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Facilitation and inhibition are asymmetric aspects of attention that differentially affect response times (RTs), accuracy and neuroelectric activity in many experimental tasks. Both vary as a function of stimulus context, with stimulus repetitions, for example, often resulting in facilitation in terms of speed, accuracy or reduced neural activity. Although inhibition has been extensively studied in the Go/NoGo task, facilitation has been overlooked. Twenty healthy adults performed an adapted Go/NoGo task which manipulated levels of facilitation and inhibition. Event related potential (ERP) and behavioural measures were averaged according to preceding stimulus sequences. Established Go/NoGo effects for N2 and P3 components were replicated. Behavioural and ERP measures, however, showed strong sequence effects. Correlates of facilitation included reduced P1 and N1 latencies, and topographic effects in P1, to Go stimulus repetitions. Manipulations of inhibitory load through increasing Go before NoGo stimuli resulted in incremental increases in N1, P2 and N2 latencies. Several additional ERP and RT measures showed quadratic effects, with indications of facilitation or inhibition which reversed towards the end of longer stimulus trains. The results suggest that both facilitatory and inhibitory processes underlie performance in the Go/NoGo task. As Go stimuli are typically more frequently repeated than NoGo stimuli, the two processes may be confounded when sequence effects are not considered. Additionally, analysing stimuli by context indicates that the timing of the Go-P3 latency is closely related to responses, and the prolongation of N1, P2 and N2 with increasing difficulty of inhibition supports a possible relation of these components to inhibition.

Disciplines

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Sequence effects in the Go/NoGo task: Inhibition and facilitation.

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Abstract

Facilitation and inhibition are asymmetric aspects of attention that differentially affect response times (RTs), accuracy and neuroelectric activity in many experimental tasks. Both vary as a function of stimulus context, with stimulus repetitions, for example, often resulting in facilitation in terms of speed, accuracy or reduced neural activity. Although inhibition has been extensively studied in the Go/NoGo task, facilitation has been overlooked. Twenty healthy adults performed an adapted Go/NoGo task which manipulated levels of facilitation and inhibition. Event-related potential (ERP) and behavioural measures were averaged according to preceding stimulus sequences. Established Go/Nogo effects for N2 and P3 components were replicated. Behavioural and ERP measures, however, showed strong sequence effects. Correlates of facilitation included reduced P1 and N1 latencies, and topographic effects in P1, to Go stimulus repetitions. Manipulations of inhibitory load through increasing Go before NoGo stimuli resulted in incremental increases in N1, P2 and N2 latencies. Several additional ERP and RT measures showed quadratic effects, with indications of facilitation or inhibition which reversed towards the end of longer stimulus trains. The results suggest that both facilitatory and inhibitory processes underlie performance in the Go/NoGo task. As Go stimuli are typically more frequently repeated than NoGo stimuli, the two processes may be confounded when sequence effects are not considered. Additionally, analysing stimuli by context indicates that the timing of the Go-P3 latency is closely related to responses, and the prolongation of N1, P2 and N2 with increasing difficulty of inhibition supports a possible relation of these components to inhibition.

Key words: event-related potentials, Go/NoGo, inhibition, facilitation, priming, sequence effects

1. Introduction

Inhibition and facilitation are the driving forces of selective attention (Kok, 1999), allowing us to respond with precedence to important environmental stimuli. Facilitation is defined as a processing benefit (in either RT or accuracy of responses), and inhibition as a processing decrement (in RT or accuracy), resulting from previous or simultaneous stimulation (Buckner et al., 1998, Klein, 2000, Posner and Snyder, 1975).

Converging evidence supports the existence of both inhibitory and facilitatory components of attention which are asymmetric in character (Eimer, 1999, Eimer and Schlaghecken, 2003, Ghatan et al., 1998, Klein, 2000, Koester and Prinz, 2007, Leboe et al., 2005, Posner and Snyder, 1975, Soetens, 1998, Soetens et al., 1984). Behavioural studies, for example, indicate that effects of facilitation and inhibition on RT can be experimentally separated from one another, and may follow different times courses (e.g. Kok, 1999). Also, fMRI investigations of visuospatial priming have reported that several non-overlapping regions of the prefrontal cortex are activated during negative and positive priming conditions, thought to reflect the coexistence of separate inhibitory and facilitatory networks operating in fronto-parietal cortex (Wright et al., 2006).

Inhibition deficits have been extensively researched in psychiatric disorders such as ADHD, schizophrenia, and obsessive-compulsive disorder (OCD). Because undue facilitation of attention or actions has also been implicated as contributing to repetitive thoughts and behaviours in psychiatric disorders such as OCD (Bannon et al., 2008, Bannon et al., 2002, Hartston and Swerdlow, 1999), it is important to establish

experimental tasks that can investigate the separate contributions of facilitation and inhibition to performance.

1.1 Facilitation

In serial RT tasks with relatively short response-stimulus intervals (RSIs; i.e. less than 500 ms), first-order stimulus repetitions are usually associated with facilitation effects relative to alternations (e.g. Soetens, 1998, Soetens et al., 1984, Vervaeck and Boer, 1980), possibly due to memory traces left by previous stimulus-response cycles, allowing bypassing of some processing mechanisms (Vervaeck and Boer, 1980). Effects can accumulate over trials leading to higher-order effects (Vervaeck and Boer, 1980). With longer RSIs, facilitation may reduce or reverse as participants have time to anticipate which stimuli will follow. Subjective expectancy can produce facilitation for one stimulus category and inhibition for another (Vervaeck and Boer, 1980).

