

The Level of Reaction Time Determines the ERP Correlates of Auditory Negative Priming

Susanne Mayr, Michael Niedeggen, Axel Buchner, and Guido Orgs

Institute of Experimental Psychology, University of Düsseldorf, Germany

Abstract. Responding to a stimulus that had to be ignored previously is usually slowed-down (negative priming effect). This study investigates the reaction time and ERP effects of the negative priming phenomenon in the auditory domain. Thirty participants had to categorize sounds as musical instruments or animal voices. Reaction times were slowed-down in the negative priming condition relative to two control conditions. This effect was stronger for slow reactions (above intraindividual median) than for fast reactions (below intraindividual median). ERP analysis revealed a parietally located negativity of the negative priming condition compared to the control conditions between 550–730 ms poststimulus. This replicates the findings of Mayr, Niedeggen, Buchner, and Pietrowsky (2003). The ERP correlate was more pronounced for slow trials (above intraindividual median) than for fast trials (below intraindividual median). The dependency of the negative priming effect size on the reaction time level found in the reaction time analysis as well as in the ERP analysis is consistent with both the inhibition as well as the episodic retrieval account of negative priming. A methodological artifact explanation of this effect-size dependency is discussed and discarded.

Keywords: auditory processing, negative priming, attention, ERPs, reaction times, median split analysis

Introduction

The so-called negative priming effect is conventionally defined as the difference in reaction times between trials on which a previously ignored stimulus has to be selected for responding and trials on which the same stimulus has to be selected without having been ignored on the preceding trial. Typically, reactions to previously ignored stimuli are slowed down (for a review, see Fox, 1995). The attempt to explain this phenomenon has elicited abundant empirical research within the last decades. However, it was not until recently that studies trying to identify physiological correlates of this phenomenon have appeared (Mayr, Niedeggen, Buchner, & Pietrowsky, 2003; Steel et al., 2001). Whereas Steel et al. (2001) chose a fMRI approach in a Stroop-like task, Mayr et al. (2003) measured ERP correlates in an auditory negative priming paradigm. Steel et al. (2001) found a widespread cortical network activated in the negative priming condition involving the inferior parietal, the left temporal, and the frontal lobes. This pattern of results could not directly falsify any of the possible theoretical accounts of negative priming such as the inhibition account (Tipper, 1985) or the episodic retrieval account (Neill & Valdes, 1992; Neill, Valdes, Terry, & Gorfein, 1992). The ERP study of Mayr et al. (2003) yielded two notable features. First, given that for diverse inhibition-associated tasks frontally located components

have been found (Eimer, 1993; Falkenstein, Hoormann, & Hohnsbein, 1999; Heil, Osman, Wiegmann, Rolke, & Hennighausen, 2000; Jodo & Kayama, 1992; Liotti, Woldorff, Perez, & Mayberg, 2000; Markela-Lerenc et al., 2004), an inhibition-based negative priming effect should be reflected in frontally located ERP correlates. However, the ERP data did not reveal any frontally located negative priming effect. Second, there was a negative priming correlate in a time range between 300–450 and 450–600 ms poststimulus. The probe mean amplitude of the negative priming condition was relatively more negative at parietal sites than that of the control conditions. Because of its polarity, time course, and topography this effect was interpreted as a reversed “old/new” effect. Old/new effects are usually obtained in studies on recognition memory (Rugg & Doyle, 1994). They denote a more positive-going parietal ERP derivation extending from 300 to 800 ms following the onset of an old compared to a new item. Among others, ERP old/new effects have been interpreted as the correlate of a gain in familiarity caused by intraexperimental repetition (Rugg, 1990; Rugg & Doyle, 1994). The increase in an event’s relative familiarity, in turn, has been associated with more fluent processing of an event (Johnston, Dark, & Jacoby, 1985). Therefore, the finding of reduced positivity when repeating a previously ignored stimulus, as in the negative priming condition of the Mayr et al. (2003) study, was interpreted as functionally equivalent to the less fluent processing of a novel stimulus.

Overall, the results of Mayr et al. (2003) were clearly not consistent with a frontal inhibition mechanism, but the parietal component was compatible with a memory-based retrieval mechanism and, therefore, the finding was related to a memory-based explanation of negative priming such as the episodic retrieval account. Following this approach (Neill & Valdes, 1992; Neill et al., 1992) the negative priming effect reflects the incongruity between the appropriate response to the actual probe target stimulus and the retrieved nonresponse information to the same stimulus when it was the distractor in the preceding prime episode. Note however, that the results of Mayr et al. (2003) are also compatible with a distractor inhibition account that does not assume a frontally located mechanism, for example Tipper's (2001) most recent reconciliatory account in which he conceives of an episodic retrieval process accessing tags but also inhibitory states.

Neill and Westberry (1987) reported that the negative priming effect critically depends on whether the instructions ask participants to sacrifice speed for accuracy or vice versa. Participants instructed to stress accuracy were slower overall and showed the negative priming effect, whereas participants who were told to sacrifice some accuracy for greater speed were indeed faster but did not show any negative priming effect. According to Neill and Westberry (1987) the lack of negative priming in the speed condition may be the result of an incomplete development of suppression. They suggested that there was no further need to build up inhibition after the prime motor response has been emitted.

