Mismatch Negativity in Socially Withdrawn Children

Yair Bar-Haim, Peter J. Marshall, Nathan A. Fox, Efrat A. Schorr, and Sandra Gordon-Salant

Background: *Individual differences in auditory processing have been associated with social withdrawal, introversion, and other forms of dysfunction in social engagement. The goal of this study was to investigate the characteristics of an electrophysiologic response that is seen to index early cortical auditory processing (mismatch negativity, MMN) among socially withdrawn and more sociable control children.*

Methods: *Auditory event-related potentials to standard and deviant tone stimuli were computed for 23 socially withdrawn children and 22 control subjects. We calculated MMN difference waveforms for frontal, central, and parietal electrode sites.*

Results: *Socially withdrawn children had smaller MMN amplitude and longer MMN latencies compared with more sociable control children.*

Conclusions: *The findings point to the involvement of individual differences in early cortical auditory processing in childhood social withdrawal. Reduced MMN amplitude and delayed latency may index a component of social withdrawal seen in socially withdrawn children and in depressed and schizophrenic patients. The existence of a secondary MMN generator in the frontal cortex may provide a link between the hypothesized frontal lobe involvement in childhood social withdrawal, schizophrenia, and depression and the MMN reductions seen in these conditions.* Biol Psychiatry 2003;54:17–24 © *2003 Society of Biological Psychiatry*

Key Words: Mismatch negativity, social withdrawal, temperament, evoked response potential, children, auditory

Introduction

Social withdrawal and other forms of dysfunction in social engagement are defining features in a variety of psychiatric disorders, including mood and anxiety disorders and schizophrenia. For this reason, researchers of human development have extensively examined factors that may contribute to the consistent display of social withdrawal during childhood, as well as the consequences of childhood social withdrawal for the later development of psychopathology (for a review, see Rubin and Stewart 1996).

Childhood social withdrawal can be broadly defined as the consistent disposition to display solitary behavior when encountering peers in social contexts. There may be different circumstances underlying individual differences in children's tendencies to exhibit social withdrawal, one of the most extensively studied being the possibility of a biological disposition. Most theorizing on the biological basis of stable individual differences in social withdrawal in early childhood centers on Kagan's (1994) concept of behavioral inhibition to the unfamiliar. Behavioral inhibition is typically viewed as a temperamental construct reflecting relatively stable individual differences in behavioral style (e.g., Goldsmith et al 1987). Behaviorally inhibited children are characteristically watchful and quiet in new situations, including social interactions with unfamiliar people (Kagan et al 1984).

It is now widely accepted that the prefrontal cortex (PFC) and the amygdala are two key structures in the central circuitry of emotion and emotion regulation (for reviews, see Davidson 2002; Davidson et al 2000). Individual differences in the functioning of both the PFC and amygdala are thought to play a key role in individual differences in the propensity for behavioral inhibition and social withdrawal. Kagan and coworkers have proposed that the contrast in reactions to novelty of inhibited and uninhibited children arises from variation in the excitability of neural circuits of the limbic system (e.g., Kagan and Snidman 1991). This model focuses on the central nucleus of the amygdala, which is the primary source of projections from the amygdala to subcortical sites that modulate behavioral and physiologic responses to a threatening stimulus (e.g., Davis 1992; LeDoux et al 1990). Increased activity of the central nucleus of the amygdala would be expected to result in specific patterns of activity of response systems that are influenced by the central nucleus. Indeed, evidence suggesting that inhibited children differ from uninhibited children in their autonomic and neuroendocrine profiles has been used to support the

From the Department of Psychology (YB-H), Tel-Aviv University, Ramat Aviv, Tel-Aviv, Israel; Departments of Human Development (PJM, NAF, EAS) and Hearing and Speech Sciences (SG-S), University of Maryland, College Park, Maryland.

Address reprint requests to Yair Bar-Haim, Department of Psychology, Tel-Aviv University, Ramat Aviv, Tel-Aviv 69978, Israel.

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amygdala model (for review, see Marshall and Stevenson-Hinde 2001). Other research has suggested that behaviorally inhibited children differ from uninhibited children on a measure of activation asymmetry in the frontal lobe that is derived from the electroencephalogram (EEG). Inhibited children have a tendency to show increased EEG activation over the right frontal region of the cortex compared with the left, with the pattern being reversed in uninhibited children (e.g., Fox et al 2001). This finding is seen in the framework of a model relating hemispheric asymmetry of activation in the frontal cortex to individual differences in the behavioral tendency to approach or withdraw in novel or unfamiliar situations (Fox 1991).