In fMRI studies of humans, neural activity to stimulus repetitions is usually reduced (Buckner et al., 1998, Grill-Spector et al., 2006, Guo et al., 2007, Henson and Rugg, 2003). Stimulus repetition may also be associated with non-linear effects over trials, such as plateaus or reversals in fMRI responses (Grill-Spector et al., 2006). In ERP studies, allowing greater temporal resolution, both positive-going (the ERP to the repeated item is more positive than that to the first presentation) and negative-going (the ERP to the repeated item is more negative than that to the first presentation) repetition effects are reported (Friedman and Cycowicz, 2006, Guo et al., 2007) depending on ERP components and tasks. A number of studies report dissociations in the direction of amplitude change between relatively early (150-300 ms) and later (400-700 ms) ERP repetition effects (Guo et al., 2007, Henson et al., 2004, Henson and Rugg, 2003), possibly reflecting automatic and controlled aspects of learning respectively (Guo et al., 2007). Additionally, some studies report reduced ERP

latencies in association with RT facilitation effects. P1 (Lobaugh et al., 2005, Taylor, 2002) and N1 (Lobaugh et al., 2005) latencies, for example, are shorter in visual search conjunction tasks where multiple features or colour pop-outs speed the search process, and P1 latencies are shorter when direction of eye gaze in a face picture cue is congruent rather than incongruent with a target location (Schuller and Rossion, 2004).

The amplitude of the P300 component shows graded changes with higher-order stimulus repetitions, which vary according to task and stimulus parameters (Friedman and Cycowicz, 2006, Rugg et al., 1994). In counting and button-press tasks, first-order repetitions elicit smaller P300 amplitude than first-order alternations, P300 amplitude decreases with increasing length of a sequence of repetitive stimuli, and is increased to a stimulus which discontinues a long run of repetitive stimuli (Squires et al., 1976). In the oddball task, P300 amplitude increases linearly when targets follow a longer rather than shorter string of non-targets (Gonsalvez et al., 1995, Gonsalvez and Polich, 2002, Johnson and Donchin, 1980). P3 amplitude also increases to non-targets when followed by longer than shorter strings of targets (Gonsalvez et al., 1999, Johnson and Donchin, 1980, Sams et al., 1983, Verleger, 1987). Because ERP repetition effects vary with task and stimulus parameters, it is necessary to investigate them in the Go/NoGo task specifically. Given the robust effects of stimulus context and repetition upon RT and ERP measures, it is surprising that there are apparently no previous publications examining facilitatory effects of Go stimulus repetitions in the Go/NoGo task.

1.2 *Inhibition*

The Go/NoGo task is commonly used to investigate inhibitory processes. The task requires selective inhibition of motor responses to “NoGo” stimuli. Typically, response prepotency is developed by presenting NoGo stimuli infrequently amongst Go stimuli which

require a motor response. Two ERP phenomena are frequently interpreted as correlates of frontal inhibitory mechanisms: A negative component peaking between 200-300ms over the fronto-central scalp (the N2 component) is enlarged in NoGo compared to Go conditions (Eimer, 1993, Falkenstein et al., 1999, Jodo and Kayama, 1992), and a positive component peaking between 300-600ms (the P3 component) is larger and shows a more anterior scalp distribution in NoGo than Go conditions (Fallgatter and Strik, 1999, Roberts et al., 1994).

Although several studies support the association between NoGo-N2 and inhibition (Bruin and Wijers, 2002, Jodo and Kayama, 1992, Roche et al., 2005), others have questioned the relationship (Donkers and van Boxtel, 2004, Falkenstein, 2006, Smith et al., 2006). Doubt has also been raised about the relationship between NoGo P3 and inhibition, which in some studies shows no systematic relationship with performance (see Falkenstein et al., 1999 for review). One difficulty in resolving these issues is that NoGo stimuli differ from Go stimuli in many ways (e.g. probability, sequence effects associated with probability, and response requirements). Preparatory and movement-related activity, for example, might differentially overlap Go- and Nogo ERPs thereby influencing Go-Nogo differences (Falkenstein et al., 1999, Kopp et al., 1996).

A possible alternative approach to test the inhibition hypotheses of N2 and P3 in Go/NoGo tasks would be to compare NoGo stimuli which are differentially primed by preceding stimulus sequences to vary inhibitory load, in order to overcome some of these methodological problems. There is some evidence to suggest that inhibitory load to NoGo stimuli varies as a function of preceding numbers of Go stimuli in sequences. An fMRI study, for example, found higher commission errors to NoGo stimuli preceded by larger numbers of Go stimuli (Durstun et al., 2002). It is reasonable to suppose that if N2 and P3 components are reliable indexes of inhibition, they should similarly be sensitive to manipulations of

inhibitory load. Should the N2 index inhibition and N2 amplitude be sensitive to inhibitory load, it should increase to NoGo stimuli as a function of the number of preceding Go stimuli (e.g., GGGN>GGN>GN, where G represents a Go stimulus and N represents a NoGo stimulus).

There is some evidence that ERP latencies in Go/NoGo tasks are also related to inhibition. Shorter N2 (Falkenstein et al., 1999, Garavan et al., 2002, Roche et al., 2005) and P3a/P3b (Roche et al., 2005) latencies are associated with successful than with failed inhibition of responses to NoGo stimuli. P3 latency is also sometimes longer in NoGo compared to Go conditions, interpreted as a sign of higher processing demands in the NoGo condition (Fallgatter and Strik, 1999, Salisbury et al., 2004). It therefore follows that systematic manipulations of inhibitory load may also affect ERP latencies in the Go/NoGo task, with greater inhibitory load predicted to be associated with longer latency of N2 and or P3 components. Roche et al. (2005) additionally posited that while authors primarily focus on the N2/P3 complex as being indicative of inhibitory processes in the Go/NoGo task, earlier waveform components such as the P1, N1 or P2 may play a major role in determining inhibition success. Accordingly, earlier peaking of the N2 in successful inhibition of responses may follow inhibition-related modulations in earlier components (Roche et al., 2005).

