

# Transferability of Training Benefits Differs across Neural Events: Evidence from ERPs

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## Abstract

■ Humans can show striking capacity limitations in sensorimotor processing. Fortunately, these limitations can be attenuated with training. However, less fortunately, training benefits often remain limited to trained tasks. Recent behavioral observations suggest that the extent to which training transfers may depend on the specific stage of information processing that is being executed. Training benefits for a task that taps the consolidation of sensory information (sensory encoding) transfer to new stimulus–response mappings, whereas benefits for selecting an appropriate action (decision-making/response selection) remain specific to the trained mappings. Therefore, training may have dissociable influences on the neural events underlying subsequent sensorimotor processing stages. Here, we used EEG to investigate this possibility. In a pretraining baseline session, participants completed two four-alternative-choice response

time tasks, presented both as a single task and as part of a dual task (with another task). The training group completed a further 3,000 training trials on one of the four-alternative-choice tasks. Hence, one task became trained, whereas the other remained untrained. At test, a negative-going component that is sensitive to sensory-encoding demands (N2) showed increased amplitudes and reduced latencies for trained and untrained mappings relative to a no-train control group. In contrast, the onset of the stimulus-locked lateralized readiness potential, a component that reflects the activation of motor plans, was reduced only for tasks that employed trained stimulus–response mappings, relative to untrained stimulus–response mappings and controls. Collectively, these results show that training benefits are dissociable for the brain events that reflect distinct sensorimotor processing stages. ■

## INTRODUCTION

Humans display significant performance costs when perceiving and acting on multiple stimuli concurrently (see Marois & Ivanoff, 2005; Pashler, 1994, for reviews). Such multitasking costs reflect fundamental capacity limitations in information processing and have been observed for the transference of a brief sensory event into a durable representation (sensory encoding; e.g., Raymond, Shapiro, & Arnell, 1992) and for selecting appropriate actions to sensory input (decision-making/response selection; e.g., Telford, 1931); both of which share neural underpinnings (Tombu et al., 2011). Sensory encoding limitations can be assessed using dual-task approaches. Specifically, in the attentional-blink (AB) effect (Raymond et al., 1992), the second of two targets often fails to enter consciousness if it succeeds the first by 200–500 msec in a data-limited, rapid serial visual presentation task requiring unsped responses. Similarly, response selection limitations are revealed by the psychological refractory period (PRP) effect (Telford, 1931), where decreasing the temporal interval between two speeded, data-unlimited sensorimotor tasks cause increases to response time (RT) for the second task (a cost).

Fortunately, multitasking limitations are not immutable. For example, training attenuates response selection costs (e.g., Ruthruff, Pashler, & Klaassen, 2001) at least partly because of an increase in the speed of a central, decisional stage of sensorimotor processing (Dux et al., 2009; Ruthruff, Johnston, & Van Selst, 2001). We (Garner, Tombu, & Dux, 2014) recently showed that training on a data-unlimited, speeded sensorimotor task attenuated RT costs in a PRP paradigm and also reduced AB magnitude, a rare instance of cross-task transfer of training. Furthermore, whereas reductions in response selection costs were specific to the trained stimulus–response (S–R) mappings, benefits for sensory encoding transferred to untrained stimuli. Thus, transfer of training benefits evident at one stage of sensorimotor processing (sensory encoding) was not manifested at a later stage (response selection).

Our previous findings suggest that a single training regimen may have a dissociable impact on neural processes underlying separable sensorimotor operations. However, this has yet to be empirically demonstrated. Moreover, no current evidence exists on the question of whether the transferability of training benefits across tasks is restricted to specific neural mechanisms. For example, the top–down amplification of incoming sensory signals plays an important role in encoding, whereas response

selection requires the efficient routing of stimulus-specific information to the appropriate motor plan (Zylberberg, Fernández Slezak, Roelfsema, Dehaene, & Sigman, 2010; Dux et al., 2009). It is conceivable that these two operations may respond differently to training. Gains in sensory amplification may transfer more readily to new materials than gains obtained by forming specific S–R associations.

Here, we investigate this hypothesis by examining brain activity elicited in response to trained and untrained S–R mappings. We compare performance on highly similar trained and untrained tasks within the training group, so that the untrained task serves as an appropriate active control, overcoming confounds associated with nonmatched control conditions (Boot, Simons, Stothart, & Stutts, 2013; Redick et al., 2013). With this design, we are able to identify which brain changes are attributable to task-specific training (by comparing brain activity for trained trials with untrained trials) and those that are attributable to general training effects (by comparing brain activity for trained and untrained trials with the passive control group).

The EEG signal carries highly temporally sensitive information regarding separable sensorimotor processing stages (e.g., Kutas, McCarthy, & Donchin, 1977), making it a highly suitable measure for determining whether dissociable training benefits are present for the neural events underlying distinct sensorimotor processing stages. By identifying the neural events that reflect transferable training benefits (i.e., those that transfer to untrained S–R mappings) and those that reflect task-specific training benefits, this study will provide insights into the neural mechanisms that can drive generalizable performance enhancements from cognitive training, a topic of intense interest for both researchers and the general public (see Owen et al., 2010).

If training has a dissociable influence on the neural events underlying task performance (i.e., the neural events associated with sensory encoding and response selection), and if this manifests itself in terms of whether training benefits transfer, then associated events/components in the EEG signal should display dissociable transferability to untrained material for sensory encoding and response selection. Sensory encoding in the AB paradigm has been associated with at least two distinct EEG signatures. First, failure to report the second target in the AB paradigm is associated with increased pretrial alpha event-related desynchronization (ERD; MacLean & Arnell, 2011) and is assumed to reflect attentional overinvestment, a posited cause of the AB (Olivers & Nieuwenhuis, 2006). Second, the earliest ERP component to show sensitivity to whether the second target is detected occurs around 270 msec into T2 target processing (Sergent, Baillet, & Dehaene, 2005), where the N2 component shows attenuated amplitudes for missed relative to detected targets (see Vogel, Luck, & Shapiro, 1998, for evidence of similar changes occurring for the subsequent P3 component). This is around the time that sensory information

is assumed to be translated into a durable representation coded within a capacity-limited frontoparietal network (e.g., Sergent et al., 2005), rendering limited processors unavailable to engage in sufficient processing of the second target before its representation is interrupted by subsequent distractors (e.g., Hommel et al., 2006; Chun & Potter, 1995). Therefore, there are at least two possible mechanisms via which training may result in sensory encoding benefits that transfer across S–R mappings. Training may influence anticipatory attention for all tasks, which would be reflected in changes to pretrial alpha ERD for both trained and untrained tasks, relative to a no-train control group. Alternatively (and not mutually exclusively), if training influences the translation of sensory information into a durable representation, then changes to events occurring around 270 msec into task processing (i.e., changes to the N2 component) should be observed for both trained S–R mappings and untrained S–R mappings, relative to a no-train control group.

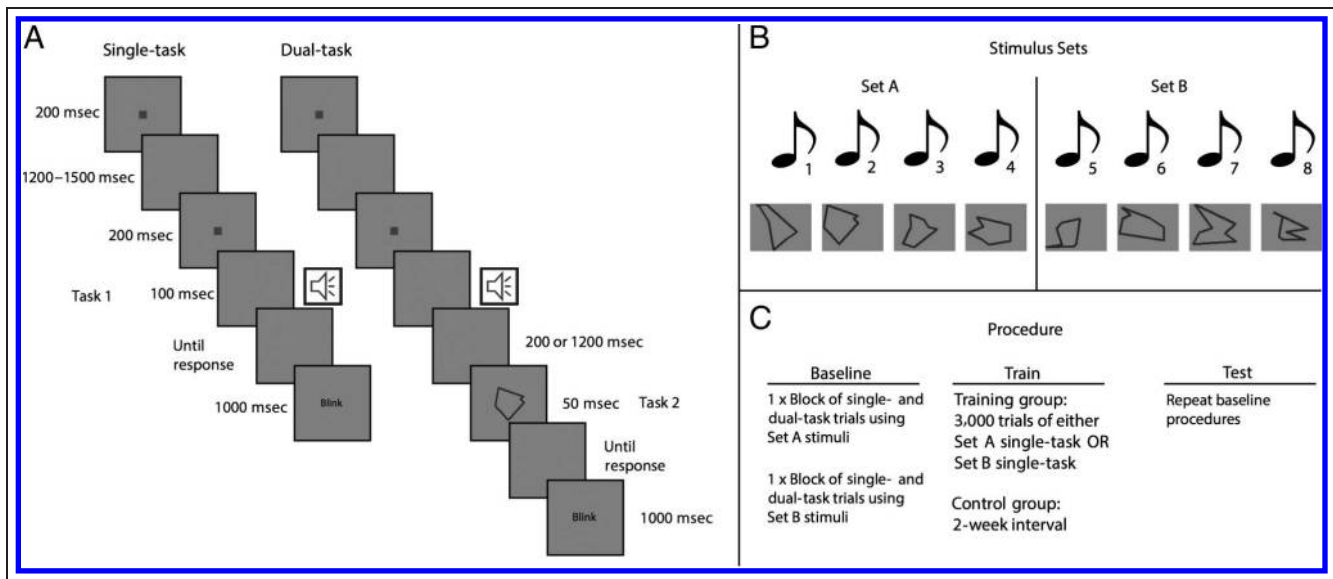
In contrast, performance costs in the PRP paradigm have been associated with the onset of the stimulus-locked lateralized readiness potential (s-LRP; Osman & Moore, 1993). The onset of this component reflects the point at which motor plans have been activated (Coles, 1989) and therefore represents the point at which decisional processes are complete. If training benefits for S–R decisions are task specific, then this should be reflected by changes to the onset of the s-LRP component for trained S–R mappings only, relative to untrained mappings and a no-train control group.