In addition to the PFC–amygdala circuitry, there are additional systems that may be involved in the physiologic differences between socially withdrawn and more sociable children. A contribution to individual differences in social behavior may also be made by normative individual variations in the transmission and processing of sensory information in the periphery or sensory cortex (or both). Such a possibility poses an intriguing question: for a given stimulus, is the nature of the partly processed sensory information reaching the amygdala and the PFC the same for a behaviorally inhibited and socially withdrawn child compared with an uninhibited sociable child?

A growing body of evidence from the adult personality literature points to the possibility that individual differences in auditory processing may contribute to individual differences in introverted and socially withdrawn behavior. Research by Stelmack and colleagues (e.g., Sasaki et al 2000; Stelmack et al 1977) has suggested that adult introverts show greater physiologic reactivity to auditory stimulation, in that the magnitude of certain components of auditory evoked response potentials (ERPs) tend to be larger for introverts than extraverts. For instance, Doucet and Stelmack (2000) reported larger N1 amplitude in introverts, and Stelmack and Michaud-Achorn (1985) found greater amplitude of the N1-P2 complex in introverts. Several other studies have found differences between introverts and extraverts in very early measures of auditory transmission. A number of research groups have reported an association between faster auditory brainstem response (ABR) latencies and adult introversion (e.g., Andress and Church 1981; Bullock and Gilliland 1993; Cox et al 2001; Stelmack and Wilson 1982; Swickert and Gilliland 1998). These findings implicate individual differences in auditory sensory processes that may not necessarily be determined by limbic mechanisms. The neural events taking place at relatively early stages of auditory processing may have implications for the later perception and interpretation of auditory information in higher brain structures. These perceptual effects may influence individuals' subjective experience of the social environment.

One aspect of stimulus processing that takes place in auditory cortex is the comparison of an incoming stimulus with prior stimuli. Novelty detection requires a memory system that assembles neural representations of events in the environment, such that changes are detected because they violate the predictions of an established neural schema. The mismatch negativity (MMN) is believed to reflect such processes (Naatanen 1995; Schroger and Winkler 1995). The MMN is an electrophysiologic response that is primarily generated in primary auditory cortex, and it is seen as indexing an attention-independent, preperceptual change detection mechanism. Because no specific task performance is required to elicit the MMN (Naatanen et al 1993), it is considered a powerful tool for examining early cortical auditory processing. The MMN is elicited to rare deviant stimuli that are embedded in a train of frequent standard stimuli and is usually assessed by calculating an ERP to the deviant stimuli and subtracting it from the analogous ERP for the standard stimuli. The resulting difference waveform shows a negativity (the MMN) that peaks around 100–200 msec from stimulus onset.

Accumulating evidence indicates a reduced MMN response in psychiatric conditions such as schizophrenia (e.g., Javitt et al 1998; Michie et al 2000; Shelley et al 1999) and depression (e.g., Ogura et al 1993). Although schizophrenia and depression are complex disorders, each involving a wide range of associated symptoms, both share a central component of social withdrawal (e.g., Deater-Deckard 2001; Goldberg and Schmidt 2001). Furthermore, Catts et al (1995) and Javitt et al (2000) reported that MMN amplitude was significantly correlated with ratings of negative schizophrenia symptoms (such as social withdrawal) but not with positive symptoms (such as hallucinations and delusions). It is therefore conceivable that the smaller MMN detected in patients suffering from these psychiatric conditions uniquely contribute to their shared component of social withdrawal. The existence of a secondary MMN generator in the frontal cortex (Naatanen and Alho 1995) may provide a link between the hypothesized frontal lobe involvement in schizophrenia and depression and the MMN reductions seen in these disorders.

Given the established associations between introversion and individual differences in auditory ERPs in adults and the reported associations between clinical syndromes involving social withdrawal and auditory ERP components, it is surprising that auditory event-related potentials have rarely been employed in the study of childhood social withdrawal. To our knowledge, the only related study was conducted by Woodward et al (2001), who reported that

10- to 12-year-old children classified as high on motor and negative emotional reactivity to sensory stimulation at 4 months of age had larger amplitudes of wave V in the ABR than did low-reactive children. High levels of negative affect in response to sensory stimulation in early infancy are associated with later behavioral inhibition (e.g., Calkins et al 1996).

