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Memory-based or afferent processes in mismatch negativity (MMN): A review of the evidence

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Abstract

The mismatch negativity (MMN) is an electromagnetic response to any discriminable change in regular auditory input. This response is usually interpreted as being generated by an automatic cortical change-detection process in which a difference is found between the current input and the representation of the regular aspects of the preceding auditory input. Recently, this interpretation was questioned by Jääskeläinen et al. (2004) who proposed that the MMN is a product of an N1 (N1a) difference wave emerging in the subtraction procedure used to visualize and quantify the MMN. We now evaluate this "adaptation hypothesis" of the MMN in the light of the available data. It is shown that the MMN cannot be accounted for by differential activation of the afferent N1 transient detectors by repetitive ("standard") stimuli and deviant ("novel") stimuli and that the presence of a memory representation of the standard is required for the elicitation of MMN.

Descriptors: Auditory event-related potential (AERP), Auditory change, Mismatch negativity (MMN), Feature-detector adaptation, N1

The Memory-Trace Explanation of the MMN

The mismatch negativity (MMN) is an electromagnetic response to a discriminable change in any regular (e.g., repetitive) aspect of auditory stimulation, which usually peaks at 150–200 ms from change onset and is elicited even in the absence of attention (Näätänen, Gaillard, & Mäntysalo, 1978). A commonly accepted interpretation of the MMN is that it is generated by an automatic change-detection process in which a disconcordance is found between the input from the deviant auditory event and the sensory-memory representation of the regular aspects of the preceding auditory stimulation (for reviews, see Näätänen, 2001; Näätänen & Winkler, 1999). It was further proposed that this change-detection process occurs preperceptually in the auditory cortices (generating the auditory-cortex subcomponent of the MMN), which in turn triggers frontal-cortex processes (generating the frontal MMN subcomponent and in part the subsequent P3a; Alho et al., 1998) underlying the possible attention switch to and conscious perception of stimulus change (Giard, Perrin, Pernier, & Bouchet, 1990; Näätänen, 1990; Näätänen & Michie, 1979; Rinne, Alho, Ilmoniemi, Virtanen, & Näätänen, 2000).

This interpretation postulates an MMN-generator process that is separate from the afferent processes triggered by *both* the frequent ("standard") and infrequent ("deviant") stimuli (or stimulus events), that is, that separate memory-related neuronal activity is involved rather than just new (or fresh) afferent elements, those activated by the deviants but *not* by the standards (see, e.g., Näätänen & Alho, 1997). Thus, according to this view, the MMN-generating neurons will be distinct from those generating the N1, the main negative deflection peaking at about 100 ms from stimulus onset, which is composed of several subcomponents (for a review, see Näätänen & Picton, 1987), and from those generating the other obligatory components of the event-related potential (ERP).

An Alternative Interpretation: The Adaptation Hypothesis

Recently, Jääskeläinen et al. (2004) proposed that the ERP response termed as the MMN might be a product of neurons that generate the obligatory N1 wave. That is, there would be no separate MMN response. According to this "adaptation hypothesis," the observable MMN wave results from subtracting the N1 response elicited by the standards from that elicited by the infrequent, deviant (or, in their terminology, "novel") stimuli.

This hypothesis is based on two of the subcomponents of the N1 response, whose characteristic behavior would, according to this hypothesis, produce the MMN wave: a posterior subcomponent "N1p," peaking at about 85 ms from stimulus onset, and an anterior subcomponent, "N1a," peaking at about 150 ms (see Loveless, Levänen, Jousmäki, Sams, & Hari, 1996; for a review,

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see Näätänen & Picton, 1987). According to the adaptation hypothesis, subtracting the (strongly attenuated or adapted) N1 elicited by standards from that to frequency deviants produces a negative displacement peaking at around 150 ms from stimulus onset (erroneously interpreted as a separate MMN component generated by change-specific neurons). This is because the adaptation effect caused by standard-stimulus repetition is smaller on the N1a response to frequency deviants than on the N1p response to these deviants, owing to the narrower frequency tuning of the N1a than N1p neurons. Thus, frequency deviants would elicit a larger N1a response than that elicited by standards, whereas the N1p response to these deviants would be larger than that to the standards only with relatively wide frequency deviations.