Despite clear effects of context on ERPs and performance, surprisingly few studies have examined sequence effects in the Go/NoGo task. Durston et al. (2002) compared fMRI activation to NoGo stimuli preceded by one, three or five Go stimuli while maintaining an overall target probability of 75%. They found an increase in errors as a function of the number of preceding Go trials, suggesting that inhibition was more difficult immediately following higher numbers of Go stimuli. Also, brain regions implicated in inhibition (inferior frontal cortex and anterior cingulate gyrus) increased their activation to Nogo trials as a

function of the number of preceding Go trials, possibly indicating that these regions maintained the task or response demands from previous Go trials, which increasingly interfered with NoGo trials. Durston and colleagues did not examine facilitation, or sequence effects upon Go stimuli.

Nieuwenhuis et al. (2003) attempted a post-hoc examination of sequence effects on a sub-set of data in a visual Go/NoGo task. They examined first-order effects only, that is effects of the immediately preceding stimulus upon the next stimulus, and reported results for only one ERP component (N2). After NoGo trials, participants reportedly responded more slowly on Go trials and made fewer commission errors to NoGo trials than after Go trials. In a 50% Nogo condition, the N2 enhancement on Nogo trials was smaller on trials following a Nogo trial than on trials following a Go trial, possibly due to transient priming of the NoGo from the previous trial, or to reductions in mismatched negativity. The effect was not statistically significant, however, possibly due to limited statistical power. The study didn't examine effects in blocks where Go stimuli were predominant and NoGo stimuli rare (the typical NoGo task), and P3 effects were not considered, thus limiting conclusions about sequence effects in the Go/NoGo task. We are not aware of any previous studies of sequence effects upon ERP latencies in the Go/NoGo task.

1.3 *The current study*

The current study examined facilitation and inhibition in healthy adults, using ERP and behavioural measures in a modified Go/NoGo task. Go stimuli were analysed by serial position to investigate facilitatory effects (in speed/accuracy) with stimulus repetition within trains. Inhibitory load to NoGo stimuli was manipulated by varying the number of immediately preceding Go stimuli in a train. We predicted that with Go stimulus repetitions, priming would result in faster responses, greater accuracy, or reductions in amplitude/latency

of ERP components. We hypothesised that with increasing numbers of Go trials immediately preceding a NoGo trial, inhibition would be more difficult and would be indexed by increases in errors, RT, ERP amplitude changes (anteriorisation of the P3 or increased N2) or longer ERP latencies.

2. Method

2.1 Participants

Participants (N=20) were Psychology students who enrolled in the study for research participation credit. Thirteen were females. Eighteen were right-handed, and two were left-handed, according to self-report. All participants had normal or corrected to normal vision. Participants were asked to refrain from consuming products containing caffeine for two hours prior to testing. They reported to be free from past or present psychiatric or neurological disorders, head injuries involving unconsciousness, and were screened for psychopathology using the Brief Symptom Inventory (Derogatis and Melisaratos, 1983).

2.2 Stimuli

Stimuli were presented individually on a computer screen in white on a black background. They comprised a warning stimulus (exclamation mark), a Go stimulus (tick), and a NoGo stimulus (X). Two further stimuli were included for future comparisons of task-switching performance with clinical groups and are not examined here (X-Go and dedicated NoGo), following at the end of stimulus trains. Stimuli occurred in sequences or trains of 4-8 stimuli. (See Fig. 1). Trains commenced with a warning stimulus (WS), followed by between 1-4 Go stimuli (G-GGGG), followed by a NoGo stimulus (X). The X-NoGo stimulus was followed on 50% of trials by a repetition of the X-stimulus (because participants were required to respond to X-repetitions, this stimulus is termed X-Go) and on 50% of trials by a square

(dedicated Nogo stimulus). Thus a NoGo (N) stimulus occurred in each train, but because N was preceded by one or more Go-stimuli, the overall ratio of Go:NoGo stimuli was 14:4 or 69%:31%. Train types were equiprobable and presented in random sequence. Stimulus duration was 200 ms. ISI varied randomly between 1-3 s (mean 2 s) and trains were separated by relatively long intervals which varied randomly between 4-6 s. Overall 635 stimuli were presented and the duration of the experiment was approximately 17 minutes.

INSERT FIG. 1 ABOUT HERE

2.3 Procedure

Participants gave written informed consent before commencement and the University of Wollongong Ethics Committee approved the research protocol. During the experiment participants were comfortably seated in a dimly lit sound-attenuated room, 1 m from the computer screen, with a button-press device fixed to a chair arm next to their dominant hand. Participants made responses using the index finger of their dominant hand. Participants were instructed to focus their gaze on a fixation cross on the monitor in front of them and to press a response button “as quickly and accurately as possible” to all Go stimuli. Prior to commencing the experiment, participants completed a four minute practice run.

2.4 Electrophysiological recording

The EEG was recorded from 19 scalp electrodes (F1, F2, Fz, F3, F4, F7, F8, Cz, C3, C4, T3, T4, T5, T6, Pz, P3, P4, O1, O2) and referenced online to linked ears according to the international 10–20 system (Jasper, 1958) using tin electrodes in an electrode cap (Electro-cap International). The participant was grounded by a cap electrode located midway between Fpz and Fz. Vertical EOG was recorded from tin electrodes placed 1 cm above and below the

left eye, and electrodes placed beyond the outer canthus of each eye recorded horizontal EOG. Electrode impedances were kept below 5k Ω .

