Abstract

Here we examined the relationship between inhibition of return (IOR) and response-selection conflict. In two go/no-go and spatial-cueing experiments, we measured the amplitude of the fronto-central N2 event-related potential component to estimate the degree of response-selection conflict for validly cued and invalidly cued targets. When the probability of a go target was high (Experiment 1), both the amplitude of the N2 elicited on no-go trials and the number of false alarm errors were greater on invalid-cue than on valid-cue trials. When the probability of a go target was low (Experiment 2), neither of these effects was observed and the magnitude of the IOR effect was greatly reduced. These results show that a relative response bias toward responding on invalid-cue trials contributes to the IOR reaction time effect when the required response is prepotent.

INTRODUCTION

The ability to select efficiently important and relevant information from the vast amount of information provided by our senses is important for an individual’s ability to function in the complex environments in which we live. It has been proposed that the mechanisms responsible for the inhibition of return (IOR) effect may serve to improve the sampling of visual information by inhibiting attention from returning to previously examined spatial locations (e.g., Klein, 1988, 2000; Posner, Rafal, Choate, & Vaughan, 1985). Consequently, an understanding of the mechanisms underlying IOR may provide valuable insights into how information is selected. For this reason, among others, IOR has attracted a significant amount of empirical and theoretical interest.

Posner and Cohen (1984) first discovered IOR in the context of spatial attention orienting studies employing a variant of the cue–target paradigm. In these experiments, target stimuli were preceded by spatially nonpredictive exogenous cues. When the cue–target interval was short, response times (RTs) for targets that appeared at the cued location (valid-cue targets) were shorter than those for targets appearing at other locations (invalid-cue targets). At longer cue–target intervals, however, the opposite result was obtained. It is the latter effect that was labeled IOR by Posner et al. (1985) when they proposed that it represented the operation of a mechanism that inhibited attention from returning to previously attended locations. Subsequent research has found that IOR occurs in a wide variety of experimental situations (for reviews, see Klein, 2000; Taylor & Klein, 1998).

Although the inhibition-of-attention account of IOR remains popular, there is still considerable controversy regarding the mechanisms underlying IOR, the functional significance of these mechanisms, and even the use of the label IOR (e.g., Berlucchi, 2006). Several other mechanisms of IOR have been proposed and virtually every stage of processing leading to the eventual response has been implicated (Taylor & Klein, 1998), including an inhibition of basic sensory process (Berlucchi, 2006; Posner & Cohen, 1984) or perceptual processes (Reuter-Lorenz, Jha, & Rosenquist, 1996) unrelated to attention orienting, biases in response-selection processes (e.g., Ivanoff & Klein, 2006; Klein & Taylor, 1994), or inhibition of motor processes (e.g., Tassinari, Aglioti, Chelazzi, Marzi, & Berlucchi, 1987).

Recently, researchers have turned to measures of brain activity in order to examine the processing changes underlying IOR. Event-related potentials (ERPs) have been particularly useful due to their high temporal resolution. Two recent studies (Prime & Ward, 2004, 2006) utilized ERPs to investigate the role of perceptual and motor effects in the IOR phenomenon. In these studies, the latency of the response-locked lateralized readiness potential, an electrophysiological measure motor activity, was unaffected by cue validity. This result suggested that changes in motor processing do not contribute to IOR. In contrast, the amplitudes of the perceptually related occipital P1 and/or N1 components were reduced for valid-cue targets relative to invalid-cue targets. These results are supported by the results of several other ERP studies utilizing uninformative cues (e.g., Wascher & Tipper, 2004; McDonald, Ward, & Kiehl, 1999; Hopfinger & Mangun, 1998) that have also found evidence of reduced occipital cortex activity for validly cued targets.
with long cue–target intervals (for a review, see Prime & Ward, 2006). Further evidence for a relative inhibition of perceptual processing of valid-cue targets at long cue–target intervals comes from a recent functional magnetic resonance imaging (fMRI) study that isolated the brain responses to the cues and targets by presenting these stimuli at adjacent locations but on opposite sides of the vertical meridian (Müller & Kleinschmidt, 2007). This study found that IOR was associated with reduced occipital cortex activity for validly cued targets relative to invalidly cued targets.

