Somatosensory deviance detection ERPs and their relationship to analogous auditory ERPs and interoceptive accuracy

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Abstract

Automatic deviance detection has been widely explored in terms of mismatch responses (mismatch negativity or mismatch response) and P3a components of event-related potentials (ERPs) under a predictive coding framework; however, the somatosensory mismatch response has been investigated less often regarding the different types of changes than its auditory counterpart. It is not known whether the deviance detection responses from different modalities correlate, reflecting a general prediction error mechanism of the central nervous system. Furthermore, interoceptive functions have been associated with predictive coding theory, but whether interoceptive accuracy correlates with deviance detection brain responses has rarely been investigated. Here, we measured ERPs to changes in somatosensory stimuli's location and intensity and in sound intensity in healthy adults (n = 34). Interoceptive accuracy was measured with a heartbeat discrimination task, where participants indicated whether their heartbeats were simultaneous or non-simultaneous with sound stimuli. We found a mismatch response and a P3a response to somatosensory location and auditory intensity changes, but for somatosensory intensity changes, only a P3a response was found. Unexpectedly, there were neither correlations between the somatosensory location deviance and intensity deviance brain responses nor between auditory and somatosensory brain responses. In addition, the brain responses did not correlate with interoceptive accuracy. The results suggest that although deviance detection in the auditory and somatosensory modalities are likely based on similar neural mechanisms at a cellular level, their ERP indexes do not indicate a linear association in sensitivity for deviance detection between the modalities. Furthermore, although sensory deviance detection and interoceptive detection are both associated with predictive coding functions, under these experimental settings, functional relationships were not observed. These results should be taken into account in the future development of theories related to human sensory functions and in extensions of the predictive coding theory in particular.

Keywords: Auditory, Deviance detection, Event-related potentials, Interoceptive accuracy, Somatosensory

Introduction

Automatic change detection and attention switching mechanisms are of high biological significance for survival because changes in the environment might signal danger. The neurophysiological responses mismatch negativity (MMN; or mismatch response, MMR) and P3a of event-related potentials (ERPs) have been widely used to study automatic change detection (Näätänen et al., 2007; Friedman et al., 2001), nowadays often referred to as deviance detection (e.g., Koshiyama et al., 2020). In the present study, we aimed to produce new information on somatosensory deviance detection by recording ERPs to somatosensory intensity changes and by investigating the relationships of response amplitudes to different stimulus types within and between sensory modalities (somatosensory and auditory). Furthermore, we explored whether these ERP amplitudes are related to the accuracy of interoception, i.e., information processing related to the sense of the physiological condition of the body (Craig, 2002). Because deviance detection brain responses both in the somatosensory and auditory modalities (e.g., Naeije et al., 2018; Wacogne et al., 2011) as well as interoception (e.g., Barret & Simmons, 2015) are associated with predictive coding theory (Friston, 2005), it can be expected that these measures could show covariance across participants.

According to the predictive coding theory (Friston, 2005), the brain predicts future events based on previous sensory input. When the input does not match the prediction, a prediction error occurs, and the error signal is projected upwards in the hierarchical neural network to update the predictive model (Friston, 2005). Sensory ERPs, especially MMN, are suggested to reflect prediction error (auditory: e.g., Wacogne et al., 2011; Chennu et al., 2013; for reviews, see e.g., Carbajal & Malmierca, 2018; Denham & Winkler, 2020; visual: e.g., Nirenberg et al., 2010; for a review, see Stefanics et al., 2014; somatosensory: e.g., Naeije et al., 2018, Xu et al., 2021).

MMN was originally discovered in the auditory domain (labeled as aMMN here for clarity, Näätänen et al., 1978, for reviews, see Näätänen et al., 2007; Kujala et al., 2007). It is typically elicited at a latency of 150-250 ms after the onset of deviance in an ignore oddball condition in which a rare deviant stimulus is interspersed with a repetitive standard stimulus (for reviews, see Näätänen et al., 2005; Näätänen et al, 2007; Garrido et al., 2009). aMMN can be produced in experimental designs employing changes in several different stimulus properties (for reviews, see e.g., Näätänen et al., 2011; Paavilainen, 2013).

MMN-like responses are also elicited outside the auditory modality. Here, we focus on the somatosensory mismatch response (called sMMR due to its positive polarity in some of studies, e.g., Shinozaki et al., 1998; Akatsuka et al., 2005; Strömmer et al., 2017), which is less studied than aMMN. There are also many studies on visual mismatch negativity (e.g., Astikainen et al., 2008; Stefanics et al., 2015; for a review, see Kremláček et al., 2016) and some on the olfactory mismatch response (Krauel et al., 1999; for a review, see Pause & Krauel, 2000). sMMR is typically elicited at 100–200 ms after the stimulus onset to changes in spatial location, duration, vibratory frequency, and a within-pair inter-stimulus interval of stimulus pairs (e.g., Akatsuka et al., 2005; Spackman et al., 2007; Chen, Hämmerer, D'Ostilio et al., 2014; Strömmer et al., 2017).

A mismatch response is followed by P3a, which reflects an automatic re-orienting of attention toward the change (for reviews, see Friedman et al., 2001; Escera et al., 2000; Polich, 2007). It peaks approximately 250–300 ms after stimulus onset (e.g., Light et al., 2007; Kiang et al., 2009; Hermens et al., 2010; for a review, see Knight & Scabini, 1998). P3a is elicited in an ignore oddball condition in different sensory modalities (the auditory modality [aP3a], e.g., Valkonen-Korhonen et al., 2003; Kiang et al., 2009; Kaur et al., 2011; Chen et al., 2015; Chien et al., 2018; the somatosensory modality [sP3a], e.g., Strömmer et al., 2017; Shen, Smyk, et al., 2018). Similar to

the mismatch response, P3a has also been associated with predictive coding: it can be understood as a transient expression of prediction error within predictive coding theory (Friston, 2005).

Previous studies have suggested that sMMR and aMMN are generated by their sensory modality-specific neural networks (Naije et al., 2016, 2018; Recasens et al., 2014; Butler et al., 2011; Restuccia et al., 2007) and are followed by a frontal component (Opitz et al., 2002; Rinne et al., 2005; Deouell, 2007; Chen et al., 2010; Naeije et al., 2016, 2018; Buttler et al., 2011). In contrast, the neural networks that generate a P3a response are suggested to be less sensory modality-specific; there is evidence that the interaction between the frontal lobe, hippocampus and temporal-parietal areas is the most crucial (e.g., Friedman et al., 2001; Knight, 1996; Wronka et al., 2012).

Within the auditory modality, aMMN to different changing features has been shown to differ in amplitude (Deouell & Bentin, 1998; Kathmann et al., 1999; Tervaniemi et al., 1999; Chen, Hämmerer, Strigaro, et al., 2014). For the somatosensory domain, a previous study showed that an earlier sMMR (100-200 ms post deviance) did not differ in amplitude between frequency and duration deviance conditions, but the later sMMR (170-270 ms post deviance) was elicited only in the frequency deviance condition (Spackman et al., 2007). Relationships between mismatch responses to different stimulus properties have rarely been studied. Regarding auditory modality, a correlation between frequency deviance and intensity deviance aMMN was found (Deouell & Bentin, 1998). Somatosensory mismatch responses in nociceptive and non-nociceptive somatosensory domains did not correlate either in the ignore or the attentive condition (Zhao et al., 2015). Thus far, there are no somatosensory MMR studies that examine the amplitudes of MMR elicited by different stimulus properties within the non-nociceptive domain using a within-subject design. The aim of present study was to advance knowledge in this under-researched area by studying somatosensory brain responses to intensity and location deviances in healthy adults. We

have previously reported sMMR and sP3a to somatosensory location changes in similar stimuli (Strömmer et al., 2014; Strömmer et al., 2017), but in the ignore oddball condition, sMMR/sP3a to somatosensory intensity changes have not been investigated.

Animal models have suggested that the neural mechanism underlying deviance detection brain responses is similar in the somatosensory and auditory modalities; stimulus-specific adaptation contributes to deviance detection in both modalities (e.g., von der Behrens et al., 2009; Musall et al., 2017). A similar cellular level mechanism could lead to associations between deviance detection ERP amplitudes recorded in different sensory modalities, but this has rarely been investigated. Zhao et al. (2015) have investigated somatosensory and auditory mismatch response amplitudes using attentive and ignore conditions. They found that there is a correlation of mismatch response amplitudes between the two attentional conditions within the auditory and within the somatosensory modalities, but there was no correlation between sMMR and aMMN amplitudes either in the ignore condition or the attentive condition. This may indicate that the two sensory modalities are not consistently sensitive to changing features, but more research is required to confirm this. Notably, investigations of P3a amplitudes across auditory and somatosensory modalities in an ignore condition are absent.

In addition to the processing of external stimuli, it is also crucial to be able to process information about the inner state of the body, which is referred to as interoceptive processing. As defined by Sherrington (1948), the concept of interoception refers to the sensations originating from the viscera. Since then, interoception has been defined in various ways. According to Craig (2002, 2003, 2009), interoception contains sensations that arise from within the body, e.g., heartbeats, gastrointestinal functions, respiration, thirst, hunger, sexual arousal, sensual touch, temperature, and pain, which include crucial information to maintain bodily homeostasis and allostasis (e.g., Craig,

2002; Barret & Simmons, 2015). In some studies, the concept of interoception is defined even more broadly, also including proprioceptive and somatosensory information processing (e.g., Ceunen et al., 2016; Horváth et al., 2021; Vaitl 1996). Similar to exteroceptive information processing, the mechanism of interoceptive information processing has also been explained using the predictive coding framework (e.g., Barret & Simmons, 2015; Gu et al., 2013; Seth, 2013).

The objective accuracy in detecting internal bodily sensations is referred to as interoceptive accuracy, and the research regarding it is mostly focused on individual sensitivity to cardiac signals (Garfinkel et al., 2015; e.g., heartbeat discrimination task; Whitehead et al., 1977; Brener & Kluvitse, 1988).

Herman et al. (2021) refer to somatosensory and olfactory processing as proximal exteroceptive senses as opposed to distal exteroceptive senses, such as auditory and visual senses. The relationship between interoceptive and exteroceptive information processing remains an understudied research area. Regarding proximal exteroceptive senses (somatosensory and olfactory), it is suggested that the boundary between interoception and exteroception is not clear (Herman et al., 2021), which aligns with the broad definition of interoception in which somatosensory processing is included in the interoception (e.g., Vaitl 1996; Ceunen et al., 2016; Horváth et al., 2021). There are some functional magnetic resonance imaging (fMRI) and behavioral studies that have demonstrated that the accuracy of the olfactory sense is positively associated with interoceptive accuracy (Krajnik et al., 2015; Koeppel et al., 2020). fMRI responses elicited when attending to interoceptive stimuli and somatosensory stimuli have been found to be partly overlapping but still dissociable (Herman et al., 2021). In some studies, the associations have been found only at the trend level (an ERP study investigating neural gating in the interoceptive and somatosensory stimuli: Jelinčić et al., 2021), and in the study by García-Cordero et al. (2017),

modulations of heartbeat-evoked potentials indicated differential attentional mechanisms for interoceptive and somatosensory processing. In behavioural studies, the relationship between interoceptive accuracy and touch detection was not found in one study (Garfinkel et al., 2016), but another study demonstrated that a better performance in the heartbeat detection task is positively correlated with false-positive responses in the tactile perception task (Durlik et al., 2014). In sum, the results for the relationship between proximal exteroceptive processing and interoceptive processing have not been consistent.