Our research examines whether individual differences in social withdrawal in childhood are associated with variation in early processing in the auditory cortex. Specifically, we hypothesize that socially withdrawn children will tend to exhibit reduced amplitude of the MMN. Although the specific underlying mechanisms and functional significance of a reduced MMN response are not entirely clear, a smaller MMN is thought to signify a deficit in preattentive processing of auditory stimuli that may feed forward to affect the perception of the sensory environment (Picton et al 2000). In addition, based on Stelmack's research with introverts (Doucet and Stelmack 2000), we examined whether there were group differences in the amplitude of the P1–N1 complex to the same tone stimuli presented in the MMN protocol.

Methods and Materials

Participants

Two groups of 7- to 12-year-old children (mean age 9.50 years, SD = 1.75 years) were selected from a larger group of 158 children who had participated in a longitudinal study on the psychophysiologic correlates of social and emotional development at the University of Maryland (for details, see Fox et al 1996, 2001). Children were primarily of middle-class background, living with their families in and around the Washington, DC, area. One group consisted of 23 socially withdrawn children (12 boys) who had shown high frequencies of solitary behaviors in previous assessments of sociability at both 4 and 7 years of age (mean age 9.38 years, SD = 1.68). The control group consisted of 22 children (8 boys) who were not socially withdrawn during the previous assessments (mean age 9.62 years, SD = 1.86). There was no significant difference in the mean age of the two groups ($p > .65$). Informed consent was obtained from parents before the beginning of the study procedures. The study was approved by the University of Maryland Research Ethics Committee.

Assessment of Social Behavior

At both 4 and 7 years of age, each child had been seen for 30 min in a play session with three unfamiliar children of the same gender and age (see Fox et al 2001). Behaviors during free play were coded with the Play Observation Scale (Rubin 1989). Each 10-sec interval of free play was coded for children's social participation and the cognitive quality of play. Independent observers coded the play sessions, and kappas ranged from .81 to .94 for the full variable matrix. Two indices of social withdrawal behavior were used to select participants for the study: solitary-passive behavior (summing the proportion of coding intervals spent in solitary-exploratory or solitary-constructive play) and social reticence (the sum of onlooking and unoccupied behavior, Coplan et al 1994). Children in the top quartiles of either of these indices at both 4 and 7 years of age were included in the socially withdrawn group. A control group consisted of children who were never in the top quartile of either social withdrawal index at both 4 and 7 years of age.

Mismatch Negativity and P1–N1 ERP Components

STIMULI. The MMN protocol consisted of 600 standard tones and 100 deviant tones (100-msec duration, 10-msec rise and fall times, 75-dB sound pressure level), with a stimulation rate of 2.22 Hz (stimulus onset asynchrony [SOA] = 450 msec onset to onset). Each deviant tone was preceded by three or more standard tones. The stimuli were presented binaurally in two blocks, each of 300 standard tones and 50 deviant tones. The first block used 1000-Hz standard tones and 1100-Hz deviant tones, and the second block used the reverse configuration of 1100-Hz standard tones and 1000-Hz deviant tones. There was a 20-sec pause between blocks.

EEG RECORDING PARAMETERS AND WAVEFORM SCORING. We collected EEG data while participants viewed a silent cartoon. Participants were instructed to ignore the acoustic stimuli. To standardize their level of attention, all participants were told that they would have to give specific feedback about the cartoon at the end of the experimental session. The EEG was recorded from nine scalp sites (Fz, F3, F4, Cz, C3, C4, Pz, P3, P4) plus the left and right mastoids, using Lycra stretch caps (Electro-Cap, Eaton, Ohio) with sewn-in electrodes according to the 10–20 system of electrode placement. Sampling rate was 1024 Hz with bioamplifier filter settings of .1-Hz high pass and 100-Hz low pass. One bipolar electrooculogram (EOG) channel was recorded from above and below the left eye using similar bandpass settings. The EEG signals were amplified by a custom bioamplifier from SA Instruments and were digitized onto a PC using an Iotech (Cleveland, Ohio) DaqBook A/D converter (5 V input range) and HEM Snap-Master data acquisition software. Further processing and analysis of the EEG signal was carried out using software from James Long Company (Caroga Lake, New York). Artifactual EEG (± 150 uV) was automatically removed from further analysis. Across all participants, the mean number of trials remaining after artifact rejection was 563 (SD = 63) for the standard stimulus and 94 (SD = 11) for the deviant stimulus. The socially withdrawn and control groups did not significantly differ in the number of artifact scored trials for the standard and deviant tones. Blinks in the EOG signal were regressed out of the EEG using a procedure based on methods described in the literature (e.g., Lins et al 1993; Miller and Tomarken 2001). The EEG channels were collected referenced to Cz, and were referenced again offline to average mastoids. Before derivation of the ERPs, the EEG signal was subjected to digital filtering between 2–10 Hz. ERPs were calculated relative to a 100-msec prestimulus baseline.