Similarly, experimental manipulations leading to an increase in the overall reaction-time level have been found to increase the effect size of negative priming in terms of reaction-time differences between negative priming and control trials. Yee, Santoro, Grey, and Woog (2000) observed increased negative priming when the target selection required conceptual instead of perceptual stimulus processing, the former of which yielded significantly larger reaction times than the latter.

If we accept that the response-time based negative priming effect may be positively related to the absolute reaction-time level in reaction time experiments, it seems reasonable to examine the influence of the absolute response-time level on ERP correlates of negative priming as well. If the ERP correlate found by Mayr et al. (2003) is causally related to the processes resulting in reaction-time negative priming effects, this ERP effect should be larger in conditions where reaction-time negative priming is increased. This was done in the present study, which was planned as a replication of Mayr et al. (2003) but with increased statistical power ($N = 30$ instead of $N = 15$). We ran the negative priming experiment as in Mayr et al. (2003) but additionally analyzed the ERP data separately for slow and fast responses.

Methods

Participants

EEG recordings and behavioral data were obtained from 35 persons who were naïve as to the purpose of the experiment. Five data sets were excluded from the analyses because of extremely strong alpha activity. The remaining 30 participants (15 female, 15 male, aged between 19 and 36, $M = 25.7$ years, $SD = 4.27$) had no history of neurological disorders or hearing disabilities.

Stimuli and Procedure

The stimuli were digitized sounds, three of which were played by musical instruments (piano, guitar, and cornet), and three were animal sounds (frog, bird, and hen). Each sound was 300 ms long including rise and fall times. The sounds were presented via headphones (KOSS KSC 50) at approximately 70 dB SPL.

A monaural 20 ms metronome click cue indicated the ear (left or right) at which the to-be-attended sound would be presented. After a 750 ms cue-target interval, the prime pair of sounds (the target sound to the cued ear, the distractor to the other ear) was presented. Participants reacted by pressing the "animal" or "instrument" key on a VSG 2/5 response box (Cambridge Research Systems), depending on the category of the target. A sagittal response key arrangement prevented spatial compatibility effects between the target's location and the required response. Reactions had to be accomplished within 1500 ms after stimulus onset. Following a 1500 ms interval after the reaction, the probe cue was presented to the ear opposite to that of the prime cue. The temporal parameters and the participants' task were identical for the prime and probe sound pairs. Participants received no feedback about the correctness of their responses. They initiated the presentation of the next prime-probe pair by an arbitrary key press.

Three basic types of prime-probe trials defined the experimental conditions: In 72 negative priming (NP) trials, the ignored prime was repeated as the to-be-attended probe sound; the remaining two sounds differed. In 72 standard control (SC) trials, all prime and probe sounds were different. In order to control for ERP effects evoked by stimulus repetition, we introduced 72 repetition control (RC) trials in which the previously attended sound became the to-be-ignored sound in the probe; the remaining two sounds differed.

The 216 negative priming, standard control, and repetition control trials were parallel in that the response categories of the prime target and prime distractor were different (Buchner, Zabal, & Mayr, 2003). Because of the nature of the negative priming trials, the same was true for the prime and probe response categories. To control for response bias, 216 filler trials were included in which the prime and probe target categories were identical. Examples of the stimulus

Figure 1. Stimulus configuration examples for the different experimental conditions. The target sound is printed in **bold** font, the distractor sound in normal font. The single letters “L” and “R” indicate the ear (left, right) to which the sound was presented. Technical details of auditory presentation are given in the text.

| | <i>Prime</i> | <i>Probe</i> |
|----------------------------------|------------------------------|------------------------------|
| <i>Negative priming:</i> | L: Frog R: Piano | |
| <i>Standard control:</i> | L: Bird R: Piano | |
| <i>Repetition control:</i> | L: Bird R: Trumpet | L: Frog R: Trumpet |
| <i>Filler trials repetition:</i> | L: Trumpet R: Bird | |
| <i>Filler trials standard:</i> | L: Piano R: Bird | |

configuration are given in Figure 1. The sequence of 432 trials (72 negative priming, 72 standard control, 72 repetition control, and 216 filler) was randomized for each participant.

The instructions emphasized correctness, but reactions were also to be made as quickly as possible. Participants were told that exceeding the 1500 ms time-out interval would count as an error. To reduce false classifications of sounds, about 50 trials were presented in a training session preceding the main experiment.

EEG Recording and Data Analysis

An elastic cap with predefined electrode positions (Falk-Minow-Services, Munich) was mounted on the participant's head. The 30 active silver-silver chloride electrodes were referenced to linked mastoids, with impedance kept below 5 k Ω . Vertical and horizontal EOGs were recorded to control for ocular artifacts. Biosignals were recorded continuously (EEG-8 amplifiers, Contact Precision Instruments, London), sampled at 250 Hz, and online band pass-filtered (0.03 to 200 Hz). Offline, EEG data were segmented according to the sound onset in each trial (–200 to 1000 ms epoch length), filtered (0.5–40 Hz, –48 dB cut-offs), and baseline corrected (–200 to 50 ms). Single EEG sweeps containing muscular or ocular (vEOG, hEOG) artifacts were excluded from the analysis by help of a semi-automated routine. The remaining sweeps were averaged according to the stimulus type (prime, probe), experimental condition, and electrode position. ERP responses evoked in filler trials, as well as the ERPs evoked by primes will not be discussed in detail, but note that prime ERPs did not differ between experimental conditions. For each participant, ERP data were subjected to a median-split: Averages were calculated for *fast* (below the participant's median reaction time) as well as for *slow* (above the participant's median reaction time) trials.