Therefore, the aim of the study was to investigate whether sensorimotor training on single-task S–R mappings has distinct effects on EEG signals related to anticipatory attention, sensory encoding, and response selection. In particular, we were interested in whether training benefits could transfer to untrained S–R mappings. Given that single-task practice results in performance benefits for both the AB and PRP (Garner et al., 2014), we focused on training-related changes to the EEG signal elicited by single-task trials. In addition, we examined the influence of training on multitasking costs as revealed by dual-task trials administered in a PRP paradigm (see Figure 1).

## METHODS

### Overview

First, in a baseline session, participants were tested on two blocks of trials while EEG recordings were taken. Each block consisted of a unique set of single-task trials, involving a four-alternative-choice auditory-manual task, intermixed with a unique set of dual-task trials, where a four-alternative-choice visual-manual task was presented subsequent to the auditory-manual task. Second, after this baseline session, participants in the training group practiced one of the single tasks over a 2-week period



**Figure 1.** Procedure for single- and dual-task trials used in the baseline and test sessions (A). Training trials followed the same procedure as single-task trials, except that no instructions regarding blinking were presented. Two stimulus sets were created (B) to form two blocks of single- and dual-task trials. Participants in the training group then trained on single-task trials using either Set A or Set B; hence, at test, one set contained trained S–R mappings, whereas the other contained untrained S–R mappings (C).

(3,000 trials). Therefore, one of the single tasks became the trained S–R mappings, and the other became the untrained S–R mappings. Thus, the untrained task served as an active control task for the training group. Finally, in the posttraining test session, the baseline procedures were repeated. The control group attended only the baseline and test sessions, with a 2-week interval between the two. Detailed procedures for the baseline, training, and test sessions are provided below.

## Participants

Participants were recruited if they were aged 18 years or over and right-handed; had normal or corrected-to-normal vision; and had no history of psychiatric or neurological illness, injury, or disorder. Participants were randomly allocated to either the training group or the control group. The training and control groups were matched for age, years of education, and sex (see Table 1). All participants received 10 AUD per hour for participation. In addition, those assigned to the training group were also able to earn bonus dollars for accuracy and for beating RT deadlines (~15 AUD per participant) during training sessions. All procedures were cleared in accordance with the ethical re-

**Table 1.** Participant Demographics

Group	<i>N</i>	Age	Sex	Years of Education
Training	12	24.1 (3.6)	4 M	16.8 (2.3)
Control	12	24.7 (2.8)	5 M	17.5 (3.2)

Total number of participants (*N*), mean age, sex, and mean total number years of education for the training and control groups. Standard deviations are presented in parentheses. M = male.

view processes of the University of Queensland Human Research ethics committee and within the guidelines of the National Statement on Ethical Conduct in Human Research.

Training-related improvements on speeded sensorimotor tasks are typically large (e.g., Pashler & Bayliss, 1991); therefore, we estimated that such changes should be represented by a medium-to-large population effect size ( $f = 0.3$ ; Cohen, 1988). A power calculation revealed that, to achieve 80% power to detect a significant 2 (Group)  $\times$  2 (Session) interaction, 24 participants would be required (Faul, Erdfelder, Lang, & Buchner, 2007). Twenty-seven participants (17 women, 10 men) were recruited for the study; however, three were excluded during the baseline session because they did not meet the criteria required to move from the practice trials to the experimental trials (see below).

## Materials

All tasks were programmed in Matlab R2009b (MathWorks, Natick, MA) using the Psychophysics Toolbox 3.0.9 extension (Brainard, 1997; Pelli, 1997). Baseline and test sessions were carried out on a Pentium IV 3-GHz desktop computer and presented on a 21-in. CRT monitor (NEC Accusync 120, Tokyo, Japan), and sounds were presented using a Focusrite Saffire 6 USB soundcard. Training was conducted with a 21-in. Sony Trinitron CRT monitor and a Macintosh 2.5-GHz minicomputer.

Eight complex tones were taken from those used previously by Dux, Ivanoff, Asplund, and Marois (2006) and were trimmed from 200 to 100 msec in duration. Eight shapes (see Figure 1B) were randomly generated using

Matlab R2009b. All visual stimuli were presented at approximately 1° of visual angle.

### **Baseline and Test (EEG) Session Tasks**

The two experimental blocks each consisted of 100 single-task trials and 200 dual-task trials. As four tones and four shapes were required for each of the two experimental blocks, two stimulus sets were used (referred to as Set A and Set B; see Figure 1B). Presentation of single- and dual-task trials was randomized within each block. For single-task trials, participants were presented with a single tone and were required to select the corresponding manual response (on a standard QWERTY keyboard). Participants were required to respond with their left and right index and middle fingers, using the keys C, V, B, or N for Set A tones or the keys G, H, J, or K for Set B tones. Tones were pseudorandomized with the constraint that each tone appeared an equal number of times for each trial type and that no tone could be presented more than three times in succession. For dual-task trials, tones were followed by one of the four shapes after either a short 200-msec (100 trials) or long 1200-msec (100 trials) SOA. Shape presentation was pseudorandomized using the same constraints that were applied to tone selection. Participants were required to select the manual response corresponding to the shape as quickly and accurately as possible, once the tone task had been completed. The response keys for the Set A and Set B shape tasks were chosen so that they sat adjacent to the left and right of the four keys used to respond to the corresponding Set A or Set B tones. Thus, participants were required to use their left and right ring and pinky fingers, using the keys Z, X, M, or , < for Set A shapes or the keys D, F, L, or ;: for Set B shapes. Thus, participants were encouraged to limit their blinking to the 1-sec interval at the end of each trial where a “blink” message was presented centrally on the screen.

Before the experimental trials, participants practiced responding to all the stimuli from Set A until 18 of 20 correct tone responses and 18 of 20 correct shape responses had been achieved. Although the shape task was never presented in isolation in the experimental blocks, it was presented alone in the practice phase so that participants could learn the S–R mappings. Participants then practiced dual-task trials (using Set A stimuli) until 18 of 20 correct trials were achieved. This procedure was then repeated for Set B stimuli. Three participants failed to achieve 18 of 20 after six iterations of 20 trials and were not included in any further experimental procedures.

Each experimental trial began with a dark gray fixation dot presented on a light gray background. After 200 msec, the fixation dot was replaced by a blank interval for a random duration lasting between 1250 and 1750 msec. The fixation dot then reappeared for a further 200 msec before tone presentation (tone duration = 100 msec). For dual-task trials, the fixation dot remained on screen for

the duration of the SOA; after which, the shape was presented for 50 msec before being replaced with the fixation dot that remained on screen until participants responded to both the tone and the shape. Once a given trial had been completed, the instruction “blink” appeared for 1000 msec (Figure 1A). Importantly, participants were unable to tell, before the appearance of the shape task, whether the trial would be a single-task trial or a dual-task trial.

To make comparisons between the training and control groups for trained and untrained S–R mappings, tasks performed by the control group were matched according to which S–R mappings the equivalent training group participant had trained on. For example, if the first participant of the training group had trained on S–R mappings using the tones from stimulus Set A (see Figure 1B and C), then performance of the first participant of the control group on the same S–R mappings for Set A tones was used for the control comparison for trained items. In turn, as the untrained S–R mappings for the first participant of the training group used stimulus Set B, then the first participant of the control group’s performance on Set B trials was included for the control comparison for untrained items. This selection procedure was completed for every participant in the training group and the control homologue.

### **Training Session Tasks**

The goal of the training procedures was to improve RT performance on the S–R mappings that had been allocated as the trained trials. In total, participants completed 3,000 training trials, administered in six sessions containing 500 trials each, with no more than one session occurring on any given day and with a mean separation of 1.67 days. Training trials were presented using the same procedure as that for the single-task trials in the baseline and test sessions, with the exception that participants did not receive instruction to blink at the end of each trial. Each tone was presented 125 times within a session, with the constraint that no tone could be presented for more than three subsequent trials. The training procedures were run according to protocols used in previous training studies (Garner et al., 2014; Dux et al., 2009). The first session began with an overview of the training program, whereas the remaining sessions began with visual feedback (in the form of a line graph) of mean RTs achieved over the previous training blocks. To encourage quick and accurate responses, participants were provided with performance feedback at the end of every 250 trials. If participants scored over 90% correct and met their RT target on over 90% of the trials, a bonus dollar was awarded. If participants maintained that accuracy and met their RT target on over 95% of trials, a new RT target was calculated, and a further two bonus dollars were awarded (\$3 in total). RT targets were derived using the mean and standard deviation of the previous block’s



RT. The 75th percentile was calculated and employed as the new RT target. Along with the performance feedback presented at the end of each block, the total number of dollars awarded for that block, the total number of dollars earned overall, and the RT target for the next block were also presented.

