

Running head: ERP Correlates of Tactile Negative Priming

Modulations of Event-Related Potentials by Tactile Negative Priming

Ann-Katrin **Wesslein**¹, Ewald **Naumann**²,

Charles **Spence**³, & Christian **Frings**²

¹ University of Tübingen, ² University of Trier, ³ University of Oxford

The authors declare that they have no conflict of interest.

Number of characters: 16,378 (max 19,500)

Corresponding author:

Ann-Katrin Wesslein

University of Tübingen, Department of Psychology

Schleichstraße 4

72076 Tübingen

Email: ann-katrin.wesslein@uni-tuebingen.de

22

23 **Abstract**

24

25 Negative priming (NP) refers to the finding that responses to previously-irrelevant stimuli are
26 impeded relative to responses to new stimuli. To date, NP has been demonstrated in the visual,
27 auditory, and tactile sensory modalities with both inhibitory processes and retrieval-based
28 processes contributing to the effect. In order to gain deeper insights into the role of both
29 processes, event-related potentials (ERPs) have been measured during NP tasks with visual and
30 separately with auditory stimuli. The specific patterns of ERP correlates are mixed, yet it can
31 generally be concluded from previous research that amplitudes of both the N2 and the P3 reflect
32 important components of NP. We present the first study to assess the ERP correlates of NP in
33 the tactile modality. We observe a significant modulation of the P3 but not of the N2, thus
34 providing tentative support for the existence of modality-specific differences in the ERP
35 correlates of negative priming.

36

37 *Keywords:* negative priming; touch; event-related potentials; selective attention; sensory
38 modalities

39

1. Introduction

Our brains are constantly processing tactile information concerning the stimuli that touch our skin. However, not all of these incoming inputs are going to be equally relevant all of the time. Indeed, it is well-established that distractor processing at a given moment in time can potentially influence subsequent stimulus processing and modulate the actions that are elicited. One common approach to the study of the after-effects of distractor processing is the negative priming (NP) paradigm [1; 2]. In identity-based NP, a trial typically consists of a sequence of one prime and one probe, each comprising a target and a distractor stimulus. In some trials, the probe target represents the same stimulus as the prime distractor (ignored-repetition, IR). This leads to a performance decrement as compared to performance in those trials in which no prime stimuli are used as either the probe target or probe distractor (control, C). Nowadays, it is widely agreed that both inhibitory and retrieval-based processes contribute to the NP effect [3; 4].

Based on the reported behavioural results, NP in an identity-based task seems to be comparable in vision, audition, and touch. Nevertheless, previous results need not necessarily imply that the underlying processes are also similar. On the one hand, NP was found to take place at an amodal level in an audio-visual experiment [5]. On the other, modality-specific effects have been observed in a visuo-tactile variant of the NP task [6].

Another approach to investigating mechanisms underlying identity-based NP is the investigation of event-related potentials (ERPs). Surprisingly, however, those have only been measured during visual and auditory identity-based NP tasks. In some visual identity-based NP studies, an increased P3 [7; 8] or LPC [9] have been obtained in ignored-repetition as compared to control trials. In other studies, however, the behavioural NP effect has been accompanied by a reduced left posterior P3 amplitude and reduced frontal lobe LPC amplitude [10-12]. The amplitude of the N4 has also been found to be more negative in IR as

compared to C trials [9; 11; 13]. Intriguingly, the same holds for auditory NP: In two experiments, a relatively more negative LPC was found in ignored-repetition as compared to two types of control trials [14; 15]. In addition, an enhanced posterior N2 amplitude was reported in several studies using slightly different variants of the identity-based (and location-based) NP task [10; 16-19]. Summing up, it would seem that ERP correlates of identity-based NP are not related to sensory processes (which depend on the modality of stimulus presentation) but rather to late processes that are associated with stimulus evaluation [20].

1.3. The Present Study

In this study, we set out – for the first time (to the best of our knowledge) – to measure the ERP correlates in addition to behavioural measures of identity-based NP in the tactile modality. A modulation of N2 components was expected [based on 9; 10; 16-19] as well as a modulation of P3 components [based on 7; 8; 10-12; 14; 21]. Hence, we focused the ERP analyses on the time window of the N2 and P3/LPC.

2. Methods

The current study was carried out according to the principles of the Declaration of Helsinki, on the basis of informed consent.

2.1. Participants

Forty students (12 male) with a median age of 21 years (ranging from 18 to 38 years) served as participants, all having normal or corrected-to-normal vision and a normal sense of touch. All of the participants had given written informed consent. The data from the recording of the electroencephalogram (EEG) of one participant were missing due to a technical failure. The data from this participant were therefore excluded from the data analyses. The experiment was approved by the local ethic committee of the University of Trier.