Two time windows were determined as regions of interest (300–400 ms, 550–730 ms). Localization of the latter window was based on the temporal characteristics of the parietal effect found by Mayr et al. (2003). The exact determination was adjusted to the grand averages that revealed a similar effect but in a somewhat shifted time slot. The early window was exclusively based on the grand averages that revealed the most distinct difference between negative priming (NP) and the control conditions (SC and RC) in this temporal interval. Mean amplitudes within these windows were computed for each participant, experimental condition, electrode, and as a function of whether the reaction time in a trial was fast (below median) or slow (above median). Topographical effects were analyzed by splitting the electrodes according to spatial variables, that is, caudality (anterior, medial, posterior) and laterality (left, central, right).

Design

The main independent variable for the behavioral data was the experimental condition (negative priming, standard control, repetition control). For the ERP data, the design also included the lateral electrode position (left, central, and right laterality) as an independent variable. This 3 \times 3 design with experimental condition and laterality was considered separately for the three levels of the caudality variable (anterior, medial, posterior) and the two different time windows (300–400 ms, 550–730 ms). Both the reaction times and the ERP data were also analyzed for fast (below-median) and slow (above-median) reactions. A multivariate approach was used for all repeated measures analyses. Beside the exact F statistic, partial η^2 is reported as an effect size measure. For *post hoc* tests comparing two levels of an independent variable, d_z is reported as standardized effect size measure.

Results

Reaction Times and Errors

Overall, the mean reaction times were largest for the negative priming condition, intermediate for the standard control, and fastest for the repetition control condition. However, the difference between the latter two conditions was small (see Figure 2). A MANOVA with experimental condition as independent variable showed that the differences among the three conditions were statistically significant, $F(2, 28) = 14.74$, $p < .001$, $\eta^2 = .51$. *Post hoc* comparisons confirmed that reaction times were significantly longer in the negative priming condition as compared to the standard control, $\Delta 29$ ms, $t(29) = 5.10$, $p < .001$, $d_z = 0.93$, and to the repetition control, $\Delta 34$ ms, $t(29) = 4.66$, $p < .001$, $d_z = 0.85$. Standard and repetition control conditions did not dif-

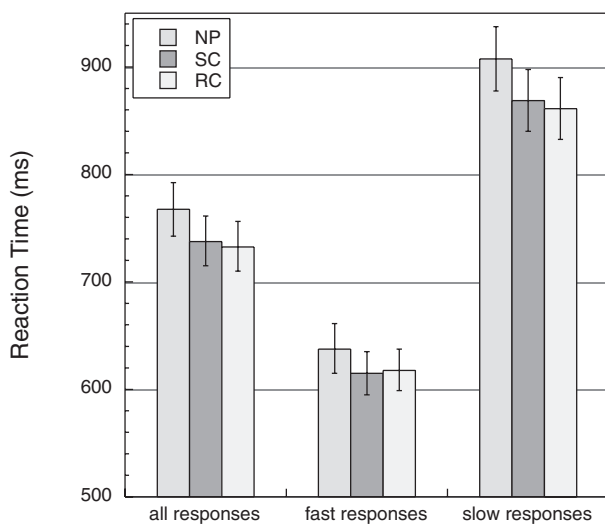


Figure 2. Bar diagrams summarizing the behavioral data. Mean reaction times for the overall responses (left), the fast responses (below median, middle), and the slow responses (above median, right) are shown for the negative priming (NP), the standard control (SC), and the repetition control (RC) condition, respectively. Error bars depict the standard errors.

fer significantly, $\Delta 5$ ms, $t(29) = 0.76$, $p = .456$, $d_z = 0.14$. All reaction time analyses were also run with median reaction-time data, which did not affect the statistical conclusions.

Error rates also differed significantly among the experimental conditions, $F(2, 28) = 3.75$, $p = .036$, $\eta^2 = .21$, with 5.37% ($SE = 1.00$) errors in the negative priming condition, 3.36% ($SE = 0.63$) errors in the standard control, and 3.14% ($SE = 0.57$) errors in the repetition control, respectively. *Post hoc* tests revealed that significantly more errors were committed in the negative priming condition compared to the standard control, $\Delta 2.01\%$, $t(29) = 2.72$, $p = .011$, $d_z = 0.5$, and compared to the repetition control, $\Delta 2.23\%$, $t(29) = 2.56$, $p < .016$, $d_z = 0.47$). The difference between the standard control and the repetition control was not significant, $\Delta 0.22\%$, $t(29) = 0.43$, $p = .668$, $d_z = 0.08$. The error data ran parallel to the reaction time data in that longer reaction times were accompanied by more errors. Therefore, a speed-accuracy trade-off can be ruled out.

In order to analyze the priming effect as a function of reaction time level, trials were split, separately for each participant, on the basis of the individual median reaction time (see Figure 2). On average, fast (below median) responses took 624 ms ($SE = 20$), whereas slow (above median) responses had a latency of 879 ms ($SE = 29$). Trivially, this difference was significant, $F(1, 29) = 488.52$, $p < .001$, $\eta^2 = .94$. The significant interaction between reaction time level and experimental condition, $F(2, 28) = 4.29$, $p = .024$, $\eta^2 = .23$, was far more relevant for our concerns because it implies that priming effects were modulated by the overall reaction-time level, which necessitates detailed

analyses of the negative priming effects for fast and slow reaction times.

