Chapter I: Background and Purpose of the Research

1. Sensory Gating

1.1. What Sensory Gating is

Sensory gating to irrelevant sensory input is a safeguard filtering mechanism of the central nervous system. It helps prevent incoming irrelevant sensory information from entering into the higher cortex and ensures normal information processing (Braff & Geyer, 1990). Sensory gating is seen as the ability of the nervous system to modulate its sensitivity to incoming stimuli (Adler, Olincy, Waldo, Harris & Griffith, et al., 1998). Successful sensory gating is an important property of the normal functioning brain. Deficits in sensory gating can result in an overflowing of irrelevant stimuli into the higher cortex, which may lead to brain dysfunction. These deficits are associated with behavioral disorders and psychotic symptoms (McGhie & Chapman, 1961).

There are two aspects to sensory gating: (1) the capacity to cease to respond or to significantly reduce the magnitude of an individual response to incoming irrelevant stimuli (gating out); and (2) the capacity to re-respond when a novel stimulus is presented or a change occurs in ongoing stimuli (gating in) (Boutros, Zouridakis & Overall, 1991). Since sensory gating activity may be as early as the P50 (40 to 75 msec) and as late as the N100 (Smith, Boutros & Schwarzkopf, 1994), and is also reflected in the mismatch negativity (MMN; 160 to 200 msec from stimulus onset), this definition reflects a complex multicomponent “sensory gating” concept (Boutros, Zouridakis & Overall, 1991).

Experimental evidence showed that individuals with schizophrenia have abnormal sensory gating (Adler, Pachtman, Franks, Pecevich, & Waldo, et al, 1982). The processing of sensory input appears to require at least two stages: a stimulus identification stage followed by a stimulus
evaluation stage (Freedman, Waldo, Bickford-Weimer, & Nagamoto, 1991). They suggested that while schizophrenic patients can usually identify the stimuli in their environment, they may have difficulty in evaluating stimuli. The increased sensory response to irrelevant stimuli in schizophrenics appears to be due to poor selective sensory inhibition, resulting in the flooding of sensory input into higher cortex.

1.2. Measuring Sensory Gating

P50 is a type of mid-latency auditory evoked responses, and it appears in the electroencephalograph (EEG) about 50 (40-75) msec after administrating an auditory stimulus. It is considered to be a stable parameter to measure sensory gating when used in a paired-tone paradigm. During the test, the subject is given a pair of single-sound stimuli: S1 (also called conditioning stimulus) and S2 (also called test stimulus). Both of them are the same in intensity, frequency and pitch. Typically there is a 500 ms inter-click interval between the two stimuli and 5 - 10s (usually 8-10s) inter-pair intervals between pairs.

The capability of sensory gating is measured by a ratio of the P50 amplitudes of S2 and S1, or it is by the difference between the amplitudes of S2 and S1, or the ratio of N40 – P50 of S2 and S1. Low ratio or large difference is thought to represent the better capability of sensory gating (Freedman, Adler, Waldo, Pachtman & Franks, 1983). Evidence shows that normal subjects can reduce the amplitude of S2 to 10-20% of the amplitude of S1, and schizophrenic subjects only reduce the amplitude of S2 by 10-20% (Freedman et al., 1983).

1.3. Mechanisms of Sensory Gating

Up to now, the mechanisms of sensory gating are not clear, but prior studies suggest that they are related to the following nervous and biochemical systems. We must consider the source
localizations of the P50 component that are used in the evaluation of sensory gating of S1 and S2. The P50 appears to be from multiple brain sources.

1.3.1. The Left and Right Superior Temporal Gyri

A major source of the P50 component is thought to be in the left and right superior temporal gyri (e.g., Edgar, Huang, Weisend, Sherwood & Miller, et al., 2003). For instance, intraoperatively recorded P50 (Ligeois-Chauvel., Musolino, Badier & Marquis, 1994) and chronic subdural electrodes (Lee, Lueders, Dinner, Lesser & Hahn, et al., 1984) exhibited near-field potentials localized in the primary auditory cortex. Since EEG is thought not to “lend itself to identification of the presumably lateralized cortical generators of P50 (p. 1595)”, Edgar et al. (2003) assessed the M50, thought to be analogous to the P50, with magnetoencephalography (MEG). They (see also Thoma, Hanlon, Moses, Edgar & Huang, et al. 2003) reported that the M50 was localized in the left and right hemisphere superior temporal gyri, with no mean differences between the two hemispheres but with between and within individual differences between the two hemispheres. When compared with P50 recorded from Cz, the latencies for P50 and M50 were identical, supporting their hypothesis that the P50 and M50 reflect superior temporal gyri activity. Yet, they did not find significant correlations between the P50 and M50 S1, S2, or S2/S1 ratios. They noted that this lack of significant correlations is “surprising” and “might suggest that the MEG and EEG responses are unrelated or that additional generators contribute to P50 (p. 27)

1.3.2. The Hippocampus

P50 may also be generated in the hippocampus. Multiple depth electrode recordings in neurosurgical patients recorded a P50 wave in the hippocampal formation (Goff, 1980; Wilson, Babb, Halgren, Wang & Crandall, 1984; Freedman et al., 1991). Data from animal models
suggest that the P50 is generated in the CA3/CA4 pyramidal neurons of the hippocampus (Bickford-Weimer, Nagamoto, Adler, Egan & Johnson, et al, 1990). More recently, Grunwald, Boutros, Pezer, Oertzen and Fernandez et al. (2003) found that sensory gating may be a multistep process, with an early phase subserved by the temporo-parietal and prefrontal cortex and a later phase mediated by the hippocampus.

Two gating theories have been proposed to explain the P50: (1) “active gating theory” (Freedman et al 1991), and (2) “passive gating theory” (Volkov & Galazyuk, 1992). The active gating theory suggests that the S1 (in the paired-tone paradigm) evokes local excited pyramidal neurons in the hippocampus, and leads to a positive wave in P50. It also evokes local inhibitory pyramidal neurons in the hippocampus, and these neurons release an abundance of an inhibitory neurotransmitter into the synapses. When S2 carries no new information, it arrives at the hippocampus, where these inhibitory neurons are still in active state and inhibitory neurotransmitter are still present in the synapses. Freedman et al. (1991) proposed that although S2 can result in a response, the inhibitory factors restrict the response to S2, and therefore, the P50 shows the decreased wave to S2. The passive gating theory (Volkov & Galazyuk, 1992) thought that S2 arrives when the neurons are in the refractory periods, but it was unbelievable because there are decreased amplitudes in P50 to S2. If this explanation were true, there would be no wave in P50 for S2. In fact, most neurons need only a few milliseconds to restore their ionic equilibrium and reset to their internal energy.

1.3.3. The Prefrontal-Thalamic Inhibitory System

Almost every sensory signal, except olfaction, entering the brain must pass through a thalamic “gate” before it is relayed to the other parts of the brain, including the hippocampus and the cortex. Thus, the thalamus is a relay station of information processing between the inferior
central nervous system and the superior cortex. The prefrontal cortex (PFC) plays an important role in regulating the flow of information to the primary sensory cortex. Neural inhibition by prefrontal regions has been reported in a variety of mammalian preparations. A net inhibitory output to both subcortical (Edinger, Siegel & Troiano, 1975) and cortical regions has been documented (Alexander, Newman & Symmes, 1976; Skinner & Yingling, 1977). This prefrontal-thalamic inhibitory system also provides a potential mechanism for the suppression of irrelevant inputs and the selection of relevant ones at an early stage of sensory processing. Patients with prefrontal lesions are less able to filter out the irrelevant sensory inputs and can not sustain attention (Knight, Staines, Swick & Chao, 1999). Thus, damage to the prefrontal cortex disrupts inhibitory modulation of sensory inputs to primary sensory cortex (Knight, Scabini & Woods, 1989, Yamaguchi & Knight, 1990). Knight et al. (1999) examined auditory gating in schizophrenic patients with those with dorsolateral prefrontal damage and to age-matched controls. Normal controls had a normal suppression of the second stimulus in an auditory pulse pair. Individuals with schizophrenia showed an inhibitory failure in the auditory gating paradigm. This failure to suppress is observed for both an early latency component generated in the auditory cortex (P35) and a later component (P50). The data suggest that prefrontal cortex dysfunction may underlie or contribute to the auditory gating deficit in schizophrenics.

1.3.4. Catecholamine System

Catecholamine plays an important role in sensory gating. Studies with normal participants showed that catecholamine decreases sensory gating (Braff & Huey, 1988). Psychotic patients treated with neuroleptics showed improved function of sensory gating (Spohn, Lacoursiere, Thompson & Coyne, 1977). Studies of animals show the same effects of catecholamines on sensory gating, both at the behavioral level and the single neuron level (Swerdlow, Vaccarino,
Amalric & Koob, 1986). The other evidence is that when normal subjects were administered drugs which increase catecholaminergic neurotransmission, the result was a temporary abnormal sensory gating, similar to that observed in schizophrenics (Braff & Huey, 1988). Furthermore, the disrupted P50 suppression found in schizophrenia, as well as schizotypy, is related to a reduced dopaminergic activity, most likely in the prefrontal cortex (Oranje, Wied, Verbaten & Kahm, 2002).

1.3.5. Nicotinic Cholinergic System

Hippocampal neurons can rapidly inhibit the response to repeated sensory stimulation, and the inhibitory function has been linked to the alpha-7-nicotinic acetylcholine receptor subunit (Luntz-Leybman, 1992). Alpha-bungarotoxin, a type of antagonist of alpha-7-nicotinic receptor, labels the GABA-containing interneurons in all regions of the hippocampus (Adams, 1999). Many hippocampal interneurons are depolarized by activation of alpha-7-nicotinic receptor subunit. Alpha-bungarotoxin blocks the inhibitory response. All of these provide evidence that the sensory gating need the activation of the a7 nicotinic receptor (Luntz-Leybman, 1992). More recently, genetic studies found that the chromosome l5ql4 locus of the alpha-7-nicotinic receptor gene was associated with the P50 sensory gating abnormalities (Freedman, Olincy, Ross, Waldo & Stevens, 2003). This finding provides the evidence that P50 sensory gating abnormal may have a genetic factor.

2. Schizotypy and Sensory Gating

2.1. Schizotypal Personality Disorder

Schizotypy includes the following signs and symptoms: ideas of reference, odd beliefs, unusual perceptual experiences, odd thinking and speech (e.g., vague, circumstantial,
metaphorical, overelaborate or stereotyped), suspiciousness, inappropriate or constricted affect, odd behavior, lack of close friends, and social anxiety (American Psychiatric Association, 1994). At a clinical level, schizotypal personality disorder (SPD) is defined as a lifelong personality disorder characterized by nine traits: ideas of reference, excessive social anxiety, magical thinking, unusual perceptual experiences, eccentric behavior or appearance, no close friends or confidants, odd speech, constricted affect, and suspiciousness (Raine, Lencz & Mednick, 1995). Gruzelier (1996) thought that the nature of schizotypy includes three main factors: (1) psychotic-like cognitive and perceptual unreality experiences often referred to as magical thinking and perceptual aberrations, as is found in two widely used schizotypy scales (Chapman & Chapman, 1980); (2) ‘negative’ interpersonal characteristics including social withdrawal and emotional constriction; and (3) cognitive disorganization, activation, and nonformity.

There is now abundant evidence of affinities between schizotypy and schizophrenia ranging over phenomenology, genetics, biology, cognition, treatment response, and outcome (O’Flynn, Gruzelier, Bergman & Siever, 2003). Longitudinal studies have confirmed that schizotypy may be a forerunner of schizophrenia (Chapman, Chapman, Kwapi, Eckblad & Zinser, 1994). Biological relatives of schizophrenic patients manifest a high rate of SPD and related symptoms (Clementz, Grove, Katsanis & Iacono, 1991; Silverman, Siever, Horvath, Coccaro & Klar, et al., 1993). Individuals who meet the diagnostic criteria for SPD in young adulthood, or who show some of its symptoms, are at a heightened risk for developing schizophrenia (Angst & Clayton, 1986; Wolff, Townshend, McGuire & Weeks, 1991). Thus, SPD represents the prodromal phase of schizophrenia in some individuals. The genetic and developmental links between schizophrenia and SPD suggest that the two disorders share some underlying neuropathological determinants (Walker & Gale, 1995).
Schizotypy includes sets of positive, negative and disorganisational signs and symptoms (Liddle, 1987). The factors of schizotypy are to some extent in parallel with three recognized subgroups of schizophrenic symptoms (Andreasen & Olsen, 1982; Arndt, Alliger & Andreasen, 1991). These factors include “distortion of reality”, “positive symptoms” and “negative symptoms”. Compared to schizophrenia, the schizotypal personality could be defined essentially by negative symptoms (Giraldez, Caro, Rodrigo, Pineiro & Gonz, 2000). Their research confirmed the existence of a significant relationship between psychometric schizotypy and cognitive deficits. They also found that the negative factor of schizotypy is related to a large number of deficits in executive frontal and verbal functions, including concept formation, planning and mental flexibility, working memory and executive functions.