2.2. Design

Essentially, the experiment consisted of a one-factorial 2 (Trial type: ignored-repetition, control) with repeated measures. Based on previous studies on distractor processing in touch [e.g., 22], the mean accuracy was our main behavioral measure. To measure the electrical activity of the brain, the EEG was recorded (see below).

2.3. Apparatus and Materials

The experiment was conducted in individual sessions on a PC with 24'' CRT screen using a standard computer keyboard. The experiment was run in E-Prime 2.0 software. Vibrotactile stimuli were delivered by means of two tactors (Model C-2, Engineering Acoustic, Inc.). Each tactor was 1.17'' in diameter and 0.30'' thick and they were fastened to the side of the participant's right/left palm.

Four vibrotactile stimuli with different rhythms were used throughout the experiment. The first vibration consisted of a continuous 500-ms pulse. The second vibration comprised two 150-ms pulses, each followed by a 100-ms pause. The third vibration consisted of five 50-ms pulses each followed by a 50-ms pause, and the fourth vibration consisted of a 400-ms pulse followed by a 100-ms pause. On a given trial, two stimuli were simultaneously presented to the participant's right and left hand. Each stimulus represented the target on some trials and the distractor on others.

Note that stimulus identity was varied orthogonally to the factor of Trial type, so it cannot account for any differences regarding IR and C trials. The same holds for the location of the target which was manipulated between-participants in the experimental factor of Target location.

2.4. Procedure

Participants were tested in a completely light- and sound-proofed room. The participant's hands were positioned palms-down in front of their body with the tactors fastened onto the inside of each palm by means of Velcro strips. Participants received white noise over

headphones. They were instructed to indicate the vibration presented to their right (left) hand as rapidly and accurately as possible on each trial by pressing the key associated with the respective vibration, while ignoring the stimuli presented to the other hand (i.e., the distractors).

The experimental session comprised two learning phases and three practice phases, as well as one experimental phase. The learning phases served to familiarize the participants with the mapping of the vibrotactile stimuli onto the response keys “C”, “D”, “M”, and “K”. To this end, the presentation of a vibration accompanied with a visual image showing a bird’s-eye view of the right and left hand with the correct motor response (i.e., the respective finger was colored green). During the *practice phases*, participants internalized the stimulus-response mapping by receiving feedback as to whether their response on a given trial had been correct or not. Throughout the phases, task difficulty was increased successively.

In the experimental phase, the participants initiated each prime-probe sequence by pressing the space bar, eliciting the presentation of a white fixation cross at the center of the screen presented against a black background (300 ms). The screen color then changed to blue/white to indicate the target location in the subsequent prime and probe trials (between-participants); the fixation cross remained on the screen. After 300 ms, the screen color changed back to black until 1,500 ms had elapsed, and the two vibrotactile prime stimuli were repeatedly delivered to the participant’s right and left palm until a response was detected or until 7,000 ms had elapsed. After a delay during which again a white fixation cross was presented centrally on a black screen for a random interval of 500-1,500 ms, the probe display started with the presentation of the vibrotactile stimuli (similar to the prime). Then, a black screen was presented for 500 ms, succeeded by the instruction to press the space bar to initiate the next prime-probe sequence. The experimental phase comprised 144 prime-probe sequences, namely 72 IR trials and 72 C trials.

2.5. Preparation of the Analyses

2.5.1. Behavioural Analysis

For all behavioural analyses, performance during the probe was analysed. All trials with erroneous prime responses were excluded from the analyses (i.e., 35.7 % of all prime-probe sequences).

2.5.2. ERP Recording and Analysis

EEG was measured with an Acti-Cap system (Brain Products GmbH, Munich) from 32 electrodes (FP1, FP2, F7, F3, Fz, F4, F8, FC5, FC1, FCz, Fc2, FC6, T7, C3, Cz, C4, T8, Cp5, CP1, CP2, CP6, P7, P3, Pz, P4, P8, PO9, PO10, O1, Oz, O2) according to the 10-10 electrode reference system [23] including the mastoids (TP9, TP10). Measurement reference was FCz; AFz served as ground. All impedances of the EEG electrodes were below 5 k Ω . Signals were amplified with 32-channel BrainAmp amplifiers (input impedance: 10 M Ω ; Brain Products GmbH, Munich) in AC mode. The pass-band was set from 0.016 to 500 Hz (-12 dB/octave roll-off); the signals were digitized at 1,000 Hz and stored to hard disk for later analysis.