The association between interoception and ERPs to distal external stimuli has been less studied. Some studies have demonstrated the positive relationship between interoceptive accuracy and the amplitude of visual P3 elicited by emotional (Pollatos et al., 2005; Pollatos, Gramann, et al., 2007; Herbert et al., 2007) and neutral (Pollatos, Matthias, et al., 2007) stimuli. In addition, a higher interoceptive accuracy is connected to a larger N170 amplitude to fearful faces and a smaller P3 amplitude in response to neutral, fearful, and sad faces (Georgiou et al., 2018).

The aims of the present study were to examine i) whether sMMR and sP3a are elicited in the intensity and location deviance conditions within the somatosensory modality and whether the amplitudes of the brain responses are correlated between these somatosensory conditions; ii) whether ERP amplitudes of the auditory and somatosensory deviance detection components are related; and iii) whether somatosensory and auditory information processing measured by deviance detection ERPs and interoceptive accuracy measured by the heartbeat discrimination task are related.

Based on the predictive coding theory (Friston, 2005) and a possibly similar cellular mechanism (stimulus-specific adaptation; von der Behrens et al., 2009; Musall et al., 2017), the following could

be hypothesized: If one has a nervous system that responds with a high ERP amplitude to one type of deviance (ERPs reflecting prediction error, i.e., aMMN/sMMR and P3a), responses to other types of deviance within the same sensory modality should also be high (Spackman et al., 2007). Furthermore, if sensory modalities reflect predictive processing with consistent sensitivity between the modalities, prediction error-related ERPs should correlate in amplitude between the sensory modalities; however, the situation may also be more complex. Even if there are similarities in the neural mechanism underlying deviance detection ERPs in the somatosensory and auditory modalities, the neural networks that generate mismatch responses are sensory modality-specific (e.g., Butler et al., 2011). Therefore, it may be also possible that the somatosensory and auditory ERPs do not show associations between the response amplitudes.

Based on the concept of predictive coding as a general mechanism applying to both exteroceptive and interoceptive information processing (Barret & Simmons, 2015), it is possible that somatosensory and auditory deviance detection and heart rate detection are related. This is supported by previous studies that have demonstrated a relationship between interoceptive accuracy and visual ERPs (Pollatos et al., 2005; Pollatos, Matthias, et al., 2007; Herbert et al., 2007). There is evidence that somatosensory and interoceptive processing partly activates the same areas of the brain (Hermans et al., 2021), and some definitions of interoception include somatosensory information processing as a sense included in interoception (e.g., Horváth et al., 2021). Auditory information processing is clearly a (distal) exteroceptive sense. Therefore, we expect that interoceptive accuracy might be more likely related to somatosensory ERPs than to auditory ERPs; however, we consider the relationship between interoceptive accuracy and deviance detection brain responses more unlikely than the relationship between deviance detection ERPs within a somatosensory modality and between somatosensory and auditory modalities because the neural

mechanisms for somatosensory and auditory deviance detection and interoceptive sensations are different.

In the present study, we focused on the amplitudes of mismatch and P3a responses as targets of the investigation. The amplitudes of these responses showed moderate to robust test-retest reliability (e.g., Pekkonen et al., 1995; Kathmann et al., 1998; Tervaniemi et al., 1999; Schröger et al., 2000; Kujala et al., 2001; Hall et al., 2006; Lew et al., 2007; Biagianti et al., 2017; Roach et al., 2020). In most studies in which both amplitude and latency have been analyzed, the test-retest reliability is greater for amplitude than for latency (e.g., Roach et al., 2020; Lew et al., 2007; Hall et al., 2006). Latencies are also difficult to define for the responses that do not show clear peaks in an individual subject's data (as in Strömmer et al., 2017, for somatosensory P3a). Therefore, to provide a better comparability to previous studies and an accurate measure, we applied only ERP amplitudes in the analysis of the present study.

Materials and methods

Participants

A sufficient sample size for the present study was estimated by conducting a priori power analysis in G*Power (Faul et al., 2007). A repeated measures ANOVA with two within-subjects factors, both with two levels, was selected as the statistical test for each measure. For all the tests, the statistical power $(1 - \beta) = 0.80$, the significance level of alpha = 0.05, and the nonsphericity correction = 1. The effect size was set on the specification as in SPSS (IBM SPSS Statistics; IBM Corporation, NY, USA) and determined based on previous studies for each ERP component of

interest (Strömmer et al., 2017; sMMR: η^2_p= 0.174, sP3a: η^2_p = 0.416, aMMN: η^2_p = 0.521). The calculations indicated that the largest number of participants, 20 participants, is required for the sMMR. The sample size for the correlation analysis was also estimated with a priori power analysis. To observe a moderate association (r = 0.5; Dancey & Reidy, 2007) between the variables of interest, the calculation with the statistical power (1 - β) = 0.80, the significance level of alpha = 0.05, and a correlation ρ H0 = 0 showed that 29 participants are required. Following the results of the abovementioned calculations, we planned to recruit at least 29 participants for the whole study.

The participants were healthy volunteers recruited via notice board advertisements and email lists of the University of Jyväskylä. They participated in this study as a healthy control group of a larger research project exploring depression-related changes in exteroceptive and interoceptive information processing. The experiment was conducted in accordance with the Declaration of Helsinki. Ethical approval for the study was obtained from the ethical committee of the Central Finland Health Care District. All the participants gave written informed consent prior to their participation.

All participants were aged between 18 - 60 years, right-handed, and had no history of psychiatric or neurological conditions or alcoholic or narcotic addictions. They also did not use any medication (central nervous system agents) that could affect the central nervous system. The information related to the inclusion and exclusion criteria was collected by a phone interview and a questionnaire. Participants had normal or corrected-to-normal vision and normal hearing. Participants' hearing thresholds were measured using a SA-51 audiometer (Mediroll Medico-Technical Limited). The ears were measured individually. An exclusion criterion was a hearing threshold above 20 dB for 1000 Hz sounds.

The data were collected from 34 participants (8 male). The mean age was 33.38 (*SD* = 14.02) years, ranging between 19 and 60 years.

Somatosensory brain activity measurements could not be conducted for one participant due to cardiac arrhythmia. Somatosensory location deviance data from two participants, somatosensory intensity deviance data from four participants, and auditory data from one participant were omitted due to technical problems during the EEG recording. The interoceptive accuracy measurement could not be run with one participant due to cardiac arrhythmia. Due to technical problems, interoceptive accuracy data from two participants were excluded. The final study included 31 participants in the somatosensory location deviance experiment, 29 participants in the somatosensory intensity deviance experiment, 33 participants in the auditory intensity deviance experiment, and 31 participants in the interoceptive accuracy measurement. To include all possible participants in the analyses, the number of participants varied according to the available data.

Procedure

This study included three types of measurements: EEG recording, heartbeat discrimination task, and questionnaires. On the first measurement day, the subjects participated in the EEG recording; on the second measurement day, the performance of the heartbeat discrimination task was measured. In addition, participants were asked to fill in a background information form and questionnaires.

During the EEG recording, the participants sat in a chair in an electrically shielded, soundproof, dimly lit room. They were monitored via a video camera by the experimenter. The participants were instructed to avoid body and head movements and facial expressions to minimize the occurrence of

muscle artefacts. They were also instructed to concentrate on watching a silent movie on the screen and to ignore the somatosensory and auditory stimuli.

Stimulation in the EEG measurement

EEG was measured to changes in somatosensory and auditory stimuli. The somatosensory brain responses were studied under two conditions: the location deviance detection condition and the intensity deviance detection condition. The order of the somatosensory conditions was randomized across the participants, but there was always an auditory experiment between the two somatosensory conditions. The somatosensory stimulation was generated with a constant current stimulator (Digitimer Ltd, model DS7A, Welwyn Garden City, UK). Each stimulus duration was 200 μs. The electrical stimuli were delivered through flexible metal ring electrodes that were moistened with conductive jelly to reduce impedance. To prevent conductivity between the two electrodes in the same finger, a piece of gauze was placed on the finger between the electrodes. In the location deviance condition, the stimuli were delivered to the left forefinger and little finger by stimulating the cathode around the proximal phalanx and the anode around the distal phalanx. In the intensity deviance condition, the stimulation was delivered similarly but only to the right forefinger. The stimuli were delivered to different hands in the somatosensory location deviance condition and intensity deviance condition to ensure that the stimulated fingers were not numb at the beginning of the latter of these two somatosensory experiments.

The subjective somatosensory thresholds were determined before the EEG recording, and the somatosensory stimulus intensities were adjusted for each participant independently for the left forefinger and little finger and right forefinger. An individually adjusted stimulation was applied

because it is difficult to find a fixed somatosensory stimulus intensity that is not noxious for someone and still discernible for all participants. In the location deviance condition, the stimulus intensity was set at 1.5 times the individual somatosensory threshold, and in the intensity deviance condition, it was set at 1.5 times (lower intensity) and at twice (higher intensity) the threshold. These coefficients for the intensity deviance condition were found to be sufficient to elicit differential responses between the stimulus types in pilot recordings; however, in the pilot recordings, we had few participants, and therefore the statistical analysis was not conducted.

During the auditory brain activity measurement, the sinusoidal sounds of 1000 Hz in frequency and 100 ms in duration (with a 10-ms onset and offset time) were presented from a loudspeaker located approximately one meter above the participant. The intensity of the sounds varied at either 80 dB or 60 dB (sound pressure level, SPL). SPLs were measured with a sound level meter (type 2235, Brüel and Kjaer, Naerum, Denmark) with A-weighting.

In the somatosensory and auditory experiments, the stimuli were presented in an oddball condition in which a repeated standard stimulus was occasionally replaced by a deviant stimulus. The order of the stimuli was pseudorandomized: at least two standard stimuli were presented between consecutive deviant stimuli. In the somatosensory experiments, the stimuli were presented in two different oddball conditions. In the location deviance condition, the deviant stimuli were presented to different fingers, and in the intensity deviance condition, the deviant stimuli were of different intensities but presented to the same finger. Both experimental conditions consisted of two different stimulus blocks, and the stimulus features were counterbalanced between them. In the location deviance condition, in one block, the standard stimulus was delivered to the forefinger and the deviant stimulus to the little finger. In the other block, the assignment to the standard and the deviant stimulus was reversed. In the somatosensory intensity deviance condition, in the increment

block, the standard stimulus was of a lower intensity and the deviant stimulus of a higher intensity. In the decrement block, these intensities were reversed.

In the auditory experiment, there were standard and deviant sounds of different intensities (60 dB, 80 dB). In the auditory increment condition, the standard stimulus was 60 dB (SPL), and the deviant stimulus was 80 dB (SPL). In the decrement condition, these intensities were reversed. Hence, in the somatosensory experiments, both locations and both intensities were applied, and in the auditory experiment, both intensities were applied as standard and deviant stimuli for all participants in a randomized order across participants. In the somatosensory intensity deviance and location deviance experiments, there were 1680 stimuli in total (deviant stimulus probability 14 % as in our previous study: Strömmer et al., 2017), and in the auditory experiment, there were 2000 stimuli in total (deviant stimulus probability 10% as in our previous study: Ruohonen et al., 2020).

