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Comparison of sensory gating to mismatch negativity and self-reported perceptual phenomena in healthy adults

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Abstract

To better understand the possible functional significance of electrophysiological sensory gating measures, response suppression of midlatency auditory event related potential (ERP) components was compared to the mismatch negativity (MMN) and to self-rated indices of stimulus filtering and passive attention-switching phenomena in an age-restricted sample of healthy adults. P1 sensory gating, measured during a paired-click paradigm, was correlated with MMN amplitude, measured during an acoustic oddball paradigm (intensity deviation). Also, individuals that exhibited less robust P1 suppression endorsed higher rates of "perceptual modulation" difficulties, whereas component N1 suppression was more closely related to "over-inclusion" of irrelevant sounds into the focus of attention. These findings suggest that the ERP components investigated are not redundant, but correspond to distinct—possibly related—pre-attentive processing systems.

Descriptors: Auditory evoked potentials, Sensory gating, Attention, Auditory perception, Auditory stimulation

Event-related potentials (ERPs), because of high temporal resolution and independence from overt behavioral responses, have been used in the investigation of brain processes that underlie pre-attentive sensory processing, including stimulus filtering and selection for involuntary attention switching (Hillyard, Teder-Salejarvi, & Munte, 1998). Sensory gating, for example, is a scalp-recorded electroencephalographic measure that reflects reduced neural activity in response to increased stimulus redundancy (Adler et al., 1998)—the latter typically modeled as pairs (Adler et al., 1982) or trains (Erwin & Buchwald, 1986) of acoustic clicks. Abnormal sensory gating of midlatency auditory ERP components, particularly P1 (P50), has been interpreted to reflect the neural basis of stimulus filtering difficulties associated with certain clinical conditions (Freedman et al., 2002). However, the contribution of sensory gating measures to a more basic understanding of pre-attentive stimulus processing in the general population has been hampered by controversy concerning these measures' specific behavioral correlates (Jin et al., 1998; Light & Braff, 2000) and incomplete characterization of their relationship to other relevant ERPs including mismatch negativity.

The prevailing application of P1 sensory gating measures has been in the study of neuropathology associated with schizophrenia. These patients often complain of sensory inundation and inappropriate orienting to irrelevant stimuli (McGhie & Chapman, 1961). The paired-click ERP paradigm was originally developed to produce an endophenotypic measure of brain function that might prove less complex genetically than schizophrenia itself (Freedman, Adler, & Leonard, 1999), and further to allow for invasive study of the neural mechanisms of intact and impaired sensory gating in an animal model (Adler, Rose, & Freedman, 1986; Bickford-Wimer et al., 1990). These lines of research have provided convergent information on schizophrenia-related neuropathology (Adler et al., 1998), culminating in the identification of abnormal α7-nicotinic receptor function as a critical neurochemical deficit (Freedman et al., 1997; Stevens & Wear, 1997). Despite this, the presumed relationship between behavioral observations regarding stimulus filtering difficulties and P1 suppression abnormalities has not been conclusively demonstrated (Jin et al., 1998; but see Light & Braff, 2000).

Sensory inundation and inappropriate attention switching are certainly not restricted to schizophrenia patients. Other conditions with particularly prominent deficits, and for which P1 sensory gating has been investigated, include traumatic brain injury (Arciniegas et al., 2000; Knight, Staines, Swick, & Chao, 1999), migraine (Ambrosini, De Pasqua, Afra, Sandor, & Schoenen, 2001), attention-deficit hyperactivity disorder (Olincy et al., 2000), and autism (Kemner, Oranje, Verbaten, & van Engeland,

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2002). However, as many as 25% of healthy individuals also endorse "feelings of being flooded/inundated by real sounds" or not being able to "focus attention on one real sound or voice to the exclusion of others" (Bunney et al., 1999). ERP measures of sensory gating have also been shown to vary within the general population (Siegel, Waldo, Mizner, Adler, & Freedman, 1984; Waldo et al., 1994). Thus, the present study was designed to investigate the functional significance of P1 sensory gating by testing for covariation with quantified behavioral observations regarding auditory stimulus filtering and inappropriate attention switching in a sample of healthy adults. Assessment of behavior for this investigation was based on the recently developed Sensory Gating Inventory, a self-report rating scale consisting of items derived from factors labeled "perceptual modulation," "distractibility," and stimulus "over-inclusion" (Hetrick & Smith, in press).