The limbic circuit plays a role in the expression of positive symptoms (Carpenter, Buchanana, Kirkpatrick, Tamminga, Thaker & Breier, 1992), whereas the frontal lobe dysfunction is associated with a behavioral syndrome that includes ‘negative’ features (Wolkin, Sanfilipo, Wolf, Angrist & Brodie, et al, 1992). SPD and schizophrenia have similar abnormalities in frontal and motor circuits, but significant differences in limbic circuitry structure and/or function. Limbic circuit structure and function have not been examined in SPD (Raine, Sheard, Reynolds & Lencz, 1992; Tien, Costa & Eaton, 1992). It is supposed that the extent to which the limbic circuitry is disrupted will determine whether the transition is made from SPD to schizophrenia (Walker & Gale, 1995).

There are several major questionnaires that assess schizotypy, including the Schizotypal Personality Questionnaire (SPQ; Raine, 1991) and the Personality Syndrome Questionnaire (PSQ; Gruzelier, Jamieson, Croft, Kaiser & Burgess, 2001a). Raine (1991) developed the SPQ to measure DSM-III-R (American Psychiatric Association, 1987) schizotypal personality disorder.
The questionnaire consists of total score and nine subscales, each of which corresponds to one of the nine symptoms of schizotypal personality disorder that the DMS-III-R manual recommends for use in diagnosis. The PSQ measures the psychometric schizotypy and separates schizophrenic-like beliefs and behaviors into “active”, “withdrawn” and “unreality” syndromes. This scale was developed by Gruzelier and associates at Imperial College, London, England. While it has been used in various studies, including P50 sensory gating, it has not been published. In the present study, the SPQ was used to select participants.

2.2. Sensory Gating in Schizotypy

Similar to research findings in schizophrenia, there are three identified inhibitory deficits in schizotypy: (1) P50 sensory gating, (2) Prepulse inhibition (PPI), and (3) antisaccade control.

P50 suppression deficit was observed in individuals with schizotypal personality disorder (Cadenhead, Light, Geyer & Braff, 2000). Cadenhead and associates suggested that sensory gating deficit observed in schizotypy was similar to those in individuals with schizophrenia and their relatives. Croft, Lee, Bertolot and Gruzelier (2001) found that college students with higher schizotypy exhibited either poor P50 suppression or increased sensitization to the second of two paired clicks. Furthermore, they found that in contrast to other positive features (such as odd speech, cognitive acceleration and bizarre behavior) and other negative features (such as constricted affect, withdrawal and social anxiety), subjects who reported more abnormal perceptual experiences and magical ideation had poorer P50 suppression. Their results suggested that the P50 suppression deficits found in schizophrenia and schizotypy are not merely fortuitous phenotypic markers of schizophrenia, but are related to core symptoms of mental disorder.

Prepulse inhibition (PPI) of the startle response is another operational measure of sensorimotor gating (Braff, Callaway, Geyer, Glick & Bali, 1978; Braff, Grillon & Geyer, 1992;
Kumari, Soni, Mathew & Sharma, 2000). Individuals with schizotypy also have reduced PPI when compared to normal comparison subjects (Cadenhead, Geyer & Braff, 1993; Cadenhead, Swerdlow, Shafer, Diaz & Braff, 2000b). Cadenhead et al. (2000b) found that schizophrenic patients, non-schizophrenic relatives of schizophrenic patients and individuals with schizotypy demonstrated greater PPI deficits in the right eye-blink than the left eye-blink response. This suggests that the inhibitory gating deficits of patients with schizophrenia spectrum disorders is associated more with left hemisphere modulation of prepulse inhibition.

Measures of saccade control (rapid redirection of gaze to locations of interest), especially those associated with behavior inhibition, effectively differentiate individuals with schizophrenia from comparison groups. Schizophrenic patients’ saccadic performance is characterized by an increased proportion of antisaccade errors, which indicates they cannot inhibit eye movements (Fukushima, Fukushima, Morita & Yamashita, 1990a). The non-psychotic first-degree biological relatives of schizophrenia and schizotypal subjects also generate an increased proportion of antisaccade errors (Brenner, McDowell, Cadenhead & Clementz, 2002; Ross, Harris, Olincy, Adler & Freedman, 1998).

Comparing three identified inhibitory deficits in 21 schizotypal personality individuals, Cadenhead, Light, Geyer, McDowell and Braff (2002) found that seven with schizotypal personality disorder had deficits on each of three paradigm, five with schizotypal personality disorder showed both P50 and antisaccade deficits, which are significantly correlated. These results indicated that P50 and antisaccade are in the same endophenotype and prepulse inhibition differs from other two.

In sum, individuals with schizotypy exhibit sensory gating and inhibition deficits that are thought to be associated with the frontal lobe, and possibly it is more evident in the left
hemisphere. Of great interest is that smoking tobacco may facilitate sensory gating among schizophrenics and health individuals. This is discussed in the next section.

3. The Effects of Tobacco on Sensory Gating

Tobacco facilitates early sensory gating and subsequent information processing (Heishman, 1994). Crawford, McClain-Furmanski, Castagnoli and Castagnoli (2002) found that sensory gating is significantly greater for smokers than never-smokers in a healthy young male sample. Smokers exhibited chronic effects in the fronto-central region, and these effects may reflect persistent dopaminergic activation due to the inhibition of monoamine oxidase observed in smokers.

The tobacco alkaloid nicotine is proposed to affect sensory gating by two ways (Crawford et al., 2002). The first one is sensitive to nicotine levels, nicotine attaches the nicotinic receptor on dopamine (DA) neurons and result in the release of DA (Cooper, Bloom & Roth, 1996). But this way is short lived since nicotine receptors deactivate rapidly. The second way is related to monoamine oxidase (MAO) level. MAO is the major enzyme that regulates the central DA level in brain (Cooper, Bloom & Roth, 1996). Long-time inhibition of MAO will lead to more DA excitement in CNS. These two ways provide more DA in the CNS, and then in turn DA enhances the sensory gating. Crawford et al. suggested that chronic smoking contributes to greater synchronization of neurons in response to auditory stimuli in the frontal and central region, and these may reflect persistent dopaminergic activation due to the inhibition of monoamine oxidase observed in smokers.

Dopamine is the neurotransmitter most often associated with the pathophysiology of schizophrenia. Nicotine appears to modulate the function of this neurotransmitter system,
suggesting that this modulation may explain the high prevalence of nicotine use among individuals with schizophrenia (Dalack, Healy & Meador-Woodruff, 1998). The prevalence of cigarette smoking among persons of schizophrenia may be 40-90%, higher than among those with other psychiatric diagnoses and as much as three times higher than the prevalence in the general population (Diwan, Casrine, Pomerleau, Meador-Woodruff & Dalack, 1998). In Glynn and Sussman’s study (1990), about 28% of the participants said they smoked in part because of associated reduction of psychiatric symptoms. Several of these patients reported increased psychiatric symptoms during withdrawal from tobacco. DA-related abnormalities may contribute to sensory gating deficits observed in schizophrenics and their near-relatives (Adler et al., 1998; Freedman et al., 1991). Smoking tobacco normalizes this deficit, but only temporarily.

4. Hypotheses and the Purpose of this Study

The aims of this study were to evaluate sensory gating, as accessed by the P50 paradigm, in individuals with and without schizotypy. Since the participants were recruited from a college population, where the schizotypal personality disorder may be less prevalent, participants represented the upper and lower 1/3s of schizotypy as assessed by Raine’s Schizotypy Personality Questionnaire (SPQ; Raine, 1991). Several studies, as reviewed previously, have reported deficits in sensory gating among individuals with schizotypy. To my knowledge, published studies have not assessed the modulating effects from smoking tobacco among this group. The following hypotheses were evaluated:

It was hypothesized that individuals with schizotypal personalities would have poorer sensory gating than those without them. Among individuals with schizotypy, those who smoke would have better sensory gating than those who do not smoke. Also, among those without
schizotypy, smokers would demonstrate better sensory gating. Like schizophrenics, after abstaining, it was expected that schizotypal smokers would show increased sensory gating due to smoking. Thus, it was hypothesized that smoking would enhance the sensory gating in individuals with schizotypal personality. An interaction was predicted between conditions and groups: condition should show a different effect in different groups.

The P50 has usually been studied at midline sites, especially Cz in schizophrenics, and almost no attention has been given to other regions or to possible lateralized effects. It was observed that individuals with schizotypy have right-sided eye blink prepulse inhibition deficits (Cadenhead et al., 2000b) and smoking can enhance the prepulse inhibition asymmetry (right greater than left). The present study tested whether this observation is extended to the P50 paradigm by comparing midfrontal (F3, F4), fronto-central (FC3, FC4), central (C3, C4), centro-parietal (CP3, CP4) and parietal (P3, P4) sites. It was hypothesized that individuals with schizotypy will show greater P50 deficits in the left hemisphere, and smoking can enhance this asymmetry (left greater than right).

There were two parts in the study: (1) online screening and (2) EEG study of low and high schizotypy groups who smoked and did not smoke. The next two chapters present the methods and results for these two parts. The final chapter is the discussion.
Chapter II: Preliminary Online Screening of Participants

1. Methods

1.1. Participants

There were 613 participants who took part in the online preliminary screening survey. There were 231 men (37.7%) and 364 women (59.4%) with an additional 18 participants (2.9%) who failed to indicate gender. Their average age was 19.01 years (range: 17 to 21, SD = 1.23). Among them, as seen in Figure 1, 298 (48.6%) were freshmen, 208 (33.9%) sophomores, 59 (9.6%) juniors, and 42 (6.9%) seniors. Six failed to report. There were 494 (80.6%) participants who reported being white, non-Hispanic. Of the others, 33 (5.4%) reported being African-American, 35 (5.7%) Asian, 15 (2.4%) Hispanic, 1 (0.2%) American Indian, 7 (1.1%) Asian Pacific Islanders, and 20 (3.3%) reported others. Eight (1.3%) did not report. The distribution is presented in Figure 2.

Institutional Review Board approval was obtained (Appendix A) with approval of consent forms (Appendices B and C). Participants read a consent form and agreed with it before they began the online survey. They received class extra credit for participation. They were mainly recruited from an introductory psychology class.

1.2. Questionnaires

In addition to asking for basic demographic information (age, gender, ethnic background), the following questionnaires were administered.

(1) Schizotypal Personality Questionnaire (SPQ; Raine, 1991). This questionnaire (see Appendix D) consists of 74 items that are answered either yes or no. In addition to a total score, the subscales are ideas of reference (9 items), excessive social anxiety (8 items), odd beliefs or
magical thinking (7 items), unusual perceptual experiences (9 items), odd or eccentric behavior (7 items), no close friends (9 items), odd speech (9 items), constricted affect (8 items), and suspiciousness (8 items). The total SPQ score was used to separate participants into four groups for the subsequent EEG study. In the original sample of California undergraduates, the mean was 23.5 for English undergraduates (Hall & Habbits, 1996). Coefficient alpha for the total scale scores was .90, with ranges from .71 to .78 for subscales (Raine, 1991). Test-retest reliability for two months was .82 (Raine, 1991). Based on their responses, participants were chosen for the EEG study.

(2) Personality Syndrome Questionnaire (PSQ; Gruzelier et al 2001a). This scale (Appendix E) was developed by Gruzelier and associates at Imperial College, London, England. While it has been used in various studies, including P50 sensory gating, the instrument itself has not been published. PSQ measures the psychometric schizotypy and separates schizophrenic-like beliefs and behaviors into “active”, “withdrawn” and “unreality” syndromes. While administered in this study, it will not be reported herein.