In the somatosensory experiments, the Stimulus Onset Asynchrony (SOA) in the stimulus presentation was randomly set at either 406, 456, or 506 ms. In the auditory experiment, SOA was randomly set at either 530 ms, 580 ms, or 630 ms. The SOAs were selected based on our previous studies reporting on somatosensory MMR and/or P3a (Strömmer et al., 2014; Strömmer et al., 2017) and auditory MMN (Ruohonen et al., 2020). In the somatosensory and auditory experiments, the stimulus presentation was controlled using E-Prime 2.0 software (Psychology Software Tools Inc., Sharpsburg, MD, USA).

EEG acquisition and EEG data processing

The EEG was recorded using a high-impedance amplifier (NeurOne Bittium Biosignals, Ltd.) and a 128-channel Sensor Net (Electrical Geodesics Inc., HydroCel GSN 128, 1.0). The sampling rate was 1000 Hz, and data were filtered online from 0.1 to 250 Hz. During the recording, the data were referenced to a vertex electrode (Cz).

The EEG data were analyzed with Brain Vision Analyzer 2.1. software (Brain Products GmbH, Munich, Germany). An average of all the channels was calculated and applied as a new reference. The data were filtered with a low cut-off at 0.1 Hz and a high cut-off at 30 Hz, a roll-off of 24 dB/octave, and a notch filter of 50 Hz. Ocular correction via an independent component analysis (ICA) as implemented in the Brain Vision Analyzer was applied. The representations for horizontal and vertical eye movements were manually selected from the ICA components based on a visual inspection. Channels with excessive noise were interpolated with a spherical spline model. The mean number of interpolated channels was 6.14 (*SD* = 2.33). The intensity deviance and location deviance somatosensory data were segmented into 500 ms segments from 100 ms prior to stimulus onset to 400 ms after stimulus onset, and the auditory data were segmented into 600 ms segments from 100 ms prior to stimulus onset to 500 ms after stimulus onset. For somatosensory and auditory data, a pre-stimulus onset time of 100 ms was determined as a baseline for a baseline correction. The EEG segments with a signal amplitude difference larger than 100 μV within a 100-ms period in any recording channel were omitted from the analysis. Segments with a difference of more than 50 μV between two consecutive time points (i.e., within 1 ms) and low activity periods (< 0.5 μV of change within a 100-ms range) were also excluded.

Averages of amplitudes for responses to standard stimuli were calculated only for stimuli immediately preceding the deviant stimuli because this procedure allows for the same number of segments and a similar signal-to-noise ratio for standard and deviant responses. Trial numbers did

not differ significantly between the standard and deviant trials in any of the conditions (all $p \geq .066$) (See Supplementary Table 1 in Electronic Supplementary Material).

The channels and time windows for sMMR and aMMN were defined based on previous literature (Strömmer et al., 2017; Ruohonen et al., 2020) and visual observations of the grand averaged waveforms. For the somatosensory intensity deviance MMR and location deviance MMR, the applied time window was 150−190 ms after stimulus onset. For intensity deviance sMMR (right hand), the amplitude values were extracted from a frontocentral channel cluster of five electrodes (channels 7, 13, 29, 30, and 31, See Supplementary Figure 1 in Electronic Supplementary Material), and for location deviance sMMR (left hand), the amplitude values were extracted from a frontocentral channel cluster of five electrodes (channels 87, 104, 105, 110, and 111, See Supplementary Figure 1 in Electronic Supplementary Material). For aMMN, the mean standard and deviant response amplitude values were calculated for the latency of 140−180 ms after stimulus onset over a frontal channel cluster of five electrodes (channels 5, 6, 11, 12, and 16 in the EGI 128-channel system, See Supplementary Figure 1 in Electronic Supplementary Material).

For the sP3a and aP3a, the time windows applied in previous studies (sP3a: Strömmer et al., 2017; Shen, Smyk, et al., 2018, and aP3a: Chien et al., 2018; Light et al., 2007; Kiang et al., 2009; Hermens et al., 2010; Kaur et al., 2011; Chen et al., 2015) fitted to our data. Based on the previous literature and visual inspection of the maximum amplitude values of the grand averaged data, we selected a time window of 200–300 ms after stimulus onset for sP3a and 220−320 ms after stimulus onset for aP3a. For both somatosensory location deviance and auditory P3a, the amplitude values were extracted from a frontocentral channel cluster of four electrodes (channels 7, 31, 80, and 106; as in Strömmer et al., 2017 for sP3a, See Supplementary Figure 1 in Electronic Supplementary Material). For somatosensory intensity deviance P3a, the amplitude values were extracted from a

frontocentral channel cluster of four electrodes (channels 6, 7, 106, and 112, See Supplementary Figure 1 in Electronic Supplementary Material).

Assessment of interoceptive accuracy

Interoceptive accuracy was assessed using a heartbeat discrimination task (Whitehead et al., 1977; Brener, & Kluvitse, 1988). The participants were seated in a chair in an electrically shielded and soundproof room. Prior to the task, they were instructed about the procedure of the task and asked to sit as still as possible to minimize motion artefacts, to breathe normally, to place hands on the armrests, and to not to take their pulse manually during the task. Participants were monitored via a video camera.

During the heartbeat discrimination task, participants were presented with 100 trials, each of which included a sequence of 10 tones (800 Hz, 100 ms) that were either simultaneous or non-simultaneous with participants' heartbeats. The tones were presented through a loudspeaker with an individually adjusted volume at a fixed delay interval following the heart R-waves. The delays were either 200 milliseconds (simultaneity condition) or 500 milliseconds (non-simultaneity condition). The use of these delay intervals was validated by Wiens and Palmer (2001) and applied in previous studies examining heartbeat discrimination skills (e.g., Wiens et al., 2000; Eshkevari et al., 2014).

In the heartbeat discrimination task, half of the trials were simultaneous, and half were non-simultaneous with participants' heartbeats. The order of the trials was randomized. At the end of each trial, a question asking whether the tones had been simultaneous with heartbeat sensations or not was presented on the screen. The participants responded by pressing one of two buttons on a

numeric keyboard (yes/no). The two response buttons were counterbalanced across the participants. One practice trial was performed prior to the proper task to familiarize the participant with the task and to ensure the instructions were understood. The whole experiment consisted of four blocks of 25 trials, between which the participants had a break and a possibility to change their position and stretch.

During the heartbeat discrimination task, the electrocardiogram (ECG) measurement was taken using nonpolarizable Ag-AgCl adhesive disposable electrodes (Kovidien Kendall H92SG). The electrodes were attached to the right mid-clavicle and lower-left rib cage. In addition, the ground electrode was placed on the right abdominal area below the ribs. The position of the electrodes was adjusted so that a clear R-wave of the QRS complex was detectable in the ECG signal. ECG activity was recorded analogously to the electroencephalogram with a high-impedance amplifier (NeurOne Bittium Biosignals, Ltd.). An in-house software script (Arduino) was used to detect individual R-waves and to control the presentation of the heartbeat locked tones (as in Lyyra & Parviainen, 2018).

The interoceptive accuracy scores were calculated as the total number of correct responses in the heartbeat discrimination task with a maximum possible score of 100. The higher the scores, the better the interoceptive accuracy.

Statistical analysis

The data were analyzed using IBM SPSS Statistics 24.0 (IBM Inc., Armonk, NY, USA). For the somatosensory and auditory intensity deviance, a repeated measures ANOVA with stimulus type

(standard, deviant) and intensity (high, low) as within-subject factors was conducted separately for auditory and somatosensory intensity deviance MMR and P3a. For the somatosensory location deviance, a repeated measures ANOVA with stimulus type (standard, deviant) and location (forefinger, little finger) as within-subject factors was conducted separately for MMR and P3a. When a significant interaction effect (stimulus type × intensity or stimulus type × location) was found, follow-up tests were performed using the paired samples t-test to compare the standard and deviant responses separately for the intensities/locations (i.e., for the intensities: low intensity standard vs. low intensity deviant and high intensity standard vs. high intensity deviant). This analysis allowed for ruling out the effect of the physical features of the stimuli on the deviance detection brain responses. Possible main effects of stimulus type and its interaction effects with other variables are reported. P-values smaller than 0.05 were considered significant. Effect size estimates are described as partial eta squared (η^2_p) scores for ANOVA and Cohen's *d* for *t*-tests.

To examine correlations, the mismatch responses and P3a responses were calculated as a differential response by subtracting responses to the standard stimulus from responses to the deviant stimulus (averaged over the intensities or the locations). Correlation between somatosensory intensity deviance and location deviance mismatch response was not investigated because there was no MMR for somatosensory intensity deviance. Somatosensory intensity deviance P3a was significant only for the high intensity stimuli (standard and deviant responses compared for high intensity stimuli). Relationships between somatosensory location deviance and somatosensory intensity deviance P3a (high intensity stimuli) differential responses as well as relationships between somatosensory and auditory differential responses were examined using Pearson's *r*. Regarding somatosensory and auditory intensity deviance P3a responses, the correlation was calculated between responses elicited for high intensity stimuli for both somatosensory and auditory P3a to keep the signal-to-noise ratio similar. To examine the relationships between brain responses

(differential responses) and the interoceptive accuracy score, Pearson's correlation coefficients were calculated. Multiple correlations were controlled by applying a false discovery rate (FDR) (Benjamini & Hochberg, 1995). FDR adjusted p-values are reported.

Before examining the correlations, the outliers were removed from the data. The data values were identified as outliers if they lied outside the following ranges: 3rd quartile + 1.5 × interquartile range and 1st quartile – 1.5 × interquartile range as implemented in IBM SPSS Statistics 24.0. The criteria for outlier exclusion were decided a priori.

Results

Somatosensory location deviance MMR and P3a

For somatosensory location deviance MMR amplitude, a repeated measures ANOVA showed a significant main effect of stimulus type (standard, deviant) but no interaction effect with the location (forefinger, little finger) (Table 1). The paired samples t-test comparing deviant and standard stimulus responses averaged over the locations showed that the responses were larger for the deviant stimuli compared to the standard stimuli, $t(30) = 2.56$, $p = .016$, $d = 0.52$. The mean amplitude difference was 0.29 μV, $SD = 0.63$, 95 % *CI* [0.06, 0.52] (Figure 1).

For somatosensory location deviance P3a amplitude, a repeated measures ANOVA showed a significant main effect of stimulus type but no interaction effect with the location (Table 1). The paired samples t-test comparing deviant and standard stimulus responses averaged over the locations showed that the responses were larger for the deviant stimuli compared to the standard

stimuli, $t(30) = 3.46$, $p = .002$, $d = 0.86$. The mean amplitude difference was 0.53 μV, $SD = 0.84$, 95 % *CI* [0.22, 0.84] (Figure 1).