Amplitude and response suppression of auditory ERP component N1 (N100) has also been studied in the context of stimulus filtering and passive attention switching (Escera, Alho, Winkler, & Näätänen, 1998; Näätänen, 1992) including investigations of impaired function in schizophrenia (Adler et al., 1982; Boutros, Belger, Campbell, D'Souza, & Krystal, 1999; Clementz & Blumenfeld, 2001; Ford, Mathalon, Kalba, Marsh, & Pfefferbaum, 2001). Current theory posits N1 amplitude as nominally proportional to a sound's *temporal* novelty—that is, how long ago it occurred, or the inverse of the interstimulus interval—as opposed to *semantic* novelty—the substantive difference from other sounds (Cowan, 1995; Näätänen, 1992). Like P1, this wave is elicited most robustly by the onset of a discrete stimulus against a static acoustic background, and decreases in magnitude as stimuli are repeated at progressively shorter interstimulus intervals (Davis, Mast, Yoshie, & Zerlin, 1966; Nelson & Lassman, 1968). *Unlike* P1, N1 magnitude (Picton & Hillyard, 1974) and suppression in the paired-click paradigm can be modulated by voluntary attention to the stimulus (Jerger, Biggins, & Fein, 1992; White & Yee, 1997) and by brain state (Kisley et al., 2003; Kisley, Olincy, & Freedman, 2001). These findings suggest that P1 and N1 suppression measured in the paired-click paradigm correspond to nonidentical processing systems. This idea was tested in the present study by assessing covariation of P1 and N1 gating measures. The possible relationship of N1 suppression to behavioral phenomena was also investigated.

Amplitude of the mismatch negativity (MMN), another preattentive ERP, indexes semantic novelty of a stimulus, irrespective of interstimulus interval (Näätänen, 1992; Picton, Alain, Otten, Ritter, & Achim, 2000). For example, this wave is elicited when the spectral, temporal, or intensity characteristics of an ongoing stimulus train suddenly change or "deviate." Among its proposed correlates, the MMN has been hypothesized to represent a neural "call to attention" to potentially important sensory stimuli—presumably a precursor to an orienting response (Näätänen, 1992; Schroger, 1996); other theories relating MMN to cognitive function are summarized by Cowan (1995). Within this theoretical framework, suppression of wave P1 due to stimulus redundancy and elicitation of MMN in response to stimulus novelty can be viewed as complementary neural functions, both of adaptive value in the allocation of attention: the filtering of background stimuli and the involuntary (i.e., passive, reflexive) switch of attention to significant stimuli, respectively.

The empirical relationship between P1 sensory gating and the MMN in healthy brain function remains unclear because they have not been studied simultaneously in a single group. A number of clinical conditions, including schizophrenia (Javitt, 2000; Michie, 2001) and frontal lobe syndrome (Alho, Woods, Algazi, Knight, & Näätänen, 1994), are characterized by impairments in both P1 suppression and MMN generation, suggesting a possible functional link between the neural systems that underlie these scalp-recorded ERPs. Also, P1 amplitude was shown to increase in response to pitch deviance during a MMN oddball paradigm (Boutros & Belger, 1999; Boutros et al., 1999). The final goal of the present study was to investigate more directly the possible relationship between P1 sensory gating and MMN generation in normal brain function by testing for covariation of these measures across individuals.

The present investigation was conducted with a sample of healthy, medication-free adults taken from a relatively narrow age range (18–35 years old) with gender as a controlled variable. This was done to avoid detection of spurious correlations due to participant demographics known to affect the measures employed. For example, in addition to the psychiatric and neurological conditions discussed above, P1 sensory gating is known to be impaired in bipolar disorder (Franks, Adler, Waldo, Alpert, & Freedman, 1983), anxiety-spectrum disorders (Neylan et al., 1999; Skinner et al., 1999), and Parkinson's disease (Teo et al., 1997). Psychoactive substances including medications and illicit drugs have also been shown to modulate sensory gating (Adler et al., 1994; Boutros et al., 2000; Light et al., 1999; Nagamoto et al., 1996; Patrick & Struve, 2000). The MMN is also sensitive to many of these factors (see reviews by Csepe & Molnar, 1997; Gene-Cos, Ring, Pottinger, & Barrett, 1999; Pekkonen, 2000). Although P1 sensory gating does not appear to vary systematically with age beyond adolescence (Rasco, Skinner, & Garcia-Rill, 2000), both involuntary attention switching and MMN generation exhibit age dependence within adult groups (e.g., Gaeta, Friedman, Ritter, & Cheng, 2001; Pekkonen, 2000). Finally, because women endorse higher rates of stimulus filtering difficulties than men (Hetrick & Smith, in press) and exhibit significantly less P1 suppression in the paired-click paradigm (Hetrick et al., 1996), all correlations in this study were controlled for gender.

Method

Procedures were approved by the University of Colorado at Colorado Springs Institute Review Board. Participants gave written informed consent before participating, and received monetary compensation or academic extra credit upon completion of the study.

Subjects and General Procedures

Healthy adults between 18 and 35 years of age participated. Exclusion criteria for all participants included current diagnosis of psychiatric or neurological illness, current psychoactive drug use, and past traumatic brain injury (including loss of consciousness lasting at least 5 min after head injury). Hearing was tested at 1000 Hz (binaural, method of limits), and participants were excluded if they exhibited a 5 dB or greater hearing loss. Fifty-two participants met qualifications for the study: mean age 22.1 years ($SD = 4.3$); 11 male; 7 smokers (at least one cigarette per day). Because nicotine's demonstrated modulatory effect on sensory gating lasts less than 60 min (Adler et al., 2001; Adler, Hoffer, Griffith, Waldo, & Freedman, 1992), recordings were not conducted until at least 1 hr after any participant had smoked. None

of the smokers were recorded during a withdrawal period that exceeded normal daily routine.