(3) Handedness Questionnaire (Annett, 1967). This questionnaire (Appendix F) consists of 12 items with five possible categories: always use left hand (1), usually use left hand (2), equally left or right (3), usually use right hand (4), or always use right hand (5). This resulted in possible scores from 0 to 60. Those scoring 48 or above were considered right-handed if they reported writing with their right hand. Based on their responses, right-handed participants were chosen for the EEG study.

(4) Family history of Schizophrenia and related disorders in first relatives (Crawford, unpublished). From a list of potential disorders (Appendix G), the participants were asked if their parents or siblings had any of them.
(5) Medical Screening Questionnaire (Crawford, unpublished). This questionnaire (Appendix H) asked for the medical history of the participants to determine if they were eligible for participating in the EEG study.

(6) Smoking History Questionnaire (Crawford & Wan, unpublished). This questionnaire (Appendix I) asked for the smoking history of the participants, whether they were smokers at the present time, how long they smoked, and how many they smoked everyday. Based on their responses, participants were chosen for the EEG study.

(7) Alcohol Usage Questionnaire (Crawford, unpublished). Participants were asked since September, 2003, on the average how many drinks they typically had if they drank alcohol. If they drank in the past two weeks, they were asked on the average how many drinks per day they had and on how many occasions they consumed five or more drinks at one sitting over the past two weeks (Appendix J).

1.3. Procedure

The present screening session was advertised on the Experimetrix internet site to all students in undergraduate psychology courses who wished to participate in experiments for extra credit. Once students indicated an interest, they read a consent form (Appendix B) and went on to the questionnaire if they indicated their consent. Filling out the questionnaires took less than one hour. Data was downloaded into an Excel spreadsheet and then transferred to SPSS for statistical analyses.

2. Results

2.1. Schizotypal Personality Questionnaire
The mean SPQ score across all participants (2 missing) was 20.7 (SD = 12.48), ranging from 0 to 61. The distribution of SPQ scores is shown in Figure 3. The mean SPQ score for the men (Mean = 22.13, SD = 13.59) was significantly higher than for women (Mean = 19.82, SD = 11.67), \( t(593) = 2.19, p < .05 \).

2.2. Handedness Questionnaire

Across all 613 participants, the handedness scores ranged from 12 to 60 (2 missing) with a mean of 50.81 (SD = 9.4). The graph in Figure 4 shows the distribution of handedness scores. The mean handedness score for the men (49.46, SD = 9.51) was significantly lower than the women (51.73, SD = 9.45), \( t(593) = 2.86, p < .005 \).

2.3. Family history of schizophrenia and other mental disorders in first blood relatives

From a list of potential disorders, such as schizophrenia, bipolar and depression, participants were asked if their parents or siblings have one or more.

Among the 613 participants, 15 (2.45%) reported that they were adopted and did not know their family history. Among the rest, 57 (9.3%) reported that their mothers had been diagnosed with depression, schizophrenia, bipolar or other mental disorder, 37 (6.04%) reported their fathers, 57 (9.3%) reported their siblings and 58 (9.46%) reported their uncles or aunts had such diagnosis.

A more detailed analysis found that 39 reported that their mothers had been diagnosed with depression, 20 reported fathers, 19 reported sisters, 14 reported brothers and 34 reported uncles or aunts have been diagnosed with depression. Five reported that their mothers were diagnosed as bipolar disorder, 8 reported fathers, 3 reported sisters, 6 reported brothers and 7 reported uncles or aunts were diagnosed as bipolar disorder. One reported that their mother was diagnosed with schizophrenia, 8 reported uncles or aunts as having schizophrenia. Twelve participants
reported that their mothers were diagnosed with other psychiatric disorders, 9 reported fathers, 4 reported sisters, 1 reported brothers and 9 reported uncles or aunts were diagnosed other psychiatric disorders.

2.4. Smoking History Questionnaire

Of the participants, 349 (56.9%) reported that they had smoked tobacco cigarettes at some time. Of the men, presently 73.6% (N = 170) do not smoke, 7.8% (N = 18) smoke but not daily, and 17.6% (N = 64) smoke daily. Of the women, presently 74.5% (N = 271) do not smoke, 8% (N = 29) smoke but not daily, and 17.6% (N = 64) smoke daily. There was no significant gender difference. The mean age for starting to smoke was 16.02 (SD = 1.73, range: 11 to 21 years). The mean number of cigarette per day was 5.58 (range: .5 to 27, SD = 6.21). Of those who reported smoking, the men (N = 61, Mean=4.25, SD= 6.25) and the women (N = 93, Mean = 3.84, SD = 5.26) reported similar cigarette consumption per day.

Of the participants, 270 (44.04%) reported that they had smoked cigars. Presently, 71 participants (11.58%) reported that they smoke cigars, but not daily, 7 participants (1.14%) reported that they smoked cigars daily. Ages they began smoking cigars ranged from 10 to 20 years of age (Mean = 16.5, SD = 2.4).

Of the participants, 101 (16.48%) reported that they have smoked a pipe. Presently, 14 participants (2.28%) reported that they smoked a pipe but not daily, while 4 participants (.65%) reported that they smoked a pipe daily. Ages they began smoking a pipe ranged from 10 years old to 19 years of age (Mean =16.6, SD = 2.06).

Of the participants, 87 participants (14.19%) reported that they have chewed tobacco. Presently, 13 participants (2.12%) reported that they chew tobacco but not daily, 11 participants
(1.79%) reported that they chew tobacco daily. Ages they began smoke pipe ranged from 13 years old to 19 years old (Mean = 16.5, SD = 1.47).

2.5. Alcohol Usage Questionnaire

Since September 2003, 590 participants (Men: 227; Women: 363) reported drinking alcohol. Of those who drank alcohol, the average drinks per participant were 3-4 drinks, among them, men (mean 5-6 drinks per time) reported drinking more heavily than women (mean of 3-4 drinks per time).
Chapter III: EEG Assessment of Sensory Gating

1. Methods

1.1. Participants

Participants were 39 strongly right-handed undergraduates, 18 men and 21 women, with an average age of 18.87 years (range: 18 to 21, SD = 1.01). Among them, 21 were freshmen, 11 sophomores, 4 juniors, and 3 seniors. Of the participants, 31 reported being white and non-Hispanic, 3 African-American, 4 Asian, and 1 failed to report ethnicity. They reported normal hearing and no known history of medical, neurological or psychiatric problems. They reported they did not ingest any prescription (except birth control) or over-the-counter drugs, alcohol or illicit drugs, for at least 1 day before the experiment. They received class extra credit for participation.

1.2. Experimental Design

There were four experimental groups selected to have high or low schizotypy characteristics, as measured by the SPQ (Raine, 1991), and within each of these groups, cigarette smokers or non-smokers. Condition of abstaining (overnight or no less than 4 hours before) and smoking (one cigarette) were compared in smoker groups. In the non-smoker groups, they were tested twice without smoking. The design is presented below.

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<th>Condition 1</th>
<th>Condition 2</th>
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<td><strong>Low Schizotypy</strong></td>
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<td>participants</td>
<td>Smoker (N=9)</td>
<td>Abstain</td>
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<td>Non-smoker (N=10)</td>
<td>Control</td>
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<td><strong>High Schizotypy</strong></td>
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<tr>
<td>participants</td>
<td>Smoker (N=10)</td>
<td>Abstain</td>
</tr>
<tr>
<td></td>
<td>Non-smoker (N=10)</td>
<td>Control</td>
</tr>
</tbody>
</table>
1.3. Inclusionary Criteria

The following inclusionary criteria were employed.

(1) Schizotypy Personality Questionnaire (SPQ; Raine, 1991). To be included, participants had to fall in the upper or lower 1/3 of Raine’s SPQ. The upper cutoff of SPQ score was 25 (66.8% and above) and the lower cutoff was 13 (33.4% and below).

(2) Handedness Questionnaire (Annett, 1967). On a scale from 0 to 60 on the Annett handedness scale, those scoring 48 or above were considered right-handed.

(3) Medical Screening Questionnaire (Crawford, unpublished). To be included, participants had to report no major medical or psychiatric histories, including no concussions or hearing problems.

(4) Smoking History Questionnaire (Crawford & Wan, unpublished). Participants who reported smoking every day or no smoking presently were assigned within the low and high schizotypy groups.

1.4. Experimental Procedure

After receiving an explanation of the study, participants signed a consent form (Appendix C). All participants were assessed for exhaled Carbon Monoxide level. Smoking abstinence was confirmed by measuring exhaled carbon monoxide (CO ppm) with a Vitalograph Breath CO carbon monoxide monitoring device (Vitalograph Inc, Lenexa, KS). It has been demonstrated that the CO can be directly related to the percentage of carboxyhemoglobin in an individual’s blood (Jarvis et al., 1986). Clinical research has shown that the best guidelines are (ppm): 0 -10: non-smoker; 11 - 20: light smoker; 21 - 100: heavy smoker. If there was evidence of recent tobacco intake (>10 ppm), the subject was to be rescheduled for a subsequent day. All
participants were below 10. Participants also filled out the Differential Attentional Processes Inventory (Crawford, Brown & Moon, 1993; not reported in the thesis) and the medical, smoking, and alcohol use questionnaires which are described in Chapter 2.

The study was conducted in the Neurocognition Laboratory located in the Department of Psychology, Virginia Tech. The EEG room was equipped with a Neuroscan 30-channel workstation. It was next to an experimental room, with a mirror between the rooms. All participants were fitted with a cap (Electrocap Inc.) with 30 electrodes (impedance < 5KΩ), referenced to the nose, plus vertical (above and below left eye) and horizontal EOG electrodes. Skin underlying the electrodes was cleaned with Nuprep and electrodes were then filled with Electrogel.

Participants sat alone in an upright comfortable chair in the experimental room. Sixty sec
eyes open and eyes closed resting EEG was recorded before the P50 paradigm during the first
condition and after the P50 paradigm during the second condition. During the P50 paradigm,
they were instructed to attend to a stationary focus point on a computer monitor screen at eye
level, 80 cm in front of them. Two identical presentations were given. Smokers were tested while
abstaining and then after smoking. The non-smokers were given the presentations twice in the
same manner as the smokers, but without smoking.

After condition one, all participants left the experimental room. Smokers were asked to
smoke their usual brand of cigarette. Non-smokers waited for a similar length of time.
 Afterwards, their carbon monoxide was measured. They were immediately hooked up for the
EEG recording and the EEG quality was verified before presenting the P50 paradigm in
condition two.
After the EEG study, the cap was removed, and the face and hair was cleaned of any recording gel. After this, they participated in two computer tasks, the ANT test (Fan, Mccandliss, Sommer, Raz and Posner, 2002) and the Stroop Test; these are not reported in the thesis. Finally, all questions were answered and a copy of the consent form was given the participant. If interested, participants were shown their EEG recordings.

1.5 Paired-Click Paradigm

In each condition, 40 pairs (S1 and S2) of 1 ms, 1000 Hz sinusoidal tones (1ms rise/fall; 70dB) were delivered by the Neuroscan stimulus generation system through speakers placed at ear height 50 cm to the left and right of the participant’s head. There was a 512 ms inter-click interval and 10s inter-pair interval. The total time was 8-9 minutes. In order to maintain a consistent level of attention across participants, they were instructed to maintain gaze on a cross “+” in the computer screen in front of them about 80cm in front of them.

1.6. EEG Recording

Continuous EEG (0.1 to 100 Hz, 500 Hz sampling rate; gain of 150) was recorded and digitized by Neuroscan® SynAmps amplifier and Scan® version 4.0 software. EEG data was collected from 30 channels (FPz, FP1, FP2, Fz, F3, F4, F7, F8, FCz, FC3, FC4, FC7, FC8, Cz, C3, C4, C7, C8, CPz, CP3, CP4, CP7, CP8, Pz, P3, P4, P7, P8, O1, O2) with tin electrodes.

