

# Mindset induction effects on cognitive control: A neurobehavioral investigation



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## ABSTRACT

Messages about how much our abilities can change – or “mindset” messages – affect learning, achievement, and performance interpretations. However, the neurocognitive mechanisms responsible for these effects remain unexplored. To address this gap, we assessed how a mindset induction influenced cognitive control brain activity. Participants were randomly assigned to read that intelligence was either malleable (growth-mindset condition) or immutable (fixed-mindset condition) before completing a reaction-time task while electroencephalogram was recorded. Findings revealed that inducing a growth mindset resulted in enhanced attention to task-relevant stimuli, whereas inducing a fixed mindset enhanced attention to responses. Despite enhanced attention to responses in the fixed mindset group, this attention allocation was unrelated to adaptive performance adjustments. In contrast, the growth mindset induction produced a relatively strong coupling between error-related attention allocation and adaptive post-error performance. These results suggest that growth- and fixed-mindset messages have differential effects on the neural dynamics underlying cognitive control.

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## 1. Introduction

Individuals are exposed to different messages about the nature of abilities on an everyday basis, from the teacher comforting a student (“It’s OK, not everyone can be a math person”), to the parent praising a child (“You really are a smart boy”), to the sports announcer commenting on a player’s skill (“Wow, what a natural!”). These kinds of messages convey implicit assumptions about the malleability of different abilities and may contribute to the attitudes individuals hold about the nature of their abilities – or “mindsets” as they are called in contemporary psychological research. Decades of research suggest that mindsets substantially influence learning, motivation, and achievement (Dweck, 1999, 2006; Gunderson et al., 2013; Mueller & Dweck, 1998; Rattan, Good, & Dweck, 2012). A consistent finding is that a belief in the malleability of self-attributes – a growth mindset – is associated with better performance and perseverance, especially when individuals are faced with challenging tasks. Although the psychological and motivational outcomes associated with mindsets are well understood, researchers know relatively little about the neurocognitive

processes that mediate the impact of mindsets on achievement outcomes. In this study, we examined how standard mindset inductions (messages about abilities) influence cognitive control.

### 1.1. Mindsets/implicit theories

People generally hold one of two beliefs (or “mindsets”)<sup>2</sup> about the malleability of self-attributes such as intelligence; the “growth mindset” construes intelligence as malleable and improvable; the “fixed mindset” understands intelligence as an absolute entity that cannot be changed (Dweck, Chiu, & Hong, 1995). A large body of literature has documented that these different mindsets lead to different attributions, goals, and experiences of performance situations (see Burnette, O’Boyle, VanEpps, Pollack, & Finkel, 2013 for a review). For instance, individuals who hold the growth mindset believe that successful performance is largely driven by effort, whereas fixed-minded individuals believe success is determined mostly by natural ability (Dweck, 2006). These basic assumptions of where ability comes from are thought to bias individuals’ goals for achievement. Whereas growth-minded individuals typically focus on learning to *master* a given task,

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<sup>2</sup> Note that we consider the terms “mindsets” and “implicit theories” as interchangeable. A growth mindset corresponds to an “incremental theory” whereas a fixed mindset corresponds to an “entity theory”.

fixed-minded individuals typically focus more on *doing well* and gaining positive evaluation for their performance (e.g., Dweck & Leggett, 1988). These opposing ideas of ability and performance are often related to notable differences in achievement, especially during challenging task environments. More growth-minded individuals increase their motivational effort to “bounce back” from task setbacks and mistakes; fixed-minded individuals – who interpret their mistakes as proof that they lack ability – disengage from the task at hand and remain stuck (Dweck et al., 1995).

Due to the implicit nature of mindsets – most people do not consciously think about whether abilities can change – they can be manipulated with rather modest interventions. For instance, developmental studies find that different types of praise can foster a fixed mindset if it is directed toward ability (“You’re so smart!”), whereas a growth mindset is encouraged if *effort* is praised (“You worked really hard!”; Gunderson et al., 2013; Mueller & Dweck, 1998; Pomerantz & Kempner, 2013). Academic “mindset interventions” have shown that students who are told that the brain can grow like a muscle and that success depends upon effort and learning obtain higher grades compared to those in control ‘study session’ groups (Blackwell, Trzesniewski, & Dweck, 2007; Dweck, 2008). Finally, mindsets can be induced simply after reading a scientific article that describes intelligence as being immutable and stable (fixed mindset induction) or as being a malleable characteristic that is developed through learning and effort (growth mindset induction; Bergen, 1991; Chiu, Hong, & Dweck, 1997; Hong, Chiu, Dweck, Lin, & Wan, 1999). Studies such as these indicate that subtle mindset messages can have noticeable effects on attitudes and motivation, which may transfer to longer term outcomes like GPA (e.g., Yeager & Dweck, 2013; Yeager & Walton, 2011).

## 1.2. Cognitive control-related brain activity and behavior

Although changes in several observable and behavioral outcomes such as grades and self-reported attitudes have been documented in mindset induction studies, the potentially intervening brain mechanisms that underlie these effects are largely unknown. Cognitive neuroscience methods that reveal the neural and behavioral ‘signatures’ of processes involved in attending to, maintaining, and accomplishing performance goals offer a set of tools to address this gap in understanding (Miller & Cohen, 2001). Accordingly, the current study was designed to integrate mindset research with cognitive neuroscience by delivering brief mindset messages to individuals just before they completed a reaction-time task (i.e., the Eriksen flanker task; Eriksen & Eriksen, 1974) during which event-related brain potentials (ERPs) were recorded. In particular, we focused on how mindset induction influenced four cognitive control related ERPs – the N2, P3, ERN, and Pe.

The N2 is a stimulus-locked ERP that reaches maximal amplitude 200–400 ms following a stimulus at frontocentral electrode sites (Folstein & Van Petten, 2008; van Veen & Carter, 2002). Although the N2 is time-locked to the stimulus, it is often linked with processes associated with *responses*, including response conflict (Yeung, Botvinick, & Cohen, 2004; Yeung & Cohen, 2006; Yeung, Ralph, & Nieuwenhuis, 2007) and response inhibition (Falkenstein, Hoormann, & Hohnsbein, 1999). The N2 amplitude is sensitive to response conflict such that its amplitude is enlarged on trials with incongruent stimuli (e.g., “<<>>”) relative to trials with congruent stimuli (e.g., “>>>”). According to the conflict-monitoring theory, the N2 reflects initial conflict detection between competing response tendencies (Yeung et al., 2004). Following this initial conflict detection process, visual information is subjected to a more

detailed analysis, reflected by the P3.<sup>3</sup> The P3 is a positive ERP component observed at centroparietal and parietal recording sites between 200 and 600 ms post-stimulus. A large body of evidence indicates the P3 is a neural index of attentional, perceptual and memory-updating processes facilitated by motivationally relevant stimuli such as target stimuli in reaction time tasks (Donchin, 1981; Donchin & Coles, 1988; Nieuwenhuis, Aston-Jones, & Cohen, 2005; Nieuwenhuis, De Geus, & Aston-Jones, 2011).

ERPs time-locked to responses offer further insight into response selection and correction processes. Most of the research has examined response-locked ERPs following errors. The error-related negativity (ERN) is a negative ERP reaching peak amplitude within 50–100 ms following an error in simple reaction-time tasks such as the flanker task (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Gehring, Liu, Orr, & Carp, 2012). Like the N2, it is maximal at fronto-central electrode sites, and converging evidence from several areas implicates the anterior cingulate cortex (ACC) as a generator of the ERN (for a review see Gehring et al., 2012). The ERN is thought to reflect the brain’s earliest signal that an error has occurred (Bartholow et al., 2005), such that it registers response conflict between the actual and intended outcome (Yeung et al., 2004), or conveys a reinforcement-learning signal to the ACC (Holroyd & Coles, 2002). The ERN is often observed even when participants are not fully aware they have made a mistake (Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001; see Wessel, 2012 for a review).