2.5 Data analysis

Mean RTs for correct responses, and percentage of errors to each stimulus type were calculated for each participant. Extreme scores (over two standard deviations from the participant's condition mean) were excluded from the analysis (Ratcliff, 1993), which represented 90 individual Go trials, or .01% of RT data overall. To examine facilitation effects, mean RTs and errors were analysed using an ANOVA with Stimulus type (G,GG,GGG,GGGG) as a repeated measures factor. Two planned contrasts were employed: A linear contrast determining whether Go stimulus repetitions were related linearly to RT, and a quadratic contrast comparing mid-train effects with early and late effects in trains to assess non-linear changes (e.g., floor or ceiling effects, or U-shaped functions) which have been reported in previous stimulus repetition paradigms.

To examine the effect of the number of preceding Go stimuli upon accuracy to NoGo stimuli, commission errors were analysed using a repeated measures ANOVA with NoGo as a function of the number of preceding Go stimuli within a train (GN, GGN, GGGN, GGGGN). As described above, planned contrasts assessed linear and quadratic effects of the number of preceding Go stimuli on NoGo stimuli.

The ERP epoch was defined as 100 ms prestimulus to 800 ms poststimulus. ERP data were amplified with EEG and EOG gains of 20,000 and 5000 respectively, digitised at a sampling rate of 512 Hz with a bandpass down 3 dB at 0.01 and 35 Hz, and filtered offline with a low pass zero phase shift filter at 30 Hz, 48 dB/ octave. Electrophysiological data were corrected for excessive eye movement using the Semlitsch (1986) procedure. Epochs containing artefacts exceeding +/- 100 μ V were automatically rejected using Neuroscan

software. The average number of trials entering the analysis by stimulus type was G: 57, GG: 45, GGG: 33, GGGG: 23 and NoGo: 54. Peak quantification involved the automatic identification of the maximum voltage within defined latency ranges, with manual confirmation. Five components were quantified from the individual participants' waveforms, with peak amplitudes determined relative to the 100 ms pre-stimulus baseline. Peaks were detected in specified channels where they generally showed maximal amplitude in the grand mean waveforms. For P1, this was electrode Pz and the search window was 50-120 ms. For N1, this was electrode O2 and the search window was 90–160 ms. For P2, this was electrode Pz from 150 to 210 ms. For N2, this was electrode Fz from 180 to 400 ms, and for P3 this was electrode Pz from 290 to 600 ms. Search windows were based on visual inspection of the grand mean waveforms.

For data analysis we focussed on 9 sites (F3, Fz, F4, C3, Cz, C4, P3, Pz, P4), an approach established in previous studies (e.g. Broyd et al., 2005, Dimoska and Johnstone, 2007, Dimoska and Johnstone, 2008, Dimoska et al., 2003, Johnstone et al., 2007, Johnstone et al., Johnstone and Clarke, 2009, Smith et al., 2004, Smith et al., 2006, Smith et al., 2008, Thomas et al., 2007, Watson et al., 2005) which reduces the number of statistical comparisons made while optimally allowing for differences in the anterior-posterior and hemispheric dimensions (Picton et al., 2000, Smith et al., 2004, Watson et al., 2005). Additionally, for N1 we included analyses at occipital electrodes (O1, O2) where this component was maximal.

ERP latencies were recorded as the time during the search window of maximal amplitude at the site where the component was quantified, and relative amplitude measures for all 11 electrodes were taken at the same post-stimulus latency (Picton et al., 2000).

ERP amplitudes were examined using ANOVAs. Firstly, the facilitation hypothesis was examined using a Stimulus type (G, GG, GGG, GGGG) x Sagittal plane (frontal,

central, parietal) x Lateral plane (left, midline, right) repeated measures design. For N1 amplitude, an additional ANOVA was conducted at occipital electrodes (O1, O2) with factors of Stimulus type (G, GG, GGG, GGGG) x Lateral plane (left, right). To compare ERPs to Go versus NoGo stimuli, ERPs to G-GGGG were averaged to form mean Go stimulus ERPs, which were compared to NoGo-X stimuli in a Stimulus type (Go versus NoGo) x Sagittal plane (frontal, central, parietal) x Lateral plane (left, midline, right) repeated measures ANOVA. For N1 amplitude, an additional ANOVA was conducted at occipital electrodes (O1, O2) with factors of Stimulus type (Go versus NoGo) x Lateral plane (left, right). To examine the effect of the number of preceding Go stimuli upon ERPs to NoGo stimuli, latencies and amplitudes were analysed using a repeated measures ANOVA with NoGo as a function of the number of preceding Go stimuli within a train (GN, GGN, GGGN, GGGGN) x Sagittal x Lateral factors as above. Finally, for N1 amplitude, an additional ANOVA was conducted at occipital electrodes (O1, O2) with factors of Stimulus type (GN, GGN, GGGN, GGGGN) x Lateral plane (left, right).

Two planned contrasts were performed on the within-subjects, Laterality factor (i) Left vs. Right electrodes, to determine between-hemisphere differences, and (ii) Midline vs. the mean of the Left and Right electrodes, to test for midline vs. lateral/peripheral topographic differences. The contrasts for the Sagittal factor were: Frontal vs. Parietal electrodes, to determine if components were frontally or parietally maximal; and Central vs. the mean of the Frontal and Parietal electrodes, to determine if components were relatively smaller/larger centrally. As the contrasts were planned and there were no more of them than the degrees of freedom for an effect, no Bonferroni-type adjustment was necessary (Tabachnick and Fidell, 1989). Also, single degree of freedom contrasts are not affected by violations of symmetry assumptions common in repeated measures analyses, and thus do not

require Greenhouse–Geisser type corrections. Following data analysis, ERP data were normalized using the vector scaling procedure (McCarthy and Wood, 1985), and interactions involving topography are reported only if they remained significant after normalization. All reported analyses and figures are of unscaled data.