After completing a survey on perceptual and attentional phenomena (Behavioral Measure), participants underwent two different 30-min ERP recording paradigms, alternately counterbalanced and separated by 5 min. Sensory gating was assessed for all 52 participants with a paired-click paradigm, and MMN for 37 participants by an intensity-deviation oddball paradigm. Fifteen participants were recorded in a time-deviation oddball paradigm that was part of a separate study not described here.

Behavioral Measure

To characterize individuals' daily experience regarding passive attention switching and associated perceptual phenomena quantitatively, an abbreviated version of the Sensory Gating Inventory (Hetrick & Smith, in press) was administered. Participants assigned Likert ratings (from 1 = *never* to 5 = *always*) to 17 questionnaire items.[1] Each item used here was selected from the full inventory by the following criteria: (a) the item was either restricted to the auditory modality or not modality specific, (b) an increasing Likert rating corresponded to an increasing degree of impairment, and (c) the item was not clearly redundant with another item. Each item was assigned to one of the three following dimensions previously identified by factor analysis (Hetrick & Smith, in press): *Perceptual Modulation* (e.g., "I have feelings of being flooded by sounds."), *Distractibility* (e.g., "I have trouble focusing because I am easily distracted."), and *Over-Inclusion* (e.g., "I seem to always notice when automatic appliances turn on and off—like the refrigerator or the heating and cooling system."). An average Likert rating for each participant was computed for each of these three dimensions.

Electrophysiology

Electrophysiological activity was recorded with a NuAmps multichannel amplifier system and Scan 4.2 software (Neuroscan, Sterling, VA). Sounds were presented binaurally through headphones while participants sat in a reclining chair watching a silent, closed-captioned movie. Because stress can modulate sensory gating (Johnson & Adler, 1993; White & Yee, 1997) all movies were selected for minimal arousal value by the subjective criterion of being appropriate for young children. ERP paradigms were programmed and presented with Eprime (Psychology Software Tools, Inc., Pittsburg, PA).

Before recording began, disposable Ag/Ag-Cl electrodes (Vermed, Bellows Falls, VT) were affixed at Cz, Fz, right and left mastoids, directly superior and lateral to the left eye, and the tip of the nose. Ground was affixed to the forehead. During recording, signals were amplified 5000 times, filtered between 0.05 and 100 Hz, and sampled at 1000 Hz. All electrodes were referenced to nose, and impedances maintained below 10 kΩ. Individuals that fell asleep during recording were excluded from further analysis ($n = 2$).

Sensory gating paradigm and analysis. Pairs of acoustic clicks (0.5 s interclick interval) were presented every 9 s throughout the recording session at 60 dB hearing level (HL) against a silent acoustic background. The evoked response at electrode Cz to the first and second clicks of the pair, termed "conditioning" and "test" responses, respectively, were analyzed separately as follows: single trial ERPs, epoched from 100 ms before to 300 ms after each click, were re-referenced to right mastoid, baseline corrected and subject to artifact rejection. Specifically, if the signal on any channel exceeded $\pm$ 75 μV during an epoch, that trial was excluded from further analysis. Individuals that had fewer than 75 conditioning or 75 test trials remaining after artifact rejection were excluded from further analysis ($n = 3$).

For analysis of P1 sensory gating, average waveforms computed from the remaining single trials were bandpass filtered between 10 and 75 Hz (96 dB/octave slope for both corners). This filter was applied both forward and in reverse to eliminate phase distortion. Component P1 in response to the conditioning click was defined as the largest positive waveform peak between 45 and 75 ms. Component P1 for the test click was determined the same way, with the restriction that the peak latency must be within $\pm$ 10 ms of the conditioning latency. If no peak satisfied these criteria, or if the largest peak within this window was below the prestimulus baseline (i.e., less than zero), the P1 test amplitude was taken as zero. Component amplitude was measured relative to a 50-ms prestimulus baseline. The amplitude of wave P1 to the second click of a pair was then compared with P1 magnitude evoked by the first click. Specifically, a ratio of the magnitudes, the test/conditioning (T/C) ratio, was computed to quantify response suppression. A T/C ratio close to 0 indicates robust suppression (very small test response compared with conditioning response) and a T/C ratio of 1 indicates essentially no suppression (test and conditioning responses comparable in magnitude).

For analysis of N1 sensory gating, average waveforms were bandpass filtered between 1 and 30 Hz (48 and 96 dB/octave slope, respectively). N1 was defined for the conditioning click as the largest negative trough between 75 and 125 ms. Test N1 was similarly identified, with the added restriction that the trough must be within $\pm$ 20 ms of the conditioning latency. N1 amplitude was measured relative to the 100 ms prestimulus baseline. Sensory gating was then quantified by computing the T/C ratio, or ratio of magnitudes for test N1 to conditioning N1. Participants for which the conditioning N1 trough was *above* baseline were excluded from further analysis ($n = 2$).