Somatosensory intensity deviance MMR and P3a

For somatosensory intensity deviance MMR, the main effect of stimulus type (standard, deviant) was non-significant, but a significant main effect of stimulus intensity was found with no interaction effect with stimulus type (Table 1 & Figure 2). The paired samples t-test comparing high intensity and low intensity stimulus responses averaged over the stimulus types showed that the responses were larger for the high intensity stimuli compared to the low intensity stimuli, $t(29) = 2.87$, $p = .008$, $d = 0.59$. The mean amplitude difference was 0.29 μV, $SD = 0.54$, 95 % *CI* [0.08, 0.49].

For somatosensory intensity deviance P3a, a main effect of stimulus type, a main effect of intensity, and an interaction effect of stimulus type × intensity were found (Table 1). The interaction effect of stimulus type × intensity was investigated with paired samples t-tests, which showed that the responses were larger for the deviant stimuli compared to the standard stimuli for the high intensity stimuli, $t(28) = 2.89$, $p = .007$, $d = 0.75$. The mean amplitude difference was 0.65 μV, SD = 1.20, 95 % CI [0.19, 1.10]. There was no statistically significant difference between responses to low intensity standard and deviant stimuli (Figure 2).

Auditory intensity deviance MMN and P3a

For auditory intensity deviance MMN, a main effect of stimulus type (standard, deviant) was observed but no interaction effect with the stimulus intensity (Table 1). The paired samples t-test comparing deviant and standard stimulus responses averaged over the intensities showed that the responses were larger for the deviant stimuli compared to the standard stimuli, $t(32) = -8.64$, $p < 0.001$, $d = 1.44$. The mean amplitude difference was -1.72 μV, $SD = 1.15$, 95 % CI [-2.13, -1.32] (Figure 3).

For auditory intensity deviance P3a, a main effect of stimulus type was found, but there was no interaction effect with the intensity (Table 1). The paired samples t-test comparing deviant and standard stimulus responses averaged over the intensities showed that the responses were larger for the deviant stimuli compared to the standard stimuli, $t(32) = 5.40$, $p < 0.001$, $d = 1.07$. The mean amplitude difference was 0.89 μV, $SD = 0.95$, 95 % CI [0.55, 1.23] (Figure 3).

Relationships between somatosensory location deviance and somatosensory intensity deviance ERPs

Because somatosensory intensity deviance elicited no MMR, correlations could not be calculated between MMR responses elicited in the somatosensory location and intensity deviance conditions.

Somatosensory intensity deviance P3a was elicited only for the high intensity stimuli. Therefore, a correlation between somatosensory intensity deviance P3a for the high intensity stimuli and somatosensory location deviance P3a (averaged over the locations) was calculated. The correlation between somatosensory intensity deviance P3a for the high intensity stimuli and somatosensory

location deviance P3a, $r = .577$, $p = .025$, $n = 28$, did not reach significance when the outliers were removed (without outliers $r = .290$, $p > .999$, $n = 25$) (Figure 4A).

Relationships between somatosensory and auditory ERPs

There was no statistically significant correlation either between somatosensory location deviance MMR and auditory intensity deviance MMN, $r = .031$, $p > .999$, $n = 30$ (without outliers: $r = .015$, $p > .999$, $n = 26$) (Figure 4B) or between somatosensory location deviance P3a and auditory intensity deviance P3a, $r = .253$, $p > .999$, $n = 30$ (without outliers: $r = .235$, $p > .999$, $n = 26$) (Figure 4C).

The correlation between somatosensory and auditory intensity deviance P3a (high intensity stimuli only) was not statistically significant, $r = .247$, $p > .999$, $n = 29$ (without outliers: $r = -.029$, $p > .999$, $n = 27$) (Figure 4D).

Relationships between interoceptive accuracy and ERPs

Interoceptive accuracy ($M = 65.03$, $SD = 11.62$, $n = 31$) was calculated as the total number of correct answers in the heartbeat discrimination task in which the maximum possible score was 100. The correlation between interoceptive accuracy and somatosensory location deviance MMR, $r = .364$, $p = .662$, $n = 29$ (without outliers $r = .330$, $p > .999$, $n = 25$) did not reach significance (Figure 5A).

There was no correlation between somatosensory location deviance P3a and interoceptive accuracy, $r = .133$, $p > .999$, $n = 29$ (without outliers $r = .070$, $p > .999$, $n = 26$) (Figure 5B). Moreover, there was no correlation between somatosensory intensity deviance P3a (high intensity stimuli) and interoceptive accuracy, $r = .219$, $p > .999$, $n = 27$ (Figure 5C).

Regarding auditory intensity deviance ERPs, there was no correlation either between aMMN and interoceptive accuracy, $r = .068$, $p > .999$, $n = 30$ (Figure 5D), or between aP3a and interoceptive accuracy, $r = -.098$, $p > .999$, $n = 30$ (without outliers: $r = -0.107$, $p > .999$, $n = 29$) (Figure 5E).

Discussion

The purposes of the present study were to investigate whether somatosensory deviance detection ERPs are elicited for both intensity deviance and location deviance and whether there are correlations between these responses or between the auditory and somatosensory responses. Correlations could be expected if ERPs for different sensory features reflect a prediction error with a consistent magnitude. In addition, correlations between somatosensory and auditory ERPs and interoceptive accuracy were investigated. We found somatosensory MMR and P3a responses to changes in stimulus location as well as auditory MMN and P3a responses to changes in sound intensity. In the somatosensory intensity deviance condition, sMMR was not elicited, and sP3a was elicited only for high intensity stimuli. Regarding the correlations, we found no relationship between the ERP amplitudes or between the ERPs and interoceptive accuracy.

For the somatosensory location deviance, the sMMR and sP3a responses were qualitatively similar in latency and topography to our previous study, where a similar stimulation was applied (Strömmer et al., 2017). Contrary to our expectations, we did not find a somatosensory intensity deviance

MMR. Previous studies have not applied the ignore condition in investigations of somatosensory intensity deviance detection. The somatosensory stimuli of a higher intensity elicited a larger response amplitude regardless of the stimulus type (standard, deviant), which indicates that the response reflected only stimulus saliency rather than deviance detection. Because the low intensity stimulus was adjusted for each participant individually to be 1.5 times stronger than the sensory threshold, it is not probable that a too low stimulus intensity was the reason for the absent sMMR. Furthermore, the intensity applied in the low intensity stimuli was the same as the stimulus intensity in the somatosensory location deviance condition in which both sMMR and sP3a responses were elicited. The difference between standard and deviant intensity was large enough because the high intensity stimulus elicited larger amplitudes than the low intensity stimulus (the significant main effect of intensity). According to our results, it seems that intensity is not a suitable stimulus feature for studying a somatosensory prediction error as indexed by sMMR.

Even if we did not find somatosensory intensity deviance MMR, somatosensory intensity deviance P3a was found, though only when the responses to the high intensity standard and deviant stimuli were compared. Our results thus suggest that attention switching towards somatosensory intensity changes is dependent on stimulus saliency. Here, the high intensity stimuli, which elicited the P3a, were presented with an intensity of twice the individual somatosensory threshold, while an intensity of 1.5 times the individual somatosensory threshold did not elicit a significant P3a.

It can be speculated that one possible explanation for why the sMMR was elicited to location but not to intensity deviance conditions might be due to the somatotopic organization of the primary somatosensory cortex (SI), which means that adjacent locations on the skin activate adjacent neurons in the neural tissue (Penfield & Boldrey, 1937). The somatotopic organization of SI could have an impact, especially on location deviance sMMR, because it is proposed to be generated by

the neural activity in the somatosensory cortices (e.g., Chen et al., 2010; Naeije et al., 2016, 2018; Buttler et al., 2011; Spackman et al., 2010; Xu et al., 2021), and the somatotopy contributes to the neural processing of the spatial location of the somatosensory stimulus (Penfield & Boldrey, 1937). It seems that even if the intensity of the somatosensory stimulus is also encoded mostly in the SI, the somatosensory intensity encoding is not based on the same kind of representational map as somatotopic maps for somatosensory location encoding (e.g., Muniak et al., 2007; Bensmaia, 2008). It is highly probable that in the location deviance condition, somatotopic representations of the forefinger and little finger in SI were activated by the stimulation delivered to these two fingers, while in the intensity deviance condition, only a representation of the forefinger was activated.

In addition to location deviance sMMR (e.g., Kekoni et al., 1997; Shinozaki et al., 1998; Akatsuka et al., 2007; Restuccia et al., 2007; Restuccia et al., 2009; Chen, Hämmerer, D'Ostilio, et al., 2014; Strömmer et al., 2014; Strömmer et al., 2017; Tarkka et al., 2016; Naeije et al., 2016, 2018; Shen, Smyk, et al., 2018; Shen, Weiss, et al., 2018; Hautasaari et al., 2019; Xu et al., 2021), a somatosensory mismatch response has been demonstrated in an ignore condition by changes in various other stimulus properties, such as duration (Spackman et al., 2007; Spackman et al., 2010, Butler et al., 2011; Chen, Hämmerer, D'Ostilio, et al., 2014; Chen et al., 2018), vibratory frequency (Kekoni et al., 1997; Spackman et al., 2007), and a within-pair inter-stimulus interval of stimulus pairs (Akatsuka et al., 2005) that are not related to spatial location or to the somatotopic organization of SI. Therefore, the present findings concerning the differences between MMR to somatosensory location deviance (existing) and intensity deviance (no significant response found) should not be explained only by the difference of how these stimulus features recruit the cortical somatotopy of fingers.

The findings of the present study that intensity deviance sMMR was not elicited at all and intensity deviance sP3a was elicited only for the high intensity stimuli are partly in discrepancy with previous attentive studies in which somatosensory intensity deviance ERPs (P300, MMR, N250) have been found (Kida et al., 2003; Iwadate et al., 2005; Ostwald et al., 2012); however, one possible explanation for the discrepancy is that we applied an ignore condition, while the previous studies used an attentive task. In addition to the attentive condition, Kida et al. (2003) used an ignore condition in their study, and in this case, somatosensory N250 and P300 were not elicited, which aligns with the finding of the present study regarding somatosensory intensity deviance MMR. Our results suggest that the preattentive processing of intensity changes in somatosensory modality may not be sufficient to support deviance detection.

Regarding the auditory intensity deviance condition, we were able to replicate our previous findings in healthy participants (Ruohonen & Astikainen, 2017; Ruohonen et al., 2020). Regarding the time window and scalp distribution, aP3a found in the present study was largely in accordance with previous studies in which an ignore oddball condition had been applied (Light et al., 2007; Kiang et al., 2009; Hermens et al., 2010; Kaur et al., 2011).

It is not known why in the auditory but not in the somatosensory modality intensity changes elicited a mismatch response and P3a. In both auditory and somatosensory intensity deviance conditions, the percentage difference (29 %) in the stimulus intensity was the same between lower and higher intensity stimuli. According to our result, it seems that the auditory system may prioritize stimulus probability encoding, whereas the somatosensory system may encode intensity regardless of the probability. Thus, could it be that the somatosensory system is more dedicated to intensity processing than deviance detection and that the system prioritizes intensity information over change detection? This might be related to the different contributions of these sensory modalities to

adaptive behaviors. There is a dearth of previous studies investigating the difference between somatosensory and auditory intensity deviance ERPs. Future studies are needed to address this issue.