On the basis of the observed modulations in the amplitude, but not the latency, of occipital cortex activity, Prime and Ward (2006) proposed that IOR arises from a relative slowing of response-selection processes on valid-cue trials due to a reduction in the signal-to-noise ratio of the perceptual input to the decision-making process. However, there are several findings regarding IOR that are difficult to account for solely in terms of changes in the signal-to-noise ratio of the target. For example, the magnitude of IOR is affected by the number of response alternatives (Pratt, Adam, & O’Donnell, 2005), the presence of a nonresponding effector (Ivanoff & Klein, 2001), and stimulus–response probability (Ivanoff & Klein, 2004). Such results indicate that part, or all, of the IOR effect is associated with changes in response-selection processes.

In the present study, we utilized the fronto-central N2 ERP component to examine response-selection processes. This ERP component has a negative peak with a latency between 250 and 350 msec and several studies utilizing dipole localization techniques have indicated an anterior cingulate cortex (ACC) source (e.g., Bekker, Kenemans, & Verbaten, 2005; Nieuwenhuis et al., 2003; van der Wildenberg, & Riddervold, 2003; van Veelen & Carter, 2002). Although the exact role that the ACC plays in response selection is currently a popular topic of debate (e.g., Botvinick, Cohen, & Carter, 2004; Paus, 2001), there is strong evidence that the amplitude of the N2 is sensitive to the degree of conflict between response alternatives. For example, in the Eriksen flanker task (Eriksen & Eriksen, 1974), the amplitude of the N2 is enhanced when the flankers are mapped to a different response than the target item (van Veelen & Carter, 2002). The N2 is also sensitive to response conflict arising from differences in the relative frequency of response alternatives. When a task requires choosing between high-frequency and low-frequency response alternatives, the amplitude of the N2 is enhanced for target stimuli signaling the low-frequency response (Nieuwenhuis et al., 2003). Furthermore, the N2 is sensitive to changes in response criteria. In an experiment examining speed–accuracy tradeoffs, Band, Riddervold, and van der Molen (2003) found that the amplitude of the N2 was enhanced when participants were instructed to emphasize speed at the expense of accuracy.

Here we tested the proposal by Klein and Taylor (1994) that IOR arises from a "reluctance to respond" to valid-cue targets by observing the effect of cue validity on N2 amplitude in a go/no-go task. Participants were required to give a speeded, simple response to “go” targets and to withhold the response from “no-go” targets. In this task, the go and no-go response alternatives compete and give rise to response-selection conflict. In Experiment 1, we used a high proportion (75%) of go stimuli. In this situation, participants are highly prepared to make the go response. This high degree of response preparation increases the difficulty of withholding the response on no-go trials and increases response-selection conflict (Nieuwenhuis et al., 2003). Consequently, a large N2 should be observed on no-go trials. If participants are biased against responding to validly cued targets (“reluctance to respond”), the degree of preparation of the go response will be reduced on validly cued trials. Therefore, the amount of response conflict will be reduced on valid-cue relative to invalid-cue trials. Hence, the amplitude of the N2 on no-go trials should be reduced on valid-cue relative to invalid-cue trials in Experiment 1. In Experiment 2, we used a low proportion (25%) of go stimuli in order to reduce response preparation for the go response. If the response-selection biases play a causal role in generating the IOR effect, both IOR and the effect of cue validity on the N2 should be reduced in this experiment.