The MMN was scored from the deviant-minus-standard difference waveform as the most negative point in the range of

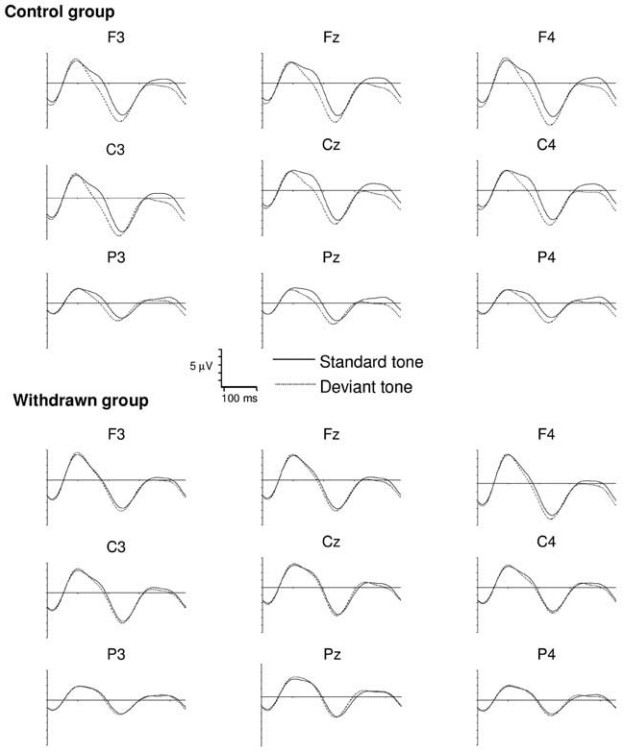

Figure 1. Grand mean evoked response potential waveforms for the standard (solid line) and deviant (dashed line) tones for the control and the socially withdrawn groups.

100–300 msec after stimulus onset. The amplitude and latency of the MMN were scored. In addition, the amplitude of the P1 (the first major positive-going peak) and N1 (the following negativity) components in the ERPs to the deviant and standard stimuli were scored, which allowed the computation of the magnitude of the P1–N1 response (P1 amplitude minus N1 amplitude).

Results

The tone stimuli resulted in ERP waveforms characterized by a large P1 peak at an average of around 110 msec after stimulus onset, followed by a broad negativity peaking at an average of around 240 msec (Figure 1). In previous developmental work, this relatively late negativity has been termed the N1b to differentiate it from earlier-occurring variants of the N1, namely, the adult N1 (around 100 msec) and the N1a (in the 90–160 msec range) that is sometimes seen in older children (Sharma et al 1997). Although appearing in a latency range similar to the N1b, we refer to the observed negativity as the N1.

The difference waveform revealed an MMN response that peaked at an average of around 200 msec after stimulus onset (Figure 2; for a similar finding in children see Kurtzberg et al 1995).