We also investigated relationships between amplitudes of ERPs within somatosensory modality and between somatosensory and auditory modalities. We expected to find a relationship between amplitudes of deviance detection responses to different types of changes in the somatosensory modality, but contrary to our expectations, we found no relationship between somatosensory intensity deviance P3a (high intensity stimuli) and somatosensory location deviance P3a when the outliers were removed. Because somatosensory intensity deviance stimuli elicited no MMR, a relationship between somatosensory location deviance and somatosensory intensity deviance MMR could not be determined.

It is notable that in our study, both high and low intensity stimuli were applied as standard and deviant stimuli in separate stimulus blocks. This allowed for analyzing the mismatch response and P3a by comparing responses to physically identical stimuli as standard and deviant. This is important because the physical features of the stimuli most likely significantly affect the responses. Quite surprisingly, most previous studies have not taken into account this either in the study design or in the analysis (see, however, Xu et al., 2021; Ostwald et al., 2012 for somatosensory deviance detection responses).

We hypothesized that amplitudes of somatosensory and auditory deviance detection responses might be related because these two sensory systems both reflect prediction error and are based on a similar cellular mechanism (stimulus-specific adaptation; von der Behrens et al., 2009; Musall et al., 2017). Contrary to our expectations, there was no relationship between somatosensory location

deviance MMR and auditory intensity deviance MMN. Our finding regarding the mismatch response is congruent with the findings by Zhao et al. (2015), showing no correlation between amplitudes of somatosensory and auditory mismatch responses. Somatosensory and auditory mismatch responses are generated by sensory modality-specific neural networks (e.g., Butler et al., 2011), which might partly explain the result that no correlation in the deviance detection ERP amplitudes was found between these modalities.

Regarding P3a responses, we found no relationship either between somatosensory location deviance P3a and auditory intensity deviance P3a or between somatosensory and auditory intensity deviance P3a (high intensity stimuli). Based on lesion studies, it has been suggested that the P3a component is at least partly modality non-specific (Friedman et al., 2001); however, a correlation in P3 amplitudes has been found between auditory, visual, and olfactory modalities (Olofsson et al., 2008). In the study by Olofsson et al. (2008), an attentive condition was used, whereas in the present study, an ignore condition was applied. In addition, in the present study, the sensory modalities were different from those in the study by Olofsson et al. (2008). Thus, these studies cannot be directly compared, but our data tentatively suggest that ignored changes do not elicit P3a responses with a consistent sensitivity in the somatosensory and auditory modalities. To the best of our knowledge, there are no previous studies investigating the relationship of P3a amplitudes between somatosensory and auditory modalities in an ignore oddball condition, and therefore more research is needed.

In sum, we did not find linear relationships between the ERPs suggested to reflect a prediction error; however, the relationship, if it exists, may not be linear. It is possible that prediction errors are binary responses; the deviance is either detected or not regardless of the amplitude. Future studies should investigate brain responses at a single subject level. For instance, it could be

determined whether subjects who have significant somatosensory deviance detection ERPs also have significant auditory deviance detection ERPs. In our study, the number of trials was too small, and the signal-to-noise ratio was too low to investigate brain responses at the level of an individual subject.

Finally, we investigated whether the interoceptive accuracy is related to the proximate and/or distant sensing of the environment. We expected interoceptive accuracy to more likely be related to somatosensory (proximal sense) ERPs than to auditory (distal sense) ERPs because somatosensory and interoceptive processing partly activates the same areas of the brain (Hermans et al., 2021), and according to the broad definitions of the interoception, somatosensory processing is even included in interoception (e.g., Horvath et al., 2021). Contrary to our expectation, somatosensory location deviance MMR and P3a, somatosensory intensity deviance P3a, and auditory intensity deviance MMN and P3a showed no correlation with interoceptive accuracy. In a fMRI study by Herman et al. (2021) in which brain activation partly overlapped in somatosensory and interoceptive processing, the interoceptive and somatosensory tasks were more equated than deviance detection ERPs and the interoceptive task in our study. As a measure, the heartbeat discrimination task is significantly different from deviance detection ERPs elicited in the ignore condition. ERPs that we measured to changes in the somatosensory and auditory stimuli are elicited automatically in the brain, but our interoception task did not measure automatic responses to interoceptive stimuli but rather the behavioral responses that were conveyed through an active decision-making process and motor response. Furthermore, the interoceptive stimuli we applied (heartbeat) cannot be as easily manipulated as external stimuli. In addition, we investigated interoceptive accuracy utilizing a task that is not related to deviance detection. The heartbeat discrimination task is based on the detection of the cardiac signals at rest, whereas deviance detection ERPs were measured by delivering

somatosensory and auditory stimuli. These differences between ERPs and behavioral responses in an interoceptive discrimination task might partly explain our result that no relationship was found between ERPs and interoceptive accuracy. Future studies should develop new research designs in which the measures of the detection of interoceptive stimuli and the detection of auditory and somatosensory stimuli would be more equated.

Our results showing no linear association between the ERPs and interoceptive accuracy seem to be in contradiction with previous studies, which have shown that interoceptive accuracy is related to ERPs elicited by visual stimuli (Pollatos et al., 2005, Pollatos, Gramann, et al., 2007; Herbert et al., 2007; Georgiou et al., 2018; Pollatos, Matthias, et al., 2007). In the late 1800s and early 1900s, bodily changes and physiological processes (i.e., interoception) were associated with emotion processing (James, 1884; Lange, 1912). Interoceptive accuracy has been shown to be positively correlated with the amplitude of the P300 response elicited by emotional pictures (Pollatos et al., 2005; Herbert et al., 2007). A high interoceptive accuracy has also been associated with large N170 amplitudes to fearful faces and small P300 amplitudes to neutral, fearful, and sad faces (Georgiou et al., 2018). Regarding neutral exteroceptive stimuli, a better interoceptive accuracy has been shown to be related to the higher amplitude of a P300 response elicited by visual target stimuli (deviant red crosses among standard white crosses) (Pollatos, Matthias, et al., 2007). Because the stimuli of previous studies investigating the relationship between interoception and ERPs have been visual and emotional, it is difficult to compare their findings to ours. In the study by Pollatos, Matthias, et al. (2007), the visual stimuli were neutral, but in this study, an attentive oddball condition was applied. In the present study, the stimuli were somatosensory and auditory, and an ignore oddball condition was used. In previous studies (Pollatos et al., 2005; Herbert et al., 2007; Georgiou et al., 2018; Pollatos, Matthias, et al., 2007), interoceptive accuracy has been measured by a heartbeat tracking task in which individuals count the number of their heartbeats during given periods of time

(Shandry et al., 1981), while in the present study, we used a heartbeat discrimination task in which individuals report whether their heartbeats are simultaneous or non-simultaneous with external stimuli (Whitehead et al., 1977). These tasks seem to pose different cognitive demands; while heartbeat tracking requires attention to visceral sensations (e.g., Ring et al., 2015), in the heartbeat discrimination task, attention must be focused on visceral sensations and exteroceptive stimuli at the same time, which requires the capacity to integrate interoceptive and exteroceptive information (Suzuki et al., 2013). The discrepancy between the previous results and those of the present study could be explained at least partly by differences in stimuli, attentional conditions, and heartbeat detection tasks. To the best of our knowledge, this is the first study to investigate the relationship between interoceptive accuracy and the automatic deviance detection brain responses elicited by neutral exteroceptive stimuli in an ignore oddball condition.

Even if interoception is sometimes thought to encompass somatosensory processing (e.g., Horvath et al., 2021), there is a scarcity of previous studies in which the relationship between interoception and somatosensory ERPs has been investigated. Instead, the relationship between somatosensory perception measured by attentive behavioral tasks and interoceptive accuracy has been explored (Garfinkel et al., 2016; Durlik et al., 2014). Garfinkel et al. (2016) found no correlation between the accuracy of touch detection and the correct responses in the heartbeat discrimination task, which aligns with the finding of the present study regarding the non-existent relationship between somatosensory ERPs and interoception.

There are some limitations to the present study. First, there was an uneven gender distribution with the significant majority of the participants being female, which must be taken into account when generalizing the results. The second limitation is the relatively small sample size. Particularly for the correlation analysis, the sample size may have been too small. Without outliers, all the

correlation coefficients were small (and the p-values large; *p*s = 1.000), but the correlations might have been observed with a significantly larger sample size. There is also a third limitation concerning the deviating stimulus features in the somatosensory and auditory experiments. We chose to investigate somatosensory ERPs in the intensity deviance condition to produce new knowledge because sMMR/sP3a responses to somatosensory intensity changes elicited in the ignore oddball condition has not been reported. The location deviance condition was selected because it is well-known that it can elicit sMMR and sP3a responses to similar stimuli we applied (Strömmer et al., 2014; Strömmer et al., 2017). Instead, in the auditory modality, we applied only intensity changes with similar stimuli as we have applied in our previous study (Ruohonen et al., 2020). It might have been advantageous to include the auditory location deviance condition in this study because it would have enabled us to investigate the relationship between somatosensory and auditory location deviance ERPs. The fourth limitation is that the number of trials and probability for the deviant stimuli were different across somatosensory and auditory experiments, and these numbers were more favorable for auditory than somatosensory stimuli (lower probability of the deviant stimulus and higher trial number for the auditory than the somatosensory condition). The condition differences were caused because we followed the same parameters that had been applied in our previous studies (auditory: Ruohonen et al., 2020; somatosensory: Strömmer et al., 2017). This provided a better comparability for the previous studies, and by applying the same parameters, we could expect to observe significant deviant-related modulations in the ERP components. Importantly, in the present study, the aim was not to directly compare the amplitudes of ERPs between somatosensory and auditory modalities but to investigate correlations between amplitudes.

Another limitation is that the intensities of somatosensory stimuli were set at 1.5 and 2 times the individual somatosensory threshold, but the intensities of auditory stimuli were not assigned according to the individual hearing threshold. We decided to adjust the somatosensory stimuli

according to the individual sensory threshold because it is difficult to find a fixed somatosensory stimulus intensity that is not noxious for someone and still discernible for all participants. The same problem is not that easily related to auditory stimuli. Here, auditory stimuli were 60 dB and 80 dB (SPL) in intensity, and an exclusion criterion was a hearing threshold above 20 dB for 1000 Hz sounds. Thus, the auditory stimuli were of a much higher intensity in relation to the hearing thresholds compared to the somatosensory stimuli in relation to the somatosensory thresholds. This could have affected the observation of significant deviance detection responses but would not necessarily prevent finding correlations between significant ERP components.

The final limitation is related to the heartbeat discrimination task. A large portion of the participants performed the heartbeat discrimination task at or just above the chance level. Hence, the task was not an optimal measure for interoceptive accuracy because it may have been too difficult. This may have hindered us from observing significant correlations between interoceptive accuracy and ERPs. In addition, in this study, interoceptive accuracy was measured by a behavioral task, while deviance detection was studied applying automatically elicited ERPs. EEG was not recorded during the heartbeat discrimination task. Future studies should investigate relationships between heartbeat evoked potentials and ERPs elicited by somatosensory and auditory stimuli.