**EXPERIMENT 1**

**Methods**

An example of the stimulus display and trial sequence is shown in Figure 1. Participants viewed a computer monitor from a distance of 57 cm and were instructed to maintain fixation on a centrally located fixation cross during the experimental blocks and to blink between trials. The screen background was black, and two gray square outline boxes (1.5° × 1.5°) centered 4.5° were displayed at all times to the left and right of fixation. After a 250-msec intertrial interval, each trial began with a 400-msec offset of the fixation cross. Participants were informed that the offset of the fixation cue indicated a safe time to blink. The cue followed the fixation offset by 1050 msec. The cue consisted of a 150-msec brightening.
of one of the two square boxes and was equally likely to occur at either location. One hundred fifty milliseconds after cue offset, a white disk 0.75° in diameter was presented at fixation for 100 msec. This reorienting event was intended to redirect attention back to fixation from the cued location (Prime, Visser, & Ward, 2006). After a variable stimulus onset asynchrony (SOA) of 400 to 800 msec, the target was presented for 900 msec. The total cue–target SOA ranged between 700 and 1100 msec. Targets were presented with equal probability within one of the two peripheral boxes (chance coincidence of cue and target locations). Trials on which cue and target occurred at the same location were classified as **valid-cue** trials, whereas trials on which cue and target occurred at opposite locations were classified as **invalid-cue** trials. The target stimulus could be either a “go” stimulus (a 0.75° white square) or a “no-go” stimulus (a white X shape, 0.75° × 0.75°). The go stimulus was presented on 75% of trials and the no-go stimulus was presented on the remaining 25% of trials. Participants were instructed to press a response key whenever they detected the onset of the square “go” target stimulus and to refrain from responding when a “no-go” stimulus was presented. Both speed and accuracy were stressed in the instructions. Trials were presented in 65 short blocks of 16 trials each, for a total of 1040 trials. Participants were allowed to rest as long as they wished between blocks.

**Electrophysiological Recording and Processing**

The electroencephalogram (EEG) was recorded from the left and right mastoids and 64 standard 10–10 scalp sites with active Ag/AgCl electrodes (BioSemi Active Two system) mounted on an elastic cap. Eye position was monitored by both the horizontal and vertical electro-oculogram (EOG). EEG and EOG channels were low-pass filtered at 67 Hz and digitized at 256 Hz. After acquisition, the EEG channels were referenced to the average of the left and right mastoids and high-pass filtered at 0.05 Hz (half power cutoff). Trials containing blinks, eye movements, and EEG artifacts were removed prior to ERP averaging by applying automated artifact detection routines. Trials were excluded if ocular artifacts were detected in the interval from cue onset until the end of the target-locked epoch. In addition, trials with errors and those with RTs outside the range of 100 to 900 msec were excluded from the analysis. ERPs were calculated separately for valid-cue and invalid-cue trials at all electrodes sites time-locked to the presentation of the target. After averaging, the ERPs were digitally low-pass filtered (30 Hz half-amplitude cutoff) to eliminate high-frequency noise and were high-pass filtered (2 Hz half-amplitude cutoff) to remove low-frequency overlap from the cue and reorienting event. The ERPs were baseline corrected to the mean voltage of the 100-msec pretarget interval. After averaging, ipsilateral and contra-lateral ERP waves for each condition were calculated by averaging corresponding left and right hemisphere electrodes based on the visual field of the target. Electrodes on the midline were averaged across left and right visual field targets. The effect of cue validity on ERP amplitude was assessed for the occipital P1 and N1 components (PO7, PO8) and the fronto-central N2 (Fz) and P3 (Cz) components. Mean amplitudes of these components were measured in 40-msec windows, calculated separately for each condition, centered around the peak of each component in the grand-average ERP.

Eighteen individuals participated in Experiment 1 (6 men, 4 left-handed). An average of 81.18% of trials was included in the analysis. The grand-average residual horizontal EOG activity remaining after trials with ocular artifacts were excluded was less than ~2.5 μV, corresponding to a shift in eye position of less than ~0.2° of visual angle (Lins, Picton, Berg, & Scherg, 1993).

**Results and Discussion**

**Behavioral Measures**

Mean RTs on valid-cue and invalid-cue trials are presented in Table 1. The expected IOR effect was observed—Participants were significantly faster in responding to go targets on invalid-cue trials (395 msec) than on valid-cue trials (424 msec) \[F(1, 17) = 35.7, p < .001, \eta_p^2 = 0.677\]. Analysis of false alarm errors on no-go trials revealed that participants made more errors on invalid-cue trials (4.9%) than on valid-cue trials (2.8%) \[F(1, 17) = 7.8, p < .02, \eta_p^2 = 0.314\]. The fact that more errors were made on the faster invalid-cue trials than on the slower valid-cue trials is consistent with the proposal that there is greater readiness to respond to invalidly cued targets. Misses and anticipations on go trials were rare (~1%) and the differences between error rates on valid-cue (1.4%) and invalid-cue (1.1%) trials were not significant \[F(1, 17) = 3.8, p > .05, \eta_p^2 = 0.183\].