MMN oddball paradigm and analysis. Tones (1000 Hz, 50 ms duration, 5 ms rise/fall) were presented continuously in a train at an offset-to-onset interstimulus interval of 500 ms. "Standard" tones at 45 dB HL were randomly replaced, on average every 15th trial, by a "deviant" tone at 55 dB HL. Single-trial evoked responses, epoched from 100 ms before to 300 ms after tone onset, were baseline corrected and trials for which any channel exceeded $\pm$ 75 μV were discarded. Individuals that had fewer than 75 deviant trials remaining after artifact rejection were excluded from further analysis ($n = 2$). Average waveforms were computed separately for standard and deviant tones, and bandpass filtered from 1 to 30 Hz (48 and 96 dB/octave slope at these corner frequencies). Difference waveforms were computed by subtracting standard from deviant waveforms, point by point. For each participant, MMN was identified on electrode Fz as the most negative trough between 140 and 200 ms poststimulus, with simultaneous positivity on a channel computed as the average of left and right mastoids (Alho, Paavilainen, Reinikainen, Sams, & Näätänen, 1986). Because all minima occurred later than 140 ms, it is not anticipated that increased N1 amplitude elicited by the 10 dB intensity increment confounded MMN amplitude measurement. Peak amplitude was measured relative to a 100-ms prestimulus baseline.

[1]The 17 items taken from the Sensory Gating Inventory (Hetrick & Smith, in press) are available from the authors.

Table 1. *Summary of ERP Measures for Entire Sample*

Variable	*M*	*SD*	Range
Component P1, paired-click paradigm ($n = 47$)			
Conditioning latency	62.6 ms	2.9 ms	54 to 74 ms
Test latency	62.0 ms	3.0 ms	52 to 73 ms
Conditioning amplitude	3.78 μV	1.93 μV	0.85 to 7.88 μV
Test amplitude	1.48 μV	1.06 μV	0.00 to 4.09 μV
T/C ratio	0.40	0.25	0.00 to 1.17
Component N1, paired-click paradigm ($n = 45$)			
Conditioning latency	104.2 ms	9.8 ms	80 to 124 ms
Test latency	104.5 ms	13.6 ms	78 to 125 ms
Conditioning amplitude	8.57 μV	4.56 μV	1.39 to 25.98 μV
Test amplitude	3.07 μV	2.41 μV	0.00 to 8.97 μV
T/C ratio	0.35	0.25	0.00 to 0.94
Component MMN, oddball paradigm ($n = 35$)			
Latency	162.8 ms	12.4 ms	141 to 194 ms
Amplitude	6.29 μV	2.67 μV	1.70 to 13.62 μV

Post hoc, component P1 was also analyzed for this oddball paradigm. To accomplish this, standard and deviant single trial responses were processed in a manner identical to that described in the preceding section for component P1 analysis. P1 amplitude and latency was then measured separately for the standard and deviant average waveforms. As a measure of P1 *enhancement* caused by stimulus novelty, the ratio of deviant P1 amplitude to standard P1 amplitude was computed (deviant-to-standard ratio).

Statistical Analysis

Significance of response suppression in the sensory gating paradigm was assessed by comparing P1 and N1 T/C ratios to 1 by one-distribution *t* test. Partial Pearson's correlation coefficients, controlled for gender, were computed to examine relationships between ERP and behavioral measures. To protect against Type I errors, the number of statistical tests was limited by restricting ERP-to-ERP comparisons to the three summary measures of interest (P1 T/C ratio, N1 T/C ratio, and MMN amplitude). However, all ERP measures (including component amplitudes and latencies) were compared to the behavioral indices (Perceptual Modulation, Distractibility, and Over-Inclusion). All significance tests were two-tailed at the .05 level.

Results

Summary ERP measures for P1 and N1, measured from the evoked responses recording during the sensory gating paradigm, and the MMN waveform, from the oddball paradigm, are provided in Table 1. Mean P1 T/C ratio of 0.40 was different than 1, $t(46) = -16.66$, $p < .001$, indicating significant suppression of this wave in response to stimulus repetition. Mean N1 T/C ratio of 0.35 was also different than 1, $t(44) = -17.19$, $p < .001$. Grand average waveforms computed from the sensory gating and MMN paradigm recordings are shown in Figure 1. Example waveforms for four different individuals are shown in Figure 2. Note different time and voltage scales for the various ERP components.