In conclusion, we detected MMN/MMR and P3a brain responses to changes in somatosensory location and auditory intensity, but for changes in somatosensory intensity, only the P3a response was detected for the higher somatosensory stimulus intensity. These results suggest that intensity processing may be fundamentally different in the auditory and somatosensory modalities. While somatosensory deviance detection occurs for location, in the somatosensory intensity encoding, encoding of stimulus properties instead of rarity of the stimuli is prioritized. Auditory deviance detection occurs for intensity. Contrary to our expectations, we found no evidence of relationship

between deviance detection in somatosensory location and intensity or between the auditory and somatosensory deviance detection. The deviance detection either in the proximal sensory modality or in the distal sensory modality did not correlate with interoceptive accuracy. Our results suggest that somatosensory and auditory modalities do not reflect predictive processing with consistent sensitivity, but people may have specific sensory modalities and stimulus features for which they show sensitivity in deviance detection. Furthermore, no evidence of a linear association was found between interoceptive information processing and both proximate and distant sensory processing of the environment. This information can affect the future development of theories related to human sensory processing.

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Declaration of competing interest

The authors declare no conflict of interest.

Electronic Supplementary Material

-ESM 1 Supplementary table 1 (supplementarytable1.pdf)

The table contains the averaged number for trials included in the analysis of the somatosensory location deviance, somatosensory intensity deviance, and auditory intensity deviance conditions.

- ESM 2 Supplementary figure 1 (supplementaryfigure1.pdf)

The figure contains a map of EGI 128-Channel Net (HydroCel Geodesic Sensor Net) and the electrode clusters applied in the analyses.

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Table 1. Results of the repeated measures ANOVA for somatosensory and auditory mismatch (MMR/MMN) and P3a responses. F- and p-values and partial eta squared (η^2_p) for effect size estimates. Significant effects in bold. NA = not applicable.

Variable	Stimulus type	Location	Intensity	Stimulus type × location	Stimulus type × intensity
Somatosensory location deviance					
MMR	**F(1, 30) = 6.54** **p = .016** **η^2_p = 0.179**	F(1, 30) = 0.05 p = 0.820 η^2_p = 0.002	NA	F(1, 30) = 0.10 p = 0.752 η^2_p = 0.003	NA
P3a	**F(1, 30) = 12.00** **p = 0.002** **η^2_p = 0.286**	F(1, 30) = 3.67 p = 0.065 η^2_p = 0.109	NA	F(1, 30) = 3.02 p = 0.093 η^2_p = 0.091	NA
Somatosensory intensity deviance					
MMR	F(1, 28) = 1.72 p = 0.200 η^2_p = 0.058	NA	**F(1, 28) = 8.25** **p = 0.008** **η^2_p = 0.228**	NA	F(1, 28) = 1.63 p = 0.212 η^2_p = 0.055
P3a	**F(1, 28) = 6.57** **p = 0.016** **η^2_p = 0.190**	NA	**F(1, 28) = 13.72** **p = 0.001** **η^2_p = 0.329**	NA	**F(1, 28) = 6.96** **p = 0.013** **η^2_p = 0.199**
Auditory intensity deviance					
MMN	**F(1, 32) = 74.63** **p < 0.001** **η^2_p = 0.700**	NA	F(1, 32) = 0.01 p = 0.938 η^2_p < 0.001	NA	F(1, 32) = 0.45 p = 0.507 η^2_p = 0.014
P3a	**F(1, 32) = 29.107** **p < 0.001** **η^2_p = 0.476**	NA	F(1, 32) = 1.267 p = 0.269 η^2_p = 0.038	NA	F(1, 32) = 2.011 p = 0.166 η^2_p = 0.059

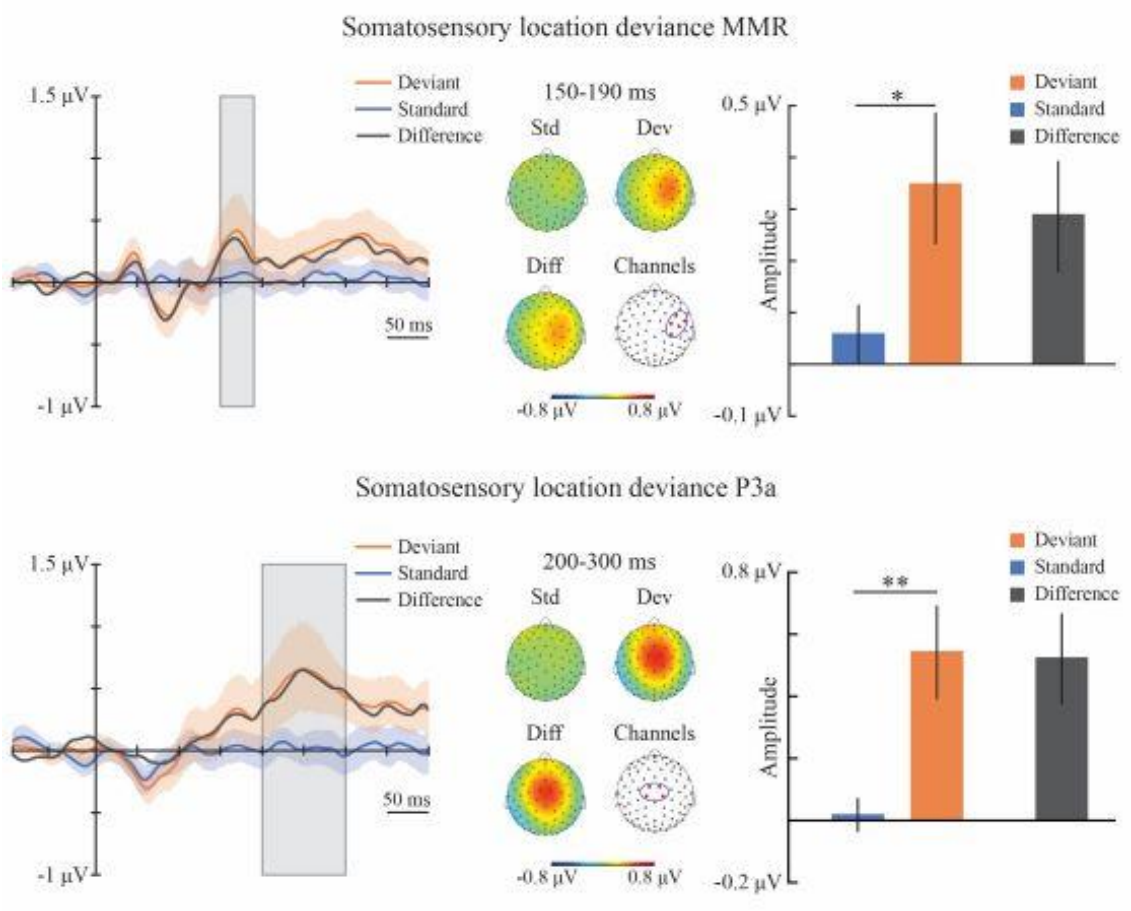

Somatosensory location deviance MMR
1.5 µV
-1 µV
50 ms
Deviant
Standard
Difference
150-190 ms
Std
Dev
Diff
Channels
-0.8 µV
0.8 µV
0.5 µV
-0.1 µV
Amplitude
*
Deviant
Standard
Difference
Somatosensory location deviance P3a
1.5 µV
-1 µV
50 ms
Deviant
Standard
Difference
200-300 ms
Std
Dev
Diff
Channels
-0.8 µV
0.8 µV
0.8 µV
-0.2 µV
Amplitude
**
Deviant
Standard
Difference

Figure 1.

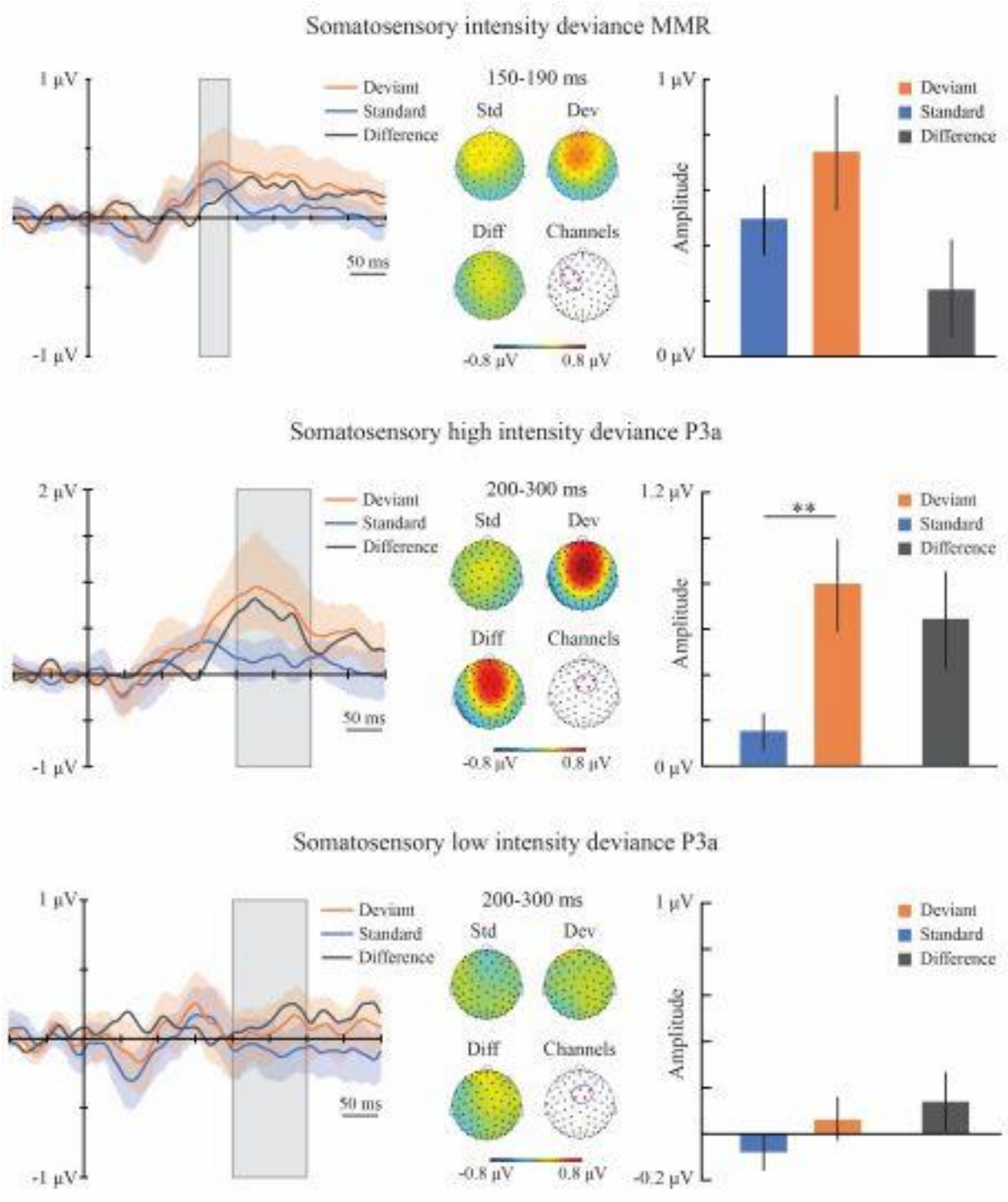

Somatosensory intensity deviance MMR
1 µV
-1 µV
50 ms
Deviant
Standard
Difference
150-190 ms
Std
Dev
Diff
Channels
-0.8 µV
0.8 µV
1 µV
0 µV
Amplitude
Deviant
Standard
Difference
Somatosensory high intensity deviance P3a
2 µV
-1 µV
50 ms
Deviant
Standard
Difference
200-300 ms
Std
Dev
Diff
Channels
-0.8 µV
0.8 µV
1.2 µV
0 µV
**
Amplitude
Deviant
Standard
Difference
Somatosensory low intensity deviance P3a
1 µV
-1 µV
50 ms
Deviant
Standard
Difference
200-300 ms
Std
Dev
Diff
Channels
-0.8 µV
0.8 µV
1 µV
-0.2 µV
Amplitude
Deviant
Standard
Difference

Figure 2.