1.7. EEG Data Analysis

EGG data was processed for artifacts, epoched and averaged using Scan® version 4.0 software. For the P50 data, the continuous EEG was epoched offline from -50 msec to 462 msec. Epochs were detrended and baselined to the pre-stimulus 50 msec period. To eliminate gross eye movement, muscle, and movement artifact, epochs were submitted to automatic artifact rejection (±50 mV for eye channels, Fp1, Fp2, F3 and F4). Then they were visually verified with further
observed muscle, movement and eye artifacts removed. Separate averaged evoked potentials (EPs) were created for the S1 and S2 stimuli in each of the two conditions for each participant. To better image the early components, we applied a two-pole Butterworth digital filter (band-pass 10-70 Hz, 24 dB per octave roll-off) to the averaged EPs.

The averaged evoked potentials were exported and analyzed off-line with Vision Analyzer® software (Brain Products, Germany). A semi-automatic program identified the N40 (30-60 ms) and P50 (45-75 ms) peak amplitudes and latencies for the following regions: frontal (F3, Fz, F4), fronto-central (FC3, FCz, FC4), central (C3, Cz, C4), centro-parietal (CP3, CPz, CP4), and parietal (P3, Pz, P4). The peaks were verified by the experimenter, evaluated by the experimenter’s major professor, and adjusted when necessary. The experimenter was blind to the schizotypy and smoking status of the participants. This data was exported to an Excel format.

1.8. Statistical Analyses

Using the Statistic Package of Social Science (SPSS), analyses of variance (ANOVA) were used for statistical tests for the EEG data, and where appropriate, p values were adjusted with the Greenhouse-Geisser epsilon correction for nonsphericity. Reported are the uncorrected degrees of freedom and the probability level after correction. T-tests were used for all post hoc comparisons. ANOVAs or t-tests were used for the other measures.

2. Results from Questionnaires

2.1. Schizotypal Personality Questionnaire

For the HiS/S group (High Schizotypy/Smoker), SPQ scores of 10 participants (6 men, 4 women) ranged from 22 to 49, the mean score was 34.2 (SD=8.9). In the LoS/S group (Low Schizotypy/Smoker), SPQ scores of 9 participants (5 men, 4 women) ranged from 0 to 11, the
mean score was 4.78 (SD=3.77). In the HiS/NS group (High Schizotypy/Nonsmoker), SPQ scores of 10 participants (4 men, 6 women) ranged from 38 to 56, the mean score was 45.9 (SD=5.82). In the LoS/NS group (Low Schizotypy/Nonsmoker), SPQ scores of 10 participants (3 men, 7 women) ranged from 1 to 7, the mean score was 2.4 (SD=1.84). The means are graphed in Figure 5.

To assess SPQ differences between groups, a Schizotypy (2) * Smoker (2) ANOVA was performed. As expected, there was a significant difference between High Schizotypy (Mean = 40.05, SD = 9.47) and Low Schizotypy (Mean = 3.53, SD = 3.08) groups, F (1, 34) = 389.45, p < .001. There was a significant difference between Smoker (Mean = 20.26, SD = 16.55) and Non-smoker (Mean = 24.15, SD = 22.7) groups, F (1, 34) = 6.37, p < .02.

There was an unexpected Schizotypy * Smoker interaction, F (1, 34) = 14.51, P < .001. Comparison of these four groups found that the HiS/S group reported significantly more schizotypy characteristics than LoS/S and LoS/NS groups, respectively, ts (17 and 18) = 9.18 and 11.06, p < .001. The HiS/NS group was significantly higher than the other three groups, respectively, ts (17 or 18) = 3.48, 18.04 and 22.54, p < .005. Thus, the HiS/NS group reported more schizotypy characteristic than the HiS/S group.

2.2. Handedness Questionnaire

Across the 39 selected participants, the mean handedness score was 55.33 (from 48 to 60, SD = 3.24). For the HiS/S group, the mean Handedness score of 10 participants was 55.9 (SD=2.96). In the LoS/S group, the mean Handedness score of 9 participants was 57.11 (SD=3.06). In the HiS/NS group, the mean Handedness score of 10 participants was 53.6 (SD=3.86). In the LoS/NS, the mean Handedness score of 10 participants was 54.9 (SD=2.33).
An ANOVA showed that Smokers (Mean = 56.47, SD = 2.99) were significantly more righthanded than the non-smokers (Mean = 54.25, SD = 3.18), F (1, 34) = 5.14, p < .05.

2.3. Family History of Schizophrenia and Related Disorders

In the HiS/S group, six reported that one or more of their first blood-relatives have been diagnosed with depression, bipolar or schizophrenia, whereas one in the HiS/NS group reported this. Three in LoS/NS group reported such things.

2.4. Smoking History Questionnaire

Among 39 participants, 20 participants reported smoking tobacco cigarettes presently. The ages they began smoking tobacco cigarettes were from 13 to 19 years (Mean = 16.32, SD = 1.46). The number of the cigarettes they smoked were from .5 to 20 per day (Mean = 4.44, SD = 5.96). Within the two groups who presently smoked, the mean smoking number per day of those with High Schizotypy (Mean = 6.15 SD = 7.49) was not significantly different from the Low Schizotypy (Mean = 1.61, SD = 2.15). Note that the standard deviation was greater among the high than low schizotypy groups. Seventy percent in each group reported smoking on the average, 2 or fewer cigarettes per day.

Smokers also filled out the Fagerström’s Smoking History Questionnaire (Fagerström, 1987). The total score was from 0 to 6 (Mean = 1.11, SD = 1.66). High schizotypy individuals (Mean = 1.8, SD = 2.04) reported more additive behaviors than low schizotypy individuals (Mean = .33, SD = .5), t (17) = 2.09, p = .052.

2.5 Carbon Monoxide Level

A Schizotypy (2) * Smoker (2) * Pre/Post Condition (2) mixed factorial ANOVA, with repeated measures for the last factor was conducted for measured carbon monoxide level. As expected, there was a significant Smoker * Condition interaction, F (1, 34) = 51.87, p < .001.
There was also a significant Condition effect, F (1, 34) = 555.23, p < .001. Smokers had a significantly higher CO level than non-smokers, F (1, 34) = 32.92, p < .001. As expected, smokers increased their CO level (ppm: means parts per million) significantly after smoking, t (17) = 7.21, p < .001 (Pre: M = 2.94, SD = 2.46; Post: M = 9.33, SD = 5.05), whereas non-smokers did not (Pre: M = 1.40, SD = .60; Post: M = 1.50, SD = .61).

3. Results from EEG data

Most studies that use the P50 paradigm assess sensory gating only assess Cz, although a few also assess Fz. I wished to assess the degree to which midline sensory gating, as well as P50 amplitude and latency, differed across regions and from surrounding left and right hemispheric sites. Furthermore, rarely has hemisphere difference of sensory gating been assessed. To assess the hypotheses, separate ANOVAs were then computed for midline and lateral sites. Below three separate sections address P50 amplitude, P50 latency, and N40 – P50 sensory gating.

3.1. Sensory Gating

Sensory gating value was calculated by S2 (P50 – N40)/S1 (P50 – N40). The lower the value, the better the sensory gating.

3.1.1. Sensory Gating Analyses across all Sites

A Condition(2) * Region(5) * Location(3) * Schizotypy (2) * Smoker(2) mixed ANOVA, with repeated measures for the first three factors, was conducted.

There was a significant Condition effect, F (1, 33) = 4.832, p < .05. As shown in Figure 6, sensory gating was significantly greater in Condition 1 (Mean = .62, SD = .30) than Condition 2 (Mean = .84, SD = .55).
There was a significant Location effect, $F(2, 66) = 6.56, p < .01$. Shown in Figure 7, the midline sites across all participants (Mean = .60, SD = .24) showed better sensory gating than the left (Mean = .86, SD = .63) and right (Mean = .74, SD = .34) hemisphere, ts (38) = 3.53, 3.74, respectively, $p < .001$. No significant difference between the left and right hemisphere was observed.

### 3.1.2. Sensory Gating Analyses in Midline Sites

A Condition (2) * Region (5) * Schizotypy (2) * Smoker (2) mixed ANOVA, with repeated measures for the first two factors, was conducted for the midline sites. There was a significant Region effect, $F(4, 136) = 2.70, p < .05$. Fz (Mean = .65, SD = .35) showed significantly poorer sensory gating than FCz (Mean = .59, SD = .36) and Cz (Mean = .53, SD = .21), ts (38) = 3.68 and 3.12, $p < .005$. Pz (Mean = .67, SD = .38) showed significantly less sensory gating than Cz and CPz (Mean = .55, SD = .24), ts (38) = 2.45 and 2.45, $p < .02$. See Figure 8.

### 3.1.3. Sensory Gating Analyses for Left and Right Hemispheres

Condition(2) * Hemisphere(2) * Schizotypy(2) * Smoker(2) ANOVAs were performed in each region.

**Frontal (F3 and F4) Region** Hemisphere did not interact significantly with either smoking group or schizotypy group. There were no significant main effects or interactions.

**Fronto-central (FC3 and FC4) Region** There was a significant Condition effect. Condition 1 showed better sensory gating than Condition 2, $F(1, 35) = 6.27, p < .02$. There was a significant interaction between Schizotypy and Smoker group, $F(1, 35) = 4.15, p < .05$. In the Low Schizotypy group, smokers (Mean = .86, SD = .29) showed less sensory gating than non-smokers (Mean = .56, SD = .27), $t(17) = 2.27, p < .05$. Whereas in the high schizotypy group, there was no significant difference between smokers and non-smokers. See Figure 9.
Central (C3 and C4) Region  Condition 1 showed better sensory gating than Condition 2, \( F(1, 35) = 4.37, p < .05 \). There was a significant interaction between Schizotypy and Smoker group, \( F(1, 35) = 6.02, p < .02 \). In the Low Schizotypy group, smokers (Mean = .99, SD = .59) showed less sensory gating than non-smokers (Mean = .51, SD = .15), \( t(17) = 2.46, p < .05 \). In the Non-smoker group, the High Schizotypy group (Mean = .77, SD = .34) showed less sensory gating than Low Schizotypy group (Mean = .51, SD = .15), \( t(18) = 2.20, p < .05 \). As seen in Figure 9, among the non-smokers, the high schizotypy group exhibited poorer sensory gating at both fronto-central and central regions, among the smokers, the low schizotypy group showed poorer sensory gating at both fronto-central and central regions.

Centro-parietal (CP3 and CP4) Region There were no significant main effects or interactions.

Parietal (P3 and P4) Region There were no significant main effects or interactions.

3.2. P50 Amplitude

The grand averages of S1 and S2 for the two conditions for each of the four groups are presented in Figures 17a, 17b, 17c and 17d (Cz is presented as the example).

3.2.1. P50 Amplitude Analyses across All Sites

A Condition (2) * Stimuli (2) * Region (5) * Location (3) * Schizotypy (2) * Smoker (2) mixed ANOVA, with repeated measures for the first four factors, was conducted. As expected, the P50 amplitude was significantly greater for S1 than S2, \( F(1, 33) = 25.67, p < .001 \). The P50 amplitude was significantly different between the five Regions, \( F(4, 132) = 10.41, p < .001 \). There was a significant Location effect for P50 amplitude, \( F(2, 66) = 26.96, p < .001 \). There was a barely significant interaction between Location and Smoker, \( F(2, 66) = 3.07, p = .053 \). There were significant two-way interactions between Stimuli and Region, \( F(4,132) = 9.04, p < .005 \),
Stimuli and Location, F (2, 66) = 24.80, p < .001, and Region and Location, F (8, 264) = 9.89, p < .005. There was a significant interaction among Stimuli, Region and Location, F (8, 264) = 7.71, p < .01. Schizotypy group was neither a significant main effect nor in any interaction. The means for the midline, left and right hemisphere sites for S1 and S2 in each condition are graphed in Figure 10.

Follow-up t-tests for assessing the interactions are provided in Appendix K. The P50 amplitude of S1 was higher than S2 in most sites in each condition, except they did not differ significantly at F3 and F4 in Condition 1. In all regions the midline P50, both S1 and S2, were significantly greater in amplitude than either the left or right hemisphere sites. The fronto-central and central regions showed significantly greater P50 amplitude for S1 than the other regions.

3.2.2. P50 Amplitude Analyses in Midline Sites

A Condition (2) * Stimuli (2) * Region (5) * Schizotypy (2) * Smoker (2) mixed ANOVAs, with repeated measures for the first three factors, was conducted for midline sites. There was significant Stimuli effect, S1 was significantly higher than S2, F (1, 34) = 43.69, p < .001. There was significant Region effect, F (4, 136) = 20.80, p < .001. There was a significant interaction between Stimuli and Region, F (4,136) = 21.18, p < .001 (see Figure 11).

