig et al., 2010). We

speculate that the majority of participants might be expected to be at least modestly responsive to negative feedback, but that only those participants who were truly high-performance monitors would be expected to show a large response to positive external feedback.

Table 3 Correlations between attentional blink (AB) task performance and the feedback-related negativities (FRNs) in the AB and time production tasks and the error-related negativity (ERN) in the flanker task

	FRN(AB)	FRN(Time)	ERN
T1 accuracy	−.07	.09	−.34[*]
Lag 8 T2 performance	.19	−.02	.17
Lag 3 T2 performance	−.25[#]	−.08	−.22

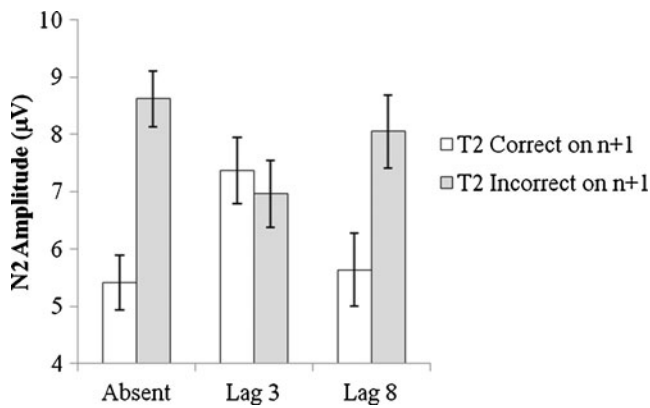$n = 54$, [#] $p < .10$ [*] $p < .05$ (two-tailed)

Fig. 5 Mean N2(AB) peak-to-peak amplitudes on the current trial (*n*), according to T2 type and T2 performance on the subsequent trial (*n* + 1). Error bars represent the standard errors of the mean differences (incorrect – correct)

Neither the ERN nor the MFN component correlated with AB magnitude. However, T1 performance was related to the performance-related modulation (difference measure) of the ERN, such that larger differences were associated with better T1 performance, and the overall MFN amplitude did predict long-lag T2 performance, such that larger ERN amplitudes were associated with better long-lag T2 performance. Thus, greater responsivity to internal feedback cues does not appear to predict the AB, but may be associated with superior target performance outside of the AB.

Individual differences in electrophysiological responses to performance feedback were associated with individual differences in AB magnitude, as hypothesized. This could happen in at least two ways. One of these is more trait-like, in that some individuals approach each experiment more or less invested than do other individuals, in terms of assigning strict cognitive control to the task at hand. This would suggest that some individuals should invest more than others throughout the task, regardless of the feedback on any given trial. Another possibility is that some individuals are more responsive to feedback on a trial-to-trial basis, and that the feedback-related N2 amplitude predicts individual differences in AB magnitude through trial-to-trial changes in cognitive control. For example, larger N2s following feedback on a given trial may increase cognitive control momentarily, and thus increase the probability that T2 performance will be incorrect on the following trial if T2 is presented at a short lag following T1 (i.e., increased probability of an AB). However, our trial-based analysis did not support this possibility. Instead, the feedback-related N2 was larger when it preceded trials on which T2 performance was incorrect rather than those on which T2 performance was correct, and only when T2 was presented at the long lag or when T2 was absent. We found no difference in the feedback-related N2 preceding trials on which T2 was presented at the short lag. This suggests that the feedback-

related N2 amplitude does not predict individual differences in AB magnitude through trial-to-trial changes in cognitive control that increase the probability of the AB. Indirectly, then, this provides some support for the idea that feedback-related N2 amplitudes may predict individual differences in AB magnitude because of general trait-like differences in responses to feedback and the rigid enforcement of cognitive control across the entire task.

The trial-based analysis did reveal that trial-to-trial changes in feedback-related N2 amplitudes did result in differences in T2 performance when T2 was presented at long lags and when T2 was absent. Thus, it appears that trial-to-trial changes in feedback-related N2 amplitudes were associated with differences in sensitivity at T2 detection when T2 was not affected by proximity to T1. Specifically, larger feedback-related N2s were associated with a reduction of T2 detection sensitivity (i.e., lower probability of hits, higher probability of false alarms) outside of the critical AB period. This is contrary to the evidence that larger FRNs are associated with improvements in performance, presumably via strengthening of cognitive control (Cohen & Ranganath, 2007; van der Helden et al., 2010). It is not clear why trial-to-trial responses to feedback influence T2 performance outside of the AB interval, whereas individual-to-individual differences do predict AB magnitude. However, this finding does underscore that these AB conditions can produce dissociable effects.

We made similar hypotheses about the relationship of the AB with both the N2 and MFN; however, only our hypothesis concerning the N2 and the AB was supported by the results. We do not wish to make the claim that this is necessarily evidence of dissociation. However, differences between the FRN and the ERN do allow us to make suggestions about the nature of the relationship between the electrophysiological responses to internal and external sources of feedback. The FRN is produced by externally generated representations of goal failures while the ERN is produced by internally generated representations of goal failures (Heldmann, Rüsseler, & Münte, 2008; Holroyd & Coles, 2002; Nieuwenhuis, Nielen, Mol, Hacjak, & Veltman, 2005; Nieuwenhuis, Slagter, Alting von Geusau, Heslenfeld, & Holroyd, 2005). Thus, when one is able to determine the outcome of performance, feedback is redundant, and an ERN is generated (Müller, Möller, Rodriguez-Fornells, & Münte, 2005).