It was further suggested that these posterior auditory-cortex neurons generating the N1p, when adapted by repetitive stimuli, gate novel or deviant stimuli into consciousness. Thus, a fast, bottom-up automatic mechanism for attention switch to novel sounds was proposed, which is based on the transient frequency-specific adaptation of posterior auditory-cortex feature-detector neurons.

Even though including some novel aspects (in particular the assumption that no separate MMN kind of mechanism exists), a hypothesis of involuntary attention switch based on N1-neuron activation by auditory change is not entirely new. In fact, Näätänen (1990, 1992), in his model on automatic and attention-dependent processes in audition, proposed that sound change has two parallel routes to attention switch: (1) via MMN neurons; (2) via such feature-specific N1 neurons (generating the supratemporal N1 subcomponent at the N1p latency; see Loveless et al., 1996; Näätänen & Picton, 1987) that are activated by deviants but not by standards (i.e., a qualitative sound change activates the "Transient-Detector System," which then engages the executive mechanisms of attention control; Näätänen, 1990, 1992).

More recently, Ulanovsky, Las, and Nelken (2003), using single-cell recordings of neurons in the cat auditory cortex, proposed the involvement of new afferent elements in attention switch to sound change (novelty detection), as also proposed by Jääskeläinen et al. (2004). Neurons in the cat primary auditory cortex responded more strongly to a rarely presented sound than they responded to the same sound when it was frequently presented. This result was obtained both with frequency and intensity deviants. Interestingly, some neurons even showed hyperacuity for frequency deviants—a frequency resolution one order of magnitude higher than that of the receptive-field widths in the primary auditory cortex. According to the authors, these phenomena resulted from stimulus-specific adaptation in the primary auditory cortex, which, they suggested, provided a single-neuron correlate of the MMN and thus were involved in novelty (deviance) detection. They therefore interpreted their results as indicating that the A1 neurons, in addition to processing the acoustic features of sounds, may also be involved in sensory memory and novelty detection. In contrast, no evidence for a separate MMN type of response was obtained.

However, no recordings were made from the secondary or association cortices in which previous studies (Csépe, Karmos, & Molnár, 1987, 1988a, 1988b; Pincze, Lakatos, Rajkai, Ulbert, & Karmos, 2001, 2002) have found an MMN type of activity in cat. This activity originated from a locus different from that of the N1 response. For example, Pincze et al. (2001) concluded that the MMN was generated in the rostroventral part of the secondary auditory cortex, well separated from the sources of the P1 and N1 components. Moreover, other animal data provide convincing evidence for the functional separability of the N1 and MMN neurons. For instance, Javitt, Steinschneider, Schroeder, and Arezzo (1996), using intracranial monkey recordings, found that an NMDA-receptor antagonist MK-801 eliminated the MMN kinds of responses to frequency and intensity deviants but left the afferent responses intact. Based on such data, Javitt et al. (1996) confirmed and extended Näätänen's (1984, 1990) suggestion of the selective release (involving all other frequencies except for that of the stimulus itself) from tonic inhibition of a frequency-specifically organized neuronal population in the auditory cortex as the basic mechanism of memory-trace formation for tone frequency. This transient selective release from inhibition (lasting for a few seconds) caused by the first same-frequency tone stimuli of a block would enable this neuronal population to generate the MMN response to a subsequent different stimulus.

Can the Adaptation Hypothesis Account for the Existing MMN Data?

Because of its close correspondence to behavioral discrimination accuracy as well as its attention- and task-independent elicitation, the MMN has prompted new perspectives both in basic and clinical/applied cognitive brain research (for reviews, see Näätänen & Escera, 2000; Näätänen & Winkler, 1999) and has been involved in more than 1000 articles in international refereed journals. Therefore it is necessary carefully to reconsider the traditional explanation of the mechanism involved when it is challenged.