Follow up tests found that for S1, Cz (Mean = 4.14µV, SD = 2.31) was significantly higher than Fz (Mean = 2.91µV, SD = 1.60), FCz (Mean = 3.82µV, SD = 2.05), CPz (Mean = 3.25µV, SD = 2.12) and Pz (Mean = 2.53µV, SD = 1.75), ts (37 or 38) = 5.85, 2.49, 7.15 and 8.42, p < .02. FCz was significantly higher than Fz, CPz and Pz, ts (37 or 38) = 7.83, 2.73 and 5.88, p < .01. CPz was significantly higher than Pz, t (37) = 7.18, p < .001. For S2, Cz (Mean = 1.86µV, SD = 1.30) was significantly higher than Fz (Mean = 1.64µV, SD = .89), CPz (Mean = 1.61µV, SD = 1.29) and Pz (Mean = 1.45µV, SD = 1.23), ts (37 or 38) = 2.10, 3.19 and 3.68, p < .05. FCz
was significantly higher than Fz and Pz, ts (38) = 3.42 and 2.99, p < .005, CPz was significantly higher than Pz, t (37) = 3.01, p < .005.

3.2.3. P50 Amplitude Analyses for Left and Right Hemisphere

Because differences were expected to occur more so in the fronto-central and central regions, based on prior research, a Condition (2) * Stimuli (2) * Hemisphere (2) * Schizotypy (2) * Smoker (2) mixed ANOVAs, with repeated measures for the first three factors, was conducted for each region.

**Frontal (F3 and F4) Region** Hemisphere did not interact significantly with either smoking group or schizotypy group.

**Fronto-central (FC3 and FC4) Region** There was a highly significant Hemisphere * Schizotypy interaction, F (1, 35) = 8.31, p < .007. As seen in Figure 12, across conditions and stimuli, the High Schizotypy individuals did not differ significantly in P50 amplitudes in the left and right hemispheres (FC3: M = 2.40µV, FC4: M = 2.51µV), whereas the low schizotypy individuals had significantly greater P50 amplitude in the left than right hemispheres (FC3: M = 2.37µV, FC4: M = 1.85µV). High schizotypy individuals showed more P50 amplitude in the right hemisphere than did the low schizotypy individuals. There was a trend for a significant interaction between condition, schizotypy group, and smoking group, F (1, 35) = 3.62, p = .065.

**Central (C3 and C4) Region** There was a significant interaction between Hemisphere and Smoker, F (1, 35) = 8.48, p < .006. The smokers showed similar left and right P50 amplitude (Left: M = 2.42µV, Right: M = 2.39µV), whereas the non-smokers showed a greater right than left P50 amplitude (Left: M = 1.92µV, Right: M = 2.13µV). There was a significant interaction between Condition, Hemisphere, and Schizotypy, F (1, 35) = 4.07, p = .05. Only in Condition 2 was there a significant interaction between Hemisphere and Schizotypy, F (1, 37) = 5.97, p < .02;
this is similar to that seen for both conditions at the fronto-central region (see Figure 13 upper graph).

**Centro-parietal (CP3 and CP4) Region** There was a significant interaction between Hemisphere and Smoker, $F(1, 35) = 5.93, p < .02$. Like the central region, the smokers showed a greater left than right P50 amplitude (Left: $M = 2.01\mu V$, Right: $M = 1.78\mu V$), whereas the non-smokers showed the opposite asymmetry with a greater right than left P50 amplitude (Left: $M = 1.70\mu V$, Right: $M = 1.89\mu V$). There was a non-significant trend for an interaction between condition, hemisphere, and schizotypy, $F(1, 35) = 3.69, p = .063$ (see Figure 13 middle graph).

**Parietal (P3 and P4) Region** There was a significant interaction between Hemisphere and Smoker, $F(1, 35) = 4.93, p < .04$. The smokers showed similar P50 amplitude in both hemispheres (Left: $M = 1.86\mu V$, Right: $M = 1.81\mu V$), whereas the non-smokers showed asymmetry with a greater right than left P50 amplitude (Left: $M = 1.41\mu V$, Right: $M = 1.69\mu V$) (see Figure 13 lower graph).

### 3.3. P50 Latency

#### 3.3.1. P50 Latency Analyses across all Sites

A $\text{Condition}(2) \times \text{Stimuli}(2) \times \text{Region}(5) \times \text{Location}(3) \times \text{Schizotypy}(2) \times \text{Smoker}(2)$ mixed ANOVA, with repeated measures for the first four factors, was conducted.

The P50 latency was significantly different across five regions, $F(4, 140) = 12.33, p < .001$. As seen in Figure 14, latency became significantly longer from posterior to anterior sites. This suggests that the P50 component moves from posterior to anterior areas of the brain, something that would need to be assessed through source localization methods. Unlike the P50 amplitude there was no significant main effect for location. There were two significant interactions: Condition, Region, Schizotypy and Smoker group, $F(4, 140) = 4.62, p < .01$, and Condition,
Region, Location, Schizotypy and Smoking Group, F (4, 140) = 2.33, p < .05. These will be addressed in the following two sections.

### 3.3.2. P50 Latency Analyses in Midline Sites

A Condition (2) * Stimuli (2) * Region (5) * Schizotypy (2) * Smoker (2) mixed ANOVA, with repeated measures for the first three factors, was conducted for midline sites. There was a significant Region effect, F (4, 140) = 8.24, p < .001. As shown in Figure 14, from posterior to anterior sites there was a progressive increase in P50 latency. Fz (Mean = 63.56ms, SD = 5.28) was significantly longer than Cz (Mean = 62.31ms, SD = 5.50), CPz (Mean = 61.97ms, SD = 5.85) and Pz (Mean = 61.79ms, SD = 6.57), ts (38) = 3.11, 3.25 and 3.35, p < .005. FCz (Mean = 63.14ms, SD = 4.97) was significantly longer than Cz, CPz and Pz, ts (38) = 3.83, 3.73 and 3.13, p < .005.

### 3.3.3. P50 Latency Analyses for Left and Right Hemisphere

Condition (2) * Stimuli (2) * Hemisphere (2) * Schizotypy (2) * Smoker (2) mixed ANOVAs, with repeated measures for the first three factors, were conducted for each region.

**Frontal (F3 and F4) Region** There was a significant interaction between Condition, Schizotypy and Smoker group, F (1, 35) = 5.36, p < .05. In HiS/S and LoS/NS groups, Condition 1 (Mean = 63.1ms and 60.5ms) was shorter than Condition 2 (Mean = 65.55ms and 64.05ms). In LoS/S and HiS/NS groups, Condition1 (Mean = 64.25ms and 63.89ms) was longer than Condition 2 (Mean = 62.4 and 61.83). There was a significant interaction between Stimuli, Hemisphere and Smoker group, F (1, 35) = 5.46, p < .05. Among smokers, left hemisphere (Mean = 62.79ms) was shorter than right hemisphere (Mean = 64.04ms) for S1, but for S2, left hemisphere (Mean = 64.55ms) was longer than right hemisphere (Mean = 62.99ms). Among non-smokers, left and right hemispheres were almost same (see Figures 15 and 16).
Other Regions  An ANOVA between left and right hemisphere in each region was performed along with the other factors. Hemisphere did not interact significantly with either smoking group or schizotypy group.
Chapter IV: Discussion

1. Overview of Findings

With respect to the hypotheses of the study, it was found that: 1) Sensory gating, as assessed by S2 (P50-N40)/S1 (P50-N40), was greater at frontal-central and central regions in comparison to mid-frontal and parietal regions. 2) Furthermore, sensory gating was significantly greater at midline than left or right hemispheres. 3) Condition 1 showed better sensory gating than Condition 2. 4) The High Schizotypy group showed poorer sensory gating than the Low Schizotypy group among non-smokers. 5) Smokers showed poorer sensory gating than non-smokers in the Low Schizotypy group.

In terms of P50 amplitude, it was found that: 1) FCz and Cz showed the highest P50 amplitude, greater than all other sites. 2) S1 had higher P50 amplitude than S2. 3) The low schizotypy individuals had significantly greater P50 amplitude in the left than in the right fronto-central region, but the high schizotypy individuals showed more P50 amplitude in the right hemisphere than did the low schizotypy individuals. 4) Smokers showed a greater left than right P50 amplitude in centro-parietal region, whereas the non-smokers showed the opposite asymmetry with a greater right than left P50 amplitude in central, centro-parietal and parietal regions.

In terms of P50 latency, it was found that: 1) The P50 latency became significantly slower from posterior to anterior sites. 2) In HiS/S and LoS/NS groups, Condition1 was faster than Condition 2. In LoS/S and HiS/NS groups, Condition1 was slower than Condition 2. 3) Among smokers, left hemisphere latency was shorter than right hemisphere for S1, but for S2, left
hemisphere was slower than right hemisphere. Among non-smokers, left and right hemisphere latencies were almost the same for S1 and S2.

2. P50 Sensory Gating

2.1. The Location of Sensory Gating

Since the early work by Freedman and his associates (1983), often an underlying but rarely tested assumption has been that sensory gating should be assessed at Cz where it was presumed to be greatest. Yet, Crawford et al. (2002) reported that greater differences were found at Fz in their examination of smokers and never-smokers (without known psychopathologies). In the present study, FCz and Cz showed greater sensory gating ratio than more anterior and posterior regions. Thus, midline fronto-central and central regions may be the best sites to assess P50 sensory gating.

2.2. Condition 1 vs. Condition 2

Condition 1 showed better sensory gating than Condition 2 across all participants in fronto-central and central regions. This was an unexpected finding. The difference between smokers and non-smokers was that, for smokers, they had an abstaining condition (overnight or no less than 4 hours before) and smoking condition (after smoke one cigarette immediately); whereas for non-smokers, they just received the same measurement twice without smoking. As discussed in greater detail below, smoking (condition 2) had no impact upon sensory gating among the smokers.

Habituation over blocks can reduce sensory gating (Brenner, Edwards, Carroll, Kieffaber & Hetrick, 2004). Arousal due to a painful stimulus or an enhancing task may decrease sensory gating (White & Yee, 1997; Johnson & Adler, 1993; Yee & White, 2001). Getting up and
leaving the room after Condition 1 may have aroused the participants. Whether this or habituation to the testing environment, contributed to the significant reduction in sensory gating in Condition 2 is unknown. Further research may need to address the fragility of consistent sensory gating levels over time and conditions.

2.3. Interaction of Schizotypy Groups and Smoking Status

In this study, there was a significant interaction between smokers and schizotypy groups, but no main effect for smoker or schizotypy groups. Among the non-smokers, the high schizotypy group exhibited poorer sensory gating at both fronto-central and central regions than did the low schizotypy group. This would be expected based upon prior research (Cadenhead et al., 2000a; Croft et al., 2001). By contrast, among the smokers, the high schizotypy group showed better sensory gating at both fronto-central and central regions than the low schizotypy group. Smokers showed poorer sensory gating than non-smokers in fronto-central and central sites in the Low Schizotypy group. Whereas in the High Schizotypy group, there was no significant difference between smokers and non-smokers. Like individuals with schizophrenia, it may be that smoking tobacco helps “normalize” sensory gating and serves as a “self-medication” among high schizotypy individuals. This needs to be investigated in further research.

A P50 suppression deficit was observed in patients with schizotypal personality disorder (Cadenhead, Light, Geyer & Braff, 2000). Croft et al. (2001) reported that schizotypy university students exhibited either poorer P50 suppression or increased sensitization to the second of two paired clicks. The finding in non-smokers is similar to their result, but among the smokers, the difference is opposite. The Low Schizotypy/Smoker group is an interesting group. When we screened the participants from internet, this group was the hardest one to get because the number of available candidates was so small.
Both Cadenhead et al. and Croft et al. failed to assess for smoking history of their participants, and thus it is difficult to compare our results with theirs. A recent study has confirmed our findings. Croft, Dimoska, Gonsalvez, and Clarke have a paper in press in which they examined P50 suppression as moderated by schizotypal beliefs and smoking. Like Crawford et al. (2002), they found P50 sensory gating was better in smokers than non-smokers. In those who did not smoke or smoked only a little, higher schizotypy participants showed poorer sensory gating, but among heavier smokers higher schizotypy participants showed better sensory gating. In the present study, we too found that among non-smokers the high schizotypy group exhibited poorer sensory gating. Furthermore, we too found that among smokers, the high schizotypy group exhibited better sensory gating. According to Fagerström’s Test for Nicotine Dependance (1987), high schizotypys reported a greater addiction to smoking than low schizotypys in the smoker group. This is a possible reason why we have the above finding. Croft et al. (in press) only reported on the Cz site, whereas in the present study we did not find these differences at Cz but rather in the fronto-central region.