Statistical analysis of the fast responses revealed statistically significant differences among the three conditions, $F(2, 28) = 6.64$, $p < .004$, $\eta^2 = .32$, which reflect an increase in mean reaction time in the negative priming condition (NP vs. SC (standard control): $\Delta 23$ ms, $t(29) = 3.66$, $p = .001$, $d_z = 0.67$, NP vs. RC (repetition control): $\Delta 20$ ms, $t(29) = 2.93$, $p = .007$, $d_z = 0.54$). The difference between the standard control and the repetition control condition was again not significant, $\Delta -3$ ms, $t(29) = -0.57$, $p = .575$, $d_z = 0.10$.

Similarly, the conditions differed significantly for the slow responses, $F(2, 28) = 14.72$, $p < .001$, $\eta^2 = .51$. However, the increase in reaction time in the negative priming condition over the control conditions was even larger for the slow responses (NP vs. SC: $\Delta 38$ ms, $t(29) = 4.94$, $p < .001$, $d_z = 0.90$, NP vs. RC: $\Delta 46$ ms, $t(29) = 4.66$, $p < .001$, $d_z = 0.85$). As for the fast responses, differences between standard control and repetition control were not significant for the slow responses, $\Delta 8$ ms, $t(29) = 0.91$, $p = .369$, $d_z = 0.17$. Note that both in terms of raw reaction-time differences and in standardized effect sizes the NP effect was much larger in the slower than in the faster reaction-time data.

Analysis of the error rates could not be conducted for the median-split data because the splitting procedure was applied to valid responses only.

ERP Data

On average, approximately 30% of the EEG sweeps had to be excluded because of artifacts or response errors. For the analysis of the whole reaction-time range, individual mean amplitudes calculated for each cell of the experimental design were based on about 50 sweeps. Accordingly, there were about 25 sweeps per cell to calculate the mean amplitudes in each of the two median-split analyses.

In the statistical analysis of the ERP data, effects were separately tested for anterior, medial, and posterior electrode clusters because the topographical distribution indicated that effects were focused at these regions. In each analysis, the experimental condition (NP, SC, and RC) and the laterality of electrode position (left, medial, and right) were considered, resulting in a 3×3 repeated measure analysis of variance. The interaction between these factors was not significant so that only main effects of experimental condition will be reported.

At first, ERP data were analyzed for the whole reaction-time range. According to our previous findings (Mayr et al., 2003) we expected negative priming to be characterized by a diminished ERP positivity extending from 300 to 600 ms at posterior leads. As shown in Figure 3, a corresponding ERP effect in a comparable time window (550–730 ms) was only marginally expressed in the data: Although the grand averaged ERP data for the neg-