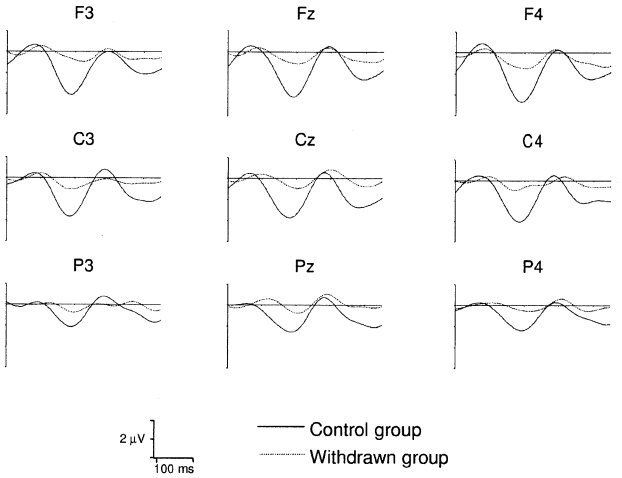

Figure 2. Grand mean tone evoked response potential difference waveforms (MMV) for the control group (solid line) and the socially withdrawn group (dashed line).

MMN Amplitude

Means and standard deviations of MMN amplitude at the different electrode sites by social group are presented in Table 1. A repeated-measures analysis of variance (ANOVA) was computed with scalp region (frontal, central, parietal) and electrode location (left, midline, right) as the within-subjects factors and social group (withdrawn vs. control) as the between-subjects factor. Where appropriate, Greenhouse–Geisser corrections were used.

There was a main effect of social group for MMN amplitude [$F(1,43) = 12.90$, $p < .001$]. Follow-up t tests showed that compared with the control group, the mean MMN amplitude of the withdrawn group was significantly smaller in magnitude at all electrode sites except for P3 (see Table 1 and Figures 1 and 2). In addition, there was a significant region × social group interaction [$F(2,42) =$

Table 1. Mean Amplitude in uV (SD) of the Mismatch Negativity for the Withdrawn and Control Groups, with the Results of t Test Comparisons and Effect Sizes

Electrode Site	Withdrawn ($n = 23$)	Control ($n = 22$)	t	w^2 (effect size)
Fz	−1.76 (1.04)	−3.22 (1.55)	3.58[c]	.21
F3	−1.51 (.91)	−3.05 (1.70)	3.81[c]	.23
F4	−1.88 (.96)	−3.36 (1.54)	3.77[c]	.23
Cz	−1.80 (.91)	−3.11 (1.73)	3.55[b]	.21
C3	−1.75 (.93)	−2.78 (1.79)	3.23[a]	.18
C4	−1.66 (1.03)	−2.86 (1.59)	4.28[b]	.28
Pz	−1.38 (.85)	−2.28 (1.31)	2.63[b]	.12
P3	−1.40 (.83)	−1.78 (1.16)	2.17	—
P4	−1.24 (.72)	−2.04 (1.03)	2.87[b]	.14

[a] $p < .05$.
[b] $p < .01$.
[c] $p < .001$.

Table 2. Mean Latency in msec (SD in Parentheses) of the Mismatch Negativity for the Withdrawn and Control Groups, with the Results of *t* test Comparisons and Effect Sizes

Electrode Site	Withdrawn (n = 23)	Control (n = 22)	t	w^2 (effect size)
Fz	214 (49)	187 (36)	2.14[a]	.08
F3	210 (54)	191 (30)	1.44	—
F4	213 (46)	192 (30)	1.87	—
Cz	212 (43)	176 (34)	3.10[b]	.16
C3	213 (49)	184 (28)	2.40[a]	.10
C4	212 (51)	179 (30)	2.61[a]	.11
Pz	219 (43)	170 (40)	3.97[c]	.25
P3	207 (54)	185 (35)	1.59	—
P4	199 (50)	191 (42)	.55	—

[a] $p < .05$.
[b] $p < .01$.
[c] $p < .001$.

5.11, $\epsilon = .78$, $p < .05$]. Three follow-up repeated-measures ANOVAs within each scalp region (frontal, central, parietal) showed that while the socially withdrawn group had significantly smaller MMN magnitude within each of the three regions, the magnitude of the group difference was smaller in the parietal region compared with the frontal and central regions. Also, there was a significant main effect of region ($F(2,42) = 16.38$, $\epsilon = .78$, $p < .001$). Contrasts revealed that the MMN was significantly smaller in magnitude in the parietal region compared with the frontal [$t(44) = 4.62$, $p < .001$] and central [$t(44) = 5.40$, $p < .001$] regions. Finally, there was a main effect for electrode location [$F(2,42) = 4.14$, $\epsilon = .91$, $p < .05$) such that MMN amplitude was larger for midline electrodes (Fz, Cz, Pz) compared with electrodes located over the left hemisphere [$t(44) = 2.81$, $p < .01$]. There was no difference in MMN amplitude between the left and right hemispheres.