Not as expected, this study did not show that Condition 2 (smoking) produced better sensory gating than Condition 1 (abstaining) among smokers. Thus, the acute effect of smoking on sensory gating is not shown in this study. Prior research (e.g., Adler et al., 1998; Freedman et al., 1991) showed that smoking tobacco decreased sensory gating deficits observed in schizophrenics and their near-relatives, but only temporarily. Earlier in our Neurocognition Laboratory, like the present study, Crawford et al. (2000) reported no acute effects of tobacco smoking on sensory gating. However, in contrast to the present study, Crawford et al. (2002) found that sensory gating was significantly greater for smokers than never-smokers. The difference between those two studies is that they selected the subjects from the normal population without considering the
schizotypal personality, but in this study, subjects came from the lower and upper 1/3 Raines’ SPQ sample. Furthermore, their participants were older and heavier smokers than those in the present study. Like Crawford et al., the present study did not find an acute nicotinic effect from smoking. Thus, any differences are chronic in nature. Among the possible contributing candidates, Crawford et al. argue for Monoamine oxidase (MAO) A and B influences. In the brains of smokers decreased MAO A and B may result in decreased rates of clearance of brain biogenic amines (such as Dopamine), and then lead to the chronic effect on sensory gating (Fowler, Volkow, Wang, Pappas, Logan, et. al, 1996). Crawford, McClain-Furmanski, Castagnoli, and Castganoli reported at the 10th World Congress of Psychophysiology in 2002 that better sensory gating correlated with lower MAO-B as measured in blood platelets. In the present study we did not assess MAO-B.

2.4. Left vs. Right Hemisphere

The P50 sensory gating has usually been studied at midline sites, especially Cz in various clinical populations and normal groups, and almost no attention has been given to lateralized effects. It was observed that individuals with schizotypy have right-sided eye blink prepulse inhibition deficits that are thought to be associated with left hemisphere deficits (Cadenhead et al., 2000b). Furthermore, they found that smoking could enhance the prepulse inhibition asymmetry (right greater than left). The present study tested whether this observation was extended to the P50 paradigm by comparing midfrontal (F3, F4), fronto-central (FC3, FC4), central (C3, C4), centro-parietal (CP3, CP4) and parietal (P3, P4) sites. It was hypothesized that individuals with schizotypy would show greater P50 deficits in the left hemisphere. Contrary to expectation, this study did not show any sensory gating differences between two hemispheres
across the low and high schizotypy groups, but it did in P50 amplitude, which will be discussed in the following section.

It has been suggested that EEG may not be able to identify the presumably lateralized sensory gating due to technical reasons (Thoma, Hanlon, Moses, Edgar & Huang, et al. 2003). MEG studies (Edgar et al., 2003; Thoma, et al. 2003) showed that the M50 was localized in the left and right hemisphere superior temporal gyri, with no mean differences between the two hemispheres but with between and within individual differences between the two hemispheres. In the next two sections, P50 amplitude and latency will be discussed.

3. P50 Amplitude

3.1. The P50 Components

The midline P50 amplitudes across all regions, both for S1 and S2, were significantly greater than either the adjoining left or right hemisphere sites (e.g., Cz, C3 and C4). The FCz and Cz showed significantly greater P50 amplitude, both S1 and S2, than Fz, CPz and Pz across all participants. There was no moderating effect from condition. According to Edgar et al. (2003), due to the orientation of the EEG current flow in the superior and inferior direction, the assumed sources in the left and right hemisphere generate maximum electrical potential distributions near the vertex (Cz) for surface recordings. Thus, the midline region around the vertex show a combined activity from both lateralized sites. The present data reflects this: maximal P50 amplitude was at fronto-central and central midline regions. Yet, as seen below, some laterality differences in P50 amplitude were found.

3.2. S1 vs. S2
As expected, the P50 amplitude of S1 was significantly greater than S2 in all midline sites. When we considered the lateral sites this was also the case except for F3 and F4 in Condition 1. As we know, sensory gating is the function of the brain to filter out the irrelevant information and filter in the relevant information. S1 can be considered as the novel information or the “relevant” information in each trial. S2 can be seen as the repeated information or the “irrelevant” information. When it reaches the brain, the brain’s activity to it was reduced automatically. This is in contrast to the participants’ experience. Participants perceived the two tones as the same intensity.

These data supports the existence of the filtering function of the brain again. There are two aspects to sensory gating: (1) the capacity to cease to respond or to significantly reduce the magnitude of its response to incoming irrelevant stimuli (gating out); and (2) the capacity to re-respond when a novel stimulus is presented or a change occurs in ongoing stimuli (gating in) (Boutros, Zouridakis & Overall, 1991).

3.3. Left vs. Right Hemisphere

There were no significant interactions between schizotypy and smoking groups, but there were some main effects.

**Low and High Schizotypy Comparisons.** While no brain asymmetries were observed for sensory gating, P50 amplitude asymmetries were observed. At only the fronto-central region, the low schizotypy individuals had significantly greater P50 amplitude in the left (FC3) than right (FC4) hemisphere, whereas the high schizotypy individuals showed no significant asymmetry. The high schizotypy individuals showed more P50 amplitude in the right (FC4) hemisphere than did the low schizotypy individuals. Similar significant asymmetries were shown in C3 and C4, but only in Condition 2. In an M50 study (Thoma et al., 2003) comparing schizophrenics and
normal comparisons, there were no significant laterality differences in M50 source strength between the groups. The present findings suggest that further research needs to assess potential underlying hemispheric source strength differences in individuals with and without schizotypy, possibly also using the M50 for better localization.

**Smoker and Non-Smoker Comparisons.** While no brain asymmetries were observed for sensory gating, P50 amplitude asymmetries were observed. There were significant interactions between group and hemisphere at central, centro-parietal and parietal regions. Smokers showed no significant asymmetries in central and parietal regions, but did show greater left than right P50 amplitude at the centro-parietal region. By contrast, the non-smokers showed greater P50 amplitude in the right than left hemispheres at central, centro-parietal, and parietal regions. In the left hemisphere in all three regions, the smokers had more P50 amplitude than did the non-smokers. Since there were no significant interactions with condition, these asymmetries seem to be chronic effects that are not impacted by whether they are abstaining or smoking. The unsolved question is whether these asymmetry differences are due to chronic smoking or to differences that were present prior to their starting to smoke. Furthermore, the larger P50 amplitude could be evidence of greater reactivity, or even overreactivity, in the left hemisphere to the auditory stimuli. If so, this suggests a potential physiological deficit in the left hemisphere.

Hemispheric differences in certain EEG band frequencies between smokers and non-smokers have been noted in the literature, with changes after acute cigarette smoking (e.g., Gilbert, Meliska, Welser & Estes, 1994; Pritchard, 1991). Since the P50 component of the ERP reflects underlying gamma band oscillations (Crawford et al., 2002), an examination of research addressing gamma (about 32 – 48 Hz) is of particular interest. Crawford et al. (2002) reported significantly greater stimulus-bound gamma oscillations at FCz and Cz for smokers but did not
examine laterality differences. Their participants were much “heavier” smokers (20 cigarettes or more per day for at least five years) than this study’s participants (Mean = 4.44 per day). If these P50 amplitudes are reflective of underlying source strengths of the two hemispheres, in the temporal superior gyri and possibly other brain regions, then the present data argue for differences in source strengths between smokers and non-smokers. The above discussed M50 study of laterality differences in individuals with and without schizophrenia may have been confounded by smoking histories since schizophrenics are significantly more likely to smoke than the general population.

4. P50 Latency

4.1. The P50 Latency

The P50 latency was significantly different across five regions; latency became significantly slower from posterior to anterior sites. This suggests that the P50 component moves from posterior to anterior areas of the brain, something that would need to be assessed through source localization methods. Unlike the P50 amplitude, there was no significant main effect for location: midline, left and right sites showed the similar latencies.

4.2. Condition 1 vs. Condition 2

There was a significant interaction between Condition, Schizotypy and Smoker group in the frontal region. In HiS/S and LoS/NS groups, Condition1 was shorter than Condition 2. In LoS/S and HiS/NS groups, Condition1 was longer than Condition 2. Cadenhead et al. (2000) reported that individuals with schizotypal personality disorders recruited from inpatient and outpatient facilities, did not differ from normals in levels of P50 latency and amplitude at Cz. In the present study, no latency differences were observed at Cz.
4.3. Left vs. Right Hemisphere

There was a significant interaction between Stimuli, Hemisphere and Smoker group in the frontal region. Among smokers, the left hemisphere was shorter than right hemisphere for S1, but for S2, left hemisphere was longer than right hemisphere. Among non-smokers, left and right hemispheres, latencies were almost the same. This again suggests potential differences in the underlying latencies of the sources in the left and right hemispheres among smokers and non-smokers. Speed of processing information may differ in smokers and non-smokers.

5. Future Research

Sensory gating studies have already shown that schizotypal individuals have poorer sensory gating than normal individuals (Croft, Lee, Bertolot, & Gruzelier, 2001). This study shows that in the low schizotypy group, non-smokers have the better sensory gating than smokers. These findings should be further verified in a population of heavier smokers.

There are limitations to the current study that should be considered. We limited our sample to 10 people per group. The small sample may limit the findings to generalize outside this population. Our sample included women and men; the unknown hormonal factor may influence the EEG. Furthermore, all participants were right-handed and the handedness may moderate EEG activities. Since we want to know the effect of smoking, the smoking history should be considered when we screen the participants. Heavier smokers might have different sensory gating problems. Future studies could focus on the patients or high scores of SPQ, and could divide smoking people into heavy smokers and lighter smokers.

Prior studies provide evidence that smokers have better sensory gating than non-smokers, but the acute effect of smoking on sensory gating did not show in this study. The smoking effect
on sensory gating was the chronic effect. Its relationships with the brain chemical metabolites, alpha-7-nicotine receptor and related gene study would be the interesting study topics in future research.

The P50 sensory gating has usually been studied at midline sites, especially Cz in schizophrenics, and no attention has been given to lateralized effects. Because individuals with schizotypy have right-sided prepulse inhibition deficits (Cadenhead et al., 2000b) and smoking can enhance the prepulse inhibition asymmetry (right greater than left), the present study assessed lateralized sensory gating. It was hypothesized that individuals with schizotypy would show greater P50 deficits in the right hemisphere, and smoking can enhance this asymmetry. However, this study did not show any hemispheric asymmetry effect of sensory gating, or any smoking effect on it.

P50 may not be the only measurement of sensory gating. It has been noted that schizophrenics have reduced amplitudes for the N100 and P200 components, and have significantly longer latencies for the P50 and N100 (Boutros, Korzyuko, Oliwa, Feingold, & Campbell, et al, 2004). Boutros and associates suggested that morphological abnormalities of the mid-latency auditory evoked potentials in schizophrenia patients should be taken into consideration. In the first known study addressing this, they recently examined sensory gating occurring at the N100 and P200 stages of information processing, what they refer to as a "post-P50 phase of information processing occurring during the mid-latency period" (p. 204). In comparison with healthy controls, the medicated schizophrenics showed sensory gating deficits beyond the preattentive P50 stage, specifically the early attentive (N100) and late attentive (P200). Future research needs to assess these later stages of sensory gating. With the present data, I will assess them in a later study.
Interestingly, P50 amplitude asymmetries were found in this study. This suggests that different brain asymmetries exist between smokers and non-smokers, and between low and high schizotypy individuals. The future studies could focus on the hemisphere asymmetry in different population samples. The effect of smoking on the two hemispheres also can be further studied.