Following the ERN, a slower-going positive ERP known as the error positivity (Pe) is observed 200–600 post-response at central and centroparietal electrode sites (Overbeek, Nieuwenhuis, & Ridderinkhof, 2005). The Pe has been shown to arise from a broader range of brain structures including ACC (Herrmann, Römmler, Ehliis, Heidrich, & Fallgatter, 2004; van Veen & Carter, 2002) as well as anterior insular cortex (Ullsperger, Harsay, Wessel, & Ridderinkhof, 2010). In this time window, it is thought that individuals are able to consciously register that they have committed an error, leading to a binary (error vs. correct) evaluation of the response. The Pe has subsequently been associated with conscious error awareness (Endrass, Franke, & Kathmann, 2005; Klein, Ullsperger, & Danielmeier, 2013; Murphy, Robertson, Allen, Hester, & O’Connell, 2012; Nieuwenhuis et al., 2001), evidence accumulation that an error has been made (Steinhauser & Yeung, 2010, 2012), and the motivated allocation of attentional resources to the error (Ridderinkhof, Ramautar, & Wijnen, 2009). A growing number of studies have noted the Pe shares several characteristics with the P3, including scalp distribution, temporal dynamic, and associations with attentional processes (Davies, Segalowitz, Dywan, & Pailing, 2001; Leuthold & Sommer, 1999; O’Connell et al., 2007; Overbeek et al., 2005; Ridderinkhof et al., 2009; Shalgi, Barkan, & Deouell, 2009).

Successful performance also involves adjusting behavior to task conflicts and errors (Danielmeier & Ullsperger, 2011; Forster & Cho, 2014; Schroder & Moser, 2014; Ullsperger, Danielmeier, & Jocham, 2014). Three key adjustments on trials following errors have been identified: post-error slowing (PES; Rabbitt, 1966), post-error accuracy (PEA; Laming, 1979), and post-error reduction of interference (PERI; Ridderinkhof, 2002). PES refers to the slowing of response times on post-error trials compared to post-correct trials, and is commonly thought to reflect a more cautious response strategy (Botvinick, Braver, Barch, Carter, & Cohen, 2001). However, newer studies suggest PES reflects an off-task *orienting* response to infrequent events such as errors in simple tasks (Notebaert et al., 2009).

<sup>3</sup> By “P3”, we refer to the P3b component (Polich, 2007).

PEA refers to the accuracy on trials that follow errors, and is seemingly always adaptive because accuracy is typically desirable (Schroder & Infantolino, 2013). PERI refers to a reduced conflict effect (difference in RT between incongruent and congruent trials) on trials that follow errors, relative to trials that follow corrects, and is thought to reflect increased cognitive control across the task (Danielmeier & Ullsperger, 2011; Ridderinkhof, 2002). Nearly all studies find PES, but not all studies find improved accuracy on trials following errors compared to trials following correct responses (Danielmeier & Ullsperger, 2011). Only a handful of studies have examined PERI, and not every study finds a significant reduction in the conflict effect following errors (e.g., Bombeke, Schoupe, Duthoo, & Notebaert, 2013; Carp & Compton, 2009; Orr, Carp, & Weissman, 2012; Van der Borgh, Braem, & Notebaert, in press). These adjustments have been dissociated in a number of studies, suggesting they reflect at least partially separable processes (Carp & Compton, 2009; Danielmeier & Ullsperger, 2011; Danielmeier, Eichele, Forstmann, Tittgenmeyer, & Ullsperger, 2011).

Adjustments also occur after trials with high response conflict (Gratton, Coles, & Donchin, 1992). Accuracy is enhanced and RT is reduced on incongruent trials that follow incongruent trials, relative to incongruent trials that follow congruent trials (e.g., Botvinick et al., 2001; Clayson & Larson, 2011). This conflict adaptation effect is thought to be driven by cognitive control resources recruited from the first incongruent trial (trial  $n - 1$ ) that carry over to the subsequent incongruent trial (trial  $n$ ; e.g., Botvinick, Cohen, & Carter, 2004). Relatively recent studies have also shown conflict adaptation effects in the N2 (e.g., Clayson & Larson, 2011, 2013; Forster, Carter, Cohen, & Cho, 2011; Freitas, Banai, & Clark, 2009) and P3 (Clayson & Larson, 2011).

Finally, the links between brain and behavior are also important to performance-monitoring integrity (Ullsperger et al., 2014). Findings are mixed in terms of the brain processes that relate to behavioral adjustments. Some studies find post-error behavioral adjustments are correlated with the ERN (e.g., Debener et al., 2005; Gehring et al., 1993; Themanson, Rosen, Pontifex, Hillman, & McAuley, 2012), while others find relationships with the Pe (e.g., Frank, D'Lauro, & Curran, 2007; Hajcak, McDonald, & Simons, 2003; Schroder, Moran, Moser, & Altmann, 2012), and still others find that neither the ERN nor the Pe relate to adjustments (Danielmeier & Ullsperger, 2011). Nonetheless, it is important to evaluate this link between brain and behavior in order to understand the functional significance of these ERPs (e.g., Cavanagh, Bismark, Frank, & Allen, 2011; Cavanagh & Shackman, in press; Schroder & Moser, 2014).

### 1.3. Mindsets and cognitive control

These types of ERPs and performance-monitoring adjustments could be useful in uncovering how mindsets influence information processing. Indeed, previous experimental research suggests that mindsets influence how individuals categorize and process information at a very basic level (e.g., Molden & Dweck, 2006; Molden, Plaks, & Dweck, 2006). Only two studies have used ERPs to examine how individual differences in mindset endorsement relate to information processing. In the first study, Mangels, Butterfield, Lamb, Good, and Dweck (2006) had college students answer difficult general knowledge questions. Following their responses, the students received *performance-relevant* feedback (right vs. wrong), followed by *learning-relevant* feedback (the correct answer to the question). This study found that fixed-minded individuals showed enhanced anterior frontal P3 amplitude to the initial performance feedback, compared to growth-minded individuals. However, fixed-minded individuals subsequently demonstrated a reduced left temporal negativity – an ERP associated with sustained memory and encoding (Butterfield & Mangels, 2003) – to the corrective feedback, suggesting reduced effortful encoding of the correct answer.

Critically, this reduced encoding of the correct answer predicted fixed-minded individuals' poorer performance on a surprise retest of the questions they had initially answered incorrectly. In short, Mangels et al. (2006) revealed that neural mechanisms related to feedback processing and memory encoding might underlie subsequent mindset-related differences in performance.

In a second study, Moser, Schroder, Heeter, Moran, and Lee (2011) used the simpler flanker task (Eriksen & Eriksen, 1974) to assess how mindsets influence performance-related brain activity immediately after responding (i.e., without performance feedback). Moser et al. (2011) found that individuals endorsing more of the fixed mindset showed reduced Pe amplitude, suggesting a reduced allocation of attentional resources to their errors (Ridderinkhof et al., 2009). Similar to the Mangels et al. (2006) study, reduced Pe among fixed-minded individuals was associated with these individuals' poorer performance following errors (PEA). This study revealed differences between mindsets in terms of immediate (within ~250 ms) allocation of resources to errors, which predicted subsequent performance on the next trial. These data indicated that mindsets influence attention allocation immediately following incorrect responses, and not just following performance and learning feedback (e.g., Mangels et al., 2006).

### 1.4. The current study

Although the two ERP studies suggested individual differences in mindsets relate to error- and feedback-related processes, no studies have evaluated how experimentally induced mindsets are related to these neurophysiological processes. The current study was designed to address this gap. Before participants completed the same task as Moser et al. (2011), they read a scientific article describing either that intelligence was malleable and able to change (growth mindset condition) or fixed and immutable (fixed mindset condition), a manipulation that reliably induces mindsets (e.g., Bergen, 1991; Chiu et al., 1997; Hong et al., 1999). We focused on the four ERPs discussed above to evaluate how the induction influenced both stimulus-locked and response-locked information processing as well as indices of behavioral adjustment following errors (PES, PEA, PERI).