**Event-related Potential Measures**

The go and no-go target elicited ERPs at occipital sites from Experiment 1 are shown in Figure 2A, the corresponding amplitude measures are presented in Table 2a. Amplitude measures for go targets were analyzed by separate 2 × 2 repeated measures analyses of variance (ANOVAs) with factors of validity (valid-cue, invalid-cue)
and laterality (ipsilateral, contralateral). Unlike some previous studies, no effect of cue validity was found on P1 amplitude. Although the ANOVA revealed a main effect of laterality $[F(1, 17) = 25.1, p < .001, \eta^2_p = 0.597]$, neither the main effect of validity nor the Validity $\times$ Laterality interaction approached significance (both $F$s < 1). The analysis of N1 amplitude revealed a significant main effect of laterality $[F(1, 17) = 6.7, p < .02, \eta^2_p = 0.28]$. The main effect of validity $[F(1, 17) = 2.13, p = .16, \eta^2_p = 0.112]$ was not significant. However, the Validity $\times$ Laterality interaction $[F(1, 17) = 3.2, p < .09, \eta^2_p = 0.160]$ approached significance. Subsequent analyses of ipsilateral and contralateral ERPs revealed that the effect of validity on the amplitude of the N1 approached significance for ipsilateral ERPs $[F(1, 17) = 3.82, p < .07, \eta^2_p = 0.183]$, but not for contralateral ERPs ($F < 1$). The no-go target stimuli used in this experiment had a smaller surface area and lower total luminance than the go target stimuli. Consequently, the amplitude of the N1 component was smaller for no-go targets than for go targets (Figure 2A). Furthermore, the signal-to-noise ratio of the ERP averages was lower for no-go targets than for go targets because no-go targets were only presented on 25% of trials. These factors are most likely responsible for the failure to obtain significant effects of validity on the amplitude of the N1 for no-go targets.

The fronto-central target-elicited ERPs for go and no-go trials from Experiment 1 are shown in Figure 2B.

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<th>Table 2. Mean Amplitude (µV) of (A) Occipital ERP Components and (B) Fronto-central ERP Components</th>
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| **B** | **Cue Condition** |
| **Experiment** | **Component** | **Valid** | **Invalid** |
| Experiment 1 | go-N2 | -0.25 | -0.51 |
| Experiment 2 | no-go-N2 | -0.84 | -2.16 |
| | go-N2 | -0.64 | -0.92 |
| | no-go-N2 | -0.86 | -0.92 |
Inspection of Figure 2B reveals that the anticipated N2 component was observed on no-go trials. Furthermore, the N2 wave was strongly modulated by cue validity on no-go trials. In addition, the central P3 immediately following the N2 on no-go trials was also modulated by cue validity. In contrast, on go trials, the N2 component was much weaker and the P3 component was virtually absent. These observations were corroborated by submitting measurements of N2 amplitude at Fz (Table 2B) to a 2 x 2 repeated measures ANOVA with factors of validity (valid-cue, invalid-cue) and trial type (go, no-go). The main effect of trial type confirmed that the N2 was significantly larger on no-go trials than on go trials \([F(1, 17) = 8.85, p < .01, \eta^2_p = 0.343]\), and the main effect of validity confirmed that the N2 was significantly larger on invalid-cue than on valid-cue trials \([F(1, 17) = 10.7, p < .01, \eta^2_p = 0.386]\). Furthermore, the interaction between these factors was significant \([F(1, 17) = 11.93, p < .01, \eta^2_p = 0.412]\). Subsequent planned comparisons of the effect of cue validity were performed separately for go and no-go trials. These analyses revealed that the effect of cue validity on N2 amplitude was significant for no-go trials \([F(1, 17) = 17.6, p < .001, \eta^2_p = 0.509]\), but not for go trials \([F(1, 17) = 1.12, p > .30, \eta^2_p = 0.062]\). Analysis of the amplitude of the P3 component, at Cz, observed on no-go trials revealed that the P3 was significantly larger on invalid-cue than on valid-cue trials \([F(1, 17) = 12.1, p < .01, \eta^2_p = 0.416]\). Inspection of the ERPs to go targets in Figure 2B reveals an unexpected amplitude modulation of a centrally maximal P2 peak. Analysis of the amplitude of this peak, at Cz, on go trials revealed that the amplitude was significantly larger on invalid-cue than on valid-cue trials \([F(1, 17) = 9.2, p < .01, \eta^2_p = 0.350]\). At present, the functional significance of this effect is unclear.