MMN Latency

Table 2 presents means and standard deviations of MMN latency at the different electrode sites by social group. A repeated-measures ANOVA with scalp region (frontal, central, parietal) and electrode location (left, midline, right) as the within-subjects factors and social group (withdrawn vs. control) as the between-subjects factor revealed a significant main effect for social group [$F(1,43) = 7.46$, $p < .01$]. Socially withdrawn children had significantly longer MMN latencies compared with the more sociable children; however, this main effect was qualified by a significant three-way interaction of region × scalp location × social group [$F(4,40) = 3.09$, $\epsilon = .77$, $p < .05$). Follow-up *t* tests showed that compared with the control group, the mean MMN latency of the withdrawn group was significantly slower at Fz, Cz, Pz, C3, and C4 (see Table 2 and Figures 1 and 2). No other significant main effects or interactions were found.

Correlations between MMN Amplitude and Latency

To assess covariation of MMN amplitudes and latencies, two-tailed Pearson correlations were computed between MMN amplitude and latency at each electrode site. Across groups, MMN amplitude and latency correlated significantly at Fz and F4 (rs = .36 and .45, ps < .05 and .01, respectively) but not at other electrode sites; however, when follow-up correlations between MMN amplitude and latency for these electrode sites were computed within each group, MMN amplitude and latency were significantly correlated only at F4 and for the withdrawn group only ($r = .46$, $p < .05$).

N1–P1 Amplitude

Separate repeated-measures ANOVAs for N1–P1 amplitudes were computed for the standard and deviant tones with scalp region (frontal, central, parietal) and electrode location (left, midline, right) as the within-subjects factors and social group (withdrawn vs. control) as the between-subjects factor.

There were no significant main effects or interactions involving social group for the ANOVA concerning P1–N1 amplitude to the standard stimuli. There were main effects of region [$F(2,42) = 221.01$, $\epsilon = .76$, $p < .001$] and electrode location [$F(2,42) = 9.49$, $\epsilon = .96$, $p < .001$], and a significant region × electrode location interaction effect [$F(4,40) = 18.75$, $\epsilon = .57$, $p < .001$). Follow-up contrasts showed that P1–N1 amplitude was significantly larger in the frontal region compared with the central region, and significantly larger in the central region compared with the parietal region. Additionally, P1–N1 amplitude was significantly larger at midline electrodes compared with electrode locations over the right or left sides of the scalp.

The ANOVA concerning P1–N1 amplitude to the deviant stimuli also revealed a main effect of region [$F(2,42) = 170.28$, $\epsilon = .69$, $p < .001$] and a significant region × electrode location interaction effect [$F(4,40) = 7.92$, $\epsilon = .75$, $p < .001$]. Follow-up contrasts showed that P1–N1 amplitude was significantly larger in the frontal region compared with the central region, and larger in the central region compared with the parietal region. Here, too, P1–N1 amplitude was largest over midline electrodes. No significant main effects or interactions involving social group were found.

Discussion

This study shows that socially withdrawn children had smaller MMN amplitude and longer MMN latencies com-

pared with more sociable control children. Although the precise functional association between the MMN and social withdrawal is not entirely clear, these findings point to the involvement of individual differences in auditory processing in childhood social withdrawal. Individual variation in the early stages of processing of auditory information have consequences for later stages of processing (Galbraith 2001; Picton et al 2000), which may in turn influence individual differences in the perceived qualities of acoustic stimuli. Alternatively, top-down processes may influence processing at various stages of the auditory pathway. Although this study does not provide a decisive answer regarding the neural mechanisms involved, it is plausible that certain types of acoustic processing either do not provide the cues important for social interaction or are aversive and less rewarding.