Although this is the first study of its kind, we had three predictions. First, we predicted that individuals exposed to the fixed mindset would demonstrate reduced P3 and Pe, given the previous findings of reduced sustained attention to motivationally relevant information (Chiu et al., 1997; Dweck et al., 1995; Hong et al., 1999; Mangels et al., 2006; Moser et al., 2011). Second, because attention allocation in the previous mindset-ERP studies related to poorer post-error performance, we predicted individuals in the Fixed condition would also show attenuated post-error behavioral adjustments (less PES, lower PEA, less PERI). Although we were hesitant about predicting null results, we expected no effect on the amplitude of the ERN would be apparent, given that we found no relation between the ERN and mindset endorsement in our previous study (Moser et al., 2011) and because mindset theory dictates differences in the subsequent evaluation of response outcomes (akin to the Pe), and not the initial detection of the response (ERN; Mangels et al., 2006). Because no previous studies have assessed how mindsets relate to the N2, we were agnostic as to how this component would be influenced by the induction.

## 2. Method

### 2.1. Participants

Participants were 62 undergraduates (69% women; mean age = 19.52 years) from a large Midwestern university who completed the study for course credit. Data from 18 participants were removed due to failure to follow task instruction ( $N = 5$ ), excessive EEG artifacts ( $N = 7$ ), or if they had fewer than six usable errors for

ERP analysis (Olvet & Hajcak, 2009;  $N = 6$ ). This left a final sample of 44 participants ( $M$  age = 19.70 years,  $SD = 1.46$ ; 75% women). Excluded and included participants did not differ on any of the relevant dependent variables, including pre- or post-mindset measures and article attributes ( $t_s(60) < 1.50$ ). All procedures were approved by the local Institutional Review Board.

Prior to the lab visit ( $M = 17$  days,  $SD = 7.19$ ), participants completed an online questionnaire including the theory of intelligence (TOI) scale (Hong et al., 1999) embedded with several other measures unrelated to the goals of the current study (e.g., self-esteem scale, Rosenberg, 1965). The TOI scale contains four fixed-minded statements (e.g., “You have a certain amount of intelligence and you really cannot do much to change it”) and participants rated the extent to which they agreed with each statement on a 6-point Likert-type scale (1 = Strongly Disagree, 6 = Strongly Agree;  $\alpha = .90$ ). TOI items were reverse-scored so that lower scores indicated more endorsement of a fixed mindset and higher scores indicated more endorsement of a growth mindset.

## 2.2. Mindset manipulation

Participants were seated approximately 60 cm in front of a computer monitor and were fitted with a cap for electroencephalogram (EEG) recording (see below for details). Following EEG setup, participants were randomly assigned to read standard mindset induction materials (Bergen, 1991; Chiu et al., 1997; Hong et al., 1999) that consisted of an article either describing new research suggesting that intelligence was primarily determined by genes and was unable to change (Fixed condition;  $n = 22$ ) or research suggesting intelligence was developed from stimulating environments and could be changed with effort and learning (Growth condition;  $n = 22$ ). Participants were asked to read the entire article and to remember the main points for a short memory test following the cognitive task – which served as a manipulation check – described below.

## 2.3. Task

After reading the article, participants completed a letters version of the Eriksen Flankers task (Eriksen & Eriksen, 1974; Moser et al., 2011). Participants were instructed to respond to the center letter (target) of a five-letter string using the mouse. Each five-letter string was either congruent (e.g., M M M M M) or incongruent (e.g., N N M N N). Characters were displayed in a standard white font on a black background and subtended  $1.3^\circ$  of visual angle vertically and  $9.2^\circ$  horizontally. During each trial, flanking letters were presented 35 ms prior to target letter onset, and all five letters remained on the screen for a subsequent 100 ms (total trial time was 135 ms). A fixation cross (+) was presented during the intertrial interval which varied randomly between 1200 and 1700 ms at 100 ms intervals. Performance feedback was not given. The experimental session included 480 trials grouped into 12 blocks of 40 trials during which accuracy and speed were equally emphasized. Letters making up the stimuli changed throughout the task and stimulus-response mappings were reversed prior to every other block to elicit a sufficient number of errors for reliable ERP analysis ( $\geq 6$ ; Olvet & Hajcak, 2009).

Following the flanker task, participants were given an “Article Memory and Comprehension Test” questionnaire. Participants wrote a short summary of the article (which served as a manipulation check) and rated the extent to which they found the article to be: difficult to read, credible, persuasive, and how much they agreed with the article's points using an 8-point Likert-type scale (e.g., Chiu et al., 1997). EEG cap and electrodes were then removed and participants completed a packet of questionnaires that included the TOI scale ( $\alpha = .95$ ).

## 2.4. Psychophysiological recording

Continuous encephalographic (EEG) activity was recorded using the ActiveTwo BioSemi system (BioSemi, Amsterdam, The Netherlands). Recordings were taken from 64 Ag–AgCl electrodes placed in accordance with the 10/20 system. In addition, two electrodes were placed on the left and right mastoids. Electrooculogram (EOG) activity generated by eye movements and blinks was recorded at FP1 and three additional electrodes placed inferior to the left pupil and on the left and right outer canthi (all approximately 1 cm from the pupil). During data acquisition, the Common Mode Sense active electrode and Driven Right Leg passive electrode formed the reference and ground, as per BioSemi's design specifications. All signals were digitized at 512 Hz using ActiView software (BioSemi). Offline analyses were performed using BrainVision Analyzer 2 (BrainProducts, Gilching, Germany). Scalp electrode recordings were re-referenced to the numeric mean of the mastoids and band-pass filtered with cutoffs of 0.1 and 30 Hz (12 dB/oct rolloff). Ocular artifacts were corrected using the method developed by Gratton, Coles, and Donchin (1983). Physiologic artifacts were detected using a computer-based algorithm such that trials in which the following criteria were met were rejected: a voltage step exceeding  $50 \mu\text{V}$  between contiguous sampling points, a voltage difference of more than  $200 \mu\text{V}$  within a trial or a maximum voltage difference less than  $0.5 \mu\text{V}$  within a trial.

ERP data were segmented starting 200 ms prior to the event (stimulus or response) and continued until 800 ms after the event. Each ERP component was evaluated at the recording site where it reached maximal amplitude. The N2 was

quantified as the average activity in the 265–365 ms post-stimulus window at Fz. The P3 was calculated as the average activity in the 300–500 ms post-stimulus window at electrode Pz. The ERN and its correct-trial counterpart (the correct-response negativity, CRN) were quantified as the average voltage in the 0–100 ms post-response time window at FCz. As previous research suggests a functional distinction between an early and a late Pe (Holmes & Pizzagalli, 2008; van Veen & Carter, 2002), the Pe and its correct-trial counterpart were quantified as the average voltage occurring in two successive post-response time windows (150–350 ms and 350–750 ms at electrode sites CPz and Pz, respectively; see also Moser et al., 2011). All ERPs were calculated relative to a 200 ms pre-stimulus/response window baseline correction. Per convention, only correct trials were included in the stimulus-locked ERP averages.

There were no between-group differences in the number of trials used in ERP analyses for response-locked error trials (Fixed:  $M = 26.95$ ,  $SD = 19.87$ , Range: 6–69; Growth:  $M = 23.95$ ,  $SD = 13.29$ , Range: 7–64;  $t(42) = .59$ ,  $p = .56$ ), response-locked correct trials (Fixed:  $M = 393.32$ ,  $SD = 47.40$ , Range: 282–454; Growth:  $M = 378.23$ ,  $SD = 70.21$ , Range: 182–461;  $t(42) = .84$ ,  $p = .41$ ), stimulus-locked congruent trials (Fixed:  $M = 208.50$ ,  $SD = 23.93$ , Range: 146–241; Growth:  $M = 211.27$ ,  $SD = 30.04$ , Range: 123–249;  $t(42) = .34$ ,  $p = .74$ ), or stimulus-locked incongruent trials (Fixed:  $M = 197.09$ ,  $SD = 23.00$ , Range: 140–229; Growth:  $M = 190.09$ ,  $SD = 27.76$ , Range: 98–223;  $t(42) = .91$ ,  $p = .37$ ).