As already mentioned, according to the adaptation hypothesis, the MMN reported in the literature would be identical to the N1 difference between the deviant (novel) and standard sounds. Further, the fact that the MMN usually peaks later than the N1 and that the MMN generator locus (estimated by equivalent current dipoles, ECD) is anterior to that of the N1 would be explained by a larger N1-amplitude difference between the deviant and standard for the later, anterior (N1a), than for the earlier, posterior N1 subcomponent (N1p). Thus, in fact, the MMN reported in literature would emerge as an artifact of the subtraction procedure used to visualize and quantify the MMN and would thus represent no genuine ERP component, that is, one generated by a separate neuronal process. (For a definition of an ERP component, see Näätänen & Picton, 1987.) Further, the difference in the locus of origin between the N1 and MMN reported in previous studies (e.g., Hari, Rif, Tiihonen, & Sams, 1992; Korzyukov et al., 1999; Sams, Kaukoranta, Hämäläinen, & Näätänen, 1991) would be only "illusory," the true locus for the MMN being that of the N1a. In addition, because feature detectors exist also for other auditory stimulus attributes, the adaptation hypothesis might, according to the authors, explain MMNs also for other types of auditory change.

Although both types of explanations of the MMN can account for most of the ERP responses observed when a repetitive sound is occasionally replaced by a sound differing from it in frequency, there are a number of phenomena traditionally interpreted in the framework of the MMN that cannot be explained on the basis of the adaptation hypothesis. The adaptation hypothesis predicts that (1) the MMN latency and duration correspond to those of the N1a (or N1p + N1a); (2) the MMN only occurs under conditions in which the N1 (N1a) is elicited (at least by deviant sounds); (3) no MMN can be found under conditions in which the selective adaptation of feature–specific neurons

cannot occur; (4) the MMN scalp distribution corresponds to that of the N1a; (5) experimental manipulations affect the MMN and N1a similarly; (6) the behavioral sensitivity of involuntary attention switch to frequency change should correspond to the capacity of the N1p neurons to respond to deviants when adapted by a standard of a slightly different frequency. As briefly reviewed below, there exists compelling evidence against each of these predictions.

1. The MMN Latency and Duration Do Not Match Those of the N1a

A central prediction derived from the adaptation hypothesis is that the MMN response peaks at around 150 ms (i.e., at the N1a peak latency) irrespective of the magnitude of stimulus change. In contrast, when the magnitude of frequency change was varied by Tiitinen, May, Reinikainen, and Näätänen (1994), the MMN peaked with very small frequency changes at about 200 ms and with wide frequency changes at 100–150 ms (see Näätänen & Alho, 1995). Corroborating results were obtained by Lang et al. (1990) and Winkler, Tervaniemi, and Näätänen (1997), who found that the MMN to barely discriminable frequency changes peaked at 200–300 ms from change onset. This extensive latency variation of the MMN as a function of the magnitude of stimulus change, observed for frequency as well as for other sound changes (Näätänen, Paavilainen, Alho, Reinikainen, & Sams, 1989; Näätänen, Paavilainen, & Reinikainen, 1989; Figure 1), is at odds with the relatively constant N1a latency (for a review, see Näätänen & Picton, 1987). The N1-neuron activation is quite closely time-locked to stimulus onset (irrespective of the magnitude of stimulus change), whereas the MMN latency depends on the magnitude of stimulus change. These data, of course, implicate that the N1 and MMN neurons are separate.

Moreover, in the aforementioned Lang et al. (1990) and Winkler et al. (1997) studies, the MMN duration was considerably longer (about 150–300 ms) and of larger amplitude than what could be expected if the MMN were an N1a (or N1p + N1a) difference wave only. In addition, similarly late, long-duration MMNs can also be found for several other types of deviants (e.g., for intensity deviants; Näätänen, Paavilainen, Alho, et al., 1989; for duration deviants; Näätänen, Paavilainen, & Reinikainen, 1989; see Figure 1). Such late, long-duration MMNs of often large amplitude cannot be explained by a hypothesis based on N1 feature-detector adaptation. Finally, using a protocol that separated the genuine MMN from the differential refractoriness of the N1 wave neurons, Jacobsen and Schröger (2001) showed a latency dissociation between these two contributions to the deviant-minus-standard difference wave.