Sensory gating studies always looked at the P50 amplitude, and little attention was given to P50 latency analyses. When we looked at the P50 latency, there were some significant results. The interaction between Condition, Schizotypy and Smoker group in the frontal region and the interaction between Stimuli, Hemisphere and Smoker group in the frontal region were significant. P50 latency is also the useful index in sensory gating and smoking studies. Since N100 latency abnormalities have already been examined in schizophrenia, the present study also can expand to analyze N100 amplitude and latency.
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childhood and adult life. II: Adult adjustment and the continuity with schizotypal personality


Figure 1: Year of College: Distribution of all Online Participants
Figure 2: Ethnic Distribution of all Online Participants
Figure 3: SPQ Score Distribution for All Online Participants

Std. Dev = 12.48
Mean = 20.7
N = 611.00
Figure 4: Handedness Score Distribution for all Online Participants

- Std. Dev = 9.44
- Mean = 50.8
- N = 611.00
Figure 5: SPQ Mean Scores for the Four Groups (HiS/S = High Schizotypy/Smoker; LoS/S = Low Schizotypy/Smoker; HiS/NS = High Schizotypy/Non-smoker; LoS/NS = Low Schizotypy/Non-smoker)
Figure 6: Mean Sensory Gating: Comparison of Conditions (N = 39)
Figure 7: Mean Sensory Gating: Midline Sites Have Better Sensory Gating
Figure 8: Mean Sensory Gating:
Central Region Shows Better Sensory Gating than Frontal and Parietal Region
Figure 9: Mean Sensory Gating in Fronto-central and Central Regions: Smokers and Non-smokers in Low and High Schizotypy Group
Figure 10: Mean P50 Amplitude for S1 and S2 in the Two Conditions
Figure 11: Mean P50 Amplitude for S1 and S2: Comparison of Midline Region
Figure 12: Fronto-central Hemispheres: P50 Amplitude Differences in Low and High Schizotypy Group
Figure 13: Central, Centro-parietal, and Parietal Hemisphere: P50 Amplitude Differences in Smokers and Non-smokers
Figure 14: Mean P50 Latency: Comparison across Regions (N = 39)
Figure 15: Mean P50 Latency in Frontal Hemispheres: Condition Differences across Groups
Figure 16: Mean P50 Latency in Frontal Hemispheres:
Compare Two Hemispheres for S1 and S2 in Smokers and Non-smokers
Figure 17a: Grand Average at Cz for Group 1: Condition 1 and Condition 2 (Black: S1, Red: S2)
Figure 17b: Grand Average at Cz for Group 2: Condition 1 and Condition 2 (Black: S1, Red: S2)
Figure 17c: Grand Average at Cz for Group 3: Condition 1 and Condition 2 (Black: S1, Red: S2)
Figure 17d: Grand Average at Cz for Group 4: Condition 1 and Condition 2 (Black: S1, Red: S2)
Appendix

Appendix A: IRB Approved Form

October 27, 2003

MEMORANDUM

TO: Helen J. Crawford Psychology 0436
    Li Wan

FROM: David M. Moore

SUBJECT: Expedited Approval – “Sensory Gating in Young Adults” – IRB # 03-496

This memo is regarding the above-mentioned protocol. The proposed research is eligible for expedited review according to the specifications authorized by 45 CFR 46.110 and 21 CFR 56.110. As Chair of the Virginia Tech Institutional Review Board, I have granted approval to the study for a period of 12 months, effective October 20, 2003.

Approval of your research by the IRB provides the appropriate review as required by federal and state laws regarding human subject research. It is your responsibility to report to the IRB any adverse reactions that can be attributed to this study.

To continue the project past the 12 month approval period, a continuing review application must be submitted (30) days prior to the anniversary of the original approval date and a summary of the project to date must be provided. My office will send you a reminder of this (60) days prior to the anniversary date.

Cc: File
    Department Reviewer: Dave Harrison Psy 0436
    OSP 0170
Appendix B: Consent Form 1

1. PURPOSE OF EXPERIMENT
   Today you are invited to fill out some common questionnaires that assess individual differences in attentional and personality characteristics. We are interested in the interrelationships between these questionnaires. Also, we are interested in learning more about individual differences in adults who do and do not smoke. You will also be asked to fill out short tobacco smoking history and handedness questionnaires.
   If you are interested and fit the criteria, you will be invited to return for a second study of EEG brain dynamics in the Neurocognition Laboratory in Williams Hall. We will ask both non-smokers and smokers to participate. Smokers will be asked to abstain from smoking for one night or continue smoking as usual before the experiment. During the experiment, smokers will be asked to smoke their own cigarette they usually use. Non-smokers will NOT be asked to smoke but will be asked to participate in the same tasks. For this two-hour experiment you will receive two hour's extra credit or $15.

2. PROCEDURE TO BE FOLLOWED IN THE STUDY:
   To accomplish the goals of the study, you will be asked to complete online a series of self-report instruments that assess your handedness, attentional styles, and personality styles. There are no right or wrong answers. It will take less than one hour.

3. ANONYMITY OF SUBJECTS AND CONFIDENTIALITY OF RESULTS:
   The results of this study will be kept strictly confidential. At no time will the researchers release your results to anyone without your written consent. The information you provide will have your name removed and only a subject code will identify you during analyses and any write-up of the research.

4. DISCOMFORTS AND RISKS FROM PARTICIPATING IN THE STUDY:
   There are no risks to you from participation in this study. Should the questions may remind of you of things that could make you feel uncomfortable, we recommend that you contact the local crisis center, RAFT, or the student counseling center.

5. BENEFITS OF THIS PROJECT:
   No personal benefit is promised you. Your participation in this project today will help advance the scientific knowledge of the interrelationships between responses on these questionnaires.

6. FREEDOM TO WITHDRAW:
   You are free to withdraw from this study at any time without penalty.

7. COMPENSATION:
   Participation will be totally voluntary. You will receive one hour's credit for participation in this project regardless of whether or not you complete the experiment today. Please check your course syllabi for information as to worth of this extra credit and for alternative ways by which to receive extra credit.

8. USE OF RESEARCH DATA:
   The information from this research may be used for scientific or educational purposes. It may be presented at scientific meetings and/or published and reproduced in professional journals or books, or used for any other purpose that Virginia Tech's Department of Psychology considers proper in the interest of education, knowledge, or research.

9. APPROVAL OF RESEARCH:
This research project has been approved by the Human Subjects Committee of the Department of Psychology and by the Institutional Review Board of Virginia Tech. You may print off a copy of the consent form.

10. SUBJECT'S PERMISSION:

I have read and understand the above description of the study. If I had questions, I called Li Wan or Dr. Crawford and had them all answered. I hereby acknowledge the above and give my voluntary consent for participation in this study. I further understand that if I participate I may withdraw at any time without penalty or not answer specific questions if I do not want to. I understand that should I have any questions regarding this research and its conduct, I should contact any of the persons named below:

Primary Graduate Student Researcher, Li Wan 239-6959
Supervising Faculty, Helen J. Crawford, Ph.D. 231-6520
Chair, Human Subjects Committee, D. Harrison, Ph.D. 231-6581
Chair, Institutional Review Board, David M. Moore
Asst. Vice Provost for Research Compliance 231-4991

If you agree to the above, please print off a copy of this consent form. Proceeding to the questionnaires is evidence of your informed consent to participate.
Appendix C: Consent Form 2

You have already been screened in Part 1 and passed the criteria to participate in this part of the study. This study compares brain wave activity of smokers with age-matched individuals who are non-smokers. There will be 20 in each group. Also, you represent different kinds of personality styles. Smokers are asked to abstain overnight (8-10 hours) from smoking and then to smoke one of their own cigarettes in the laboratory. Non-smokers will not be asked to smoke.

The purpose of this experiment is to examine (1) auditory evoked potentials to 70-75 dB tones presented in different patterns while attending the stimuli, and (2) assess attentional performance on several computerized tasks. The equipment is completely grounded and isolated and therefore safe; it has been approved for human use.

You are to report to the experimenter if you are on any medications or under doctor’s treatment. You are not to have ingested alcohol in the last 24 hours or any caffeine-containing drinks for at least 4 hours prior to the experiment. You are to report any recent history of using tobacco, either smoked or chewed. You are to inform the experimenter of all known medical and psychiatric problems that you have had that might interfere with the experiment.

2. PROCEDURE TO BE FOLLOWED IN THE STUDY:

To accomplish the goals of this study, the research will be carried out in Williams Hall psychology department. Participants will be assessed at the same time of day, between 8 AM and 1 PM to regulate food and nicotine ingestion and to control for possible circadian influences. You have already participated in the preliminary screening. This part of the experiment will take approximately two hours.

You will be administered questionnaires to assess your medical history and whether anyone in your family has experienced a mental illness. Next, you will be administered the Beck Depression Inventory to ensure that depression is not a confound. If you indicate you might harm yourself, confidentiality will be broken and appropriate agencies will be contacted. You may examine the questionnaires before signing this consent form.

You will be tested for tobacco intake with a Micro-Smokelyzer CO monitoring device (Bedford Scientific Ltd, Model 3A). If there is no evidence of recent tobacco (10 ppm or less), you can participate. If it is higher, we will need to reschedule you. Also we will assess your hearing level as it is important that you can hear the tone that will be presented to you. If you do not pass the latter, you will be given one hour's extra credit and dismissed.

Methods of smoke delivery for smokers: In a smoking area just outside Williams Hall, cigarette smokers will be tested after conditions of abstinence (overnight for 8-10 hours) and smoking. Smoking will require subjects to take inhalations on a lighted cigarette from your usual brand of cigarette which you will provide. The researcher will keep track of the timing of the puffs, type of cigarette and nicotine content. Immediately afterwards smokers will be tested again for tobacco intake with the CO monitoring device; this reading will be recorded for future analysis. Non-smokers exposed to the smoking area, but not asked to smoke.

EEG Study. You will be asked to put on an electrode cap which has electrodes permanently placed in the cap; the cap is like a swimming cap and may be slightly uncomfortable as it is attached to a harness that is fastened lightly around your chest to hold the cap in place. We will also place electrodes on your left and right ear lobes, and four near your eyes to measure eye movements. The auditory tones will be presented from a loudspeaker near your head. Your skin will be cleaned with a mildly abrasive cleanser and may cause slight discomfort. If you have skin allergies, we will only use alcohol as a cleanser. To insure your safety from infection, the
experimenter has thoroughly sanitized the electrodes and washed the electrode cap. The experimenter will wear clean rubber gloves while attaching the electrodes.

Following this, you will be asked to close your eyes for 60 seconds for an EEG recording, and then to open your eyes and attend to the auditory tones presented to you. This will be done twice. If you are a never-smoker, you will be tested twice and will NOT be administered any cigarettes. If you are a smoker, you will be tested twice: once when you have abstained from tobacco for overnight, and once when you have not. During the abstain condition, you will be given the tasks without smoking any cigarettes. During the smoking condition, you will smoke your own cigarette.

Afterwards, we will discuss your experiences and you will be able to see your recorded brain activity.

You are to report to the experimenter if you are on any medications or under doctor’s treatment. You are not to have ingested alcohol in the last 24 hours. You are to report any recent history of using tobacco, either smoked or chewed. You are to inform the experimenter of any skin reactions from lotions or anything else you have had in the past. You are to inform the experimenter of all medical and psychiatric problems that you have had that might interfere with the experiment.

Attention Tasks. After the completion of the EEG study, you will go to a different experimental room and be administered three computerized tasks. They are the Wisconsin Card Sorting Test, Posner's Attention Network Task, and the Tower of Hanoi. If you have done any of them in the past, we would like to be told.

3. ANONYMITY OF SUBJECTS AND CONFIDENTIALITY OF RESULTS:

The results of this study will be kept strictly confidential. At no time will the researchers release your results to anyone without your written consent. The information you provide will have your name removed and only a subject code will identify you during analyses and any write-up of the research. Should you report that you may harm yourself or others (on the Beck Depression Inventory). The researcher has the obligation to break confidentiality and report this information to the appropriate agency.

4. DISCOMFORTS AND RISKS FROM PARTICIPATING IN THE STUDY:

There are minimal risks to you from participation in this study. If you smoke a cigarette (smokers only), you may experience side effects from smoking include nausea, dizziness and hypotension. Since you are using your own regularly used cigarette brand and you are a smoker (at least 10 cigarettes per day), research shows there are negligible negative effects.