### EEG Recording and Analysis

EEG analysis focused upon single-task trials. Continuous EEG was acquired during the baseline and test sessions using the Biosemi ActiveTwo electrode system (Amsterdam, The Netherlands) from 64 scalp electrodes, digitized at a sample rate of 1024 Hz with 24-bit analog–digital conversion. A fifth-order sinc filter with a  $-3$ -dB point at 204.8 Hz was applied during analog–digital conversion to prevent aliasing. Vertical eye movements were recorded with two vertical EOG electrodes placed above and below the left eye, whereas electrodes at the outer canthus of each eye recorded horizontal movements. Data were analyzed in Matlab (R2012b) using the EEGLab toolbox v11.0.3.1b (Delorme & Makeig, 2004) with the ERPLab plugin v.3.0.2.1 (Lopez-Calderon & Luck, 2014). The data were rereferenced off-line to the average of the mastoids. Trials with incorrect behavioral responses were not included in the analyses. All electrode channels were submitted to an artifact criterion of  $\pm 100$   $\mu$ V to reject trials with excessive EOG or other noise transients. After exclusion of incorrect trials and subsequent artifact rejection, for the trained trials, 85% of the trials remained for the training group at the baseline session and 86% for the test session. For the control comparisons, 88% of the trials remained for the baseline session and 91% for the test session. For untrained trials, 85% of the trials remained for the training group at the baseline session and 88% remained for the test session. For the control comparisons, 88% of the trials remained from the baseline session and 87% remained from the test session.

### ERD Analysis

In accordance with previous research investigating the influence of pretrial attenuations of alpha power (ERD) on sensory encoding (MacLean & Arnell, 2011), the analysis was confined to the higher bandwidth of 10–12 Hz. This higher alpha range has been shown to represent task-specific processing (e.g., stimulus identification and stimulus processing), whereas the lower bandwidth of 8–10 Hz has been associated with general alertness and nonspecific factors (Klimesch, Pfurtscheller, & Schimke, 1992). As previous investigations have implicated differences between sensory encoding and the alpha power signal collected over frontal and parietal regions (MacLean & Arnell, 2011), EEG data were selected from a frontal region (taking six electrodes centered on Fz) and a parietal region (taking six electrodes centered on Pz). These data were epoched locked to the onset of the fixation dot that sig-

naled the beginning of the pretrial interval. The epochs lasted from  $-250$  to 875 msec relative to fixation onset. Calculation of alpha ERD followed the bandpass method (see Pfurtscheller & Lopes da Silva, 1999). First, epoched data were bandpass filtered (finite impulse response length of 104 msec) with a low-pass half-amplitude cutoff of 12 Hz and a high-pass half-amplitude cutoff of 10 Hz (applying the bandpass filter to the continuous data before epoching yielded the same pattern of alpha ERD results; therefore, filtering epochs did not distort the results in any way). Second, the amplitude of the filtered EEG was then squared to provide an estimate of power. To increase statistical power, the estimate was then segmented into  $7 \times 125$  msec intervals (spanning from fixation onset to 875 msec) by taking the mean power within that interval. The mean power estimate calculated from  $-250$  msec to fixation onset served as the baseline. ERD was then computed as the percent power change between the baseline period and each 125-msec interval.

### ERP Preprocessing

For the stimulus-locked analysis of the N2 component, data were binned into epochs from  $-500$  to 600 msec relative to stimulus onset. Epoched data were filtered using a second-order zero-phase shift infinite impulse response (IIR) filter with a roll-off of 12 dB/octave. A low-pass filter with a half-amplitude cutoff at 30 Hz and a high-pass filter with a half-amplitude cutoff at 0.01 Hz were applied (filtering continuous data before epoching yielded the same pattern of N2 results; therefore, filtering epoched data did not distort the N2 findings). To correct slow drifts that were present in the data, a linear detrend analysis was performed (where the best straight-line fit was removed from the epoch using the Matlab detrend function [R2012b]). Epoched data were subsequently normalized using a baseline from  $-500$  to  $-200$  msec relative to stimulus onset. This baseline period was optimal in part because of an N1 component triggered by the fixation dot (this fixation-induced component did not show any influence of training; see Figure 8). The N2 component structure was then confirmed by visual inspection of grand-averaged waveforms and associated scalp maps from the baseline session (see Figure 5). Electrodes were selected by taking the six that were centered under the peak of the grand-averaged waveform (C1, Cz, CP1, CPz, P1, and Pz; Figure 5C).

As the s-LRP continues well after the response, where it is susceptible to contamination from proprioceptive feedback activity, data were binned from  $-200$  msec relative to stimulus onset to the 70th RT percentile for each group in each condition. This value ensured that a minimum of 70% of the trials would be retained for analysis for each participant (on average, 96% [ $SD = 5\%$ ]) of trials included for each participant). Epoched data were low-pass (30 Hz) and high-pass (0.01 Hz) filtered using the IIR filter defined above (applying the filters to the continuous data

before epoching yielded the same pattern of s-LRP results; therefore, filtering epoched data did not distort the s-LRP findings). The epochs were then baseline corrected over the period from  $-200$  to  $0$  msec relative to stimulus onset. This baseline period was optimal as the LRP subtraction procedure removed any activity because of components evoked by the fixation onset. s-LRPs were obtained from sites C3 and C4 using the formula  $([C3(t) - C4(t) \text{ right hand}] - [C3(t) - C4(t) \text{ left hand}])$ ; de Jong, Wierda, Mulder, & Mulder, 1988).

## ERP Measurement

Measurement windows for amplitude and latency of the N2 component were centered on the peak of the observed waveform from the baseline session grand average and subsequently applied to individual subject data. Single-task trials elicited a negative shift over central-parietal sites with an onset of  $\sim 210$  msec, a peak at  $280$  msec, and an offset of  $\sim 390$  msec; this was identified as the N2 component. Amplitude measures were calculated by taking the mean voltage from  $-50$  to  $50$  msec relative to the peak of the component and were measured as the mean voltage (relative to baseline) in the predefined measurement window.

Determining the timing of ERP components can be difficult because of the low signal-to-noise ratio present in a single participant's ERP signal. Simulation studies have shown that the jackknifing approach (Ulrich & Miller, 2001; Miller, Patterson, & Ulrich, 1998) in conjunction with a fractional area latency measure (Kiesel, Miller, Jolicœur, & Brisson, 2008) increases statistical power for detecting latency differences between conditions while controlling for Type 1 errors. The jackknife procedure entails replacing each participant's average waveform with the subaverage waveform taken from  $n - 1$  participants for each group in each experimental condition. As jackknifed subsample scores artificially reduce the error variances computed in the ANOVA test, corrected  $F$  values are computed as  $F_c = F/(n - 1)^2$ , which compensates exactly for the reduction in error variance (see Ulrich & Miller, 2001, for the mathematical proof of this adjustment). Consequently, for area latency measures, jackknifed estimates of the mean ( $\text{mean}_{\text{jack}}$ ) and error ( $\text{er}_{\text{jack}}$ ) are reported. The fractional area latency technique (Hansen & Hillyard, 1980) defines the latency of the component as the first time point at which a certain percentage of the total area of the component has been reached. Because latency measures can be sensitive to high-frequency noise, a low-pass filter (zero-phase shift IIR filter with a roll-off of 12 dB/octave) with a half-amplitude cutoff of 13.6 Hz was applied before the calculation of the fractional area latency measures (e.g., Kappenman et al., 2012; Luck et al., 2009; Osman & Moore, 1993).

For the N2 component, latency measures were calculated as the point the component reached 50% fractional area (i.e., the halfway point for the area under the com-

**Table 2.** Epochs for s-LRP Measurement (Taken from  $-200$  msec Relative to Stimulus Onset)

Group	Baseline		Test	
	Trained	Untrained	Trained	Untrained
Training	1,206	1,232	777	881
Control	1,069	1,330	867	996

End of the s-LRP measurement windows for each group in each S-R mapping condition (milliseconds). The 20% area latency measure was taken from the area under the s-LRP spanning from stimulus onset to the end of the measurement window. As the s-LRP continues well after the response, where it is susceptible to contamination from proprioceptive feedback activity, data were binned from  $-200$  msec relative to stimulus-onset to the 70th RT percentile for each group in each condition.

ponent). Measurement windows were selected to provide maximal coverage to the jackknifed waveforms and spanned from  $240$  to  $370$  msec. The aim of the s-LRP latency analysis was to identify the point at which central processing had been completed and the activation of motor plans had begun; therefore, onset latency of the s-LRP was of primary interest. As such, onset latency of the s-LRP was defined as the 20% area latency, for the area under the s-LRP occurring between stimulus onset and the end of the epoch. The pattern of results was the same when 50% area latency, and when 20% and 50% fractional peak latency measures were applied to the data. For s-LRP latency measurement windows, see Table 2.

## Statistical Approach

To test for training-related benefits, performance on trained S-R mappings was compared with a control group (no-train group) who did not receive training. To detect transferable training benefits, performance on untrained S-R mappings was compared with the no-train control group. Given that the trained/untrained manipulation did not apply to the no-train control group, it was deemed most appropriate to conduct the analysis separately for trained and untrained tasks. Where a training benefit was observed, trained and untrained S-R mappings were compared within the training group only. This comparison assessed whether accrued benefits were greater for trained relative to untrained tasks.

For all analyses, where data violated assumptions of sphericity, adjusted values using the Greenhouse-Geisser epsilon correction are reported. For latency measures using jackknifed waveforms, the  $F$  ratio adjustment ( $F_c$ ) defined by Ulrich and Miller (2001) was applied. Statistically significant interactions were followed up with post hoc ANOVA tests, and a false discovery rate adjustment (Benjamin & Hochberg, 1995) of  $q < .05$  was applied to control for inflated Type 1 error because of multiple comparisons.