Comparison of ERP Measures

Pearson correlation coefficients, controlled for gender, were computed between the three summary ERP measures: P1 T/C ratio, N1 T/C ratio, and MMN amplitude. There was a relative lack of systematic relationship between P1 T/C ratio and N1 T/C ratio, zero-order $r(43) = -.08$, $p = .57$; partial $r(42) = -.07$, $p = .65$. On the other hand, P1 T/C ratio and MMN amplitude exhibited a significant correlation, zero-order $r(30) = -.36$, $p = .041$; partial $r(29) = -.43$, $p = .015$. The direction of this correlation indicates that higher P1 T/C ratio (i.e., less response suppression) during the paired-click paradigm was associated with lower MMN amplitude in the oddball paradigm. N1 T/C ratio and MMN amplitude were not significantly correlated, zero-order $r(29) = .22$, $p = .23$; partial $r(28) = .27$, $p = .15$.

To further explore the relationship between P1 and MMN, an unplanned post hoc analysis between these components was conducted within the oddball paradigm. Specifically, amplitude of component P1 was measured for the standard and deviant tones of the intensity-deviation paradigm, and compared to amplitude of the corresponding MMN wave. For the

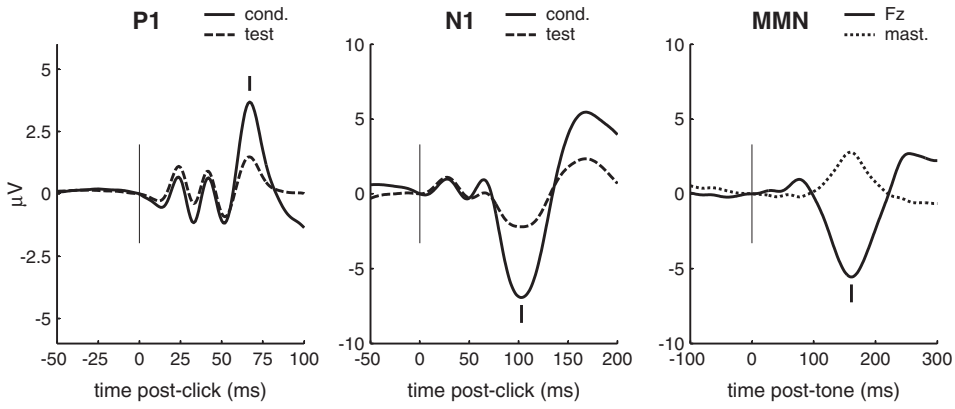

Figure 1. Grand-averaged waveforms across all nonexcluded participants. Left: Average conditioning (solid line) and test (dashed line) evoked responses recorded from Cz during paired-click paradigm, filtered to enhance middle-latency components ($n = 45$). Component P1 indicated by tick mark. Middle: Same original average waveform as left, but filtered to enhance component N1, indicated by tick mark. Right: Average difference waveform evoked during intensity-deviation paradigm ($n = 35$). MMN is identified by tick mark on electrode Fz (solid line) and accompanied by simultaneous positivity at electrode computed as average mastoids (dotted line). For all plots, positivity plotted upwards. Note change in time and voltage scales for P1, N1, and MMN waveforms.