In order to confirm that the N2 component observed here was the same as that observed previously, a dipole model of the N2 peak on invalid-cue no-go trials was calculated using BESA (www.besa.de). Modeling was performed on average referenced ERPs, across a 20-msec window around the N2 peak, using a four-shell ellipsoidal head model. A dipole model with two symmetric dipoles (Figure 3) explained most of the variance in the data for the N2 peak \((x = \pm 1.1 \text{ cm}, y = 1.9 \text{ cm}, z = 6.8 \text{ cm}; \text{RV} = 6.7%)\). This model closely corresponds to prior dipole models from studies that have investigated the relationship between response conflict and N2 amplitude (e.g., Nieuwenhuis et al., 2003; van Veen & Carter, 2002).

In a recent study, Prime and Ward (2006) utilized a sparse central and posterior electrode array to examine the effect of IOR on occipital ERP components and the lateralized readiness potential across a variety of experimental tasks. In an experiment utilizing a go/no-go task similar to the present experiment, an effect of cue validity on the amplitude of a N2 peak was observed on no-go trials at central (C1, C2) and parietal (Pz) electrodes. Due to the sparse electrode array used, it was not possible to examine the scalp distribution of the negative peak in order to determine its identity. Prime and Ward speculated that the N2 peak might be either the attention-sensitive N2b component (e.g., Mulder, Wijers, Brookhuis, Smid, & Mulder, 1994) or the anterior N2. The results of the present experiments clearly indicated that the N2 peak observed in this prior study was due to the posterior portion of the scalp distribution of the anterior N2 peak.

In the present experiment, greater response conflict, as indexed by N2 amplitude, and an increase in false alarm errors were observed on invalid-cue trials. Prior research has demonstrated that N2 amplitude is enhanced...
when the response criterion is lowered in order to emphasize speed at the expense of accuracy (Band et al., 2003). Hence, both the behavioral and electrophysiological results from this experiment are consistent with a relative bias in response-selection processes against responding to validly cued targets (or, equivalently, toward responding to invalidly cued targets). This suggests that the effect of cueing on RT may arise, at least in part, from changes in the duration of response-selection selection processes arising from a response bias. However, the relationship between the N2 index of response conflict and RT is necessarily correlational. Experiment 2 was conducted to provide evidence supporting a causal relationship between these factors.

**EXPERIMENT 2**

Prior research has demonstrated that, in a go/no-go task, IOR magnitude is reduced when the probability of a go target is low (Ivanoff & Klein, 2004). Furthermore, when speeded responses are required to infrequent go stimuli, the conflict-related N2 is reduced on no-go trials and enhanced on go trials due to the task demands of responding quickly but infrequently (Nieuwenhuis et al., 2003). In the present experiment, we reduced the probability of go stimuli to 25% in order to reduce the magnitude of the IOR effect. If the response-selection related processes indexed by the N2 play a causal role in generating the IOR effect, the effect of cue validity on N2 amplitude should also be reduced.

**Methods**

All aspects of Experiment 2 were identical to Experiment 1, with the following exception: The proportion of go and no-go stimuli was reversed so that no-go stimuli were presented on 75% of trials. Eighteen individuals participated in Experiment 2 (4 men, 4 left-handed). An average of 88.8% of trials was included in the ERP analysis. The grand-average residual horizontal EOG activity remaining after trials with ocular artifacts were excluded was less than $\frac{1}{2.5}$ $\mu$V, corresponding to a shift in eye position of less than $\frac{1}{0.2}$ of visual angle (Lins et al., 1993).

**Results and Discussion**

**Behavioral Measures**

Mean RTs on valid-cue and invalid-cue trials are presented in Table 1. The expected IOR effect was again observed; participants were significantly faster in responding to go targets on invalid-cue trials (502 msec) than on valid-cue trials (519 msec) $[F(1, 17) = 40.69, p < .001, \eta^2_p = 0.705]$. This IOR effect was approximately half as large as in Experiment 1, however (17 msec vs. 31 msec), and analysis of RT across these two experiments revealed a significant Experiment $\times$ Validity interaction $[F(1, 34) = 5.96, p < .02, \eta^2_p = 0.149]$. Unlike Experiment 1, false alarm errors on no-go trials were rare (<1%) and did not differ between cue-validity conditions ($F < 1$). Miss errors on go trials occurred more frequently on valid-cue trials (3.4%) than on invalid-cue trials (1.4%) $[F(1, 17) = 17.04, p < .001, \eta^2_p = 0.501]$. The effect of cue validity on miss errors is difficult to interpret. The finding is consistent with both a higher criterion on valid-cue trials or increased signal gain on invalid-cue trials.