## 3. Results

### 3.1. Mindset manipulation check

Inspection of written responses indicated all participants were able to accurately summarize the manipulation articles. TOI scores were submitted to a 2(Time: Online vs. Post-Induction)  $\times$  2(Mindset-Condition: Fixed vs. Growth) ANOVA. A significant Time  $\times$  Mindset-Condition interaction ( $F(1, 42) = 9.64$ ,  $p < .01$ ,  $\eta_p^2 = .19$ ) indicated that pre-induction TOI scores did not differ between conditions, but post-induction TOI scores were significantly less growth-minded in the Fixed condition (see Table 1). Article ratings (e.g., difficulty) did not differ significantly between experimental groups (Table 1).

### 3.2. Behavioral performance

Behavioral data are presented in Table 2. Consistent with previous work with this task, accuracy across the entire sample was quite high ( $M = 93.15\%$ ,  $SD = 5.25$ ), and did not differ between Mindset-Conditions (See Table 2). A 2(Accuracy: Error vs. Correct)  $\times$  2(Mindset-Condition) ANOVA indicated RTs on error trials were significantly faster than RTs on correct trials across the whole sample ( $F(1, 42) = 225.69$ ,  $p < .001$ ,  $\eta_p^2 = .84$ ), consistent with a speed-accuracy trade off. Neither the main effect of Mindset-Condition ( $F(1, 42) = 0.25$ ,  $p = .62$ ,  $\eta_p^2 = .01$ ) nor the interaction between Accuracy and Mindset-Condition ( $F(1, 42) = 0.64$ ,  $p = .43$ ,  $\eta_p^2 = .02$ ) was significant for RT.

**Post-error adjustments.** A 2(Previous-Trial Accuracy: Post-Error vs. Post-Correct)  $\times$  2(Mindset-Condition) ANOVA on correct RTs showed that correct RTs on post-error trials were longer than correct RTs on post-correct trials ( $F(1, 42) = 94.68$ ,  $p < .001$ ,  $\eta_p^2 = .69$ ), confirming the typical PES effect. The interaction between Previous-Trial Accuracy and Mindset-Condition was not significant ( $F(1, 42) = 1.10$ ,  $p = .30$ ,  $\eta_p^2 = .03$ ).

When accuracy data were submitted to this ANOVA, accuracy following errors ( $M = 94.40\%$ ,  $SD = 5.69$ ) was not significantly higher than accuracy following corrects ( $M = 93.11\%$ ,  $SD = 4.54$ ;  $F(1, 42) = 2.74$ ,  $p = .11$ ,  $\eta_p^2 = .06$ ).<sup>4</sup> This is consistent with other studies that failed to find an accuracy improvement on trials following errors (e.g., Danielmeier & Ullsperger, 2011). Again, there was no interaction between Mindset-Condition and Previous-Trial

<sup>4</sup> One outlier participant in the Fixed condition had a post-error accuracy difference score below 4 standard deviations of the sample average and was removed from post-error accuracy analyses.

**Table 1**  
Mindset induction article attributes.

Measure	Growth		Fixed		Analysis			
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>t</i>	<i>df</i>	<i>p</i>	<i>d</i>
Pre-induction TOI	4.47	1.26	4.63	.97	.47	42	.64	-.15
<b>Post-induction TOI</b>	<b>4.45</b>	<b>1.19</b>	<b>3.48</b>	<b>1.13</b>	<b>2.80</b>	<b>42</b>	<b>.01</b>	<b>.86</b>
Difficulty	1.73	0.51	1.50	.57	1.23	42	.23	.44
Credibility	6.27	1.28	6.14	1.39	.34	42	.74	.10
Persuasiveness	6.32	0.84	6.23	1.34	.27	42	.79	.08
Agreeability	6.14	1.81	5.41	1.47	1.87	42	.07	.45

Note: TOI: Implicit Theory of Intelligence scale scores (range 1–6) were coded such that higher responses indicate higher growth mindset endorsement. A positive *d* indicates that Growth mindset group score was higher than the Fixed mindset condition. The one statistically significant difference at  $p < .05$  is bolded.

Accuracy for post-error accuracy data ( $F(1, 41) = 0.14, p = .72, \eta_p^2 = .003$ ). PES was weakly associated with PEA across the whole sample ( $r(41) = -.25, p = .11$ ). However, greater PES was significantly associated with lower PEA in the Growth condition ( $r(20) = -.50, p = .02$ ) but not in the Fixed condition ( $r(19) = .15, p = .51$ ). That is, slowing down after an error was associated with poorer accuracy following an error in the Growth condition, but was not associated with PEA in the Fixed condition. Fisher's *r*-to-*Z* transformation indicated that these correlations were significantly different ( $Z = 2.13, p = .03$ , two-tailed).

The PERI effect on RTs was evaluated with a 2(Previous-Trial Accuracy: Post-error vs. Post-Correct)  $\times$  2(Current Congruency: Congruent vs. Incongruent) ANOVA. Although there was a Previous-Trial Accuracy  $\times$  Current Congruency interaction ( $F(1, 42) = 4.08, p = .05, \eta_p^2 = .09$ ), follow-up tests revealed congruency effects were actually larger following errors than following corrects (see Table 2;  $t(43) = 2.04, p < .05, d = .43$ ). This finding is in line with some recent studies that found no (Carp & Compton, 2009) or even backwards PERI effects (e.g., Bombeke et al., 2013). The interaction between Previous-Trial Accuracy, Current Congruency, and Mindset-Condition was not significant ( $F(1, 42) = .19, p = .67, \eta_p^2 = .004$ ).

**Conflict adaptation.** Conflict adjustments on RT and accuracy were evaluated using a 2(Previous-Trial Congruency: Congruent vs. Incongruent)  $\times$  2(Current-Trial Congruency: Congruent vs. Incongruent) ANOVA. Conflict adjustment analyses on RT revealed a

main effect of Previous-Trial Congruency ( $F(1, 43) = 4.42, p = .04, \eta_p^2 = .10$ ), such that trials that were congruent were followed by slower RTs compared to trials that were incongruent. A main effect of Current-Trial Congruency ( $F(1, 43) = 260.51, p < .001, \eta_p^2 = .86$ ) established the well-known flanker interference effect of increased RTs on incongruent trials (e.g., Botvinick et al., 2001). Finally, the interaction between Previous-Trial and Current-Trial Congruency ( $F(1, 43) = 14.02, p < .001, \eta_p^2 = .25$ ) indicated a significant conflict adaptation effect for RT (see Table 2). None of the interactions with Mindset-Condition were significant: Previous-Trial Congruency  $\times$  Mindset-Condition ( $F(1, 42) = .04, p = .85, \eta_p^2 = .001$ ); Current-Trial Congruency  $\times$  Mindset-Condition ( $F(1, 42) = .05, p = .83, \eta_p^2 = .001$ ); Current-Trial  $\times$  Previous-Trial Congruency  $\times$  Mindset-Condition ( $F(1, 42) = .03, p = .87, \eta_p^2 = .001$ ). An identical pattern was evident in the accuracy data, including the main effect of Previous-Trial Congruency ( $F(1, 42) = 6.25, p = .016, \eta_p^2 = .13$ ), the main effect of Current-Trial Congruency ( $F(1, 42) = 91.36, p < .001, \eta_p^2 = .69$ ), and the interaction between Previous-Trial and Current-Trial Congruency ( $F(1, 42) = 19.38, p < .001, \eta_p^2 = .32$ ). None of the interactions involving Mindset-Condition were significant: Previous-Trial Congruency  $\times$  Mindset-Condition ( $F(1, 42) = 1.41, p = .24, \eta_p^2 = .03$ ), Current-Trial Congruency  $\times$  Mindset-Condition ( $F(1, 42) = .04, p = .85, \eta_p^2 = .001$ ), Current-Trial  $\times$  Previous-Trial  $\times$  Mindset-Condition ( $F(1, 42) = .18, p = .68, \eta_p^2 = .004$ ). In sum, typical conflict-related behavioral