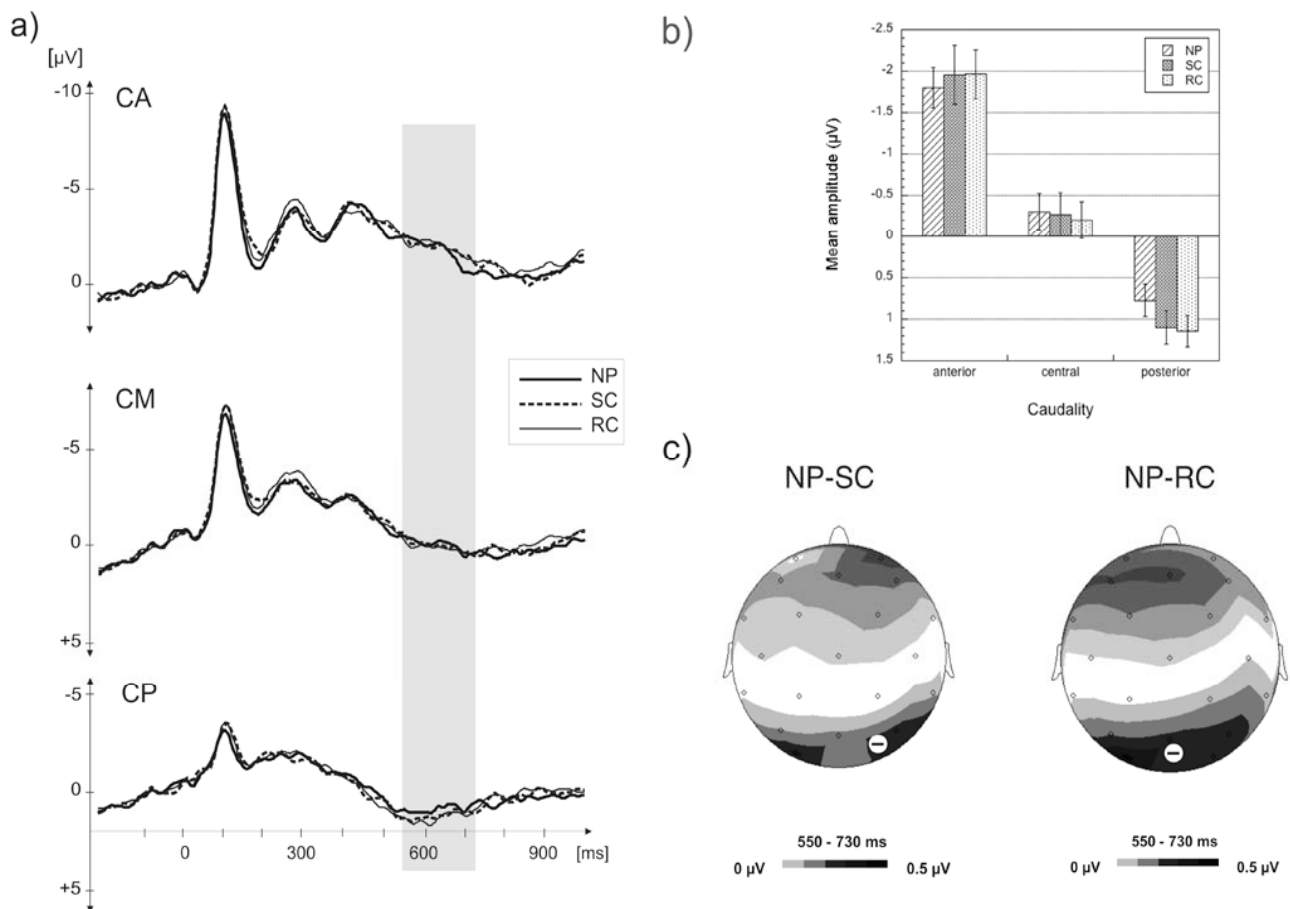


Figure 3. ERPs of the overall data are depicted as (a) grand average ERPs, (b) mean ERP amplitudes, and (c) average-referenced topographic maps. (a) The ERP grand averages are separated according to caudality for central anterior (CA), central medial (CM), and central posterior (CP) sites. Condition averages are superimposed for negative priming (NP, thick line), the standard control (SC, dashed line), and repetition control (RC, thin line). The gray bar indicates the time window analyzed (550–730 ms post-stimulus). (b) Mean ERP amplitudes are separated according to the caudality of the electrode location. Error bars depict the standard errors. (c) The map illustrates the topographical distribution of the ERP effect associated with negative priming in the aforementioned time window. The left map refers to the spatial distribution of the difference waves between negative priming and standard control; the right map refers to the spatial distribution of the difference waves between negative priming and repetition control.

ative priming condition were more negative-going at posterior electrodes in contrast to the control conditions, the difference among the experimental conditions just failed to reach the conventional level of significance, $F(2, 28) = 2.83$, $p = .076$, $\eta^2 = .17$. There were also no effects of the experimental conditions at anterior, $F(2, 28) = 0.32$, $p = .733$, $\eta^2 = .01$, or medial, $F(2, 28) = 0.16$, $p = .849$, $\eta^2 = .01$ clusters. Albeit the overall negative priming effect in the ERP data was clearly smaller in the present experiment than in that reported by Mayr et al. (2003), the current results essentially replicate those earlier findings.

Parallel to the analysis of the reaction time data, ERPs were averaged separately for fast (below median) and slow (above median) responses. Figure 4 depicts the grand averaged ERPs obtained for the fast responses. Within the late temporal epoch (550–730 ms), a minor

effect of negative priming appeared at posterior electrodes, $\Delta -41 \text{ V}$ for NP vs. SC, $\Delta -27 \text{ V}$ for NP vs. RC, but statistical analyses revealed no significant difference in this electrode cluster, $F(2, 28) = 1.41$, $p = .260$, $\eta^2 = .09$. The same was true for the anterior, $F(2, 28) = 1.12$, $p = .340$, $\eta^2 = .074$, and the medial clusters, $F(2, 28) = 0.01$, $p = .992$, $\eta^2 = .00$. Nevertheless, visual inspection of Figure 4 depicting the grand averaged ERP showed that the negative priming condition can be differentiated from both control conditions on the basis of earlier frontal ERP components. In a time range extending from 300 to 400 ms, the ERPs evoked in the negative priming condition were more positive-going as compared to the control conditions, $\Delta 0.60 \text{ V}$ for NP vs. SC, $\Delta 0.70 \text{ V}$ for NP vs. RC. Analyzing the mean amplitudes in this temporal epoch did reveal significant differences between the experimental conditions at anterior electrode clusters,