3. Results

For clarity, results are organised to address key hypotheses. To maintain the focus on our hypotheses, only effects or interactions involving stimulus type are reported.

3.1 Facilitation

3.1.1 Behavioural correlates of facilitation: Responses to repetitions of Go stimuli (G-GGGG)

Accuracy of responding to Go stimuli was high (97%) and did not differ as a function of stimulus serial position (G-GGGG). With Go stimulus repetitions (across G-GGGG), RT initially decreased then increased with further repetitions (Linear: $F(1, 19) = .77, p = .392$; Quadratic $(1, 19) = 30.57, p < .001$: Fig 2). In order to investigate whether the reversal of facilitation with increasing train length was due to anticipatory effects (e.g. due to participants learning that the maximum number of consecutive Go stimuli in a train was four), we compared RTs early in the experiment with those late in the experiment. Similar quadratic functions were evident for RTs early and late in the experiment, including for RTs in the first train.

INSERT FIG. 2 ABOUT HERE

3.1.2 ERP correlates of facilitation: ERPs to repetitions of Go stimuli (G-GGGG)

Grand average ERP waveforms at central electrodes to Go stimuli (G, GG, GGG, GGGG) are shown in Fig. 3. Visual inspection of the waveforms (and Fig. 2) shows that P3 occurs earlier to Go stimulus repetitions (GG-GGGG) than to the first Go stimuli (G) in trains.

INSERT FIG. 3 ABOUT HERE

For P1 amplitude there was a significant Stimulus (quadratic) by Laterality (left versus right) interaction, $F(1, 19) = 5.22, p < .05$ (Fig. 4). P1 amplitude showed initial reductions in both hemispheres with stimulus repetitions, however right hemisphere amplitude of P1 showed a quadratic effect with increases after further repetitions. There was a significant Stimulus (linear) by Sagittal plane (frontal vs. parietal) interaction $F(1, 19) = 6.85, p < .05$ (Fig. 4). P1 amplitude to Go stimulus repetitions increased linearly at parietal sites and decreased linearly at frontal sites. There were no significant effects for N1 amplitude. With Go stimulus repetitions, N2 amplitude showed an overall linear increase ($F(1, 19) = 10.9, p < .01$), which was qualified, however, by an additional quadratic effect, with initial repetitions producing a reduction and subsequent repetitions producing increments (quadratic contrast: $F(1, 19) = 4.42, p < .05$; fig. 4). For P3 amplitude to Go stimuli there was a significant Stimulus (quadratic) by Laterality (L/R) interaction. With Go stimulus repetitions within trains, left hemisphere P3 amplitude showed little change, however right hemisphere P3 amplitude initially reduced then reached a plateau, $F(1, 19) = 12.4, p < .01$ (Fig. 4).

INSERT FIG. 4 ABOUT HERE

ERP latencies are shown in Table 1. There were marginal reductions in P1 latency with Go stimulus repetitions, $F(1, 19) = 3.34, p = .08$. N1 latency reduced linearly to Go stimulus repetitions within trains, $F(1, 19) = 7.91, p = .01$. P3 latency showed a significant quadratic effect of stimulus, following the pattern obtained for RT (Fig. 2), with Go stimulus repetitions producing initial latency reductions followed by increases, $F(1, 19) = 7.71, p < .05$,

INSERT TABLE 1 ABOUT HERE

3.2 Inhibition

3.2.1 Go versus NoGo stimuli

Average ERPS to Go (mean G to GGGG) versus NoGo stimuli are shown in Fig. 5.

INSERT FIG. 5 ABOUT HERE

A main effect of Stimulus indicated that P1 amplitude was larger to NoGo (3.2) than Go (1.7) stimuli, $F(1, 19) = 13.73, p < .01$. A Stimulus by Laterality interaction indicated that N1 amplitude was greater at lateral (vs. midline) sites, with this difference being greater for NoGo (Lateral: Midline = 4.4: 3.5) compared to Go stimuli (lateral: midline = 2.3: 1.9), $F(1, 19) = 10.57, p < .05$. At the occipital electrodes, a main effect of Stimulus indicated that N1 amplitude was larger to NoGo (-9.4) than Go (-6.3) stimuli, $F(1, 19) = 15.12, p < .01$. N2 amplitude overall was larger to NoGo (1.7) than Go (3.2) stimuli, $F(1, 19) = 5.69, p < .05$, indicated by a main effect of Stimulus. Stimulus by Sagittal plane (linear and quadratic) interactions indicated that, although P3 amplitude was maximal at parietal sites for both Go and NoGo stimuli, P3 amplitude (locked to the time of maximum amplitude at Pz) showed a stronger frontal effect for NoGo stimuli (frontal: parietal = 2.5: 2.9) than it did for Go-stimuli (1.1: 1.8; i.e. a classic NoGo anteriorisation effect), $F(1, 19) = 4.59, p < .05$, and a greater

central (versus fronto-parietal) effect to NoGo (Central: fronto-parietal = 4.1: 2.8) than Go stimuli (2.1: 1.5), $F(1, 19) = 23.22, p < .001$. A Stimulus by Lateral plane interaction indicated that P3 amplitude to NoGo stimuli showed a stronger Right > Left effect (Right: left = 3.3: 2.8) than P3 amplitude to Go stimuli (1.6: 1.5), $F(1, 19) = 6.55, p < .05$.