2. MMN to Deviants in the Absence of the N1

An MMN can be elicited even by auditory events eliciting no N1 response. For example, stimulus omission in a sequence of brief tone pips presented at a short constant SOA elicits an MMN (Yabe, Tervaniemi, Reinikainen, & Näätänen, 1997; Yabe et al., 1998), as does the omission of the second tone of a regularly presented tone pair with a short intertone interval (Tervaniemi, Saarinen, Paavilainen, Danilova, & Näätänen, 1994). These MMNs cannot be explained by the selective adaptation of any N1 or N1a neurons, for no such neurons are activated in the absence of an external stimulus. Moreover, robust MMNs (or comparable slow positive waves; Ruusuvirta, Huotilainen, Fellman, & Näätänen, 2003; Winkler et al., 2003) to various kinds of auditory change can be recorded even in newborns in whom no well-defined N1–type of response is elicited (e.g., Alho, Sainio, Sajaniemi, Reinikainen, & Näätänen, 1990; Cheour et al., 2002). In addition, almost normal-size MMNs (reflecting the effect of prior discrimination training in wakefulness) to a change in a complex spectrotemporal stimulus could be recorded in REM sleep during which the N1 was almost completely abolished (Atienza & Cantero, 2001).

3. MMN Elicitation When Selective Feature-Specific Adaptation Cannot Occur

Stimulus repetition in a sequence of tones steadily ascending or descending in frequency (standards) elicits a distinct MMN (Figure 2; Tervaniemi, Maury, et al., 1994), even though there is no stimulus repetition among the "standard" (regular) stimuli that could cause selective adaptation. In another example of an MMN being elicited with no possibility of the frequency-specific adaptation of N1-generating neurons, Paavilainen, Simola, Jaramillo, Näätänen, and Winkler (2001; see also Saarinen, Paavilainen, Schröger, Tervaniemi, & Näätänen, 1992) used stimuli widely varying both in frequency and intensity but conforming to an abstract rule involving the conjunction of the two sound features, such as "the higher the frequency, the higher the intensity." Again, no selective adaptation could occur, for there was no stimulus repetition. Nevertheless, an MMN was elicited, in subjects ignoring these stimuli, by sounds violating this abstract rule, that is, by infrequent high-frequency tones of low intensity or low-frequency tones of high intensity. See also Gomes, Bernstein, Ritter, Vaughan, and Miller (1997) and Ruusuvirta et al. (2003). (The mechanisms of generation of these more cognitive kinds of MMNs [for a review, see Näätänen, Tervaniemi, Sussman, Paavilainen, & Winkler, 2001] of course involve other, obviously higher-order, neuronal populations than those activated by a mere frequency change.)

Finally, the MMN is sensitive to backward masking in a way similar to that in auditory sensory memory (Winkler, Reinikainen, & Näätänen, 1993). Because the standard, the deviant, and the mask stimulus were all kept constant, frequency-specific adaptation cannot explain the effect of the standard-to-mask interval on the elicitation and amplitude of the MMN.

4. The MMN Scalp Distribution and Generator Locus Do Not Correspond to Those of the N1a

All auditory-cortical N1 subcomponents are larger in amplitude above the hemisphere contra- rather than ipsilateral to the stimulated ear (for a review, see Näätänen & Picton, 1987). In contrast, the MMN to changes in simple acoustical features such as frequency, duration, or intensity is right-hemispheric predominant (and left-hemispheric predominant to phoneme changes; Näätänen et al., 1997; Shestakova et al., 2002) irrespective of the ear stimulated (Paavilainen, Alho, Reinikainen, Sams, & Näätänen, 1991). Also, the MMN scalp distribution differs for different auditory attributes (such as frequency, duration, and intensity; Giard et al., 1995; see also Frodl-Bauch, Kathmann, Möller, & Hegerl, 1997) and for simple and complex sounds (Alho et al., 1996), which is very difficult to explain with the adaptation hypothesis. Consistent with these data, Molholm, Martinez, Ritter, Javitt, and Foxe's (in press) recent functional magnetic resonance imaging (fMRI) study revealed differences between the activation patterns for frequency and duration deviants, suggesting that with the hemodynamic correlates of the MMN, one might be able to map the neuroanatomy of sensory memory in audition. For corroborating magnetoencephalography

(MEG) results, see Rosburg (2003) and Levänen, Ahonen, Hari, McEvoy, and Sams (1996).