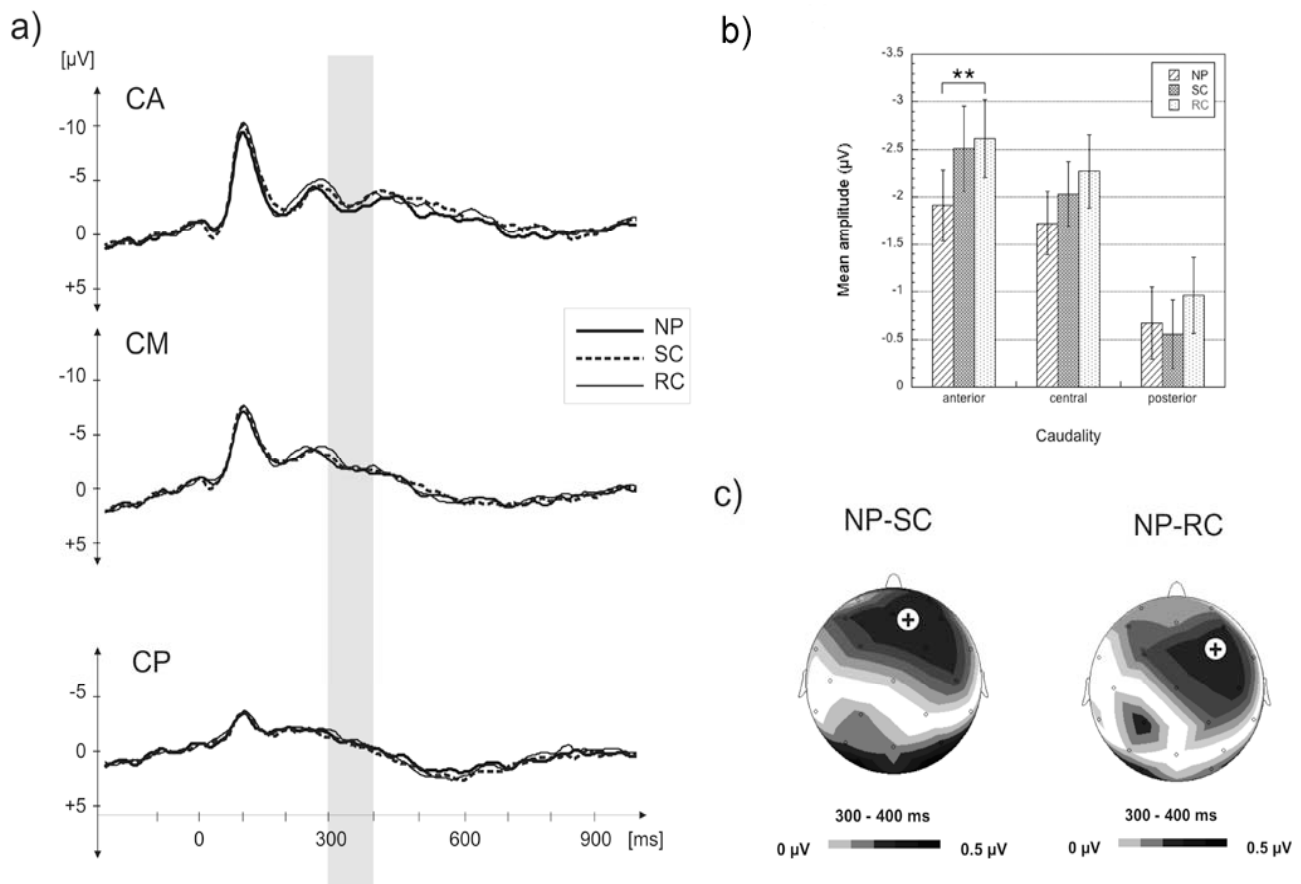


Figure 4. ERPs of the fast response (below median) data are depicted as (a) grand average ERPs, (b) mean ERP amplitudes, and (c) average-referenced topographic maps. (a) The ERP grand averages are separated according to caudality for central anterior (CA), central medial (CM), and central posterior (CP) sites. Condition averages are superimposed for negative priming (NP, thick line), the standard control (SC, dashed line) and repetition control (RC, thin line). The gray bars indicate the time window of most interest (300–400 ms post-stimulus). (b) Mean ERP amplitudes are separated according to the caudality of the electrode location. Significant amplitude differences between conditions in the aforementioned time window are indexed by asterisks ($*p \leq .05$, $**p \leq .01$) for all cases in which the superordinate ANOVA was statistically significant. Error bars depict the standard errors. (c) The map illustrates the topographical distribution of the ERP effect associated with negative priming in the aforementioned time window. The left map refers to the spatial distribution of the difference waves between negative priming and standard control, the right map refers to the spatial distribution of the difference waves between negative priming and repetition control.

$F(2, 28) = 3.69$, $p = .038$, $\eta^2 = .21$. The *post hoc* comparison confirmed the positive amplitude shift in the negative priming condition when compared to the repetition control condition, $t(29) = 2.75$, $p = .010$, $d_z = 0.50$, but was only on the verge of significance when compared to the standard control condition, $t(29) = 1.95$, $p = .061$, $d_z = 0.36$. The standard control did not differ significantly from the repetition control, $t(29) = 0.44$, $p = .66$, $d_z = 0.08$. No differences were obtained at medial or posterior leads. No differences were obtained at medial or posterior leads (for medial clusters: $F(2, 28) = 2.14$, $p = .137$, $\eta^2 = .13$, for posterior clusters: $F(2, 28) = 0.93$, $p = .405$, $\eta^2 = .06$).

Figure 5 shows the ERPs based on the slow responses (above median). In the time range extending from

550–730 ms, negative priming was characterized by a reduced positivity at posterior sites. Statistical analysis confirmed this visual impression, $F(2, 28) = 3.84$, $p = .034$, $\eta^2 = .22$, with significant *post hoc* differences between negative priming and standard control, $\Delta -0.45$ V, $t(29) = -2.18$, $p = .037$, $d_z = 0.40$, and repetition control, $\Delta -0.68$ V, $t(29) = -2.71$, $p = .011$, $d_z = 0.49$, respectively. The standard control did not differ significantly from the repetition control, $t(29) = -1.04$, $p = .309$, $d_z = 0.19$. Since the effect was focused at posterior leads – as shown in the topographical distribution in Figure 5 – no differences were obtained at anterior or medial electrode clusters (for anterior clusters: $F(2, 28) = 0.18$, $p = .833$, $\eta^2 = .01$, for medial clusters: $F(2, 28) = 0.36$, $p = .703$, $\eta^2 = .03$).