**Table 2**  
Behavioral performance measures.

Measure	Growth		Fixed		Analysis			
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>t</i>	<i>df</i>	<i>p</i>	<i>d</i>
Error RT	362.94	47.02	364.30	35.31	.11	42	.91	-.03
Correct RT	434.85	38.36	444.26	35.56	.84	42	.40	-.26
Post-error RT	478.73	63.06	498.83	56.58	1.11	42	.27	-.34
Post-correct RT	430.81	38.72	439.35	37.52	.74	42	.46	-.23
Post-error slowing	47.92	39.16	59.48	33.86	1.05	42	.30	-.32
Accuracy (% Correct)	93.72	3.09	93.15	5.25	.45	42	.66	.14
Post-error accuracy	94.96	4.34	93.82	6.90	.65	41	.52	.20
Post-correct accuracy	93.38	3.53	92.82	5.48	.40	41	.59	.13
Post-error accuracy difference	1.58	5.35	1.00	5.00	.37	41	.72	.29
Post-Error Cong RT	449.52	65.28	467.93	52.60	1.03	42	.31	-.32
Post-Error Incong RT	504.21	64.69	526.88	69.68	1.12	42	.27	-.35
Post-Correct Cong RT	411.69	36.96	420.66	39.10	.78	42	.44	-.24
Post-Correct Incong RT	453.90	38.75	460.33	37.24	.56	42	.58	-.17
Post-Error Cong Dif RT	54.68	44.97	58.94	58.78	.27	42	.79	-.08
Post-Correct Cong Dif RT	42.22	11.08	39.68	19.53	.53	42	.60	.16
PERI RT	-12.47	44.08	-19.27	59.04	.43	42	.67	.13
cC RT	407.00	38.05	415.74	39.15	.75	42	.46	-.23
iC RT	415.59	35.89	423.35	40.78	.67	42	.51	-.21
cl RT	453.16	38.87	460.37	39.10	.61	42	.54	-.19
il RT	450.95	41.25	458.07	38.47	.61	42	.55	-.18
cC Accuracy	96.60	2.39	95.78	3.88	.84	42	.40	.26
iC Accuracy	95.55	2.63	95.19	4.12	.34	42	.73	.11
cl Accuracy	90.26	5.43	89.33	8.23	.45	42	.66	.14
il Accuracy	92.18	4.19	92.33	5.96	.10	42	.92	-.03

**Table 3**  
ERP descriptive statistics. Statistically significant differences at  $p < .05$  are bolded.

Measure	Growth		Fixed		Analysis			
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>t</i>	<i>df</i>	<i>p</i>	<i>d</i>
ERN	-5.39	4.97	-5.48	7.12	.05	42	.96	.02
CRN	0.36	4.29	0.69	4.13	.26	42	.80	-.08
ΔERN	-5.75	5.69	-6.17	4.82	.26	42	.80	.08
Avg ERN	-2.51	3.67	-2.39	5.30	.09	42	.93	-.03
Early Pe	5.98	5.27	6.98	7.84	.50	42	.62	-.15
Early Pe Correct	-5.53	3.71	-4.63	3.81	.79	42	.43	.24
Early ΔPe	11.51	5.42	11.61	9.14	.04	42	.97	-.01
Early Avg Pe	0.23	3.67	1.17	4.13	.80	42	.43	-.25
<b>Late Pe</b>	<b>-4.04</b>	<b>3.19</b>	<b>2.66</b>	<b>5.01</b>	<b>2.14</b>	<b>42</b>	<b>.04</b>	<b>-.66</b>
Late Pe Correct	-8.00	3.26	-6.13	3.23	1.90	42	.06	-.59
Late ΔPe	7.96	3.31	8.80	5.48	.62	42	.54	-.19
<b>Late Avg Pe</b>	<b>-4.02</b>	<b>2.77</b>	<b>-1.74</b>	<b>3.20</b>	<b>2.53</b>	<b>42</b>	<b>.02</b>	<b>-.78</b>
Congruent N2	1.22	4.68	1.41	4.99	.13	42	.90	-.04
Incongruent N2	0.32	5.00	0.63	5.14	.21	42	.84	-.06
ΔN2	-0.91	1.37	-0.78	1.18	.32	42	.75	-.1
Avg N2	0.77	4.79	1.02	5.03	.17	42	.87	-.05
Congruent P3	10.26	2.81	8.32	3.60	2.00	42	.052	.25
<b>Incongruent P3</b>	<b>11.04</b>	<b>3.34</b>	<b>8.81</b>	<b>3.93</b>	<b>2.03</b>	<b>42</b>	<b>.049</b>	<b>.63</b>
ΔP3	-0.78	1.17	-0.50	0.89	.89	42	.38	-.28
<b>Avg P3</b>	<b>10.65</b>	<b>3.03</b>	<b>8.56</b>	<b>3.73</b>	<b>2.03</b>	<b>42</b>	<b>.048</b>	<b>.63</b>

adjustments from the flanker task were observed, but were not influenced by the mindset induction procedure.

### 3.3. ERPs

**N2.** Table 3 presents all ERPs between the two Mindset-Conditions. Consistent with past research, the main effect of Congruency ( $F(1, 42) = 19.28, p < .001, \eta_p^2 = .32$ ) indicated N2 at Fz was larger on incongruent trials than on congruent trials. Neither the main effect of Mindset-Condition ( $F(1, 42) = .03, p = .87, \eta_p^2 = .001$ ) nor the interaction between Congruency and Mindset-Condition ( $F(1, 42) = .10, p = .75, \eta_p^2 = .002$ ) approached statistical significance.

All conflict-adaptation effects for the ERPs were evaluated with a 2(Previous Congruency)  $\times$  2 (Current Congruency)  $\times$  2(Mindset-Condition) ANOVA. In terms of conflict adaptation effects on the N2, the main effect of Previous Congruency was not significant ( $F(1, 42) = 0.99, p = .33, \eta_p^2 = .02$ ), but both the main effect of Current Congruency ( $F(1, 42) = 13.29, p = .001, \eta_p^2 = .24$ ) and the interaction between Previous and Current Congruency ( $F(1, 42) = 4.23, p = .046, \eta_p^2 = .09$ ) were significant. Follow-up *t*-tests revealed that the conflict effect on the N2 (the difference between incongruent and congruent N2 amplitudes) was significant on post-congruent trials ( $M$  difference =  $-1.13, SD = 1.77; t(43) = 4.23, p < .001, d = .22$ ), but was smaller and not significant on post-incongruent trials ( $M$  difference =  $-0.47, SD = 1.80; t(43) = 1.73, p = .09, d = .09$ ), consistent with the conflict adaptation effect (e.g., Clayson & Larson, 2011). The difference between post-congruent and post-incongruent conflict effects was significant ( $t(43) = 2.07, p = .044, d = .37$ ), which is consistent with a conflict adaptation effect of the N2 (cf. Clayson & Larson, 2013; Forster et al., 2011). None of the interactions involving Mindset-Condition were significant: Previous Congruency  $\times$  Mindset-Condition ( $F(1, 42) = 0.05, p = .83, \eta_p^2 = .001$ ); Current Congruency  $\times$  Mindset-Condition ( $F(1, 42) = 0.30, p = .59, \eta_p^2 = .01$ ); Previous Congruency  $\times$  Current Congruency  $\times$  Mindset-Condition ( $F(1, 42) = 0.34, p = .56, \eta_p^2 = .01$ ).