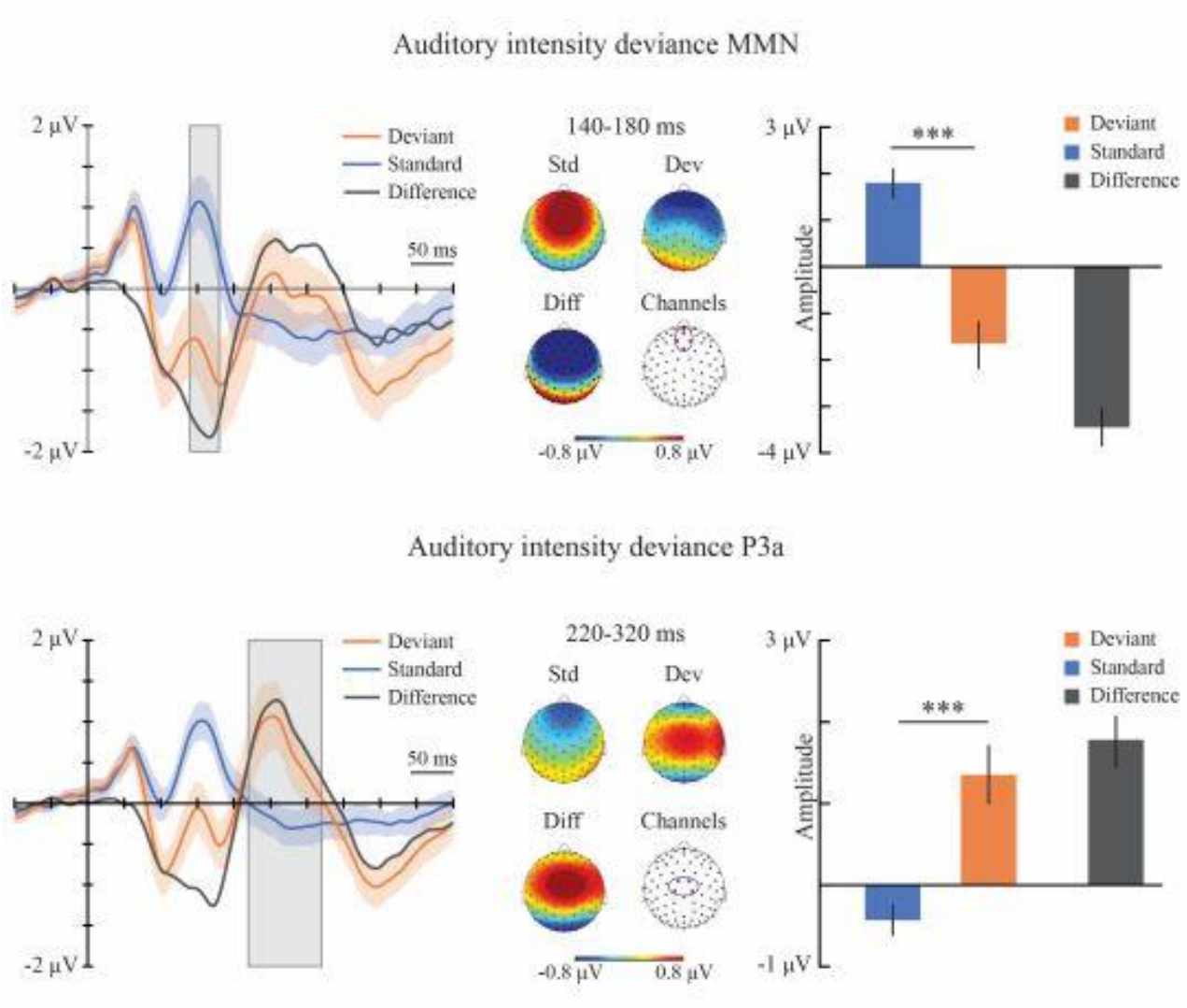

Auditory intensity deviance MMN
2 µV
-2 µV
Deviant
Standard
Difference
50 ms
140-180 ms
Std
Dev
Diff
Channels
-0.8 µV
0.8 µV
3 µV
-4 µV
***
Amplitude
Deviant
Standard
Difference
Auditory intensity deviance P3a
2 µV
-2 µV
Deviant
Standard
Difference
50 ms
220-320 ms
Std
Dev
Diff
Channels
-0.8 µV
0.8 µV
3 µV
-1 µV
***
Amplitude
Deviant
Standard
Difference

Figure 3.

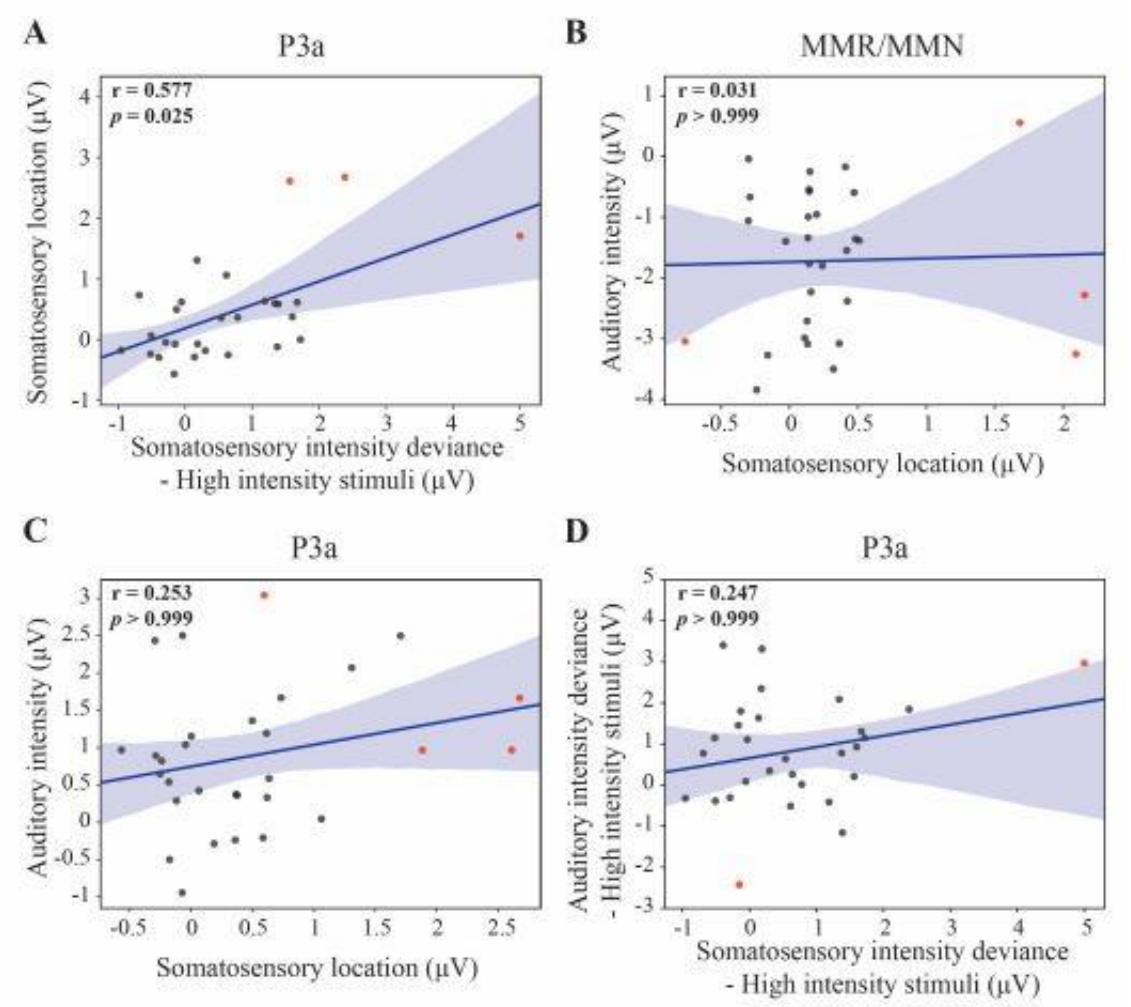

A
P3a
r = 0.577
p = 0.025
Somatosensory location (μV)
Somatosensory intensity deviance
- High intensity stimuli (μV)
B
MMR/MMN
r = 0.031
p > 0.999
Auditory intensity (μV)
Somatosensory location (μV)
C
P3a
r = 0.253
p > 0.999
Auditory intensity (μV)
Somatosensory location (μV)
D
P3a
r = 0.247
p > 0.999
Auditory intensity deviance
- High intensity stimuli (μV)
Somatosensory intensity deviance
- High intensity stimuli (μV)

Figure 4.