EEG analysis was conducted with Brain Vision Analyzer Software (Version 2.03; Brain Products). First of all, ocular activity was removed with an ocular correction ICA as implemented in the Analyzer Software. After referencing the EEG to a linked mastoid reference, the data were segmented for all 144 trials at each electrode from 200 ms before stimulus onset to 1,300 ms post stimulus onset. Further artifacts were removed by a semiautomatic procedure which identified artifacts if (a) the difference between successive measurement points exceeded 30 μ V, (b) the absolute difference in a segment exceeded 150 μ V, or (c) there was no more than 0.5 μ V difference in activity within 100 ms (flat line). Finally, artifact-free averages were computed for the control and ignored-repetition trials.

All ERP analyses were conducted following established guidelines for EEG studies [24]. As compared to peak scoring, measures derived from area scoring are less susceptible to problems elicited by low signal-to-noise-ratios which often occur when few (or differing

numbers of) trials are considered. Hence, we computed area measures as the average of the amplitudes of the measured points in a particular pre-defined time window.

3. Results

For all of the statistical analyses reported here, a significance level of $\alpha = .05$ was specified.

3.1. Behavioural Performance

To compare probe performance in ignored-repetition trials and control trials, we conducted a t -test on the dependent measures. This test revealed a significant difference, $t(38) = 2.32$, $p = .026$, $d_z = 0.37$, indicating that performance was impeded in ignored-repetition trials (mean accuracy rate = 61.65 %, $SD = 19.39$ %) as compared to the control trials (mean accuracy rate = 65.34 %, $SD = 15.98$ %).

3.2. ERP Correlates of Negative Priming

Based on a visual inspection of the grand averages and considering topographic information (see Figure 1), N2 average amplitudes were calculated at F3, Fz, and F4 in a time window from 196 to 206 ms after stimulus onset. Average P3 amplitudes were extracted in a time window from 300 to 320 ms after stimulus onset from locations P3, Pz, and P4.

-- Insert Figure 1 about here --

These amplitudes were submitted to a 2 (Trial type: ignored-repetition, control) \times 3 (Electrode location: F3, Fz, F4) repeated-measures ANOVA. Where necessary, violations of sphericity were corrected by multiplying the degrees of freedom with the Greenhouse-Geisser Epsilon (GG- ϵ) coefficient. The ANOVA for the N2 average amplitude revealed a main effect of Electrode location, $F(2, 76) = 3.38$, $p = .039$, GG- $\epsilon = .98$; $\eta_p^2 = .09$, with the amplitudes more negative at Fz and F4 compared to F3. The relevant main effect of Trial type, $F(1, 38) = 3.58$,

$p = .066$; $\eta_p^2 = .09$, revealed a tendency to less negative N2 amplitudes in ignored-repetition as compared to control trials (see Figure 1). The interaction was not significant, $F < 1$.

For the P3 average amplitude a 2 (Trial type: ignored-repetition, control) \times 3 (Electrode location: P3, Pz, P4) repeated-measures ANOVA was conducted, again revealing a main effect of Electrode location, $F(2, 76) = 3.71$, $p = .029$, GG- $\epsilon = .86$, $\eta_p^2 = .09$, with the amplitudes more positive at Pz as compared to P4 and P3. The relevant main effect of Trial type, $F(1, 38) = 4.39$, $p = .043$, $\eta_p^2 = .10$, showed less positive P3 amplitudes in ignored-repetition trials as compared to control trials (see Figure 1). The interaction was not significant, $F < 1$.

4. Discussion

The present study is the first to investigate ERP correlates of identity-based NP with tactile stimuli. That is, we recorded the EEG of participants while they were confronted with the typical prime-probe configurations. In line with our hypotheses, performance in terms of accuracy rates was significantly lower in the ignored-repetition trials as compared to the control trials (i.e., standard NP effect). With regard to the ERP correlates, we observed significant modulations of the P3 amplitudes as a function of the prime-probe configuration. Our results provide an extension of previous findings, being in line with those studies indicating that tactile sustained spatial attention affects neural activities related to later processing stages. Still, we did not analyse early components in our study, since we focused on ERP components that were reported to relate to NP. Moreover, our findings extend the insights concerning the neural correlates of identity-based NP and enable their comparison across the senses. We observed less positive P3 amplitudes in ignored-repetition trials as compared to the control trials. This is in conflict with some previous studies on the modulations of the P3 [7; 8; 9], but well in line with others [10-12; 14; 21]. With regard to the interpretation of the P3, a reduction of the P3 in ignored-repetition as

compared to control trials is at odds with the interpretation of the P3 in terms of retrieval [10] because a non-repeated stimulus should not elicit stronger retrieval than a repeated one. Yet elsewhere the P3 has been interpreted as indexing an “updating” of the mental representation of the stimulus environment [25]. In this regard, a reduced P3 in ignored-repetition trials might indicate that processing a repeated distractor requires less attention (as it has already been processed during the prime display). Note that with regard to the N2 component, no significant modulation was observed (and the observed tendency shows the opposite pattern than the one usually reported [10; 16-19]).