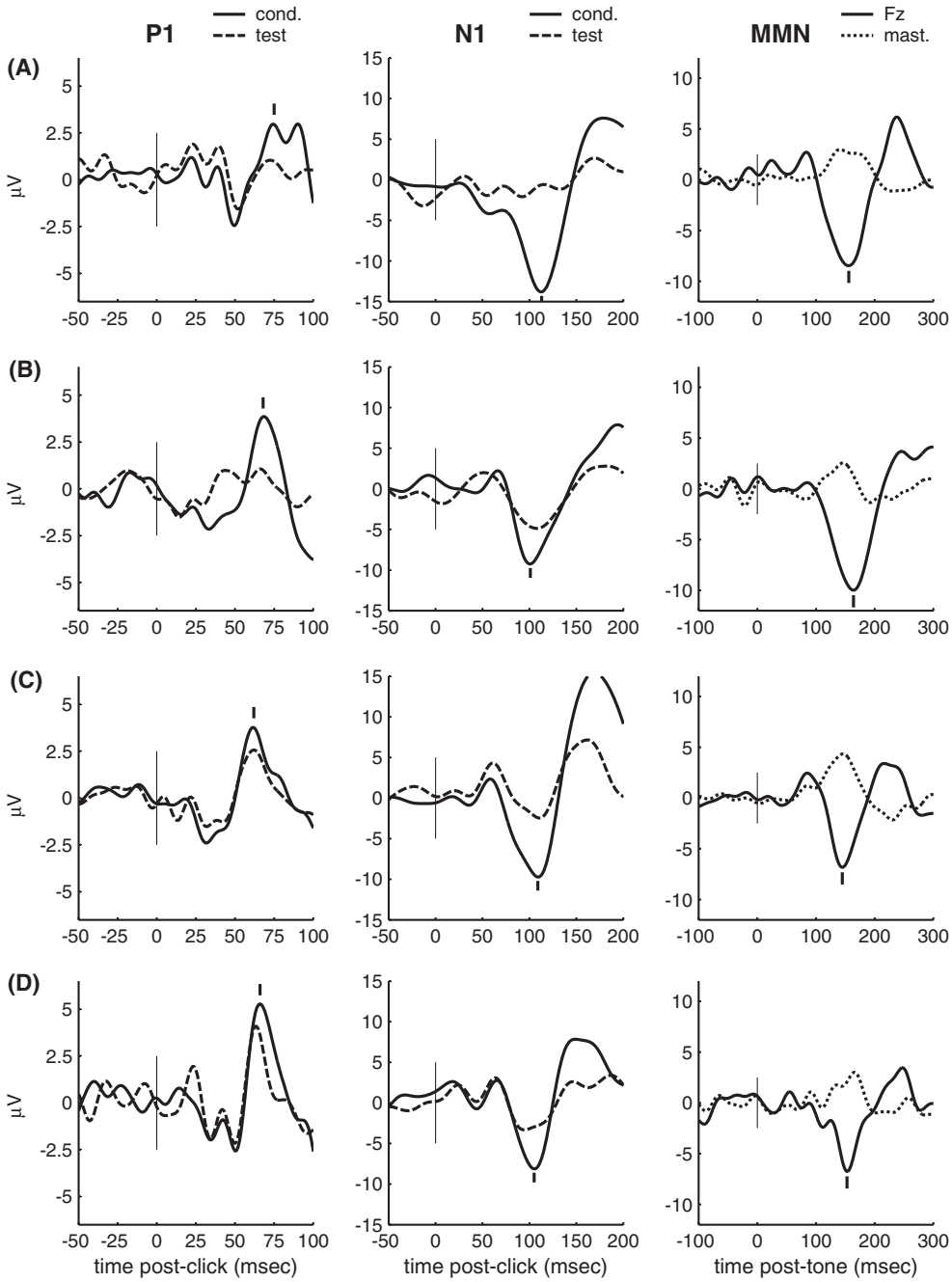

Figure 2. Example event-related potentials from 4 individuals. Electrodes and waveform parameters same as in Figure 1. Waveforms across each row correspond to a single individual: A: P1 T/C = .35, N1 T/C = .08, MMN amplitude = 8.46 μV. B: P1 T/C = .28, N1 T/C = .53, MMN amplitude = 9.99 μV. C: P1 T/C = .68, N1 T/C = .20, MMN amplitude = 6.83 μV. D: P1 T/C = .77, N1 T/C = .41, MMN amplitude = 6.76 μV.

entire sample, mean P1 amplitude was 1.24 ($SD = 0.55$) μV in response to standard tones and 1.48 (0.81) μV in response to deviant tones; these were significantly different from each other, paired $t(31) = -2.49$, $p = .018$, but neither was correlated with MMN amplitude. Furthermore, the P1 deviant-to-standard amplitude ratio ($M = 1.25$, $SD = 0.61$) was not correlated with MMN amplitude, zero-order $r(30) = -.23$, $p = .20$; partial $r(29) = -.25$, $p = .18$. Thus, P1 amplitude discriminated between the occurrence of standard and deviant stimuli, but did not systematically relate to the subsequent MMN waveform amplitude.

Comparison to Behavioral Measures

For all 52 participants, average Likert-rating summary measures were 1.68 ($SD = 0.54$) for Perceptual Modulation, 2.15 (0.69) for Distractibility, and 2.78 (0.79) for Over-Inclusion. Compared to ERP measures, the Perceptual Modulation index was significantly correlated only with P1 T/C ratio, zero-order $r(45) = .28$, $p = .034$; partial $r(44) = .28$, $p = .034$ (Figure 3, top), whereas the Over-Inclusion index was correlated only with N1 T/C ratio, zero-order $r(43) = .33$, $p = .029$; partial $r(42) = .34$, $p = .024$ (Figure 3, bottom). Thus, P1 and N1 sensory gating measures tended to covary with different behavioral phenomena. The pos-

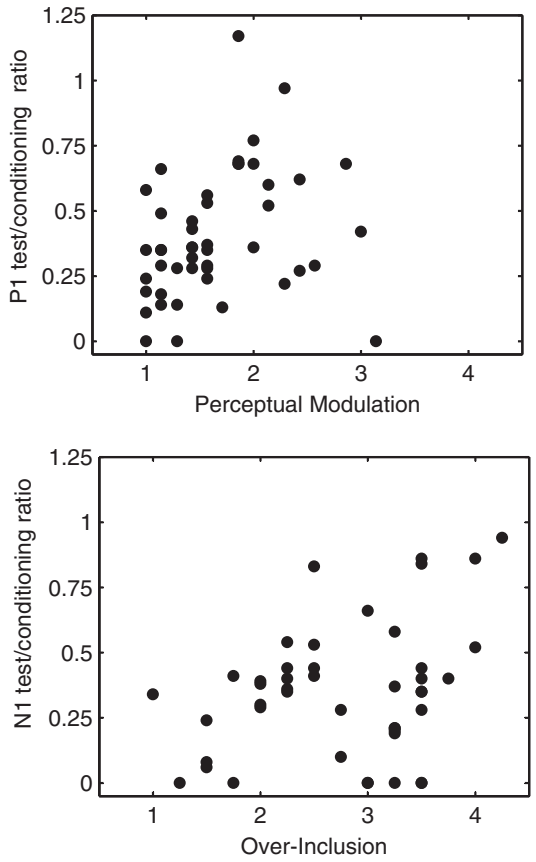

Figure 3. Relationship between behavioral and event-related potential measures. Elevated P1 T/C ratio was associated with a higher rate of reported perceptual modulation difficulties (top), whereas a higher N1 T/C ratio corresponded more closely to problems with over-inclusion (bottom).