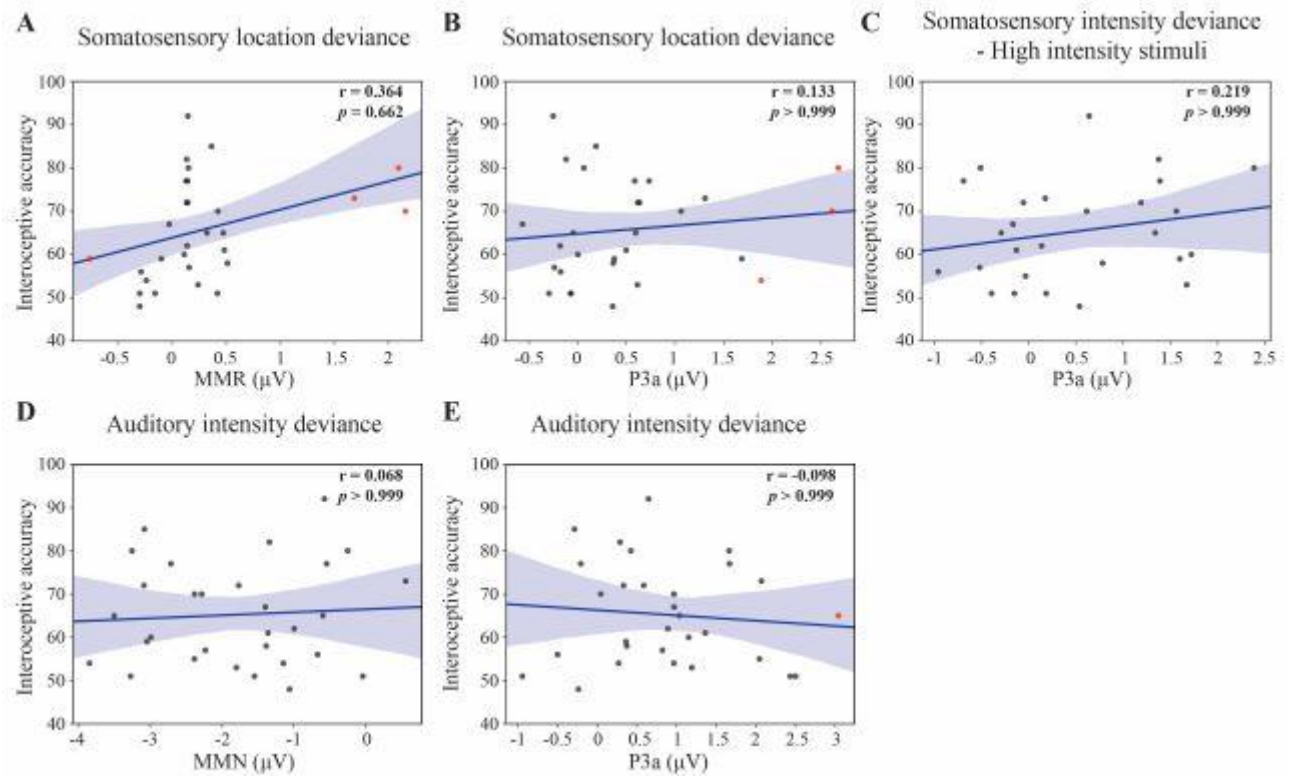

A
Somatosensory location deviance
r = 0.364
p = 0.662
Interoceptive accuracy
MMR (μV)
B
Somatosensory location deviance
r = 0.133
p > 0.999
Interoceptive accuracy
P3a (μV)
C
Somatosensory intensity deviance
- High intensity stimuli
r = 0.219
p > 0.999
Interoceptive accuracy
P3a (μV)
D
Auditory intensity deviance
r = 0.068
p > 0.999
Interoceptive accuracy
MMN (μV)
E
Auditory intensity deviance
r = -0.098
p > 0.999
Interoceptive accuracy
P3a (μV)

Figure 5.

Legends

Figure 1. Somatosensory location deviance MMR and P3a. Left: Grand-averaged waveforms to deviant and standard stimuli (averaged over the locations) and a differential waveform (deviant minus standard). Waveforms represent averages of the electrode pools applied in the analyses. The grey rectangle shows the analysis window for sMMR (150-190 ms) and sP3a (200 - 300 ms). Middle: The topographical maps of responses to deviant (dev) and standard (std) stimuli and differential response (diff) are shown as average voltages over the analysis time window for each component. The channel cluster applied in the analysis is marked in the figure (channels). Right: Bar chart represents mean amplitudes for responses to standard and deviant stimuli and differential responses (averaged over the locations and electrodes applied in the analysis) at the time window of 150-190 ms (sMMR) and 200-300 ms (sP3a). Error bars represent standard error of the mean. *$p < .05$, **$p < .01$

Figure 2. Somatosensory intensity deviance MMR and P3a. Left: Grand-averaged waveforms to deviant and standard stimuli (sMMR: averaged over the low and high intensity, sP3a: high intensity stimuli and low intensity stimuli separately) and a differential waveform (deviant minus standard). Waveforms represent averages of the electrode pools applied in the analyses. The grey rectangle shows the analysis window for sMMR (150-190 ms) and sP3a (200 - 300 ms). Middle: The topographical maps of responses to deviant (dev) and standard (std) stimuli and differential response (diff) are shown as average voltages over the analysis time window for each component. The channel cluster applied in the analysis is marked in the figure (channels). Right: Bar chart represents mean amplitudes for responses to standard and deviant stimuli and differential responses (sMMR: averaged over the intensity and electrodes applied in the analysis, sP3a: averaged over the electrodes applied in the analysis for the high intensity stimuli and low intensity stimuli separately)

at the time window of 150-190 ms (sMMR) and 200-300 ms (sP3a). Error bars represent standard error of the mean. **p < .01

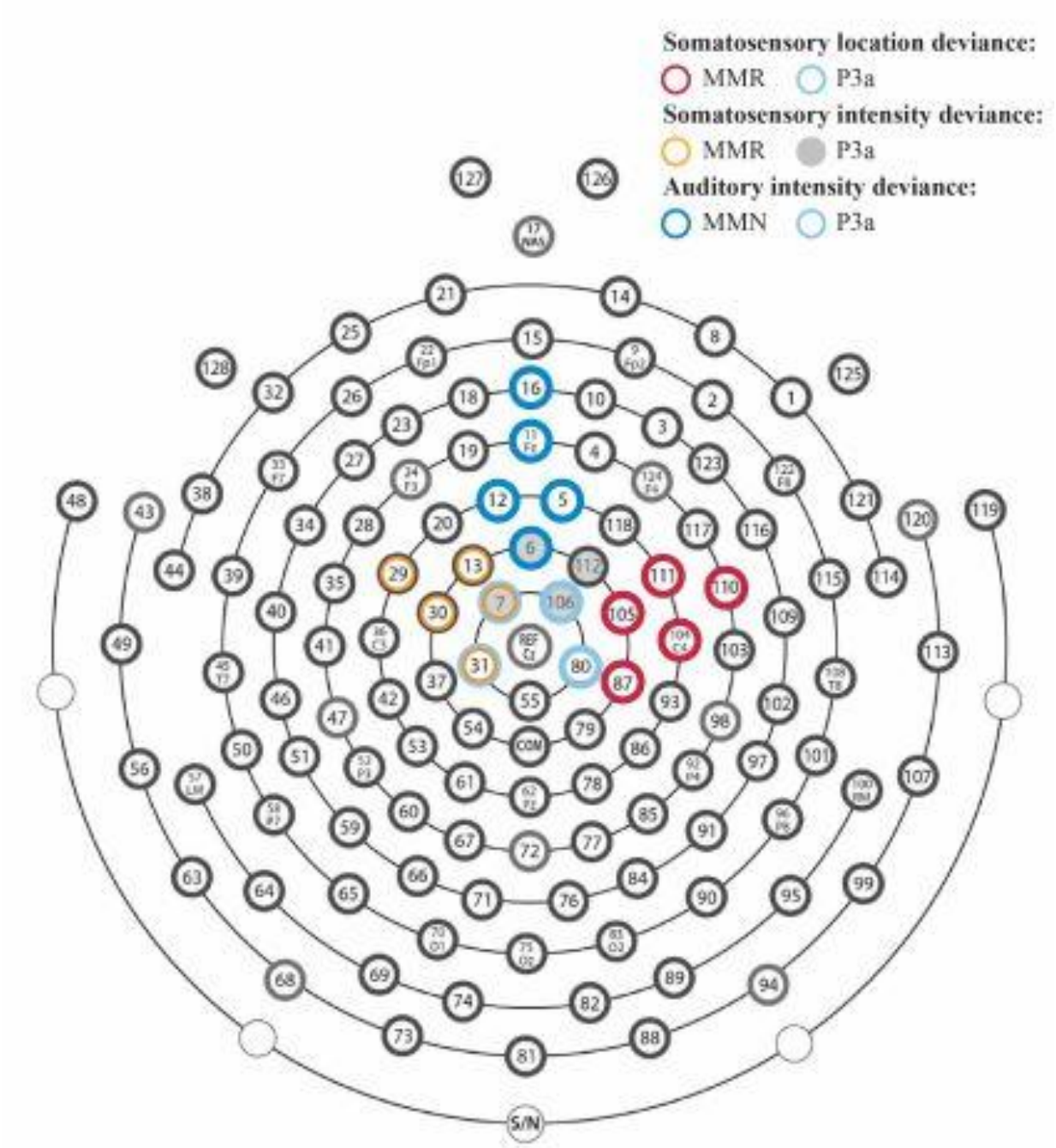
Figure 3. Auditory intensity deviance MMN and P3a. Left: Grand-averaged waveforms to deviant and standard stimuli (averaged over the intensities) and a differential waveform (deviant minus standard). Waveforms represent averages of the electrode pools applied in the analyses. The grey rectangle shows the analysis window for aMMN (140-180 ms) and aP3a (220 - 320 ms). Middle: The topographical maps of responses to deviant (dev) and standard (std) stimuli and differential response (diff) are shown as average voltages over the analysis time window for each component. The channel cluster applied in the analysis is marked in the figure (channels). Right: Bar chart represents mean amplitudes for responses to standard and deviant stimuli and differential responses (averaged over the intensities and electrodes applied in the analysis) at the time window of 140-180 ms (aMMN) and 220-320 ms (aP3a). Error bars represent standard error of the mean. ***p < .001

Figure 4. (A) Correlation (Pearson´s *r*) between somatosensory intensity deviance P3a for the high intensity stimuli and somatosensory location deviance P3a, (B) correlation between somatosensory location deviance MMR and auditory intensity deviance MMN, (C) correlation between somatosensory location deviance P3a and auditory intensity deviance P3a, (D) correlation between somatosensory intensity deviance P3a for the high intensity stimuli and auditory intensity deviance P3a for the high intensity stimuli. Outliers marked in red colour are included in the analysis. Please note that in (A), the correlation is non-significant when the outliers are removed from the analysis.

Figure 5. Correlations (Pearson´s *r*) between interoceptive accuracy and (A) somatosensory location deviance MMR, (B) somatosensory location deviance P3a, (C) somatosensory intensity deviance P3a for the high intensity stimuli, (D) auditory intensity deviance MMN, (E) auditory intensity deviance P3a. Outliers marked in red colour are included in the analysis.

Supplementary Table 1. The averaged number [range] for included trials in the analysis of the somatosensory location deviance, somatosensory intensity deviance and auditory intensity deviance conditions.
NA = not applicable

	Forefinger		**Little finger**		**High intensity**		**Low intensity**	
	Deviant	Standard	Deviant	Standard	Deviant	Standard	Deviant	Standard
Somatosensory location deviance	110.3 [64 -120]	111.3 [58 -120]	111.5 [54 -120]	110.9 [54 -120]	NA	NA	NA	NA
Somatosensory intensity deviance	NA	NA	NA	NA	112.5 [58 -120]	111.8 [60 -120]	111.5 [52 -120]	112.7 [49 -120]
Auditory intensity deviance	NA	NA	NA	NA	96.2 [64 -100]	94.3 [46 -100]	94.3 [36 -100]	95.2 [38 -100]



Measurement	ERP component	Electrode pool
Somatosensory location deviance	MMR	87, 104, 105, 110, 111
	P3a	7, 31, 80, 106
Somatosensory intensity deviance	MMR	7, 13, 29, 30, 31
	P3a	6, 7, 106, 112
Auditory intensity deviance	MMN	5, 6, 11, 12, 16
	P3a	7, 31, 80, 106

Supplementary Figure 1. Map of EGI 128-Channel Net (HydroCel Geodesic Sensor Net) and the electrode clusters applied in the analyses. Please note that in the somatosensory location deviance

condition, the stimuli were delivered to the left hand, and in the somatosensory intensity condition, the stimuli were delivered to the right hand. The ERPs were investigated from the contralateral site in relation to the stimulation.