The FRN and ERN also tend to have different scalp topographies (e.g., Gentsch, Ullsperger, & Ullsperger, 2009; Müller et al., 2005), as they did in this study, suggesting that at least some neural generators are not shared between the two components (Gehring & Willoughby, 2002; Potts, Martin, Kamp, & Donchin, 2011). Indeed, in our study we found that the ERN and FRN were not correlated and that the MFN and N2 were only modestly

correlated. Unique neural generators of the FRN—for instance, the posterior cingulate—are involved in the evaluation of these external signals—for example, evaluation of a reward, as indicated by an external stimulus (Müller et al., 2005; Nieuwenhuis, Slagter, et al., 2005). In this way, it is possible that individual differences in performance-monitoring functions unique to the evaluation of externally generated information regarding outcomes are what predict AB magnitude, rather than investment in tasks in general, or performance monitoring more generally.

It is, of course, also possible that our failure to find a relationship between MFN amplitude and AB magnitude is due to, for example, measurement error. However, both AB task performance measures and ERP amplitudes demonstrated either high or very high reliability (internal consistency), as measured by split-half (even vs. odd trials/epochs) correlations. This is consistent with previous evidence that these measures, both the AB (Dale & Arnell, in press) and FRN/ERN amplitude (Segalowitz et al., 2010), have good test–retest reliability and can be considered trait-like measures. The two measures of N2 amplitude in our study, from completely different tasks, also correlated positively and very strongly with each other, and both correlated positively with the MFN. Thus, our measures were all likely reliable measures of individual differences associated with AB magnitude and responses to both performance and feedback. Therefore, the failure to find a relationship between individual differences in MFN amplitude and in AB magnitude is not due to poor, unreliable measures of those individual differences. It is possible, however, that task-based differences in the ERN may mean that our ERN task—a flanker task in which speed was encouraged—did not capture the kind of variability in performance monitoring that might correlate with AB magnitude.

Implications for models of the AB

Our results are consistent with models of the AB, such as the boost-and-bounce (Olivers & Meeter, 2008) and threaded-cognition (Taatgen et al., 2009) models, that posit cognitive control as contributing to the production of the AB. Our results extend these models by suggesting that individual differences in cognitive control can underlie individual differences in the AB, in that those individuals who generally produce larger electrophysiological responses to performance feedback would generally enforce cognitive control more strongly, and thus have larger AB magnitudes. For example, in the boost-and-bounce model of the AB, a "boost" of attention initiated in response to T1 is followed by a cognitive control function, the "bounce," initiated by the presence of the distractor trailing T1 (Olivers & Meeter, 2008). This "bounce" prevents T2 from entering working memory when it is presented shortly after T1, during the "bounce." The threaded-cognition model (Taatgen et al.,

2009) features a similar cognitive control function, a "memory function," that impairs T2 detection. Our results suggest that individuals may differ with respect to the strength of the bounce response or memory function, so that some individuals may exhibit greater cognitive control than others in response to external stimuli that suggest that performance goals are at risk—namely, feedback and the presence of the distractor that trails T1. In this manner, individuals who more strongly enforce this "bounce"/"memory function" will be more likely to miss T2 at short lags, and thus will have larger AB magnitudes than will individuals who are weaker enforcers.

The observed relationships between electrophysiological responses to external performance feedback and the AB is also consistent with previous individual-difference studies showing that a flexible, diffuse, and less-invested processing style predicts smaller AB magnitudes, using measures such as global/local bias (Dale & Arnell, 2010), personality (MacLean & Arnell, 2010), and affect (MacLean et al., 2010), as well as with previous findings showing that performing a simultaneous additional task, such as detecting yells in music, attenuates the AB (Olivers & Nieuwenhuis, 2005). Diffuse and less-invested processing (either via individual differences or task manipulations) may work to decrease performance monitoring and its call for heightened cognitive control. In this manner, the present results are also consistent with the overinvestment hypothesis of Olivers and Nieuwenhuis (2005, 2006), which proposes that the AB results from an overinvestment of unnecessary attentional resources in the RSVP items, including the distractors, such that they strongly compete with target items.

In conclusion, we observed that electrophysiological responses to external performance feedback predicted individual differences in AB magnitude such that greater feedback-related N2 amplitudes, from both an AB task and an unrelated time production task, were associated with larger AB magnitudes, regardless of the valence of the feedback. These results support cognitive control models of the AB, according to which greater feedback responsivity is associated with the stronger enforcement of cognitive control, which in turn leads to larger AB magnitudes. However, we did not find the hypothesized relationship with electrophysiological responses to internally generated feedback (e.g., the MFN/ERN). We suggest, then, that it is possible that variability in the evaluation of externally generated information regarding outcomes is what predicts individual differences in the AB.

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