Before behavioral data analysis, RT data were subject to an outlier screening procedure for each participant

in each condition. Trials were excluded if a given RT was either <100 msec or >3 SDs above the mean for that participant in that condition and task. Excluded trials accounted for 2.2% of the data.

## RESULTS

### Single-task RTs

Analysis of single-task RTs showed a task-specific influence of training (see Figure 2). Namely, significant reductions in RT were only found for trained S–R mappings, relative to the control group and relative to the untrained S–R mappings. Performance on untrained S–R mappings was not statistically different to the control group.

To test whether the training group improved to a greater extent than the control group when performing trained trials, RTs from single-task, trained S–R mappings were submitted to a 2 (Group: training vs. control) × 2 (Session: baseline vs. test) mixed ANOVA. A significant Group × Session interaction demonstrated that participants in the training group showed a greater reduction in RTs from baseline to test (baseline mean = 1090 msec, *SD* = 250 msec; test mean = 758 msec, *SD* = 191 msec) relative to the control group (baseline mean = 1064 msec, *SD* = 304 msec; test mean = 937 msec, *SD* = 306 msec;  $F(1, 22) = 9.12$ ,  $MSE = .014$ ,  $p = .006$ ,  $\eta_p^2 = .29$ ; see Figure 2A).

To test for a training benefit on RTs for untrained trials, RTs from single-task, untrained S–R mappings were subject to a 2 (Group: training vs. control) × 2 (Session: baseline vs. test) mixed ANOVA. The Group × Session interaction was not significant ( $p = .68$ ; see Figure 2B), although a significant main effect of Session was found ( $F(1, 22) = 22.58$ ,  $MSE = .021$ ,  $p < .001$ ,  $\eta_p^2 = .51$ ). This indicates that participants in the training group did not decrease RTs for untrained trials (baseline mean = 1088 msec, *SD* = 305 msec; test mean = 870 msec,

*SD* = 262 msec) any more than a control group who received no training (baseline mean = 1126 msec, *SD* = 388 msec; test mean = 943 msec, *SD* = 324 msec).

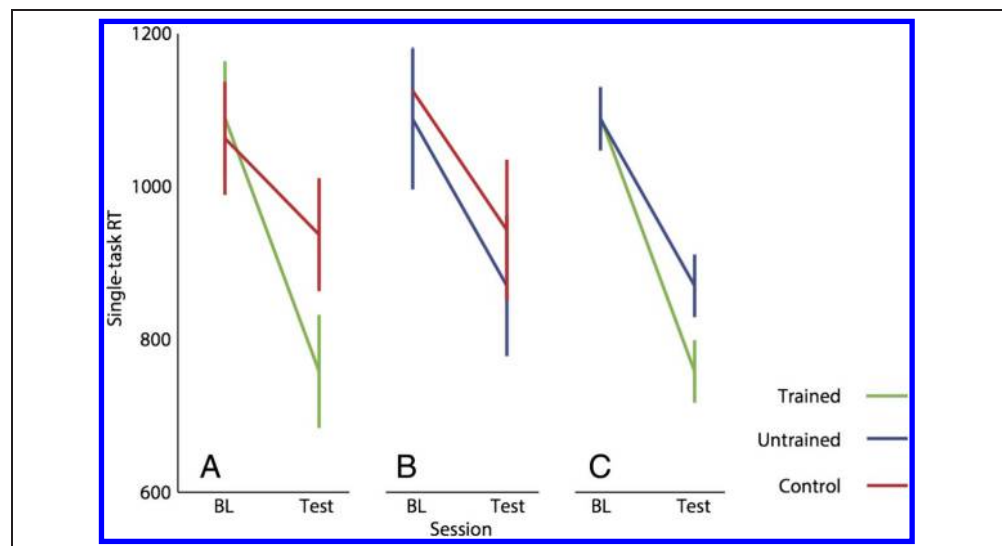
If performance improvements are task specific, then the training group's RTs for trained S–R mappings should be significantly faster than untrained S–R mappings at the test session. A 2 (Session: baseline vs. test) × 2 (S–R mapping condition: trained vs. untrained) repeated-measures ANOVA revealed a significant Session × Condition interaction ( $F(1, 11) = 9.19$ ,  $MSE = .004$ ,  $p = .011$ ,  $\eta_p^2 = .46$ ). Post hoc follow-ups revealed that RTs to trained and untrained trials were not statistically different at the baseline session ( $F(1, 11) = .003$ ,  $MSE = .013$ ,  $p = 1.0$ ,  $\eta_p^2 = .00$ ). However, RTs at the test session were significantly faster for trained S–R mappings relative to untrained S–R mappings ( $F(1, 11) = 10.10$ ,  $MSE = .007$ ,  $p = .02$ ,  $\eta_p^2 = .48$ ; see Figure 2C). These results demonstrate that data-unlimited, sensorimotor training provides an RT benefit that does not transfer to untrained S–R mappings.

### Dual-task RT Costs

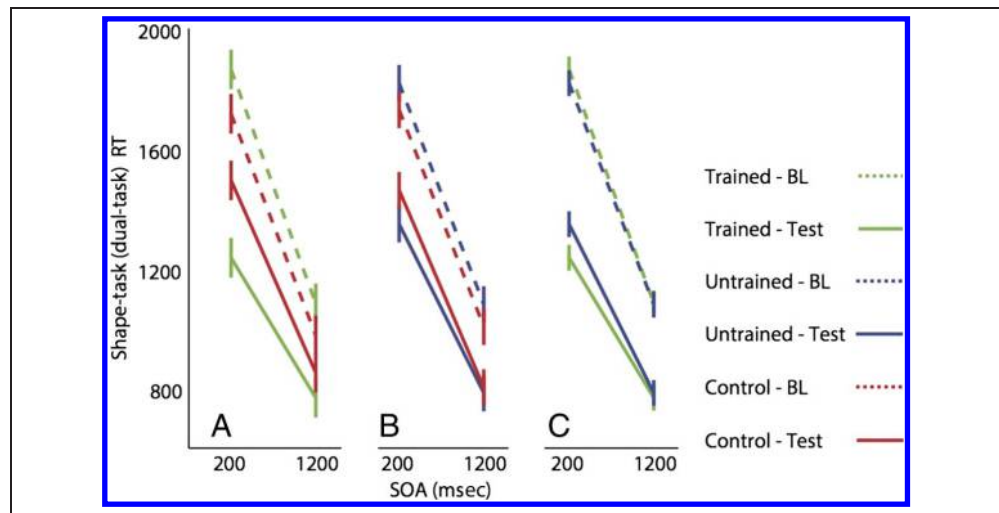
The finding that RT training benefits remain specific to trained tasks supports the hypothesis that response selection training is task specific. If this is the case, then training benefits for a task tapping response selection limitations should also be task specific. This was indeed the case, as task-specific training benefits were observed for the dual-task condition. Specifically, when the second task was performed concurrently with the trained/untrained task, reductions in dual-task RT costs were reduced when that task was paired with trained S–R mappings, relative to when it was paired with untrained S–R mappings and relative to the performance of the no-train control group (see Figure 3).

Dual-task costs were quantified by subtracting the shape task RT (Task 2) at the long SOA from the short SOA RT. To test whether these dual-task costs showed a training benefit (i.e., whether greater reductions in

**Figure 2.** RTs to single-task trials in the baseline (BL) and test sessions as a function of training. RTs for trained S–R mappings were significantly reduced at test relative to a control group (A). This difference was not observed when RTs to untrained S–R mappings were compared with those of the control group (B). RTs to trained S–R mappings were significantly faster at test relative to untrained S–R mappings (C). Error bars represent 95% confidence intervals (calculated using the Subject × Session error term; Masson & Loftus, 2003).



**Figure 3.** RTs to the shape task on dual-task trials at the baseline (BL) and test sessions as a function of training. When the shape task was presented with trained S–R mappings, the RT difference between vshort (200 msec) and long (1200 msec) SOAs was significantly reduced at test relative to a control group (A). This difference was not observed when the shape task was paired with untrained S–R mappings (B). When the shape task was presented with trained S–R mappings, the RT difference between short and long SOAs was significantly reduced at test relative to when the task had been paired with untrained S–R mappings (C). Error bars represent 95% confidence intervals (calculated using the Subject × Session error term; Masson & Loftus, 2003).



dual-task costs were found for the training group relative to the control group), these data were submitted to a 2 (Group: training vs. control) × 2 (Session: baseline vs. test) mixed ANOVA. A significant Group × Session interaction ( $F(1, 22) = 6.30$ ,  $MSE = .022$ ,  $p = .02$ ,  $\eta_p^2 = .22$ ) showed that reductions in dual-task costs were significantly greater for the training group (baseline mean = 779 msec,  $SD = 198$  msec; test mean = 467 msec,  $SD = 186$  msec) than for the control group (baseline mean = 739 msec,  $SD = 199$  msec; test mean = 641 msec,  $SD = 238$  msec).

If training benefits for response selection are task specific, then changes to dual-task costs should not differ between the training group and the control group when the shape task is paired with untrained trials. A 2 (group: training vs. control) × 2 (Session: baseline vs. test) mixed ANOVA confirmed that the two groups were comparable at both the baseline and test sessions (Group × Session interaction,  $p = .18$ ; Figure 3B).