Furthermore, the MMN has its second main generator in the frontal lobes (Alho, Woods, Algazi, Knight, & Näätänen, 1994; Dittmann-Balcar, Jüptner, Jentzen, & Schall, 2001; Giard et al., 1990; Molnár, Skinner, Csépe, Winkler, & Karmos, 1995; Näätänen & Michie, 1979; Müller, Jüptner, Jentzen, & Müller, 2002; Opitz, Rinne, Mecklinger, & von Cramon, 2002; for a review, see Alho, 1995), partly explaining the high MMN amplitudes over the frontal scalp, which are not mirrored by the N1a topography (for a review, see Näätänen & Picton, 1987).

In addition, very recently, Rosburg, Haueisen, and Kreitschmann-Andermahr (2004) found that the dipoles of the magnetic equivalents of the N1 (N1m or N100m) and the MMN (MMNm) disclosed dipole shifts with different orientations during their respective latency ranges, which militates against the assumption that the MMN is a derivative of the N1 (N1a) response.

5. The Sensitivity of the N1a to Various Experimental Manipulations Does Not Correspond to That of the MMN

Different pharmacological manipulations affect the N1 (N1a) and MMN differently. For instance, as already mentioned, the administration of an NMDA-receptor antagonist MK-801 abolished the MMN in monkey with no effect on the afferent responses such as the N1 (Javitt et al., 1996), and thus on the N1a. In addition, some drugs enhanced the N1 amplitude but attenuated the MMN amplitude, and vice versa (e.g., Umbricht et al., 2000). Furthermore, certain lesions may eliminate the MMN but leave the N1 intact (e.g., Alho et al., 1994). Also, subjects can be trained to perform an initially very difficult discrimination, which is accompanied by the emergence and growth of the MMN with no corresponding effect on the N1 or N1a (Atienza, Cantero, & Dominguez-Marin, 2002; Näätänen, Schröger, Karakas, Tervaniemi, & Paavilainen, 1993). In addition, Lopez et al. (2003) found that the MMN and its magnetic counterpart MMNm to different violations in "musical sequences" were larger in amplitude in "musical" than "nonmusical" subjects, whereas there was no N1 difference between the groups.

All these data implicate separate N1 (N1a) and MMN generators (even though their locus difference might be too small for the resolution of some of the technologies available).

6. The Behavioral Sensitivity of Attention Switch to Frequency Change Exceeds That Predicted on the Basis of the Frequency Specificity of the N1p Neuronal Population

The wide receptive fields of the posterior N1 neurons (N1p) do not permit one to explain with these neurons attention switches to minor frequency changes, which were observed in some studies. For instance, Berti, Roeber, and Schröger (2004) found that the performance in a tone-duration discrimination task was deteriorated even by occasional 1% frequency changes in this tone. This indicates that even such minor frequency changes that cannot, because of the relatively wide receptive fields, elicit an enhanced N1p response (relative to that to standards) cause an attention switch away from the task performance (see also Schröger & Berti, 2000).

The wide range of studies briefly reviewed above show that the MMN is difficult to explain by a model based on afferent neurons. Rather, higher-order, memory-related mechanisms seem to be involved. Consequently, MMN does not appear to be "an N1 response suppressed and delayed by stimulus specific adaptation (i.e., that the responses would be generated by common cortical sources)," as suggested by the adaptation theory (Jääskeläinen et al., 2004, p. 6813).

Jääskeläinen et al.'s New Experiments

The data reported by Jääskeläinen et al. (2004) do not alter the conclusions based on this literature review. Experiment 1 aimed at showing that, in contrast to the prevailing view, an MMN kind of response can be elicited even without the repetitive presentation of a preceding different (standard) stimulus. The results of Condition A (see Jääskeläinen et al., 2004, Figure 1) in that experiment indeed show a negative difference elicited by the second tone of a tone pair composed of two different tones (AB) when compared with the second-tone response in pairs with two identical tones (BB). This difference between the B-stimulus responses in the two conditions can, however, be attributed to the difference in the refractory state of those N1-generating neurons that differentially respond to the A and B tones, for B was presented 100% of the time in the control blocks, but only 50% of