Latencies: Latencies of four components were significantly faster to Go than NoGo stimuli: for N1, 119 vs. 123 ms, $F(1, 19) = 4.44, p < .05$; for P2, 192 vs. 209 ms, $F(1, 19) = 6.38, p < .05$; for N2, 303 vs. 312 ms, $F(1, 19) = 4.93, p < .05$; and for P3, 355 vs. 394 ms, $F(1, 19) = 4.92, p < .05$.

3.2.2 Inhibition as a function of the number of Go preceding NoGo stimuli

Accuracy of responses to NoGo stimuli (NoGo-X) was high (92%). As the number of Go stimuli preceding NoGo stimuli increased, there was a linear reduction in commission errors, $F(1, 19) = 13.7, p < .01$. Closer examination reveals that this effect was driven by reductions of errors only where three or four Go stimuli preceded NoGo stimuli. Average ERPS to NoGo stimuli as a function of the number of preceding Go stimuli are shown in figure 6.

INSERT FIGURE 6 ABOUT HERE

INSERT FIGURE 7 ABOUT HERE

For N1 amplitude a Stimulus (linear) by Sagittal plane (quadratic) interaction indicated that as the number of preceding Go stimuli increased, N1 amplitude to NoGo stimuli became reduced at central relative to fronto/parietal electrodes, $F(1, 19) = 5.23, p < .05$ (Fig. 7). N1 amplitude at occipital electrodes showed a Stimulus (quadratic) by Lateral (left vs. right) effect, with larger left hemisphere N1 to NoGo stimuli occurring early and late in trains, $F(1, 19) = 11.35, p < .01$. As the number of Go preceding NoGo stimuli increased,

N2 amplitude showed an overall linear reduction, $F(1, 19) = 10.27, p < .01$. This effect was qualified in that N2 amplitudes initially increased then reduced with greater numbers of Go preceding NoGo stimuli (quadratic contrast), $F(1, 19) = 6.29, p < .05$ (Fig. 7). With increasing Go stimuli preceding NoGo stimuli, N2 amplitude showed a greater central relative to fronto/parietal distribution, with this pattern reversing with further stimulus repetitions $F(1, 19) = 6.0, p < .05$ (Fig. 7). As the number of preceding Go stimuli increased, P3 amplitude to NoGo stimuli decreased, $F(1, 19) = 7.28, p < .02$ (Fig. 7). The quadratic trend suggested in Figure 7 was not significant ($p > .05$).

Latencies are shown in Table 1. With increasing Go preceding NoGo stimuli, a pattern of increasing latencies was observed for N1, P2 and N2 components; P3 did not evidence this pattern. More specifically, N1 latencies increased as a function of Go preceding NoGo stimuli, although this pattern was not statistically significant¹. P2 latency to NoGo stimuli increased marginally as the number of preceding Go stimuli increased, $F(1, 19) = 4.13, p = .056$. There was a non-significant quadratic trend to reduced P2 latency towards the end of longer stimulus trains, $F(1, 19) = 3.67, p = .07$. N2 latency to NoGo stimuli increased significantly as the number of preceding Go stimuli increased, $F(1, 19) = 12.9, p < .01$. As the number of preceding Go stimuli increased, P3 latency to NoGo stimuli initially increased then reduced with longer trains (Linear: $F(1, 19) = 5.27, p < .05$; Quadratic: $F(1, 19) = 4.95, p < .05$).

4. Discussion

An important contribution of this study is to demonstrate that RTs and ERPs to Go stimuli in the Go/NoGo task vary systematically as a function of the number of immediately

¹ In a previous version of the data analysis, where N1 was quantified at electrode Pz, the observed linear increase in N1 latencies as a function of the number of Go preceding NoGo stimuli was statistically significant, $F(1, 19) = 5.03, P < .05$.

preceding Go stimuli, with indications of facilitation, or repetition priming, present to Go stimuli preceded by other Go stimuli. We also replicated conventional (N2 and P3 amplitude) changes often attributed to inhibition in the Go/NoGo task, and demonstrated additional correlates of inhibition which vary as a function of the number of Go stimuli preceding NoGo stimuli in experimental sequences. We found evidence of increased inhibitory load where NoGo stimuli were preceded by greater numbers of Go stimuli. These systematic effects of Go repetitions on ERPs and RTs to both Go and Nogo stimuli remain hidden when ERPs are derived from averages computed across all Go/ Nogo stimuli. Additionally, to our knowledge, this is the first study to report the effects of repetition upon Go stimuli in the NoGo task, and the first ERP study of higher-order sequence effects in the Go/NoGo task.

4.1 Stimulus repetition and facilitation effects on the Go-stimulus

With Go stimulus repetitions, both P1 and N1 latencies reduced linearly, with these effects being marginal for P1 and highly significant for N1. Previous ERP studies report reduced latencies of P1 and N1 in conditions of facilitation. P1 (Lobaugh et al., 2005, Taylor, 2002) and N1 (Lobaugh et al., 2005) latencies, for example, are shorter in visual search conjunction tasks where multiple features or colour pop-outs speed the search process, and P1 latencies are shorter when direction of eye gaze in a face picture cue is congruent rather than incongruent with a target location (Schuller and Rossion, 2004). Shorter N1 latencies have also been reported in tasks requiring less attentional effort, compared to demanding tasks, interpreted as an indication that N1 latency is related to general effort at processing (Callaway and Halliday, 1982).

Increase in frontal P1 has been reported in active tasks (e.g. in tasks requiring responding and withholding responses) relative to passive tasks, interpreted as possibly due to the effects of attention or general arousal (Potts, 2004). Conversely, decrease of P1

amplitude at frontal relative to parietal sites in the current study with Go stimulus repetitions may indicate reductions in attention/arousal with stimulus repetitions. The current P1 and N1 results indicate facilitatory effects with Go stimulus repetitions including speeded processing and decrease in effortful processing during relatively early, sensory and attentional, stages of stimulus processing.