Furthermore, if training benefits for response selection are task specific, then dual-task costs within the training group should be significantly reduced when the shape task is paired with the trained S–R mappings, relative to

when the task is paired with the untrained S–R mappings. To test this, a 2 (session: baseline vs. test) × 2 (S–R mapping condition: trained vs. untrained) repeated-measures ANOVA was conducted on the training group’s dual-task cost data. A significant Session (baseline vs. test) × S–R mapping condition (trained vs. untrained) interaction was observed ( $F(1, 11) = 5.94$ ,  $MSE = .009$ ,  $p = .03$ ,  $\eta_p^2 = .35$ ; Figure 3C). The dual-task cost reduction was larger when the shape task had been paired with trained S–R mappings relative to when the shape task had been paired with untrained S–R mappings (see Figure 3C; untrained baseline mean = 738 msec,  $SD = 129$  msec; untrained test mean = 563 msec,  $SD = 154$  msec). These data show that, to show reductions in dual-task costs over and above that which can be gained by task repetition, task-specific training on S–R mappings is required.

### Accuracy

Analysis of the accuracy data (see Table 3) did not suggest any speed/accuracy trade-offs or any group differences. For the trained S–R mappings, there was suggestion of

**Table 3.** Accuracy across the Groups and Tasks

Group: Session	Tone Task	Trained		Untrained		
		Shape Task		Shape Task		
		Short SOA (200 msec)	Long SOA (1200 msec)	Short SOA (200 msec)	Long SOA (1200 msec)	
Training: baseline	.89 (.08)	.92 (.04)	.92 (.04)	.92 (.07)	.92 (.04)	.94 (.04)
Training: test	.93 (.06)	.94 (.04)	.95 (.02)	.90 (.05)	.92 (.05)	.96 (.03)
Control: baseline	.94 (.04)	.93 (.05)	.95 (.05)	.95 (.04)	.93 (.06)	.95 (.05)
Control: test	.95 (.04)	.95 (.04)	.97 (.03)	.94 (.06)	.95 (.04)	.97 (.02)

Mean proportion correct for single- and dual-task trials for the training and control groups at the baseline and test sessions. Standard deviations are presented in parentheses.

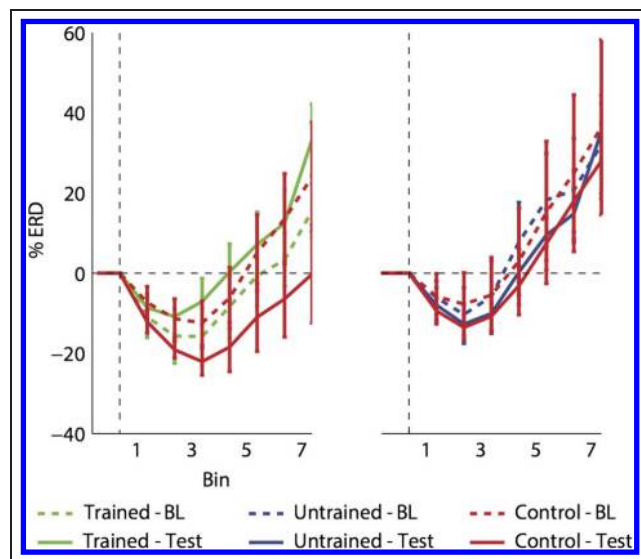


an improvement in accuracy from baseline to test (main effect of Session:  $F(1, 22) = 4.15$ ,  $MSE = .001$ ,  $p = .054$ ,  $\eta_p^2 = .16$ ) that did not interact with Group ( $p = .13$ ). It is therefore unlikely that the training group engaged in a speed/accuracy trade-off. Analysis of the accuracy data for untrained S–R mappings did not reveal any significant main effects or interactions ( $p$ s > .33), once again showing no evidence for speed/accuracy trade-offs over the baseline and test sessions.

### Pretrial Alpha ERD

Previous observations (MacLean & Arnell, 2011) have shown that alpha ERD increases during the pretrial period and that correlations between sensory encoding and alpha ERD occur throughout the pretrial period. The goal of the present pretrial alpha ERD analysis was to test for training-related changes to pretrial alpha ERD. Although alpha ERD increased over the pretrial period, there was no influence of training (see Figure 4).

To test for a task-specific training benefit on pretrial alpha ERD, estimates from correct trials involving trained S–R mappings were subject to a 2 (Group: training vs. control)  $\times$  2 (Region: frontal vs. parietal)  $\times$  6 (Electrode)  $\times$  7 (Interval) mixed ANOVA. In accordance with previous observations (MacLean & Arnell, 2011), there was a significant main effect of Interval ( $F(1.47, 32.24) = 8.59$ ,  $MSE = 39434.85$ ,  $p = .003$ ,  $\eta_p^2 = .28$ ), as alpha ERD increased across the pretrial period (see Figure 4). Importantly, there were no significant main effects or interactions involving the Group or Session factors (all  $p$ s > .06), suggesting that training did not show a task-specific influence on pretrial alpha ERD.



**Figure 4.** Alpha ERD averaged across all electrodes during the pretrial period for trials involving trained and untrained S–R mappings at the baseline (BL) and test sessions. Although there was an increase in alpha ERD over the pretrial period, alpha ERD was not influenced by training. Error bars represent 2.5 SEM.

To test whether a training benefit on pretrial alpha ERD was observed during performance of untrained trials, analysis of ERD estimates from correct trials involving untrained S–R mappings was submitted to an identical mixed ANOVA as above. The same pattern of results was revealed. A significant main effect of Interval ( $F(1.52, 33.35) = 9.54$ ,  $MSE = 65179.29$ ,  $p = .001$ ,  $\eta_p^2 = .30$ ) showed that alpha ERD increased over the pretrial period. Once again, there were no significant main effects or interactions involving either the Session or Group factors (all  $p$ s > .17), suggesting no influence of training on pretrial alpha ERD during performance of untrained trials. Given that training-related changes to pretrial alpha ERD were not found for trained trials or for untrained trials, no further analysis was conducted. Given the absence of training-related change to pretrial alpha ERD, it is unlikely that training benefits sensory encoding via anticipatory attention mechanisms.

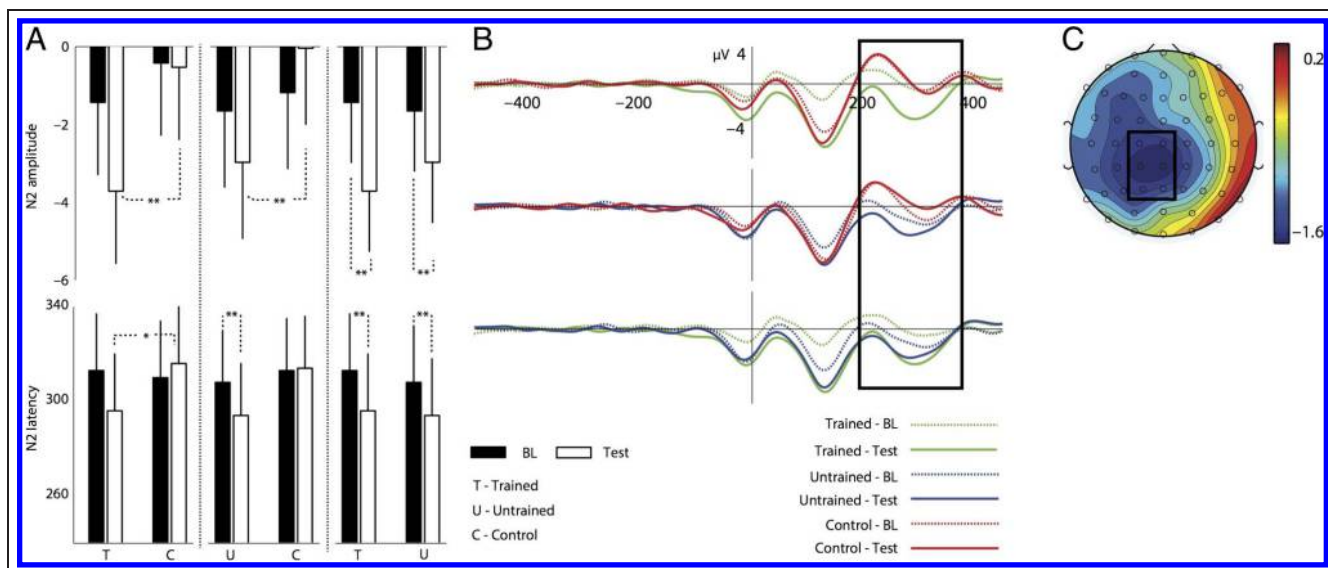
### ERP Results

#### N2 Component

Analysis of the N2 component revealed an influence of training that transferred to untrained mappings. Increased amplitude and reduced latencies of the N2 were found for both trained and untrained trials, relative to no-train controls (see Figure 5).