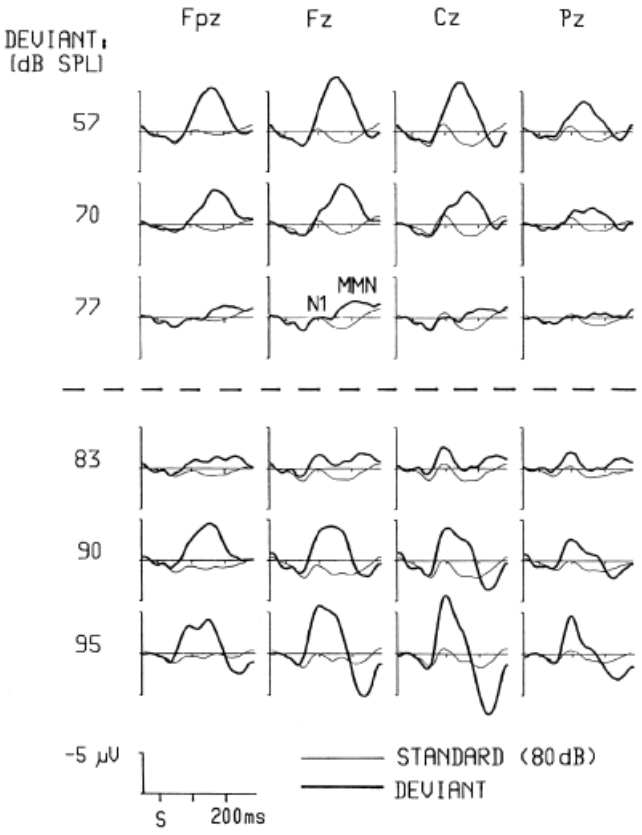

Figure 1. Grand-average frontal-pole (Fpz), frontal (Fz), vertex (Cz), and parietal (Pz) event-related potentials (ERPs) to standard stimuli of 80 dB (thin lines) and to deviant stimuli of different intensity (thick lines) as indicated on the left. The MMN amplitude is increased and latency decreased with increasing magnitude of intensity deviation. For the smallest intensity decrement, N1 and MMN can be seen as clearly separate but with further intensity reduction, MMN moves earlier, overlapping N1. With intensity increments, in turn, a similar data pattern is observed, however with the exception that the N1 generator activity now is enhanced (rather than diminished, as is the case with intensity reductions). This enhancement is proportional to the deviant-stimulus intensity. From Näätänen (1992), copyright Lawrence Erlbaum Associates Publishers, reprinted with permission.

the time in the test blocks (for supporting data, see Butler, 1968, 1972; Cowan, Winkler, Teder, & Näätänen, 1993; Näätänen et al., 1988; Picton, Woods, & Proulx, 1978).

Thus, it appears that no MMN type of activity was generated (which supports the traditional memory-representation interpretation of the MMN). To separate the differential refractoriness from the genuine MMN, the authors should have used the control protocol developed by Schröger and Wolff (1996) (and applied to the frequency MMN by Jacobsen and Schröger, 2001) or equated the probabilities of the A and B tones within a single stimulus block (i.e., mixing AA, AB, BA, and BB pairs equiprobably), as was done by Winkler (1993, p. 88) who found no MMN under these circumstances. (For compatible results, see Schröger, 1997a).

In Experiment 2, the responses elicited by test tones (''novels'': 1, 2, or 4 octaves higher than standards, in separate blocks) presented alone were compared with those elicited by the same test tones embedded in the repetitive sequence of standard tones. The N1 amplitude elicited by the three different test tones when presented alone depended on the spectral makeup of these tones, as is known for the exogenous N1 response (for a review, see Näätänen & Picton, 1987). When presented together with the standards, the octave-deviant test tone elicited two clear negative waves, one coinciding with the N1 peak elicited by the same tone alone, the other peaking ca. 130 ms later. Further, with an increasing spectral difference between the test and standard tones, the second negative peak moved earlier, finally merging with the N1 response (see Jääskeläinen et al., 2004, Figure 2). It was argued that this gradual convergence of the responses elicited by the infrequent, ''novel'' sounds alone and when presented among