**Event-related Potential Measures**

Analysis of the effect of cue validity on the go target-elicited ERPs (Figure 4A) revealed a similar pattern of results to that obtained in Experiment 1. As before, the analysis of P1 amplitude revealed only a main effect of laterality $[F(1, 17) = 16.4, p < .001, \eta^2_p = 0.491]$, neither the main effect of validity nor the Validity $\times$ Laterality interaction was significant (both $F$s < 1). The analysis of N1 amplitude revealed main effects of laterality $[F(1, 17) = 36.9, p < .001, \eta^2_p = 0.685]$ and validity $[F(1, 17) = 7.35, p < .02, \eta^2_p = 0.302]$. However, unlike Experiment 1, the effect of cue validity on N1 amplitude

![Figure 4. ERPs from Experiment 2. (A) Occipital ERPs and (B) fronto-central ERPs.](http://direct.mit.edu/jocn/article-pdf/21/5/991/1760051/jocn.2009.21105.pdf)
was observed for ipsilateral and contralateral ERPs and the Validity × Laterality interaction did not approach significance (F < 1). Although there is no obvious reason for this discrepancy between experiments, both experiments found that N1 amplitude was reduced on valid-cue trials relative to invalid-cue trials.

Analysis of P1 amplitude for no-go targets revealed only a main effect of laterality [F(1, 17) = 23.5, p < .001, \( \eta^2_p = 0.580 \)], and neither the main effect of validity [F(1, 17) = 1.8, p = .20, \( \eta^2_p = 0.096 \)] nor the Validity × Laterality interaction [F(1, 17) = 1.8, p = .2, \( \eta^2_p = 0.094 \)] was significant. The analysis of no-go target N1 amplitude revealed main effects of laterality [F(1, 17) = 27.1, p < .001, \( \eta^2_p = 0.615 \)] and validity [F(1, 17) = 11.7, p < .01, \( \eta^2_p = 0.408 \)]. In contrast to the analysis of go target N1 amplitude, the Validity × Laterality interaction was significant [F(1, 17) = 9.2, p < .01, \( \eta^2_p = 0.350 \)]. Analyses of ipsilateral and contralateral ERPs revealed that the N1 amplitude was reduced on valid-cue relative to invalid-cue trials for ipsilateral ERPs [F(1, 17) = 21.0, p < .001, \( \eta^2_p = 0.555 \)]. Although an effect of validity on N1 amplitude is visible for contralateral ERPs (Figure 4A), this effect was not significant (F < 1). This difference in results from the analysis of go trial ERPs is probably due to the weaker ERP response to the less luminous no-go stimulus.

The fronto-central ERPs from go and no-go trials (Figure 4B) reveal a different pattern of cue-validity effects than that obtained in Experiment 1. Consistent with previous results (e.g., Nieuwenhuis et al., 2003), the N2 was enhanced on go trials and attenuated on no-go trials relative to Experiment 1. However, in the present study, the between-experiment difference in N2 amplitude was not significant for either no-go [F(1, 34) = 2.65, p > .11, \( \eta^2_p = 0.072 \)] or go (F < 1) trials. Furthermore, N2 amplitude was not modulated by cue validity in the present experiment. Analysis of N2 amplitude failed to reveal main effects of trial type [F(1, 17) = 0.1, p > .8, \( \eta^2_p = 0.004 \)], validity [F(1, 17) = 1.9, p > .19, \( \eta^2_p = 0.099 \)], or an interaction between these factors [F(1, 17) = 1.0, p > .32, \( \eta^2_p = 0.058 \)]. Similarly, the analysis of P3 revealed only a main effect of trial type [F(1, 17) = 21.4, p < .001, \( \eta^2_p = 0.557 \)], indicating that P3 amplitude was significantly larger on go than on no-go trials. Although the P3 amplitude appears slightly larger on invalid-cue trials than on valid-cue trials, neither the main effect of Validity nor the Trial type × Validity approached significance (both Fs < 1). In contrast to Experiment 1, the amplitude of the P2 peak on go trials did not differ between valid-cue and invalid-cue trials (F < 1). Further research will be required to determine the cognitive significance of the effect of cue validity on the P2 peak.