To assess whether training influences a key ERP component associated with successful sensory encoding, mean N2 amplitude data evoked from trained S–R mappings were submitted to a 2 (Group: training vs. control)  $\times$  2 (Session: baseline vs. test)  $\times$  6 (Electrode) mixed ANOVA. A significant Group  $\times$  Session interaction was found ( $F(1, 22) = 10.50$ ,  $MSE = 8.08$ ,  $p = .004$ ,  $\eta_p^2 = .32$ ). The training group was not significantly different to the control group at the baseline session ( $p = 1.0$ ,  $\eta_p^2 = .05$ ; training group mean =  $-1.43 \mu\text{V}$ ,  $SD = 2.04 \mu\text{V}$ ; control group mean =  $-0.42 \mu\text{V}$ ,  $SD = 2.61 \mu\text{V}$ ). However, at the test session, the training group showed significantly larger N2 amplitudes (mean =  $-3.71 \mu\text{V}$ ,  $SD = 11.95 \mu\text{V}$ ) relative to baseline ( $F(1, 11) = 47.73$ ,  $MSE = 3.92$ ,  $p = .004$ ,  $\eta_p^2 = .81$ ) and relative to the control group (mean =  $-0.53 \mu\text{V}$ ,  $SD = 2.32 \mu\text{V}$ ;  $F(1, 22) = 12.66$ ,  $MSE = 28.76$ ,  $p = .004$ ,  $\eta_p^2 = .37$ ), who did not show a change in N2 amplitude from baseline to test ( $p = 1.0$ ,  $\eta_p^2 = .003$ ). This clearly demonstrates that training increases mean amplitude of the N2 component (see Figure 6 later in this paper).

Untrained S–R mappings were submitted to an identical ANOVA (as described above) to test whether training-related change of the N2 component generalizes to new S–R mappings. The pattern of results was the same as that found for trained S–R mappings. There was a significant Group  $\times$  Session interaction ( $F(1, 22) = 11.13$ ,  $MSE = 9.77$ ,  $p = .003$ ,  $\eta_p^2 = .34$ ). The training group and the control group were not significantly different at the baseline session ( $p = 1.0$ ,  $\eta_p^2 = .01$ ; training group mean =  $-1.65 \mu\text{V}$ ,



**Figure 5.** Stimulus-locked ERP (B) for the baseline (BL) and test sessions as a function of training. A negative-going shift was observed over central-parietal sites (C, boxed region shows selected electrodes) peaking at 280 msec (N2, boxed region in B). Mean amplitude (A, top) was significantly increased at test for trained and untrained S-R mappings relative to the control group. Timing of the component was also significantly faster at test for trained and untrained S-R mappings (A, bottom). Error bars represent 95% confidence intervals (calculated using the Subject  $\times$  Session error term; Masson & Loftus, 2003). \* $p < .02$ , \*\* $p < .005$ .

$SD = 1.60 \mu V$ ; control group mean =  $-1.18 \mu V$ ,  $SD = 2.78 \mu V$ ). However, at test, the training group showed significantly larger N2 amplitude for untrained trials relative to baseline (mean =  $-2.97 \mu V$ ,  $SD = 1.90 \mu V$ ;  $F(1, 11) = 6.91$ ,  $MSE = 9.01$ ,  $p = .05$ ,  $\eta_p^2 = .39$ ) and relative to the control group (mean =  $-0.04 \mu V$ ,  $SD = 1.94 \mu V$ ;  $F(1, 22) = 14.76$ ,  $MSE = 20.94$ ,  $p = .004$ ,  $\eta_p^2 = .40$ ), who did not show a statistically significant decrease in N2 amplitude ( $F(1, 11) = 4.46$ ,  $MSE = 10.53$ ,  $p = .089$ ,  $\eta_p^2 = .29$ ). Therefore, training benefits transferred to untrained S-R mappings.

To test whether training-related changes to the N2 component were comparable for trained and untrained S-R mappings, a 2 (Session)  $\times$  2 (S-R mapping condition: trained vs. untrained)  $\times$  6 (Electrode) repeated-measures ANOVA was conducted on the data from the training group only. A significant main effect of Session was found ( $F(1, 11) = 11.69$ ,  $MSE = 1470.55$ ,  $p < .001$ ,  $\eta_p^2 = .52$ ). However, the Session  $\times$  S-R mapping condition (trained vs. untrained) interaction was not significant ( $p = .44$ ,  $\eta_p^2 = .06$ ), showing that training benefits for the N2 component were comparable across trained and untrained S-R mappings.

To test whether training influences N2 latency on trained trials, N2 latency data from correct trained trials were submitted to a 2 (Group: training vs. control)  $\times$  2 (Session: baseline vs. test)  $\times$  6 (Electrode) mixed ANOVA. A significant Group  $\times$  Session interaction was found ( $F_c(1, 22) = 6.82$ ,  $MSE = 1406.26$ ,  $p = .016$ ,  $\eta_p^2 = .24$ ; see Figure 5A). The training and control groups did not differ significantly from one another at the baseline session (training group mean<sub>jack</sub> = 313 msec,  $er_{jack} = 5.69$  msec; control group mean<sub>jack</sub> = 310 msec,  $er_{jack} = 4.67$  msec;

$p = 1.0$ ,  $\eta_p^2 = .01$ ). In contrast, at test, the training group showed significant reduction in N2 latency ( $F_c(1, 22) = 7.53$ ,  $MSE = 1921.60$ ,  $p = .022$ ,  $\eta_p^2 = .25$ ; training group mean<sub>jack</sub> = 296 msec,  $er_{jack} = 7.18$  msec) relative to the control group (control group mean<sub>jack</sub> = 316 msec,  $er_{jack} = 2.58$  msec). Therefore, training reduces the duration and increases the amplitude of the N2 component.

To investigate whether this training benefit generalizes, N2 latency measures from untrained S-R mappings were submitted to a 2 (Group: training vs. control)  $\times$  2 (Session: baseline vs. test)  $\times$  6 (Electrode) mixed ANOVA. The Group  $\times$  Session interaction was not statistically significant ( $p = .07$ ,  $\eta_p^2 = .14$ ).

To assess whether changes in N2 latency are task specific or are a transferable training benefit, training-group N2 latency estimates from trained and untrained S-R mappings were submitted to a 2 (Session)  $\times$  2 (S-R mapping condition: trained vs. untrained)  $\times$  6 (Electrode) repeated-measures ANOVA. There was a main effect of Session ( $F_c(1, 11) = 11.69$ ,  $MSE = 1470.76$ ,  $p = .006$ ,  $\eta_p^2 = .52$ ), which did not interact with whether the S-R mapping was trained or untrained ( $p = .76$ ,  $\eta_p^2 = .01$ ). This indicates that training caused reductions in N2 latency consistently for both trained and untrained S-R mappings in the training group.

Given the above suggestions that N2 latency changes may reflect a transferable training benefit, we decided to revisit the N2 latency changes for untrained S-R mappings for the training group in relation to the control group's performance. The training group showed a significant reduction in N2 latency from baseline (mean<sub>jack</sub> = 308 msec,  $er_{jack} = 10.84$  msec) to test (mean<sub>jack</sub> = 294 msec,

$er_{jack} = 0$  msec;  $F_c(1, 11) = 12.05$ ,  $MSE = 596.05$ ,  $p = .01$ ,  $\eta_p^2 = .52$ ). This effect was not present in the control group ( $p = 1.0$ ,  $\eta_p^2 = .001$ ; baseline  $mean_{jack} = 313$  msec,  $er_{jack} = 7.74$  msec; test  $mean_{jack} = 314$  msec,  $er_{jack} = 6.17$  msec). Taken together, the evidence suggests that N2 latency was reduced for trained and untrained S–R mappings in the training group, which is indicative of a transferable training benefit.

### s-LRP

It was expected that there would be a task-specific influence of training on the s-LRP, as this component is assumed to index the time taken to activate motor plans (Coles, 1989). In accordance with this hypothesis, the onset latency was reduced for trained trials relative to untrained trials and relative to no-train controls. Furthermore, there was no statistical difference between untrained trials and the no-train control group (see Figure 6).

To test for training benefits for trained trials, a 2 (Group: training vs. control)  $\times$  2 (Session) mixed ANOVA was performed for estimated s-LRP onsets from trained S–R mappings. A significant interaction was observed ( $F_c(1, 22) = 4.55$ ,  $MSE = 12437.23$ ,  $p = .04$ ,  $\eta_p^2 = .17$ ), indicating a larger reduction in s-LRP onset from baseline to test for the training group (baseline  $mean_{jack} = 592$  msec,  $er_{jack} = 180$  msec; test  $mean_{jack} = 347$  msec,  $SD = 101$  msec) relative to the control group (baseline  $mean_{jack} = 556$  msec,  $er_{jack} = 134$  msec; test  $mean_{jack} = 449$  msec,  $er_{jack} = 138$  msec; see Figure 6A).