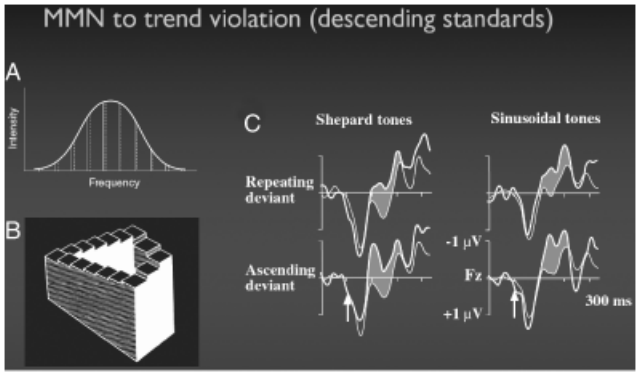

Figure 2. A: A spectrum of an individual Shepard sound that, when presented in ascending or descending sequences of 12 sounds in one semitone steps, causes a pitch to ascend or descend, respectively, in an endless manner. One Shepard sound consists of 10 frequency components, of one octave apart, with a bell-shaped spectrum. While a 12-tone series of Shepard sounds is delivered, the tone-height (which is equivalent to the sense of octave) perception is made to disappear by manipulating the sound spectrum. B: A visual analogy of the Shepard illusion, the endlessly ascending or descending stairs. C: Event-related potentials (ERP) recorded at the frontal (Fz) electrode from reading subjects to Shepard and sinusoidal tones (thin line, standard stimulus; thick line, deviant stimulus). The left column presents ERPs from a regularly descending Shepard sound sequence randomly replaced by a repeating (top) or an ascending (bottom) tone (deviant). The right column presents corresponding ERPs from an identical condition with sinusoidal tones. The arrow indicates deviant-stimulus onset and the shadowed area the statistically significant part of the mismatch negativity. Adapted, with permission, from Tervaniemi, Maury, and Näätänen (1994). Copyright Rapid Science Publishers.

standards supports the notion of a common mechanism for the N1 and the MMN. However, in light of previous studies (e.g., Tiitinen et al., 1994), the second negative peak can be identified as a genuine MMN, for the MMN gets earlier in latency with increasing deviation from the standard, finally merging with the N1. This is because, with greater stimulus deviations, mismatch between the deviant sound and the memory representation of the standard can be detected faster. Therefore, because the observable MMN response reflects the outcome of the deviance detection process (Ritter, Deacon, Gomes, Javitt, & Vaughan, 1995), the MMN peak moves earlier with increasing separation between the standard and deviant sounds. Consequently, it appears that the dependence of the peak latency of the difference wave on the magnitude of stimulus deviation resulted from that of the (genuine) MMN and not from that of the N1a. Therefore, these data rather seem to support separate N1 and MMN generator mechanisms.

It was further argued that, because the negativity to the test tones with the four-octave separation in Experiment 2 was not larger in amplitude when standards were present than when they were absent, the whole response can be explained by the activation of the N1–generating neurons. However, it is well established that the contribution from the N1 generators is considerably smaller when the tone rate is higher (i.e., when standards are present than when they are absent; Näätänen & Picton, 1987). The fact that the total negativity to the test tones (novels) was not larger when standards were present than when they were absent is probably explained by a contribution from a separate MMN generator that balanced the decreased contribution from the N1 generator. (This weakening of the N1 process when standards are present compared with when they are absent can be seen in the one-octave separation condition in which the N1 and MMN are clearly separate in time; see Figure 2 in Jääskeläinen et al., 2004.)

Schröger and Wolff (1996; see also Näätänen & Alho, 1997; Schröger, 1997b) recently developed a new protocol for disentangling the N1 and MMN components from one another. Their results showed exactly the same separation of the N1 and MMN as can be seen for the one-octave separation condition of Jääskeläinen et al. (2004), Experiment 2 (Figure 2) whose ERP results hence are fully compatible with the notion of the separate N1 and MMN mechanisms. Furthermore, the protocol that controlled for the differential states of refractoriness of the feature-specific afferent neurons demonstrated a dissociation of the N1 and MMN to frequency changes (Jacobsen and Schröger, 2001). Similar results were obtained for MMNs to changes in location, duration, and intensity (Jacobsen and Schröger, 2003; Jacobsen, Horenkamp, & Schröger, 2003; Schröger & Wolff, 1996).