The failure to obtain any significant differences in N2 amplitude in this experiment is consistent with our prediction that a reduction in the magnitude of the IOR effect would be accompanied by a reduction of the effect of cue validity on N2 amplitude. In order to more directly assess the effect of go target probability on N2 amplitude, a cross-experiment analysis was performed. An initial 2 × 2 × 2 between–within ANOVA with factors of experiment, validity (valid-cue, invalid-cue), and trial type (go, no-go) was performed. The results of Experiment 1 indicated that validity interacted with trial type and that validity only affected the N2 amplitude on no-go trials. In contrast, no effects of validity were found on N2 amplitude in the present experiment. If this difference in results was reliable, we would expect the between-experiment ANOVA to produce a significant three-way interaction and a significant interaction between experiment and validity. This pattern of results was exactly obtained, and both the three-way interaction [F(1, 34) = 11.5, p < .01, \( \eta^2_p = 0.252 \)], and the Experiment × Validity interaction [F(1, 34) = 5.1, p < .05, \( \eta^2_p = 0.131 \)] were significant. In addition, significant effects were obtained for validity [F(1, 34) = 12.4, p < .01, \( \eta^2_p = 0.268 \)], trial type [F(1, 34) = 4.9, p < .05, \( \eta^2_p = 0.126 \)], and the interaction between trial type and validity [F(1, 34) = 4.7, p < .05, \( \eta^2_p = 0.122 \)]. No other effects were significant (all ps > .05). To confirm that the between-experiment differences in N2 amplitude arose from differences in the effect of validity on no-go trials, a 2 × 2 between–within ANOVA with the factors of experiment and validity was performed on the data from no-go trials. As anticipated, this analysis revealed an interaction between experiment and validity [F(1, 34) = 7.0, p < .01, \( \eta^2_p = 0.271 \)], as well as a main effect of validity [F(1, 34) = 15.6, p < .001, \( \eta^2_p = 0.308 \)].

The present experiment demonstrates that reducing the probability of the go target both reduced the magnitude of the IOR effect and eliminated the effect of cue validity on the conflict-related N2. Although caution must always be exercised when interpreting the relationship between behavioral and ERP data, the observed covariation of effects is consistent with a causal relationship between the operation of the neural processes indexed by the N2 and the RT effects. However, significant IOR was observed in the absence of an effect of cue validity on N2 amplitude. This indicates that the processing changes giving rise to the response-selection conflict indexed by the N2 cannot be the sole cause of the IOR effect.

**GENERAL DISCUSSION**

The present results demonstrate that cue validity can modulate the response-selection conflict-related anterior N2 ERP peak. The present and previous source localizations of this peak indicate an ACC source. This is consistent with considerable evidence that the ACC is part of a conflict detection system (e.g., Botvinick et al., 2004). Thus, the current results indicate that cue validity can modulate response-selection conflict-related activity in the ACC. The observed pattern of ERP and behavioral
results is consistent with a response bias in favor of invalidly cued targets. However, the results of Experiment 2 indicate that IOR can occur in the absence of increased response-selection conflict on invalid-cue trials. Thus, response bias may only substantially contribute to IOR when the response is prepotent, such as when a simple detection response or high probability response is required (cf., Ivanoff & Klein, 2004). This is consistent with prior research indicating that IOR magnitude is greater when the required response is known before the target is presented (e.g., Prime & Ward, 2006; Pratt et al., 2005). Due to the fact that the majority of IOR studies have required subjects to make highly prepotent simple detection responses, it is likely that response bias has been a major contributor to the IOR effects observed in prior studies.