**P3.** In the 300–500 ms post-stimulus window, the main effect of Congruency ( $F(1, 42) = 16.49, p < .001, \eta_p^2 = .28$ ) indicated P3 amplitudes were larger on incongruent trials relative to congruent trials. Consistent with our first prediction, the main effect of Mindset-Condition was significant ( $F(1, 42) = 4.13, p < .05, \eta_p^2 = .09$ ), such

that overall P3 amplitude (averaged across congruency) was larger in the Growth condition compared to the Fixed condition (see Table 3 and Fig. 1). The Congruency  $\times$  Mindset-Condition interaction was not significant ( $F(1, 42) = 0.79, p = .38, \eta_p^2 = .02$ ).

In terms of conflict-adaptation effects on the P3, the main effect of Previous Congruency was not significant ( $F(1, 42) = 2.06, p = .16, \eta_p^2 = .05$ ), but the main effect of Current Congruency was significant ( $F(1, 42) = 10.73, p = .002, \eta_p^2 = .20$ ). In contrast to the conflict-adaptation effect observed for the N2, the interaction between Previous and Current Congruency was not significant ( $F(1, 42) = 0.92, p = .34, \eta_p^2 = .02$ ). None of the interactions involving Mindset-Condition and conflict-adaptation effects for the P3 were significant: Previous Congruency  $\times$  Mindset-Condition ( $F(1, 42) = 0.16, p = .70, \eta_p^2 = .004$ ); Current Congruency  $\times$  Mindset-Condition ( $F(1, 42) = 0.28, p = .60, \eta_p^2 = .01$ ); Previous  $\times$  Current Congruency  $\times$  Mindset-Condition ( $F(1, 42) = 0.000001, p = .99, \eta_p^2 = .001$ ).

**ERN.** The main effect of Accuracy ( $F(1, 42) = 56.28, p < .001, \eta_p^2 = .57$ ) in the 0–100 ms post-response window indicated that amplitudes were larger (more negative) on error trials compared to correct trials, consistent with the presence of an ERN. Neither the main effect of Mindset-Condition ( $F(1, 42) = .01, p = .93, \eta_p^2 < .001$ ) nor the Accuracy  $\times$  Mindset-Condition interaction ( $F(1, 42) = .07, p = .80, \eta_p^2 = .002$ ) was significant.<sup>5</sup>

**Pe.** In the 150–350 ms post-response time window, the main effect of Accuracy ( $F(1, 42) = 104.18, p < .001, \eta_p^2 = .71$ ) confirmed the presence of an early Pe; error trials were associated with a greater positivity compared to correct trials. In this time window, neither the main effect of Mindset-Condition ( $F(1, 42) = .002, p = .97, \eta_p^2 < .001$ ) nor the interaction between Accuracy and Mindset-Condition was significant ( $F(1, 42) = .002, p = .97, \eta_p^2 < .001$ ).

In the 350–750 ms time window, the main effect of Accuracy was again significant ( $F(1, 42) = 150.58, p < .001, \eta_p^2 = .78$ ), consistent with a late Pe (see Fig. 1). Importantly, the main effect

<sup>5</sup> Some authors have noted that the ERN begins prior to response onset (at time 0), and that earlier baselines may account for this (e.g., Davies et al., 2001). The ERN results were unchanged when a  $-200$  to  $-100$  ms pre-response baseline was used: Main effect of Accuracy ( $F(1, 42) = 71.69, p < .001, \eta_p^2 = .63$ ), interaction between Mindset-Condition and Accuracy ( $F(1, 42) = .044, p = .84, \eta_p^2 = .001$ ), main effect of Mindset-Condition ( $F(1, 42) = .064, p = .80, \eta_p^2 = .002$ ).

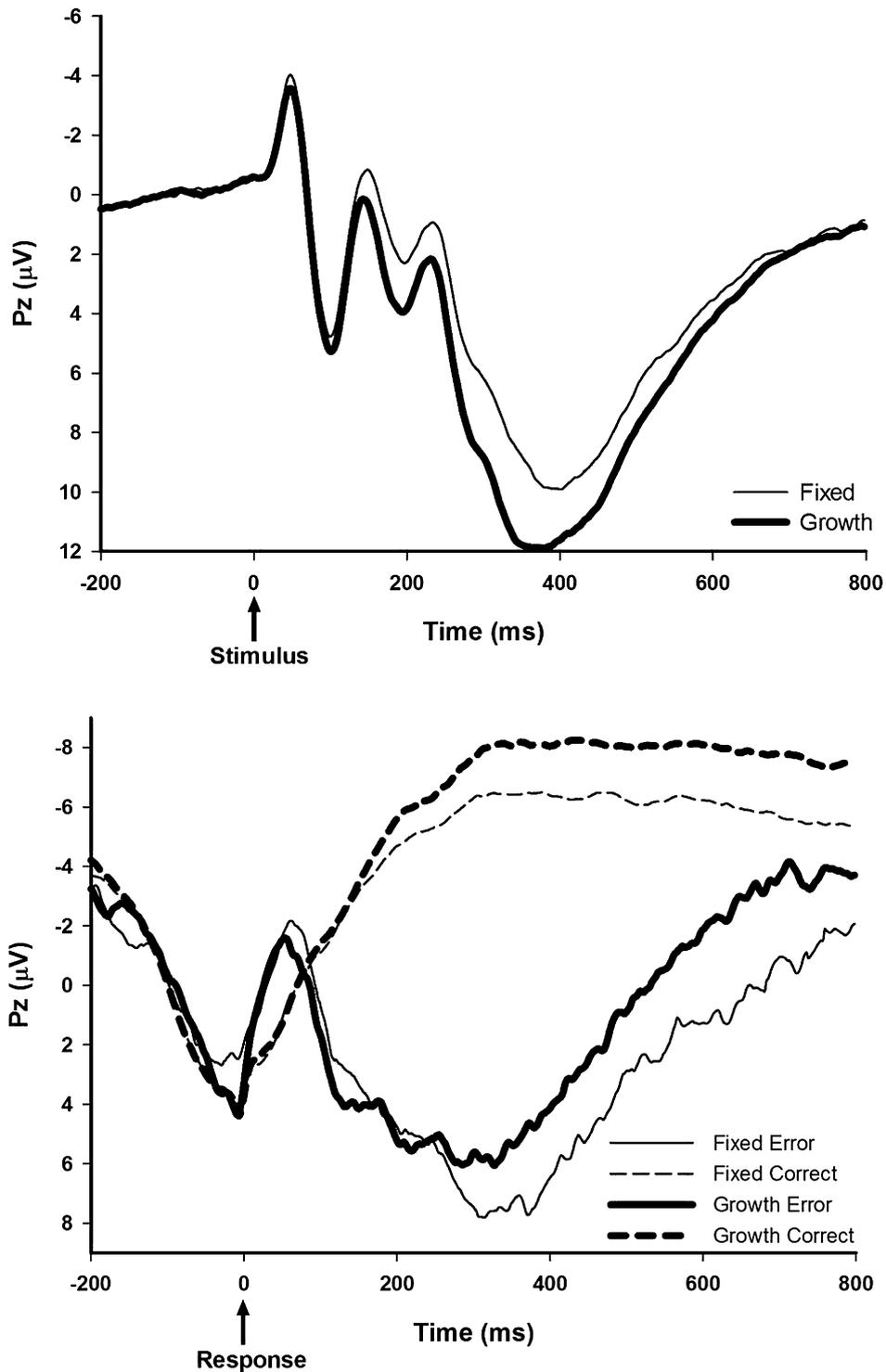
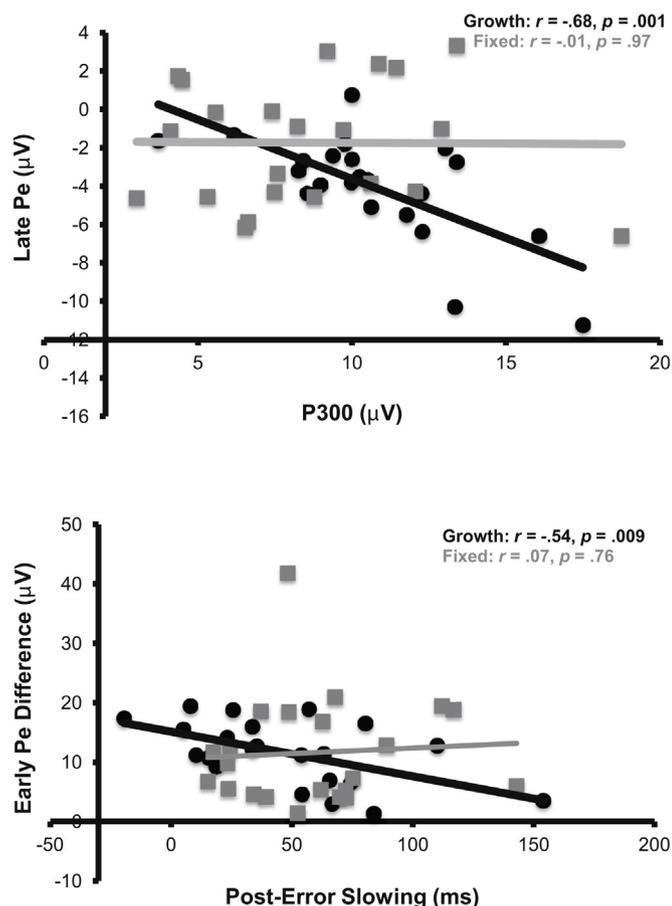