Naatanen and Winkler (1999) proposed that there is an increase in frequency specific inhibition of populations of neurons that are responsive to the frequency of the repeated standard stimulus. This inhibitory state that constitutes the memory trace is paralleled by an increase in excitability of neurons responsive to other frequencies. Thus, the group differences in MMN in this study may be interpreted as a reduction in the development of neuronal inhibition to the standard stimuli in the withdrawn group. Such an interpretation is also consistent with the notion that reduced and delayed MMN could arise from disruptions in acoustic processing in earlier stages of the auditory pathway. For example, if the integrity of the signal is compromised in its transmission through the ascending auditory pathway (e.g., Bar-Haim 2002; Bullock and Gilliland 1993; Woodward et al 2001), the comparison of the standard and deviant stimuli may be impaired and a smaller and delayed MMN will be elicited. Alternatively, abnormally rapid decay of the memory trace associated with the standard stimulus may mean that an incoming deviant elicits less of an MMN response; however, the implications for perceptual experience of the later deficit in auditory sensory memory are profound and should give rise to substantial perceptual and attentional disturbances (Cowan 1984, 1988) that are not characteristic of our sample. In addition, the MMN differences observed between the withdrawn and control groups in our study are unlikely to be due to a sensory memory deficit given the fast stimulation rate.

The reduced MMN in the withdrawn children may be due to influences on the MMN from higher centers in the forebrain. This hypothesis has yet to be systematically investigated, particularly taking into account the fact that the majority of the existing MMN literature indicates that the MMN is relatively immune to significant cognitive or attentional influences. There is some evidence for frontal cortex involvement in MMN generation, however, especially in the right frontal region (e.g., Giard et al 1990; Naatanen and Alho 1995; Opitz et al 2002). This may provide a link to understanding MMN reductions in conditions such as schizophrenia and depression, which have been shown to involve frontal cortex dysfunction. Given the previous findings of hemispheric differences in EEG activation over the frontal cortex between socially withdrawn and nonwithdrawn children (e.g., Fox et al 1995, 2001) and the finding from our study of an association between MMN latency and amplitude only for socially withdrawn children and only over the right prefrontal cortex, it is also possible that the right frontal cortical MMN generator is impaired in socially withdrawn children.

Although characteristics of auditory processing mechanisms have emerged as significant correlates of social withdrawal and introverted behavior, the subjective perceptual and emotional experiences associated with such processing patterns are as yet unclear. It is tempting to speculate that the individual differences in processing and transmission of acoustic stimuli found in socially withdrawn children, adult introverts, and patients suffering from psychiatric disorders that involve a significant component of social withdrawal may map onto behavioral characteristics such as a preference for lower environmental noise levels (Geen 1984), a preference for playing with children who are less noisy (Evans 2001), a greater desire for privacy (Weinstein 1978), and lower auditory thresholds (e.g., Goldman et al 1983; Smith 1968; Stelmack and Campbell 1974). It is also possible that a reduced and delayed MMN could be indicative of poorer auditory discrimination ability in withdrawn individuals and may also be reflected in language deficits. To our knowledge, however, there is no evidence showing that adult introverts or socially withdrawn children exhibit reduced auditory discrimination abilities. The use of detection performance measures, language assessment batteries, and the manipulation of discrimination task difficulty may be a useful strategy for future research examining the MMN in socially withdrawn children and adults.

It is conceivable that socially withdrawn children derive less pleasure and feel more anxious in noisy social situations, as a result of their characteristic auditory processing pattern. If such children perceive the acoustic stimulation in social environments as being overwhelming or unpredictable, they may consistently find noisy social interactions to be unrewarding. They could then exhibit behavioral avoidance of social situations, which could evolve into a more complex cognitive and behavioral pattern of social withdrawal. Although this is still a working hypothesis and specific mechanisms still need to be elucidated, the research reported here supplies indirect evidence for the possibility that the development and

maintenance of individual differences in social withdrawal in childhood is associated with variation in early cortical auditory sensory processing.

Finally, previous research by Stelmack and colleagues has suggested that the magnitude of certain auditory ERP components is larger for adult introverts than extraverts (e.g., N1: Doucet and Stelmack 2000; N1-P2: Stelmack and Michaud-Achorn 1985). Our findings do not support the same conclusions in children: P1–N1 amplitude did not differentiate between the socially withdrawn and control groups for the standard and for the deviant stimuli. It should be emphasized, however, that the late negativity (N1 or N1b) seen in the children in our study might be functionally different from the adult N1 (see Sharma et al 1997). Furthermore, although the tone frequencies used in our study (1000–1100 Hz) are commonplace in MMN research, differences between introverts and extraverts in P1 and N1 components have primarily been found using lower frequency stimuli (e.g., 500 Hz and 750Hz; Stelmack 1990).

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