itive direction of both correlations indicates that an elevated T/C ratio (i.e., less response suppression) was associated with relatively more self-reported difficulty concerning the behavioral phenomena. MMN amplitude was not significantly correlated with any behavioral index, but this measure was available for fewer participants than for P1 and N1. No other ERP amplitude or latency measures were correlated with any of the behavioral indices.

Because the Perceptual Modulation index included two different subdimensions, filtering difficulties (e.g., hearing "everything at once") and loudness sensitivity, a post hoc analysis was conducted to determine if P1 sensory gating was primarily correlated to one or both of these behavioral phenomena. Across the study group, P1 T/C ratio was correlated to the within-subject average of Likert ratings from Items 2, 4, 5, and 6 (related to filtering), zero-order $r(45) = .29$, $p = .044$, partial $r(44) = .30$, $p = .042$. P1 T/C ratio was not correlated to the within-subject average of Likert ratings from Items 1, 3, and 7 (related to loudness sensitivity), zero-order $r(45) = .21$, $p = .15$, partial $r(44) = .20$, $p = .18$. Thus, the correlation observed between P1 gating and perceptual modulation was primarily determined by the items associated with filtering difficulties.

Discussion

To better understand the possible functional significance of electrophysiological sensory gating measurements, response suppression of midlatency auditory ERPs in the paired-click paradigm was compared to behavioral self-ratings of stimulus filtering and passive attention switching in a group of healthy adults. A modest but significant relationship between P1 sensory gating and the perceptual modulation index was uncovered. Based on a post hoc analysis, the items from this behavioral index that were most closely associated with P1 suppression involved phenomena such as being "flooded by sounds" and hearing "everything at once." Individuals with stronger P1 suppression (i.e., lower T/C ratios) endorsed lower rates of daily acoustic inundation. This is consistent with the hypothesis that suppression of wave P1 in response to auditory stimulus redundancy reflects a pre-attentive filtering process (Freedman et al., 2002).

The observed relationship between P1 gating and behavioral ratings of perceptual modulation should be interpreted with caution due to methodological limitations of the present study. First, click-evoked responses were recorded while participants watched a silent, closed-captioned movie. The majority of P1 sensory gating literature to date report measures taken from individuals sitting or lying quietly, without the influence of stimuli that might distract attention away from the paired clicks. However, watching a silent movie is unlikely to affect P1 gating measurements because the P1 T/C ratio is insensitive to the direction of selective attention (Jerger et al., 1992; Kho et al., 2003; White & Yee, 1997). Second, only a subset of the full Sensory Gating Inventory (Hetrick & Smith, in press) was selected here for behavioral assessment of daily stimulus filtering and passive attention switching phenomena. Therefore, the three behavioral "factors" presented should not necessarily be considered independent of each other. Increased confidence in the present finding awaits replication with the full Sensory Gating Inventory.

The present study produced more evidence for the hypothesis, advanced by Boutros et al. (1999), that P1 and N1 sensory gating measured with the paired-click paradigm represent distinct phenomena. First, P1 T/C ratio and N1 T/C ratio were not correlated. This can be interpreted to imply that strength of P1 and N1 suppression varied independently from each other across this age-restricted, healthy sample. Further, N1 T/C ratio and P1 T/C ratio were correlated with different behavioral indices—over-inclusion and perceptual modulation, respectively. The majority of items in the over-inclusion index might be said to reflect heightened *awareness* of background sounds (e.g., automatic appliances turning on and off) as opposed to being *flooded* by them. The finding that individuals with relatively less N1 suppression (i.e., higher T/C ratios) endorsed increased awareness of background sounds is consistent with a role in passive attention switching to temporally novel sounds for the neural activity underlying this ERP component (Cowan, 1995; Näätänen, 1992).

Sensory Gating and the MMN

A significant correlation was found between P1 T/C ratio, measured from the paired-click paradigm, and MMN amplitude, elicited by deviation in the intensity of an ongoing train of tones in the oddball paradigm. In particular, individuals that exhibited stronger P1 suppression tended to show larger MMN waveforms. Although the underlying cause of this observed correlation remains unknown, a relationship between these variables was anticipated (see the introduction). Together with the large body of literature on the functional significance of the MMN (reviewed by Näätänen & Winkler, 1999; Picton et al., 2000), the present finding hints at a possible link between the neural cor-

relates of pre-attentive stimulus filtering and automatic detection of stimulus novelty.