The present results converge with previous behavioral findings indicating that IOR arises from changes in both perceptual and response-selection processes. Recently, Ivanoff and Klein (2006) applied signal detection theory measures of sensitivity (d') and response criterion (c) to the study IOR in the context of experiments utilizing a response signal technique that has been used frequently to examine speed–accuracy tradeoff functions (e.g., Wickelgren, 1977). Ivanoff and Klein combined the response signal technique with the cue–target paradigm by adding a response signal tone after the presentation of the target that indicated that a response was required within a 210-msec response window immediately following the tone. By varying the target-tone SOA from 120 to 480 msec, it is possible to manipulate the duration of target processing and response speed, allowing the analysis of response accuracy as a function of RT. Although this procedure substantially changes the response requirements from that of the standard cue–target paradigm by separating the informative stimulus (the target) from the imperative stimulus (the tone), the results of Ivanoff and Klein’s Experiment 1, which used a go/no-go task, are in agreement with the results of the present Experiment 1. Consistent with the effect of cue validity on the N2 component and accuracy rates observed here, an increase in the response criterion was found for valid-cue targets relative to invalid-cue targets early in target processing (i.e., when the interval between target onset and response signal onset was short). Later in target processing, a relative decrease in sensitivity was observed for valid-cue targets. This latter finding is consistent with ERP (e.g., Prime & Ward, 2004, 2006) and fMRI (Müller & Kleinschmidt, 2007) evidence that IOR is associated with reduced activity in occipital visual areas.

Previously, Prime and Ward (2006) proposed that IOR arises from a relative slowing of response-selection processes on valid-cue trials due to a reduction in the signal-to-noise ratio of the perceptual input to the decision-making process. According to this account, the rate of evidence accumulation in the response-selection decision mechanism is dependent on the signal-to-noise ratio of the target stimulus. Consequently, the time required for the response threshold to be reached is also dependent on the signal-to-noise ratio. In addition, increasing the signal-to-noise ratio would also tend to decrease errors by more effectively driving the decision process toward the correct response threshold. This account is consistent with both ERP (e.g., Prime & Ward, 2004, 2006; McDonald et al., 1999) and fMRI (Müller & Kleinschmidt, 2007) evidence that IOR is associated with a reduction of occipital cortex activity for validly cued target relative to invalidly cued targets. However, this account does not explain the present differences in the magnitude of the RT IOR effect between experiments, the pattern of false alarm errors, or the observed N2 modulations. There is no evidence that the relative signal-to-ratio between validly cued and invalidly cued targets differed between the two experiments. In fact, the overall effect of validity on the N1 amplitude for go targets was slightly larger in Experiment 2 than in Experiment 1, despite the fact that the IOR effect observed in Experiment 2 was approximately half as large as that observed in Experiment 1. Thus, this account must be modified to account for the present results.

The present results indicate that, at least when the response is prepotent, a response bias in favor of invalidly cued targets also contributes to IOR by reducing the amount of evidence required to reach the response threshold on invalid-cue trials. In addition to shortening response times, such a response bias would also increase the error rate on invalid-cue trials (e.g., Experiment 1). The observed differences in the magnitude IOR across experiments can be explained by an account that attributes IOR to both signal-to-noise ratio changes and a response bias. According to this account, the large IOR effect observed in Experiment 1 is due to both a reduction of the signal-to-noise ratio on valid-cue trials (indexed by the N1 amplitude effect) and a response bias (indexed by the false alarm and N2 amplitude effects). In contrast, the smaller IOR effect observed in Experiment 2 is due to changes in the signal-to-noise ratio only.

Although the current results indicate that a response bias may only substantially contribute to IOR when the response is prepotent, further research will be required to assess the relative contributions of changes in perceptual processing and response biases in generating IOR across a variety of tasks and situations. Differences in the relative contribution of these two effects may explain why the IOR effect on RT is sometimes accompanied by reduced errors on invalid-cue trials, increased errors on invalid-cue trials, or no difference in error rates between invalid-cue and valid-cue trials (see Ivanoff & Klein, 2006; Figure 1).

Recently, progress has been made in revealing the processes that give rise to IOR. This empirical work has motivated a number of recent attempts to provide a more thorough theoretical account of IOR (e.g., Prime & Ward,
The present work contributes to this growing body of work by showing that spatial cuing leading to IOR interacts strongly with response-selection mechanisms under conditions leading to response conflict. Ultimately, such efforts will lead to a better understanding of the mechanisms of spatial selection and response selection that allow us to function efficiently in our environment.

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