Conclusion

At the behavioural level, identity-based NP effects have been documented in vision, audition, and touch in several studies, and they appear to be comparable across the senses. Assessing ERP correlates of tactile negative priming, our study – while surely not settling the issue – might be taken tentatively to suggest that the processes underlying identity-based NP appears to differ across the senses. **This might be due to differing perceptual resolutions across the senses, due to neurobiological differences across the sensory systems, or perhaps due to the divergent structures of the visual, auditory, and somatosensory cortices (spatiotopically vs. tonotopically vs. somatotopically organized). The results of the present study cannot be used to clarify this issue**, so future research is needed in order to investigate how differences regarding temporal discrimination requirements elicited by visual, auditory, or tactile stimuli contribute to modality-specific differences. Moreover, future research is also needed to replicate our results and to assess a broader range of somatosensory ERPs including their components in identity-based NP tasks and to compare ERP modulations among different sensory modalities. For, as suggested by the present results, distractor processing might not be as similar across the senses as has been previously assumed.

5. Figure Legend

Figure 1. Upper graph: Topographic maps showing difference waves, scaled in μV . The waves reflect comparisons of ERPs on ignored-repetition trials to those seen on control trials regarding (a) the time window between 196 ms and 206 ms, reflecting the N2 component, and (b) the time window between 300 ms and 320 ms, reflecting the P3 component. Lower graph: Grand averages for the control (solid) and ignored-repetition (dotted) trials at locations F3, Fz, F4, P3, Pz, and P4. The dotted vertical line represents stimulus onset. Time windows of the N2 and P3 are highlighted in grey. X-axes show the time-frame from 200 ms before to 500 ms after stimulus onset. Y-axes are scaled in μV .

6. Author Notes

Ann-Katrin Wesslein, University of Tübingen, Department of Psychology, Schleichstraße 4, 72076 Tübingen, Germany (Email: ann-katrin.wesslein@uni-tuebingen.de), Ewald Naumann, University of Trier, Department of Psychology, Universitätsring 15, 54286 Trier, Germany, Charles Spence, Department of Experimental Psychology, University of Oxford, South Parks Road, Oxford OX1 3UD, United Kingdom, and Christian Frings, University of Trier, Department of Psychology, Universitätsring 15, 54286 Trier, Germany.

7. Funding

This work was supported by a grant from the German Research Foundation (DFG) to Christian Frings and Charles Spence (FR 2133/5-1).

8. Acknowledgements

We would like to thank Thorsten Brinkmann, Lea Arlt, Alina Frey, and Yannick Runge from the University of Trier for their help collecting the data.

9. References

- [1] Dalrymple-Alford EC, Budayr B. Examination of some aspects of the Stroop color-word test. *Percept Mot Skills*. 1966; 23: 1211-1214.
- [2] Tipper SP. The negative priming effect: Inhibitory priming by ignored objects. *Q J Exp Psychol*. 1985; 37A: 571-590. doi: 10.1080/14640748508400920
- [3] D'Angelo MC, Thomson DR, Tipper SP, Milliken B. Negative priming 1985 to 2015: A measure of inhibition, the emergence of alternative accounts, and the multiple process challenge. *Q J Exp Psychol*. 2016; 69: 1890-1909. doi: 10.1080/17470218.2016.1173077
- [4] Frings C, Schneider KK, Fox E. The negative priming paradigm: An update and implications for selective attention. *Psychon Bull Rev*. 2015; 22: 1577-1597. doi: 10.3758/s13423-015-0841-4
- [5] Buchner A, Zabal A, Mayr S. Auditory, visual, and cross-modal negative priming. *Psychon Bull Rev*. 2003; 10: 917-923. doi: 10.3758/BF03196552
- [6] Frings C, Amendt A, Spence C. When seeing doesn't matter: Assessing the after-effects of tactile distractor processing in the blind and the sighted. *J Exp Psychol Hum Percept Perform*. 2011; 37: 1174-1181. doi: 10.1037/a0022336
- [7] Ceballos NA, Nixon SJ, Tivis R. Substance abuse related P300 differences in response to an implicit memory task. *Prog Neuropsychopharmacol Biol Psychiatry*. 2003; 27: 157-164. doi: 10.1016/S0278-5846(02)00347-0
- [8] Kathmann N, Bogdahn B, Endrass T. Event-related brain potential variations during location and identity negative priming. *Neurosci Lett*. 2006; 394: 53-56. doi: 10.1016/j.neulet.2005.10.001