If training benefits for the s-LRP are task specific, then this interaction should not be observed when s-LRP onsets from untrained S–R mappings are subject to a 2 (Group: training vs. control)  $\times$  2 (Session) mixed

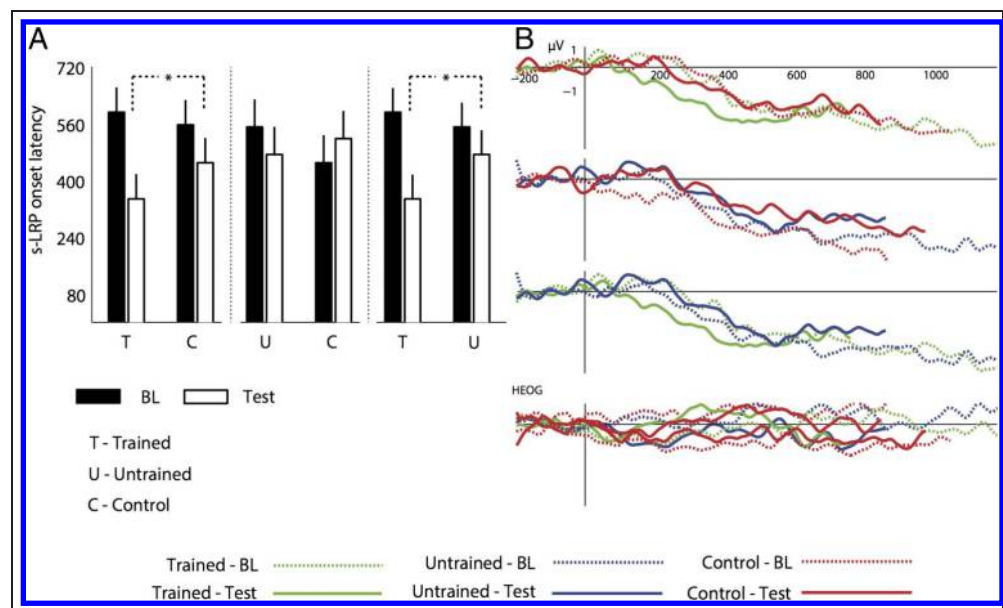
ANOVA. Accordingly, the Group  $\times$  Session interaction was not significant ( $p = .5$ ,  $\eta_p^2 = .02$ ; see Figure 6A).

Furthermore, if training benefits are task specific, then the training group should show a reduced s-LRP onset for trained S–R mappings, relative to untrained S–R mappings, when the training group’s data are analyzed in isolation. When a 2 (Session)  $\times$  2 (S–R mapping condition: trained vs. untrained) repeated-measures ANOVA was performed on the data from the training group, a significant Session  $\times$  Condition interaction ( $F_c(1, 11) = 7.01$ ,  $MSE = 11868.29$ ,  $p = .02$ ,  $\eta_p^2 = .39$ ; see Figure 6A) was observed, indicating a task-specific training benefit. Onset latencies were not significantly different between trained and untrained S–R mappings at the baseline session (untrained baseline  $mean_{jack} = 550$  msec,  $er_{jack} = 137$  msec;  $p = 1.0$ ,  $\eta_p^2 = .05$ ). However, they were significantly reduced for trained S–R mappings relative to untrained S–R mappings at the test session (untrained test  $mean_{jack} = 473$  msec,  $er_{jack} = 83$  msec;  $F(1, 11) = 8.85$ ,  $MSE = 10609.04$ ,  $p = .02$ ,  $\eta_p^2 = .45$ ). These observations demonstrate a task-specific training benefit on s-LRP onset that occurs for trained S–R mappings and does not transfer to untrained S–R mappings.

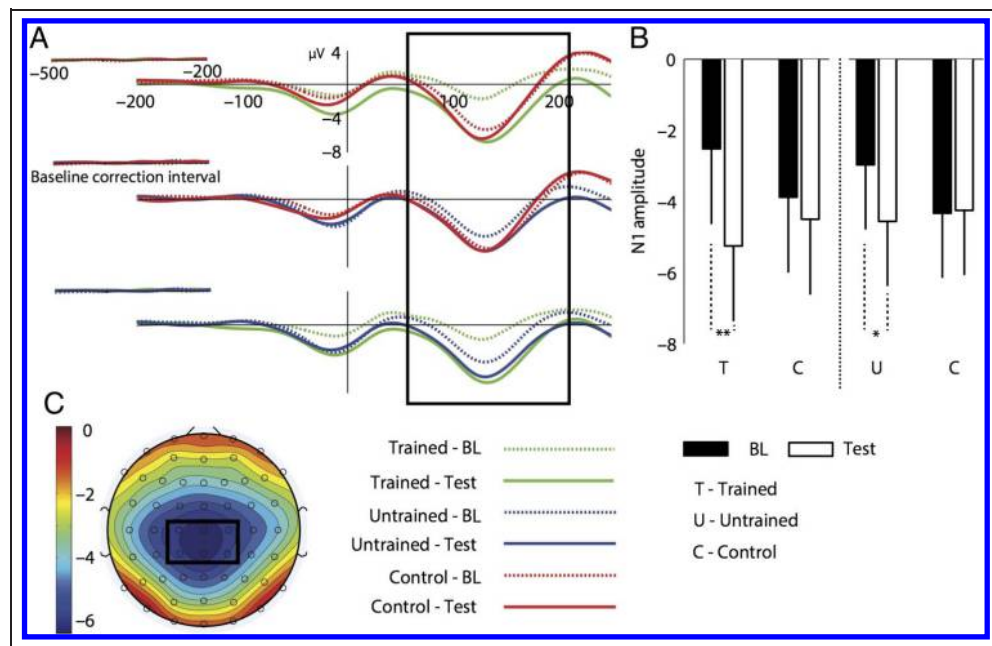
### Additional Analyses

Examination of the ERPs in Figure 5 is suggestive of a training influence on brain activity occurring before N2, that is, for the stimulus-evoked N1 component. Furthermore, the ERPs show that the fixation dot presented before the stimulus also evoked its own N1 component. We examined the influence of training on these components in an exploratory analysis to better constrain the locus of the training effect.

**Figure 6.** s-LRP (B) for the baseline (BL) and test sessions as a function of training. Estimated onset of the s-LRP was significantly faster at test for trained S–R mappings, relative to untrained S–R mappings and the control group (A). Estimated onset of the s-LRP for untrained trials was not significantly different to the control group at test. Error bars represent 95% confidence intervals (calculated using the Subject  $\times$  Session error term; Masson & Loftus, 2003). HEOG = horizontal EOG.  $*p < .05$ .



**Figure 7.** Stimulus-locked ERP (A) for the baseline (BL) and test sessions as a function of training. A negative-going shift was observed over central-parietal sites (C, boxed region shows selected electrodes) peaking at ~120 msec (N1, boxed region in C). Mean amplitude (B, top) was significantly increased (i.e., more negative) at test for trained and untrained S-R mappings relative to the control group. Error bars represent 95% confidence intervals (calculated using the Subject  $\times$  Session error term; Masson & Loftus, 2003). \* $p < .02$ , \*\* $p < .001$ .



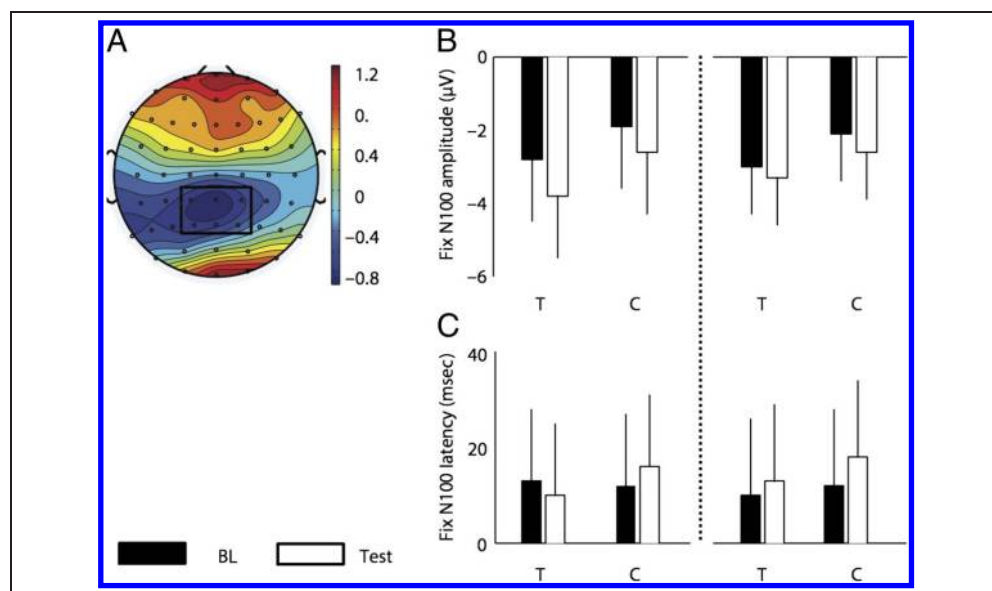
#### Stimulus-evoked N1

Analysis of stimulus-evoked N1 amplitude (occurring ~50–200 msec after stimulus onset over electrodes C1, Cz, CP1, CPz, P1, and Pz; see Figure 7A) revealed the same pattern of results that was observed for the N2. When contrasting trained S-R mappings to the control group, a significant Group  $\times$  Session interaction was observed ( $F(1, 22) = 7.03$ ,  $MSE = 11.31$ ,  $p = .015$ ,  $\eta_p^2 = .24$ ; see Figure 7B). Follow-up analysis showed that the two groups did not differ statistically at the baseline session ( $p = .11$ ,  $\eta_p^2 = .08$ ). However, the training group showed an increase in N1 amplitude from baseline to test (baseline mean =  $-2.5 \mu\text{V}$ ,  $SD = 2.15 \mu\text{V}$ ; test mean =  $-5.25 \mu\text{V}$ ,

$SD = 2.66 \mu\text{V}$ ;  $F(1, 11) = 31.80$ ,  $MSE = 8.49$ ,  $p < .001$ ,  $\eta_p^2 = .74$ ), whereas the control group did not (baseline mean =  $-3.88 \mu\text{V}$ ,  $SD = 2.72 \mu\text{V}$ ; test mean =  $-4.51 \mu\text{V}$ ,  $SD = 2.73 \mu\text{V}$ ;  $p = .33$ ,  $\eta_p^2 = .086$ ).