With Go stimulus repetitions, N2 amplitude, P3 latency and RT showed quadratic patterns, with reductions as predicted to initial repetitions in trains, and a reversal of this pattern towards the end of longer stimulus trains. As the same reversal of higher-order effects occurred with our manipulations of inhibition, these are likely due to common mechanisms associated with serial position effects, which will be discussed below. The results indicated that when effects of stimulus sequences are considered the Go-P3 latency is closely time-related to the response, supporting the relation of the P3 to overt responses (Kutas et al., 1977, Verleger et al., 2006), patterns which may be missed in typical analyses, .

In summary, Go stimulus repetitions resulted in linear decreases in P1 and N1 latencies, and reduction of frontal relative to parietal P1 amplitude, consistent with facilitation effects. RTs, right hemisphere P1, N2 and P3 amplitudes showed a quadratic pattern, with strong reductions for the first and second repetitions in trains, and effects reversing with longer stimulus trains. The quadratic patterns are consistent with facilitation for initial stimulus repetitions which diminishes with higher-order Go repetitions, possibly due to serial position effects.

4.2 Inhibitory load as a function of the number of Go stimuli preceding NoGo stimuli

Firstly, we replicated the well-known phenomena of larger N2 amplitude and anteriorisation of the P3 component to NoGo versus Go stimuli, linked to inhibition in past studies (e.g. Eimer, 1993, Falkenstein et al., 1999, Fallgatter and Strik, 1999, Jodo and

Kayama, 1992). Our analysis also demonstrated additional correlates of inhibition which vary systematically as a function of sequential context, specifically the number of Go preceding NoGo stimuli.

N1, P2 and N2 latencies to NoGo stimuli increased as a function of preceding Go stimuli, with effects for N1 being significant at electrode Pz, P2 being marginally significant, and significant effects for N2. Processing decrement (in speed or accuracy) resulting from previous stimulation is a defining feature of inhibition (Buckner et al., 1998, Klein, 2000, Posner and Snyder, 1975). Longer latencies to NoGo than Go stimuli have also been previously interpreted as a sign of higher processing demands in the NoGo-condition (Fallgatter and Strik, 1999, Salisbury et al., 2004). Previous studies have suggested that latency rather than amplitude measures were key to inhibitory processes in the Go/NoGo task, with peaks of the N2 and P3a/P3b being significantly earlier for correct than failed withholds (Roche et al., 2005).

Neither of the two previous studies examining sequence effects in the Go/NoGo task reported latency analyses (Durstun et al., 2002, Nieuwenhuis et al., 2003), however one (Nieuwenhuis et al., 2003) did report slower RT to Go stimuli following a NoGo. In the current study, increases of N1, P2 and N2 latencies suggest higher processing demands and increased inhibitory load to NoGo as a function of preceding Go stimuli (Fallgatter and Strik, 1999, Salisbury et al., 2004). The results also support previous observations that although the primary focus in inhibition research has been the N2/P3 complex, earlier waveform components such as the N1 & P2 may play a major role in inhibitory processes (Johnstone et al., 2009, Roche et al., 2005). This may indicate that an important correlate of inhibitory processing in the NoGo task (processing speed) has largely been overlooked, as previous studies have not examined ERP latencies to NoGo stimuli as a function of serial position.

Manipulating inhibitory load to NoGo stimuli may allow the study of inhibition which bypasses some of the methodological problems in comparing Go with NoGo stimuli (e.g. additional variability due to differing response requirements between stimuli). Measures traditionally linked to inhibition in the Go/NoGo task as yet provide an incomplete and somewhat controversial picture of electrophysiological indices of inhibitory processes (e.g. Falkenstein, 2006, Falkenstein et al., 1999). It is therefore of interest that in the current study, where sequence effects are taken into account, ERP latencies rather than amplitudes appear to be more sensitive correlates of inhibitory load.

As with the facilitation effects, several quadratic effects were present in the inhibition data with longer stimulus trains. With initial increases in the number of Go stimuli preceding NoGo stimuli, P3 latency increased, as did N2 amplitude, consistent with increased inhibitory load (Bruin and Wijers, 2002, Jodo and Kayama, 1992, Roche et al., 2005). As the number of preceding Go stimuli increased, however, these effects reversed, contrary to predictions. Errors initially showed no change then reduced with longer stimulus trains, contrary to predictions. The higher-order effects for P3 latency and N2 amplitude in the facilitation and inhibition manipulations showed strikingly converse patterns, suggesting that a common mechanism or mechanisms may underlie the reversal of experimental effects towards the end of longer stimulus trains.

Research in this area is extremely limited, however Nieuwenhuis and colleagues (2003) found that an N2 enhancement observed in Go trials during a frequent (80%) NoGo condition was marginally less pronounced on trials following a Go trial than on trials following a Nogo trial. First-order results of the current study are consistent with Nieuwenhuis's for first-order effects, however Nieuwenhuis didn't examine higher-order effects and we are aware of no previous Go/NoGo studies with which to compare the higher-order results.