- 287 [9] Wagner M, Baving L, Berg P, Cohen R, Rockstroh B. An ERP investigation of semantic
288 priming, repetition priming, and negative priming in schizophrenic patients. *J*
289 *Psychophysiol.* 2006; 20: 195-211. doi: 10.1027/0269-8803.20.3.195
- 290 [10] Gibbons H. An event-related potential investigation of varieties of negative priming. *J*
291 *Psychophysiol.* 2006; 20: 170-185. doi: 10.1027/0269-8803.20.3.170
- 292 [11] Gibbons H. Functional brain-electrical correlates of negative priming in the flanker task:
293 Evidence for episodic retrieval. *Psychophysiol.* 2009; 46: 807-817. doi: 10.1111/j.1469-
294 8986.2009.00819.x
- 295 [12] Stahl J, Gibbons H. Event-related brain potentials support episodic-retrieval explanations
296 of flanker negative priming. *Exp Brain Res.* 2007; 181: 596-606. doi: 10.1007/s00221-
297 007-0951-y
- 298 [13] Heil M, Rolke B. Unattended distractor-induced priming in a visual selective attention
299 task: N400 effects in the absence of RT effects. *J Psychophysiol.* 2004; 18: 164-169. doi:
300 10.1027/0269-8803.18.4.164
- 301 [14] Mayr S, Niedeggen M, Buchner A, Orgs G. The level of reaction time determines the
302 ERP correlates of auditory negative priming. *J Psychophysiol.* 2006; 20: 186-194. doi:
303 10.1027/0269-8803.20.3.186
- 304 [15] Mayr S, Niedeggen M, Buchner A, Pietrowsky R. ERP correlates of auditory negative
305 priming. *Cogn.* 2003; 90: 11-21. doi: 10.1016/S0010-0277(03)00142-2
- 306 [16] Frings C, Groh-Bordin C. Electrophysiological correlates of visual identity negative
307 priming. *Brain Res.* 2007; 1176: 82-91. doi: 10.1016/j.brainres.2007.07.093

- 308 [17] Hinojosa JA, Pozo MA, Méndez-Bértolo C, Luna D. Event-related potential correlates of
309 visual identity negative priming unbiased by trial-by-trial effects. *Brain Cogn.* 2009; 69:
310 531-537. doi: 10.1016/j.bandc.2008.11.004
- 311 [18] Groh-Bordin C, Frings C. Where has all the inhibition gone? Insights from
312 electrophysiological measures into negative priming without probe distractors. *Brain*
313 *Cogn.* 2009; 71: 92-98. doi: 10.1016/j.bandc.2009.04.005
- 314 [19] Ruge H, Naumann E. Brain-electrical correlates of negative location priming under
315 sustained and transient attentional context conditions. *J Psychophysiol.* 2006; 20: 160-
316 169. doi: 10.1027/0269-8803.20.3.160
- 317 [20] Mayr S, Buchner A. Negative priming as a memory phenomenon: A review of 20 years
318 of negative priming research. *J Psychol.* 2007; 215: 35-51. doi: 10.1027/0044-
319 3409.215.1.35
- 320 [21] Mayr S, Hauke R, Buchner A. Auditory location negative priming: A case of feature
321 mismatch. *Psychon Bull Rev.* 2009; 16: 845-849. doi: 10.3758/pbr.16.5.845
- 322 [22] Wesslein AK, Spence C, Mast F, Frings C. Spatial negative priming: In touch, it's all
323 about location. *Atten Percept Psychophys.* 2016; 78: 464-473. doi: 10.3758/s13414-015-
324 1028-9
- 325 [23] Chatrian GE, Lettich E, Nelson PL. Modified nomenclature of the "10%" electrode
326 system. *J Clin Neurophysiol.* 1988; 5: 183-186.
- 327 [24] Keil A, Debener S, Gratton G, Junghöfer M, Kappenmann ES, Luck SJ, et al. Committee
328 report: Publication guidelines and recommendations for studies using
329 electroencephalography and magnetoencephalography. *Psychophysiol.* 2014; 51: 1-21.
330 doi: 10.1111/psyp.12147

- 331 [25] Polich J, Kok A. Cognitive and biological determinants of P300: An integrative review.
332 Biol Psychol. 1995; 41: 103-146. doi: 10.1016/0301-0511(95)05130-9