The observation that stimulus deviance in an ongoing train of tones leads to enhanced P1 amplitude (Boutros & Belger, 1999; present study) and generation of a MMN, in that order, is consistent with a direct causal relationship between these ERP components. Along these lines, attenuation of wave P1 in response to stimulus redundancy in the paired-click paradigm has been interpreted as "gating out," and enhancement of wave P1 in response to stimulus novelty in the oddball paradigm as "gating in" (Boutros & Belger, 1999). The differential neural activity associated with the larger scalp-recorded P1 in the latter paradigm could then subsequently lead, through appropriate synaptic connections, to a neural "call to attention," which manifests at the scalp as a MMN (Näätänen, 1992). However, you would then expect individuals with more robust P1 enhancement in response to stimulus novelty in the MMN paradigm to be those with relatively larger MMN amplitudes. This was not found here: Neither P1 amplitude in response to intensity-deviant tones nor the ratio of deviant-to-standard P1 amplitude was correlated with MMN amplitude in the oddball paradigm. It should be considered that P1 amplitude enhancement in response to the particular deviant stimulus employed here might reflect stimulus intensity (deviants were 10 dB louder than standards) in addition to, or possibly instead of, stimulus novelty.

Other explanations for the correlation between P1 gating and MMN amplitude remain to be tested. Perhaps more efficient filtering of irrelevant sounds at an early stage of processing (reflected by stronger P1 suppression) increases the signal-to-noise ratio of neural representations at subsequent stages (Broadbent, 1958; Cowan, 1995), thus allowing for more robust detection of changes in the physical properties of a repeating stimulus, and subsequently a larger mismatch signal. Alternatively, the observed correlation might result from the influence of a common neural system on both P1 gating and MMN generation. Although past research into P1 generation and suppression has focused primarily on the temporal lobe (Adler et al., 1998; Freedman et al., 2002), recent findings also suggest a role for frontal cortex circuits in sensory gating (Grunwald et al., 2003; Knight et al., 1999; Weisser et al., 2001). Portions of both these cortical regions are also believed to be involved in the generation of MMN waveforms (Alain, Woods, & Knight, 1998; Giard, Perrin, Pernier, & Bouchet, 1990; Rinne, Alho, Ilmoniemi, Virtanen, & Näätänen, 2000).

Implication for the Study of Pre-Attentive Processing Deficits

Among the most replicable ERP impairments associated with schizophrenia are a lack of P1 suppression due to stimulus redundancy (Adler et al., 1982; Boutros, Zouridakis, & Overall, 1991; Clementz, Geyer, & Braff, 1998; Erwin, Mawhinney-Hee, Gur, & Gur, 1991; Judd, McAdams, Budnick, & Braff, 1992; Kisley et al., 2003; Yee, Nuechterlein, Morris, & White, 1998) and reduced MMN amplitude in response to deviation of virtually every stimulus dimension tested (Baldeweg, Klugman, Gruzelier, & Hirsch, 2002; Csepe & Molnar, 1997; Davalos, Kisley, Polk, & Ross, 2003; Javitt, 2000; Michie, 2001; Salisbury, Shenton, Griggs, Bonner-Jackson, & McCarley, 2002). The present finding of increased P1 T/C ratio associated with decreased MMN amplitude, within a group of healthy individuals, mirrors this pattern. Demonstration of an analogous covariation of ERP deficit severity within a group of schizophrenia patients would be consistent with overlapping causes for both deficits. By comparison, lack of covariation across patients would suggest relatively *independent* pathological processes for P1 gating and MMN impairments.

P1 sensory gating deficits associated with schizophrenia have generally been interpreted to correspond to behavioral reports of stimulus filtering difficulties and sensory inundation (Freedman et al., 2002). However, the only direct empirical test in schizophrenia patients contradicted this idea (Jin et al., 1998). Within the present sample of healthy adults, electrophysiological measures of P1 sensory gating were found to be correlated with behavioral measures of perceptual modulation, but not distractibility or over-inclusion. Indirectly, this finding supports the hypothesis that P1 gating deficits in schizophrenia correspond to improper stimulus filtering and subsequent experiences of being "flooded" by sensation, but not necessarily difficulties in passive attention switching. These phenomena have been described as "disorders of perception" and "disorders of attention," respectively (McGhie et al., 1961). The observed correlation between N1 suppression and behavioral measures of over-inclusion, though for healthy individuals, is consistent with the idea that N1 gating deficits documented in schizophrenia (Adler et al., 1982; Boutros et al., 1999; Clementz & Blumenfeld, 2001) correspond more closely to attentional difficulties. As such, parallel investigation of P1 and N1 sensory gating in schizophrenia (and other clinical syndromes associated with pre-attentive processing deficits) might provide information about pathology in separate, though related stimulus processing systems.

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