Fig. 1. Grand average ERP waveforms locked to the stimulus (top) and to the response (bottom) at electrode site Pz. Negative amplitude is plotted up.

of Mindset-Condition was also significant ( $F(1, 42)=6.41, p=.02, \eta_p^2=.13$ ), such that the late Pe (averaged across accuracy) was more positive in the Fixed condition than in the Growth condition (see Table 3 and Fig. 1). The Accuracy  $\times$  Mindset-Condition interaction was not significant ( $F(1, 42)=.38, p=.54, \eta_p^2=.01$ ).

The results to this point indicate that individuals in the Growth condition exhibited enhanced P3 amplitudes and reduced late Pe. Because P3 and Pe are thought to reflect similar processes related to the allocation of attention (e.g., Donchin, 1981; Ridderinkhof et al., 2009), we hypothesized that enhanced P3 in the Growth condition

may indicate attention was allocated at the time of the stimulus, precluding the need for further processing following the response. Consistent with this prediction, bivariate correlations indicated that larger P3 amplitudes (averaged across congruency) were significantly related to reduced late Pe (averaged across accuracy) in the Growth condition ( $r(20)=-.68, p=.001$ ). This relationship was absent in the Fixed condition, however ( $r(20)=-.01, p=.97$ ). Fisher's  $r$ -to- $Z$  transformation indicated these correlations were significantly different from each other ( $Z=2.53, p=.01$ , two-tailed; Fig. 2).



**Fig. 2.** Top: scatterplots illustrating the relationship between late Pe at Pz (averaged across accuracy) and P3 at Pz (averaged across congruency) in the two experimental conditions. Bottom: scatterplots illustrating the relationship between early error trial Pe at CPz and post-error reaction time.

### 3.4. Brain–behavior relationships

Finally, we examined whether key brain–behavior relationships differed between the mindset conditions. Multiple comparisons and correlations pose problems in cognitive neuroscience studies (e.g., Curtin & Schulz, 1998; Vul, Harris, Winkielman, & Pashler, 2009; Yarkoni, 2009) as they increase the risk of Type I error (i.e., erroneously finding a correlation below a threshold level of significance). At the same time however, overly conservative alpha restrictions (e.g., Bonferroni) may mask true effects (Type II error). We therefore restricted statistical significance to .01 for these analyses to balance Type I and II error rates. We also restricted our investigations to difference wave amplitudes (error minus correct) in order to reduce the number of correlations. Moreover, researchers have increasingly advocated using effect size estimates and confidence intervals (CIs) to evaluate psychological research findings, as opposed to null hypothesis significance testing and  $p$  values (e.g., Cumming, 2013; Simmons, Nelson, & Simonsohn, 2011). Thus, we focused on effect sizes, and most critically, to the effect size differences between the two conditions for this set of analyses.

The ERN difference wave was not associated with PES (Fixed:  $r(20) = -.19, p = .40$ ; Growth:  $r(20) = .01, p = .95$ ) or with PEA (Fixed:  $r(19) = -.21, p = .35$ ; Growth:  $r(20) = -.33, p = .14$ ). In contrast, whereas there was no relation between the early Pe difference wave and PES in the Fixed condition ( $r(20) = .07, p = .76$ ), the early Pe difference wave was negatively associated with PES in the Growth condition ( $r(20) = -.54, p = .009$ ), such that larger early Pe difference

amplitudes corresponded with less PES.<sup>6</sup> Fisher's  $r$ -to- $Z$  transformation confirmed these correlations were significantly different from each other ( $Z = 2.08, p = .04$ , two-tailed; see Fig. 2). The early Pe difference wave was not associated with PEA in the Fixed condition ( $r(19) = -.03, p = .88$ ), or the Growth condition, although the relationship was stronger in the latter condition ( $r(20) = .37, p < .09$ ) indicating larger early Pe difference was associated with greater PEA. However, these correlations were not significantly different from each other (Fisher's  $r$ -to- $Z$  transformation:  $Z = 1.27, p = .20$ , two-tailed). The late Pe difference wave was not associated with PES (Fixed:  $r(20) = -.16, p = .47$ ; Growth:  $r(20) = .10, p = .67$ ) or with PEA (Fixed:  $r(19) = .06, p = .80$ ; Growth:  $r(20) = .02, p = .92$ ). Finally, the PERI effect on RT was not associated with the ERN difference, early Pe difference, or the late Pe difference waves in either the Fixed condition ( $r_s = -.09, -.18, \text{ and } -.20, p_s = .69, .42, .37$ , respectively) nor in the Growth condition ( $r_s = .07, .13, \text{ and } -.07, p_s = .76, .57, \text{ and } .77$ , respectively).

## 4. Discussion

The current study evaluated the effects of a brief mindset induction on neurocognitive and behavioral correlates of cognitive control. Although previous studies have examined how mindset inductions/interventions influence behavioral outcomes (Blackwell et al., 2007; Chiu et al., 1997; Hong et al., 1999), and how trait mindsets relate to performance-relevant brain activity (Mangels et al., 2006; Moser et al., 2011), no previous study had considered these aspects together. The results indicate that the mindset induction had effects on both stimulus- and response-related aspects of cognitive control. Individuals exposed to the fixed mindset demonstrated reduced P3 amplitudes and enhanced late Pe amplitudes (averaged across accuracy) compared to participants exposed to a growth mindset. No differences were found with respect to the N2 or ERN. Brain–behavior correlations were less strongly coupled in the Fixed condition relative to the Growth condition. We discuss these findings in the context of current theories of mindset and cognitive control.