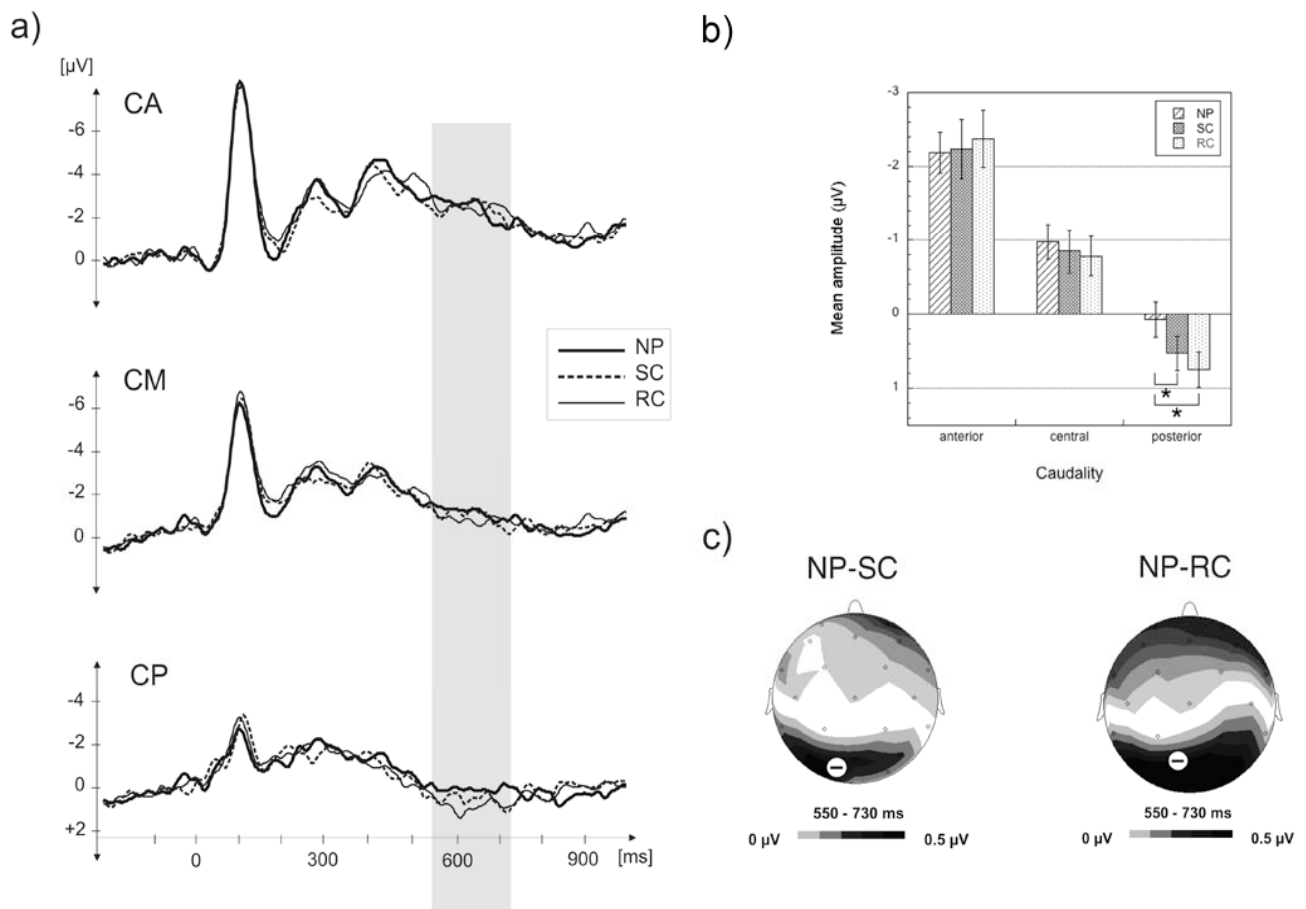


Figure 5. ERPs of the slow response (above median) data are depicted as (a) grand average ERPs, (b) mean ERP amplitudes, and (c) average-referenced topographic maps. (a) The ERP grand averages are separated according to caudality for central anterior (CA), central medial (CM), and central posterior (CP) sites. Condition averages are superimposed for negative priming (NP, thick line), the standard control (SC, dashed line) and repetition control (RC, thin line). The gray bars indicate the time window of most interest (550–730 ms post-stimulus). (b) Mean ERP amplitudes are separated according to the caudality of the electrode location. Significant amplitude differences between conditions in the aforementioned time window are indexed by asterisks ($*p \leq .05$, $**p \leq .01$) for all cases in which the superordinate ANOVA was statistically significant. Error bars depict the standard errors. (c) The map illustrates the topographical distribution of the ERP effect associated with negative priming in the aforementioned time window. The left map refers to the spatial distribution of the difference waves between negative priming and standard control; the right map refers to the spatial distribution of the difference waves between negative priming and repetition control.