One interpretation of the current results is that participants may have learned that the maximum number of consecutive Go stimuli in a train was four, and therefore anticipated a change of stimulus by the third or fourth Go repetition. This could have led to reversal of facilitation (including increased RT, P3 latency and N2 amplitude) where another Go stimulus occurred, due to expectancy violations. Conversely, where a NoGo stimulus occurred after three or more Go stimuli, if it were anticipated, this could explain the faster P3 latency, increased accuracy and reduced inhibitory load (N2 amplitude) to NoGo after longer runs of Go stimuli. This possible interpretation is tempered by the fact that some quadratic effects were apparent from the first experimental sequence, however participants completed a practice prior to commencing the task which may have allowed for learning. Some quadratic patterns were also present in amplitudes of relatively early (P1, N1 & N2) components, less commonly associated with anticipatory effects than the P3, however some studies have demonstrated that even the P1 is sensitive to task demands as well as stimulus qualities (Taylor, 2002) hence it may possibly be affected by top-down processes such as anticipation. The issue of the sensitivity of components earlier than the P3 to expectancy effects has theoretical implications for cognitive science in general and it might be worthwhile for future research to examine this issue in a more systematic manner. Further studies varying the complexity of stimulus sequences, for example, or examining contingent negative variation, may enlighten the expectancy issue.

Alternatively, the U-shaped pattern could be the product of two processes: a) a linear facilitation effect for Go stimulus repetitions or an inhibitory effect with increasing Go preceding NoGo stimuli at the beginning of trains and b) a serial position effect for later items in the stimulus train. Participants may, for example, be optimally prepared to respond

to items soon after the warning signal, with this optimal state-of-readiness diminishing during the latter stages due to changes in attentional or other resources.

With Go-repetitions, P3 amplitude to the Nogo stimulus reduced (GN>GGN>GGGN). In other experimental tasks, however, a pattern of increasing P3s to non-targets is typically observed, (TN<TTN<TTTN; Gonsalvez et al., 1999, Johnson and Donchin, 1980). In the absence of additional research, it is difficult to be certain that the P3 amplitude changes observed here reflect inhibitory load. One possibility is that both sequential (e.g., facilitation and inhibition) and serial position effects influence ERPs to stimuli when they are presented in trains, with these effects combining in an additive or interactive manner. Thus, reduced amplitudes (e.g., N1, P3) observed to NoGo stimuli later in the train might reflect decreasing stimulus salience or changes in attentional, memory or other processing resources. Although stimulus salience (Johnson, 1993) and attentional resource allocation (Polich, 2007) are known to affect amplitudes including the P300, it is unclear whether serial position is a reliable determinant of these mechanisms, making this explanation tentative at present. It is also possible that latency measures (as compared with amplitude measures) may be more sensitive and reliable indices of inhibition and inhibitory load, at least within the context of the Go/NoGo paradigm.

In summary of the inhibition results, increases in Go preceding NoGo stimuli resulted in incremental increases in N1, P2 and N2 latencies, consistent with higher processing demands and greater inhibitory load. The prolongation of N1, P2, and N2 latencies with increasing difficulty of inhibition supports the relation of N2 to inhibition, but also is consistent with hypotheses that earlier (N1, P2) components may also related to inhibition (Johnstone et al., 2009, Roche et al., 2005). The amplitude of N1, N2, P3 and the latency of P3 showed quadratic patterns. N2 and P3 amplitude also showed an overall linear reduction

as a function of increasing Go before NoGo stimuli. These results were unexpected, and suggest a reversal of predicted patterns with higher order effects towards the end of long stimulus trains. As this is to our knowledge the first ERP study of higher-order sequence effects in the Go/NoGo task, further research manipulating experimental parameters (ISI, inter-train interval, train length and sequences of stimuli) is required before conclusive interpretations can be made about higher-order effects.

Conclusions

The current study adopted a novel Go-NoGo paradigm to manipulate facilitation and inhibitory load in order to determine their ERP correlates. Our replication of well-established Go/NoGo effects suggests that the stimulus train paradigm is in some important ways comparable with other Go/NoGo presentation formats. Additionally, the results compellingly suggest that sequential context affects ERPs to both Go and NoGo stimuli. Correlates of facilitation included shorter P1 and N1 latencies and topographic effects in P1. Correlates of inhibitory load with increasing numbers of Go preceding NoGo stimuli included increased N1, P2 and N2 latencies. The current study also raises the possibility that mechanisms other than facilitation and inhibition (e.g., expectancy and serial position) influence ERPs in the Go-NoGo task. In Go/NoGo tasks where sequence effects are overlooked, facilitation and inhibition effects may be confounded, particularly given the typically unequal repetitions between Go and NoGo stimuli. The results are of importance to investigations of clinical participants, in particular those with OCD in which both inhibitory and facilitatory information-processing anomalies may occur. We are currently analysing data from clinical groups in a similar experimental design.

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Table 1: ERP latencies

Stimulus	ERP latencies				
	P1	N1	P2	N2	P3
G	97.6 (18)	125.2 (11)	193 (32)	248.9 (23)	370.5 (44)
GG	97.5 (20)	117.2 (19)	194 (34)	239.4 (23)	346.9 (34)
GGG	87.5 (17)	116.8 (12)	191.8 (31)	241.4 (24)	343.5 (41)
GGGG	91.5 (22)	115.3 (19)	192.1 (29)	239.1 (21)	359.6 (37)
GN	95.7 (22)	123.5 (14)	185.6 (19)	251 (25)	388.8 (41)
GGN	92.5 (20)	125.5 (17)	194.8 (23)	256.5 (29)	401.6 (37)
GGGN	96.8 (17)	129.5 (17)	200.3 (22)	277.4 (29)	394.3 (61)
GGGGN	94.7 (24)	129.4 (22)	197.2 (25)	276 (36)	368.7 (45)

Table 1: Mean latencies of ERP components by stimulus type, in milliseconds (standard deviations are in parentheses).

Figure 1

Figure 2

Figure 3

Figure 4

Figure 5

Figure 6

Figure 7