In estimating the locus of the MMN (using both the MEG and fMRI methodologies), Jääskeläinen et al. (2004) unfortunately included considerable neural activity originating from the N1 generator, and even more so with larger spectral differences between the standard and deviant sounds, because of the decreased overlap between the neuronal populations responding to the standard and deviant sounds. Thus, when the separability of the N1 and MMN sources was tested, in fact, N1 was compared with MMN + N1, which seriously decreased the sensitivity of the method used to determine whether there is a significant difference (which is critical when only 7 subjects were used; see Jacobsen, Schröger, Horenkamp, & Winkler, 2003). Nevertheless, the MEG data suggested a difference, that is, that the MMN locus was anterior to that of the N1 (although this effect just fell below

the level of statistical significance, $p < .07$). Consequently, the fact that no significant difference between the source loci of the MMN and the anterior N1 (N1a) in the MEG or fMRI was found does not constitute strong evidence supporting common origin of the MMN and the anterior N1. The clear trend in their MEG data rather is in line with the rest of the literature suggesting separate generator loci for the N1 (N1a) and MMN.

Particularly compelling evidence for the separate generator mechanisms for the N1 and MMN was provided by Kropotov et al.'s (2000) human intracranial recordings (see also Halgren, Baudena, Clarke, Heit, Liégeois-Chauvel, et al., 1995; Halgren, Baudena, Clarke, Heit, Marinkovic, et al., 1995; Halgren, Marinkovic, & Chauvel, 1998). They found that one area of the temporal cortex (Area 22) gave a differential response to deviants (1300-Hz tone) among standards (1000-Hz tones) in the MMN latency range, but did not respond differentially to these tones when each of them was presented alone in a sequence, and did not respond differentially to the 1000-Hz standard tone when it was (alone) presented at a slow (deviant-stimulus) or fast (standard-stimulus) rate. In contrast, frequency-dependent responses were recorded from Area 41 and responses to rarity per se (i.e., a larger response to deviants alone at their typical long intervals than that to these stimuli alone at short intervals) from Area 42 (cf. Ulanovsky et al., 2003). Hence, the existence of genuine change-dependent responses could be verified and their cortical origin separated from that of the cortical reception of the afferent input that gives rise to frequency- and rate-specific cortical afferent responses such as the N1 (N1a). For supporting evidence, see Liasis, Towell, and Boyd (1999).

Conclusions

In conclusion, the MMN cannot be interpreted as an N1 (N1a) response that is suppressed and delayed by stimulus-specific adaptation, that is, as an afferent response with no involvement of memory (encoding the regular features of the preceding stimulation). As we have shown here, none of the predictions based on this hypothesis holds in the light of the previous studies. Moreover, the data presented by Jääskeläinen et al. (2004) are compatible with the commonly accepted interpretation of a fully separate N1 (N1a) and MMN. These brain responses can be unequivocally dissociated, both in terms of cognitive function and physiological measures, as we have shown here. Therefore, we argue that the principal gate to awareness for auditory deviation sought by Jääskeläinen and his colleagues continues to be the MMN mechanism (based on separate memory-related deviance-detection neurons). This mechanism depends on the detection of an auditory regularity in the preceding sound sequence and provides a preperceptual auditory-cortex signal of auditory deviation as an output from an automatic *memory-based* comparison process. This signal can then trigger the frontal mechanisms of attention switch to, and conscious perception of, deviant and novel stimuli, thus gating them to consciousness (Näätänen, 1990, 1992; Näätänen & Winkler, 1999). However, an additional route to attention switch from auditory change appears to employ feature-detector neurons as previously proposed by several authors (Desimone, 1992; Näätänen, 1990, 1992; Ulanovsky et al., 2003), and now by Jääskeläinen et al. (2004), but this route by no means renders the one based on the comparison with a sensory-memory representation (generating the MMN) obsolete. Based on the evidence, we argue that the route reflected by the MMN is the principal route to attention switch from auditory change and one which is operational even when the new afferent elements cannot participate in serving this vital function. Nevertheless, in using the MMN measure for basic research and clinical applications, it is always important to carefully consider the possible role of these fresh afferent elements as stressed by Jääskeläinen et al. (2004).

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