Discussion

Responses were slower when an attended sound was previously ignored, that is, a typical negative priming effect was observed. The negative priming effect was observed for both fast and slow reactions. Importantly, the size of the negative priming effect was much larger for the slow responses than for the fast responses. This result is congruent with previous findings in which longer reaction times were also correlated with stronger negative priming effects (e.g., Neill & Westberry, 1987; Yee et al., 2000). Note that the response time level in this study was not manipulated – intentionally or unintentionally – by help of an experimental manipulation, such as instruction (Neill & Westberry,

1987) or target selection type (Yee et al., 2000). The *post-hoc* classification of reactions as fast and slow by means of a median split had the advantage of avoiding additional manipulations that could potentially involve additional stages in information processing, which might contaminate the ERPs.

At a descriptive level, the present results and those of Mayr et al. (2003) were parallel in that for a temporal epoch characterized by a late positive complex (of about 500 ms) the ERPs at posterior electrodes decreased in amplitude for the negative priming in contrast to the control conditions.

When the ERP data were split and analyzed separately for short and long reaction times, the late positive ERP effect exhibited a pattern that was parallel to the reaction time data. More precisely, the late positive ERP effect was very

small for short reaction times and clearly larger for long reaction times. Interestingly, reaction times in the study of Mayr et al. (2003) in which a large negative priming related ERP effect was found were also clearly larger than in the present study (790 ms vs. 746 ms). Thus, it appears that faster reaction times reduce the chances of finding a negative priming effect in both the reaction time data and the ERP data. The main difference between reaction time and ERP data obviously is that the negative priming effect is much stronger in the former than in the latter type of data to begin with. Reducing the size of the reaction time based negative priming effect still leaves it in an order of magnitude in which it can be detected with the sample sizes that are typical of experiments in that area. The ERP effect, however, may be reduced to a level at which it can no longer be detected reliably. This finding may help to explain the lack of ERP studies of the negative priming phenomenon.

Why should larger reaction times be associated with more pronounced negative priming effects at the reaction time level? We want to explicate this using both the distractor inhibition and the episodic retrieval explanation of negative priming. According to the distractor inhibition account (Tipper, 1985) the representation of the prime distractor is suppressed in order to allow for task-oriented processing of the prime target. This suppression has to be overcome in the probe situation before a task-appropriate reaction to the probe target can be carried out. A straightforward assumption is that inhibitory processes take time to develop. With longer response times inhibition of the representation of the previous distractor should be more fully developed, causing a larger delay in responding when the inhibition has to be overcome in the probe trial.

A similar argument can be put forward from the perspective of the episodic retrieval account. According to this account, negative priming is the result of retrieving the prime episode when exposed to the probe stimulus. Part of the retrieved episode is the "do not respond" information tied to the prime distractor. This response information conflicts with the need to respond to this stimulus in the probe episode. Resolving this conflict is time-consuming. However, retrieval of the prime episode is not inevitably successful. We assume that successful retrieval is positively correlated with the reaction time level. Presumably, a substantial amount of the fast responses are responses for which participants were already prepared to respond *before* stimulus presentation or analysis was completed. In these cases prime retrieval did not take place and, as a consequence, could not affect the processing. However, a certain percentage of these responses are correct guesses and will influence the analysis, in that they will reduce the reaction time level and the negative priming effect. In contrast, among the slow responses there should be fewer or none of these prepared responses but a larger percentage of completely processed trials, which should allow for a higher probability of successful prime retrieval.

In accordance with these theoretical accounts, the late ERP effect that is more distinct for slow responses would

either be the correlate of a stronger level of suppression or of a higher probability of retrieving the conflicting prime episode, or of both of these variables.

An important question is whether we need such a substantive explanation for the observed pattern of data in the first place. A number of studies have found slower reaction times to be associated with a reduced P3 amplitude as well as an increased P3 latency (e.g., Friedman, 1984; Roth, Ford, & Kopell, 1978). As Roth et al. (1978) state in their discussion, there is an ambiguity in the interpretation of amplitudes in ERP averages since a reduction can result not only from a decrease in the amplitude of individual trials but also from an increase in the variability of the latency of these trials, which is typical for slower trials compared to faster trials.

Is it conceivable that the ERP effect found for slow relative to fast responses in our experiment is simply based on the methodical problem of averaging trials of different variability in the data? We do not think that this argument can explain the present data for the following reason.

The median split resulted in larger reaction time variability and potentially more variable ERP peaks, not only for negative priming trials but also, and to a comparable degree, for the standard control and the repetition control trials (see Figure 2). The critical late positive complex must, therefore, be affected similarly for the negative priming and the control conditions. In other words, all conditions should be affected by the potentially increased variability to similar degrees so that the *difference* between conditions should stay approximately constant.

The analysis of the fast responses revealed an additional frontally located ERP effect of negative priming starting about 300 ms. Provided that the effect can be confirmed in further studies, it may indicate a second neural correlate of negative priming.

In sum, longer reaction times are associated with a larger chance of obtaining a negative priming effect in both the reaction time data and the ERP data. Second, both the distractor inhibition and the episodic retrieval account can explain this finding. Third, the observed data pattern is unlikely to be the result of a methodological artifact resulting from the averaging of more variable ERP responses.

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Susanne Mayr

Institute of Experimental Psychology
Heinrich Heine University
D-40225 Düsseldorf
Germany
Tel. +49 211 811-2270
Fax +49 211 811-5037
E-mail susanne.mayr@uni-duesseldorf.de