### 4.1. Mindset induction and conflict monitoring

The ERP differences between the mindset induction groups were specific to the P3 (averaged across congruency) and late Pe (averaged across accuracy), but no differences were found for either the N2 or ERN, putative neural markers of conflict monitoring. This level of specificity is important in that the mindset induction effects were most pronounced 'later on' during information processing in our task and suggest that mindset inductions do not primarily impact conflict-related processes. Indeed, the results are consistent with our earlier study that found that trait mindsets did not relate to the ERN (Moser et al., 2011). The N2 and ERN share similar scalp topography, waveform morphology, and have been incorporated into the conflict monitoring theory (Schroder et al., 2012; van Veen & Carter, 2002, 2006; Yeung et al., 2004, 2007; Yeung & Cohen, 2006). Whereas the N2 is sensitive to conflict influenced primarily by distracting task-irrelevant information, the ERN is most sensitive to processing of the target stimulus (Yeung & Cohen, 2006). We also did not find any evidence that the mindset induction had an effect on neural or behavioral indices of conflict adaptation (e.g., Clayton & Larson, 2013; Forster et al., 2011). In sum, we do not

<sup>6</sup> When we examined the correlation between the early Pe on error trials only with post-error RT, the difference between mindset conditions was also present: Growth:  $r(20) = -.72, p = .0001$ ; Fixed:  $r(20) = .04, p = .87$ ; Fisher's  $r$ -to- $Z$  transform:  $Z = 2.92, p = .0035$ , two-tailed).

think there is strong evidence to suspect that mindset inductions influence conflict-related processes.

#### 4.2. Mindset induction and stimulus/response evaluation

The P3 and Pe have long been considered to reflect similar processes related to stimulus/response evaluation, memory updating, evidence accumulation, and attentional orienting to motivationally significant events (e.g., Davies et al., 2001; Donchin & Coles, 1988; Leuthold & Sommer, 1999; Overbeek et al., 2005; Ridderinkhof et al., 2009; Steinhauser & Yeung, 2010; Ullsperger et al., 2010). It was therefore somewhat unexpected that these ERPs showed contrasting effects in the two mindset induction groups (i.e., reduced P3 but larger Pe in the Fixed mindset-condition). One interpretation of the results is that there is an overarching difference in processing strategies between the two mindset groups. Reduced P3 and enhanced late Pe in the Fixed condition suggests individuals exposed to the fixed mindset may have emphasized processing of the *response* over the processing of the stimulus (e.g., Bartholow et al., 2005; Ridderinkhof et al., 2009). In contrast, enhanced P3 and reduced late Pe in the Growth condition suggests that, on average, individuals exposed to the growth mindset may have prioritized the processing of the *stimulus* over the processing of the response, in terms of the allocation of attentional resources (e.g., Ridderinkhof et al., 2009).

In this way, these findings are entirely consistent with decades of previous studies showing that individuals exposed to the fixed mindset are more concerned with achievement *outcomes* than they are with mastering a given task (e.g., Chiu et al., 1997; Dweck, 1975, 1986, 1999, 2006; Dweck & Leggett, 1988; Elliot & McGregor, 2001; Elliot & Dweck, 1988; Hong et al., 1999). That is, the fixed mindset appears to promote heightened concerns with outcomes (or responses). Importantly, the difference between error and correct response-related processing in this study did not distinguish the mindset groups; the Fixed condition was associated with enhanced *overall* processing of responses in the time range of the late Pe. Indeed, mindsets and their associated goal orientations are known to influence the processing of both failures *and* successes (Diener & Dweck, 1980). The present findings, then, indicate that attention allocation to responses is promoted immediately after exposure to the fixed mindset, relative to the growth mindset.

#### 4.3. Mindset induction and brain-behavior relationships

A critical finding in the current study was that the enhanced attention to responses in the Fixed condition did not confer a behavioral advantage on subsequent trials. That is, there were no brain-behavior correlations in the fixed mindset-condition. In contrast, early Pe difference amplitudes in the Growth condition were associated with faster and more accurate responding after errors (reduced PES and enhanced PEA). Although PES has been considered an adaptive response to errors (Botvinick et al., 2001), recent work suggests its utility is dependent upon the task and context (e.g., Danielmeier & Ullsperger, 2011; Jentsch & Dudschig, 2009; Schroder & Moser, 2014), and it is not always positively related to PEA (e.g., Carp & Compton, 2009; Danielmeier & Ullsperger, 2011; Schroder et al., 2012; Torpey, Hajcak, Kim, Kujawa, & Klein, 2012). In this sample, PES may have reflected off-task orienting processing (Notebaert et al., 2009) because it was negatively related to PEA. Thus, the brain-behavior relationship patterns suggest that early attention allocation to errors was directly associated with efficient post-error behavior in the Growth condition only.

These findings are consistent with Molden and Dweck's (2006) observation that, "Following failure, any self-regulation in which [growth-minded individuals] are engaged is thus more likely to focus on determining how to bring about this [ability]

improvement." (p. 194). In this way, the brain-behavior relationships in the Growth condition converge with our previous study of trait mindsets (Moser et al., 2011). In that study, we found that growth mindset endorsement related to larger Pe difference between error and correct trials, which related to higher PEA. The current results suggest that although a growth mindset induction does not relate to enhanced Pe difference amplitude, it does promote an adaptive brain-behavior connection in a similar fashion. The results may also suggest that exposure to the fixed mindset negates the association between brain activity and adaptive adjustments. Additional research evaluating the temporal dissociation between trait and state mindsets will be important to understand precisely how mindset inductions influence the relation between brain and behavior.

#### 4.4. Limitations and conclusion

There were several limitations in the current study that should be addressed in future research. First, prior to the mindset induction, most of the participants endorsed more of a growth mindset as indicated by their online TOI scores ( $M = 4.55$ ,  $SD = 1.11$ , mode = 5.00). This may have resulted in a ceiling effect in which individuals in the Growth condition could not become more growth-minded, but individuals in the Fixed condition could become more fixed-minded. However, more frequent baseline endorsement of the growth mindset has been noted in prior studies (e.g., Rattan, Savani, Naidu, & Dweck, 2012), and many mindset manipulation studies do not report pre- and post-TOI scores (e.g., Chiu et al., 1997; Hong et al., 1999). Regardless of the pre-existing mindset endorsement, the findings indicate that messages about ability have an influence on cognitive control brain activity in a manner that is consistent with existing theory (Dweck, 1999). Future studies might consider (a) recruiting larger samples, (b) recruiting participants in the middle of the distribution (scores in the 3–4 range on the TOI), or, more ideally, (c) conducting a  $2 \times 2$  design in which extreme fixed- and growth-minded individuals are exposed to the mindset manipulations. Second, we evaluated cognitive control-related brain activity immediately following the mindset induction, and research examining these neural correlates weeks or months after the mindset induction will speak to the long-term consequences of these kinds of manipulations. Indeed, many studies *do* show lasting effects of these 'simple' interventions (for a review, see Yeager & Walton, 2011). Finally, although our sample size is similar to many between-group ERP studies (e.g., Holmes & Pizzagalli, 2008; Olvet & Hajcak, 2012; Pfabigan et al., 2013), it was still quite small, and future studies will need to replicate these effects using larger samples (cf. Yarkoni, 2009). Notably, the effect sizes revealed here ( $d$ s between .63 and .78) are similar to or larger than those in similar studies that induced mindsets with persuasive articles (e.g., Chiu et al., 1997:  $d = .32$ ; Da Fonseca et al., 2008:  $d = .84$ ; Hong et al., 1999:  $d = .70$ ). It should also be noted that a recent meta-analysis found that the effect sizes in studies in which mindsets are experimentally manipulated are significantly larger than those for studies where mindsets are evaluated naturally (Burnette et al., 2013).

These limitations notwithstanding, one important implication from the current study is that brain activity and cognitive control can be altered after reading a short article (2 pages, less than 7 min on average reading time) regarding abilities. The two experimental conditions were associated with different brain responses to both stimuli and responses, and showed different brain-behavior relationships. It has long been known that mindset beliefs and these types of messages influence academic achievement, decision-making, and effortful behaviors (Blackwell et al., 2007; Dweck, 1999). The current findings add to this knowledge base by showing that changes in basic attention and error-processing mechanisms

accompany these manipulations and may underlie how mindset messages influence performance. A critical next step is to link these neural processes with the myriad behavioral outcomes associated with fixed- and growth-mindset messages that have been noted in laboratory and naturalistic settings (e.g., Dweck, 2008; Gunderson et al., 2013; Pomerantz & Kempner, 2013; Yeager & Walton, 2011).

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