When comparing untrained S-R mappings with the control group, a significant Group  $\times$  Session interaction was also observed ( $F(1, 22) = 5.94$ ,  $MSE = 8.39$ ,  $p = .02$ ,  $\eta_p^2 = .21$ ). Again, follow-up analysis revealed that the two groups were comparable at the baseline session ( $p = .25$ ,  $\eta_p^2 = .06$ ), whereas only the training group showed a significant increase in N1 amplitude from baseline to test (baseline mean =  $-2.97 \mu\text{V}$ ,  $SD = 2.12 \mu\text{V}$ ; test mean =  $-4.56 \mu\text{V}$ ,  $SD = 2.81 \mu\text{V}$ ;  $F(1, 11) = 7.85$ ,  $MSE = 11.63$ ,

**Figure 8.** Fixation-locked ERP (A) for the baseline (BL) and test sessions as a function of training. A negative-going shift was observed over central-parietal sites (A, boxed region shows selected electrodes) peaking at ~0 msec (relative to stimulus onset). No influence of training was found for mean amplitude (B) or latency (C). Error bars represent 95% confidence intervals (calculated using the Subject  $\times$  Session error term; Masson & Loftus, 2003).





$p = .017$ ,  $\eta_p^2 = .42$ ; control group: baseline mean =  $-4.33 \mu\text{V}$ ,  $SD = 2.79 \mu\text{V}$ ; test mean =  $-4.25 \mu\text{V}$ ,  $SD = 3.01 \mu\text{V}$ ;  $p = .85$ ,  $\eta_p^2 = .003$ ).

Importantly, when N1 amplitudes for trained and untrained S–R mappings were compared directly within the training group, a statistically significant interaction was not observed ( $p = .84$ ,  $\eta_p^2 = .004$ ), indicating that N1 amplitudes increased comparably for trained and untrained S–R mappings. Thus, this exploratory analysis revealed that transferable training benefits are observed as early as the N1 component. This provides clues for the mechanisms that may mediate transferable sensory encoding benefits, which are considered further in the discussion.

#### *Fixation-evoked N1*

Analysis of this component (occurring approximately  $-80$  to  $80$  msec over CP1, CPz, CP2, P1, Pz, and P2) did not reveal any significant training effects when examining mean amplitude (Group  $\times$  Session interaction for trained S–R mappings:  $F(1, 22) = .26$ ,  $p = .61$ ; Group  $\times$  Session interaction for untrained S–R mappings:  $F(1, 22) = .07$ ,  $p = .80$ ) and latency (trained S–R mappings, Group  $\times$  Session interaction:  $F_c(1, 22) = 1.97$ ,  $p = .17$ ; untrained S–R mappings, Group  $\times$  Session interaction:  $F_c = .39$ ,  $p = .54$ ; see Figure 8). Therefore, we have no evidence for an influence of training in response to the fixation point. This further confirms that training influences brain activity that occurs in response to the target stimulus, rather than preceding events.

## **DISCUSSION**

The transferability of training benefits for the brain events underlying sensory encoding and response selection was assessed using EEG and a sensorimotor training paradigm. Participants trained on four S–R mappings, with four tones each requiring a unique button press. At test, participants showed increased amplitudes and reduced timing of the N2 component, which transferred to similar but untrained S–R mappings, relative to a no-train control group. These effects were not associated with anticipatory attention, as measured by pretrial alpha ERD. In contrast, reductions in the onset latency of the s-LRP, a component reflecting the duration of central decisional processing before the activation of motor plans, showed significantly greater reductions for trained S–R mappings, relative to controls and to untrained S–R mappings.

These findings show that sensorimotor training has dissociable impacts on the neural events underlying information processing. Events occurring a few hundred milliseconds into stimulus processing are sensitive to the influence of training, regardless of whether the trained stimulus set, or an untrained stimulus set, is presented. In contrast, neural events reflecting the duration

taken to initiate motor plans show changes for trained stimuli that do not transfer to untrained stimuli. Therefore, these findings are the first to demonstrate that the consequences of a single sensorimotor training regimen are different across dissociable brain events and, consequently, the stages of processing indexed by these events. Specifically, sensorimotor training has dissociable influences on sensory encoding and response selection. The finding that the consequences of a single training regimen are dissociable across information processing stages holds exciting promise for enhancing the efficacy of cognitive training regimens. By isolating the neural events for which the influence of training shows transfer to untrained materials, training regimens can be tailored to ensure that benefits will transfer to new tasks and situations.

The finding that training increases amplitude of the N2 component for both trained and untrained S–R mappings provides evidence for a transferable training benefit on the neural mechanisms that underpin sensory encoding. The N2 component is the first to show changes that correlate with whether the second target is detected in the AB paradigm (Sergent et al., 2005); it is also assumed to reflect mental access to the properties of a stimulus (see Hillyard & Anllo-Vento, 1998, for a review). We have previously shown that sensorimotor training benefits AB performance, even when the stimuli used in the task differ to those that were trained on and are highly familiar (Garner et al., 2014). It is therefore unlikely that the N2 changes observed here reflect other processes that the component has been linked to, such as novelty detection and cognitive control (see Folstein & Van Petten, 2008).

Given that a transferable training benefit has been observed for the neural events that are sensitive to sensory encoding demands, the next step involves determining how sensory encoding mechanisms are influenced by training to benefit novel/untrained inputs. One possibility is that training results in more expert/efficient deployment of selective attention. This would motivate a more robust, and rapid, consolidation of sensory information into a durable form. As selective attention has been shown to modulate sensory signals across tasks and modalities (e.g., Lakatos et al., 2013; Scolar, Byers, & Serences, 2012; Herrmann, Montaser-Kouhsari, Carrasco, & Heeger, 2010; Woldorff et al., 1993) and both the N1 and N2 components are sensitive to selective attention manipulations (Hillyard & Anllo-Vento, 1998; Michie, 1984), it is reasonable to suggest that training may refine deployment of selective attention. For example, once someone has trained on deploying selective attention resources to analyze incoming sensory signals within a task structure, it may be that the act of deploying selective attention within the task environment is refined, as well as the processing of the stimulus-specific information.

Pretrial alpha ERD, a measure of anticipatory attention previously associated with the AB (MacLean & Arnell, 2011), was not found to be modulated by training, despite

the finding that pretrial alpha ERD did increase over the pretrial interval. This indicates that the transferable benefit observed here and in previous work (Garner et al., 2014) emerges between stimulus onset and 270 msec into information processing, before the period (270–300 msec) that sensory information is hypothesized to be encoded via long-range frontal–parietal connections, making sensory information available for conscious report (Dehaene & Changeux, 2011; Del Cul, Baillet, & Dehaene, 2007; Sergent & Dehaene, 2004). Taken together, these lines of evidence support the above idea that data-unlimited, sensorimotor training facilitates attention mechanisms involved in the transfer of sensory signals into a conscious percept and that training of these mechanisms can transfer to facilitate encoding of untrained stimuli.

It could be argued that the observed transferable benefit is not caused by the training regimen itself but is attributable to another factor distinguishing training and control groups, such as motivational differences. Previous behavioral observations have shown that other active control tasks, such as visual search training, do not result in sensory encoding benefits (Garner et al., 2014). Furthermore, if motivational differences were contributing to the observed transferable benefits, it would be more likely that volitional processes, such as anticipatory attention, would also show benefits from training and transfer. However, the current training regimen did not affect our measure of anticipatory attention. Taken together, these findings suggest that the observed transferable benefits are most likely to be a consequence of the sensorimotor training regimen.

In contrast to the transferable training benefit found for sensory encoding, response selection benefits, as measured by reduced s-LRP onsets and RT costs on dual-task trials, are task specific. Given that the onset of the s-LRP represents the time at which central, decisional operations are complete (Coles, 1989), these findings support previous suggestions that response selection limitations are overcome by increasing the efficiency of these operations (Kamienkowski, Pashler, Dehaene, & Sigman, 2011; Dux et al., 2009; Ruthruff, Van Selst, Johnston, & Remington, 2006). These findings also show that increasing the efficiency of this central stage requires practice on the specific decision that is to be executed, suggesting that response selection efficiency is dependent on the connection strength of specific inputs and outputs.

It is interesting that training-related changes to the timing of earlier ERP components did not produce training-related timing reductions in later components. For example, the current findings show that, although the training group displayed significantly greater reductions in the timing of the N2 component for untrained items relative to a control group, onset of the s-LRP for the same untrained items was comparable with the control group. This suggests that an improvement in sensory encoding stages of sensorimotor processing is not fully sufficient to ensure an increase in decisional efficiency. Instead, the

current observations suggest that strengthened connections between specific inputs and outputs are required.

The finding that transferability of training benefits is not equal across information processing stages has implications for the design and use of cognitive training regimens. It continues to be debated whether cognitive training can produce transferable training benefits (e.g., Redick et al., 2013; Jaeggi, Buschkuhl, Jonides, & Shah, 2011; Owen et al., 2010). Despite this uncertainty, there has been a recent explosion in the use of brain training to improve cognitive function, resulting in a multimillion dollar brain training industry (see Owen et al., 2010). The current demonstration that transferability of training benefits is not the same for different information processing stages shows that some cognitive training regimens may be better at producing transferable effects than others. The present results clearly demonstrate that it is important to identify the distinct mechanisms that underpin task performance as well as how training may differentially benefit these mechanisms when designing cognitive training regimens and when claiming that benefits are transferable.

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