5. BENEFITS OF THIS PROJECT:

No personal benefit is promised you. Your participation in this project today will help advance the scientific knowledge of the interrelationships between your physiological reactivity to the auditory and prior measures taken. Comparisons will be made between smokers and non-smokers of varying personalities.

6. FREEDOM TO WITHDRAW:

You are free to withdraw from this study at any time without penalty.

7. COMPENSATION:

Participation will be totally voluntary. You will receive $15 (or one hour’ extra credit) per hour in this project regardless of whether or not you complete the experiment today. Please check your course syllabi for information as to worth of this extra credit and for alternative ways by which to receive extra credit.

8. USE OF RESEARCH DATA:
The information from this research may be used for scientific or educational purposes. It may be presented at scientific meetings and/or published and reproduced in professional journals or books, or used for any other purpose that Virginia Tech's Department of Psychology considers proper in the interest of education, knowledge, or research.

9. APPROVAL OF RESEARCH:
   This research project has been approved by the Human Subjects Committee of the Department of Psychology and by the Institutional Review Board of Virginia Tech. You will receive a copy of this consent form.

10. SUBJECT'S PERMISSION:
   I have read and understand the above description of the study. I have had an opportunity to ask questions and have had them all answered. I hereby acknowledge the above and give my voluntary consent for participation in this study. I further understand that if I participate I may withdraw at any time without penalty. I understand that should I have any questions regarding this research and its conduct, I should contact any of the persons named below:

Primary Researcher: Li Wan 239-6959
Supervisor, Helen J. Crawford, Ph.D. 231-6520
Chair, Human Subjects Committee, D. Harrison, Ph.D. 231-6581
Chair, Institutional Review Board, David M. Moore
   Asst. Vice Provost for Research Compliance 231-4991

SUBJECT'S SIGNATURE: _________________________________________
SUBJECT’S PHONE: _____________________________________________
DATE: _______________________________________________________

You will be given a copy of this consent form.
Appendix D: Schizotypy Personality Questionnaire (SPQ; Raine, 1991).

Please answer each item marking 0 for No and 1 for Yes on your opscan. Answer all items even if unsure of your answer. When you have finished, check over each one to make sure you have answered them.
1. Do you sometimes feel that things you see on the TV or read in the newspaper have a special meaning for you?
2. I sometimes avoid going to places where there will be many people because I will get anxious.
3. Have you had experiences with the supernatural?
4. Have you often mistaken objects or shadows for people, or noises for voices?
5. Other people see me as slightly eccentric (odd).
6. I have little interest in getting to know other people.
7. People sometimes find it hard to understand what I am saying.
8. People sometimes find me aloof and distant.
9. I am sure I am being talked about behind my back.
10. I am aware that people notice me when I go out for a meal or to see a film.
11. I get very nervous when I have to make polite conversation.
12. Do you believe in telepathy (mind-reading)?
13. Have you ever had the sense that some person or force is around you, even though you cannot see anyone?
14. People sometimes comment on my unusual mannerisms and habits.
15. I prefer to keep to myself.
16. I sometimes jump quickly from one topic to another when speaking.
17. I am poor at expressing my true feelings by the way I talk and look.
18. Do you often feel that other people have got it in for you?
19. Do some people drop hints about you or say things with a double meaning?
20. Do you ever get nervous when someone is walking behind you?
21. Are you sometimes sure that other people can tell what you are thinking?
22. When you look at a person or yourself in a mirror, have you ever seen the face change right before your eyes?
23. Sometimes other people think that I am a little strange.
24. I am mostly quiet when with other people.
25. I sometimes forget what I am trying to say.
26. I rarely laugh and smile.
27. Do you sometimes get concerned that friends or co-workers are not really loyal or trustworthy?
28. Have you ever noticed a common event or object that seemed to be a special sign for you?
29. I get anxious when meeting people for the first time.
30. Do you believe in clairvoyancy (psychic forces, fortune telling)?
31. I often hear a voice speaking my thoughts aloud.
32. Some people think that I am a very bizarre person.
33. I find it hard to be emotionally close to other people.
34. I often ramble on too much when speaking.
35. My "non-verbal" communication (smiling and nodding during a Y N conversation) is poor.
36. I feel I have to be on my guard even with friends.
37. Do you sometimes see special meanings in advertisements, shop windows, or in the way things are arranged around you?
38. Do you often feel nervous when you are in a group of unfamiliar people?
39. Can other people feel your feelings when they are not there?
40. Have you ever seen things invisible to other people?
41. Do you feel that there is no-one you are really close to outside of your immediate family, or people you can confide in or talk to about personal problems?
42. Some people find me a bit vague and elusive during a conversation.
43. I am poor at returning social courtesies and gestures.
44. Do you often pick up hidden threats or put-downs from what people say or do?
45. When shopping, do you get the feeling that other people are taking notice of you?
46. I feel very uncomfortable in social situations involving unfamiliar people.
47. Have you had experiences with astrology, seeing the future, UFOs, ESP or a sixth sense?
48. Do everyday things seem unusually large or small?
49. Writing letters to friends is more trouble than it is worth.
50. I sometimes use words in unusual ways.
51. I tend to avoid eye contact when conversing with others.
52. Have you found that it is best not to let other people know too much about you?
53. When you see people talking to each other, do you often wonder if they are talking about you?
54. I would feel very anxious if I had to give a speech in front of a large group of people.
55. Have you ever felt that you are communicating with another person telepathically (by mind-reading)?
56. Does your sense of smell sometimes become unusually strong?
57. I tend to keep in the background on social occasions.
58. Do you tend to wander off the topic when having a conversation?
59. I often feel that others have it in for me.
60. Do you sometimes feel that other people are watching you?
61. Do you ever suddenly feel distracted by distant sounds that you are not normally aware of?
62. I attach little importance to having close friends.
63. Do you sometimes feel that people are talking about you?
64. Are your thoughts sometimes so strong that you can almost hear them?
65. Do you often have to keep an eye out to stop people from taking advantage of you?
66. Do you feel that you are unable to get "close" to people?
67. I am an odd, unusual person.
68. I do not have an expressive and lively way of speaking.
69. I find it hard to communicate clearly what I want to say to people.
70. I have some eccentric (odd) habits.
71. I feel very uneasy talking to people I do not know well.
72. People occasionally comment that my conversation is confusing.
73. I tend to keep my feelings to myself.
74. People sometimes stare at me because of my odd appearance.
Appendix E: Personality Syndrome Questionnaire (PSQ; Gruzelier et al 2001a).

Please answer each item by choosing 0 for No and 1 for Yes. Answer all items, even if unsure of your answer. When you have finished, check over each item to make sure you have answered them all.

1. Some people say that I go into too much detail when I talk.
2. Sometimes things on the TV or radio have a hidden meaning for me.
3. I sometimes avoid going to places where there will be lots of people because I think I will get anxious.
4. I have never had an experience with the supernatural.
5. I never or rarely mistake objects or shadows for people, or noises for voices.
6. I frequently find my thoughts are racing.
7. I am never or rarely perceived as slightly eccentric.
8. I am enthusiastic about getting to know other people.
9. People sometimes find it hard to understand what I am saying.
10. People sometimes find me aloof and distant.
11. I am sure I am being talked about behind my back.
12. I nearly always express my thoughts clearly when I speak.
13. I am aware that people notice me when I go out for a meal or to see a film.
14. I never or rarely get nervous when I have to make polite conversation.
15. I believe in telepathy (mind reading).
16. I have never sensed some person or force around me when alone.
17. People sometimes comment on my unusual mannerisms or habits.
18. I prefer to keep to myself.
19. I never find thoughts fall away mid way through thinking or speaking.
20. I sometimes jump quickly from one subject to another when speaking.
21. I am generally good at expressing my feelings by the way I talk and look.
22. I never or rarely feel that other people have got it in for me.
23. I always keep to the point when speaking and never go off at tangents.
24. People rarely or never drop hints about me or say things with a double meaning.
25. I hardly ever get nervous when someone is walking behind me.
26. Sometimes every thought I have immediately suggests an enormous number of ideas.
27. I am sometimes sure that other people can tell what I am thinking.
28. When looking at a person or myself in the mirror, I have never seen the face change right before my eyes.
29. Sometimes other people think that I’m a little strange.
30. I rarely have trouble finding or using the right word to express what I want to say.
31. I am usually quiet when I am with other people.
32. When I catch a train I hardly ever arrive at the last minute.
33. It is rare for me to forget what I am trying to say.
34. I rarely laugh and smile.
35. I am sometimes concerned that friends or co-workers are not really loyal and trustworthy.
36. No-one has ever said that I express myself too elaborately for others to take in what I am saying.
37. Common objects or events have seemed to be special signs for me.
38. I don’t usually get anxious when meeting people for the first time.
39. I don’t believe in clairvoyance (fortune telling).
40. I often hear a voice speaking my thoughts aloud.
41. Things people say frequently don’t register.
42. I am often thought of as a very bizarre person.
43. I find it easy to be emotionally close to other people.
44. I dislike doing things in which I have to act quickly.
45. It is unusual for me to ramble on too much when speaking.
46. My “non-verbal” communication (smiling and nodding during conversation) is good.
47. I never feel I have to be on guard with friends.
48. I sometimes see special messages in advertisements, shop windows, or the way things are arranged around me.
49. I never or rarely feel nervous when with a group of unfamiliar people.
50. Other people can feel my feelings when they are not there.
51. I have never seen things that were invisible to other people.
52. I feel that there is no-one I am really close to outside my family, no-one I can confide in or talk to about personal problems.
53. Some people find me a bit vague and elusive during a conversation.
54. I often find that I can’t sit still.
55. I am good at returning social courtesies and gestures.
56. I never or rarely pick up hidden threats and put downs from what people say and do.
57. I sometimes find it difficult to put together what people are saying to understand their meaning.
58. When shopping I hardly ever get the feeling that other people are taking notice of me.
59. I feel very uncomfortable in social situations involving unfamiliar people.
60. I have never had experiences with astrology, seeing the future, UFO’s, ESP or a sixth sense.
61. I rarely take on more activities than I have time for.
62. Everyday things often seem unusually large or small.
63. It’s worth taking trouble to write letters to friends.
64. I sometimes use words in unusual ways.
65. I tend to avoid eye contact when conversing with others.
66. My words are sometimes so mixed up that they don’t make sense.
67. I have found that its best not to let other people know too much about me.
68. When I see other people talking to each other I often wonder if they are talking about me.
69. I have such a wide range of interests that I often don’t know what to do next.
70. I would feel very anxious if I had to give a speech in front of a large group of people.
71. I have never felt that I am communicating with another person telepathically (mind reading).
72. My sense of smell sometimes becomes unusually strong.
73. I tend to keep in the background on social occasions.
74. I sometimes find that I say one thing and mean just the opposite.
75. I don’t tend to wander off the topic when having a conversation.
76. I never or rarely feel that others have it in for me.
77. I sometimes feel that other people are watching me.
78. I sometimes feel speeded up.
79. I sometimes suddenly feel distracted by sounds I am not normally aware of.
80. I attach high importance to having close friends.
81. I sometimes feel that people are talking about me.
82. My thoughts are sometimes so strong that I can almost hear them.
83. I often have to keep an eye out to stop people taking advantage of me.
84. I feel that I cannot get close to people.

Please indicate which hand you habitually use for each of the following activities by choosing the appropriate number. "1"=Always Left, "2"=Usually Left, "3"=Left or Right Equally,"4"=Usually Right, "5"=Always Right
1. To write a letter legibly
2. To throw a ball to hit a target
3. To hold a racket in tennis, squash or badminton
4. To hold a match while striking it
5. To cut with scissors
6. To guide a thread through the eye of a needle (Or guide needle on to thread)
7. At the top of a broom while sweeping
8. At the top of a shovel when moving sand
9. To deal playing cards
10. To hammer a nail into wood
11. To hold a toothbrush while cleansing your teeth
12. To unscrew the lid of a jar

Family History: We would like to know if any of your first degree family members have been diagnosed with a major psychiatric disorder (e.g., schizophrenia, bipolar, depressed). Leave the questions blank if they have not; write in disorder if they have had one. You will participate in the experiment regardless of your family history, but we need to know this to better understand your responses.
1. I am adopted and I do not know.
2. Mother (blood relative)
3. Father (blood relative)
4. Sisters (blood relative)
5. Brothers (blood relative)
6. Uncles or aunts (blood relatives)
7. Your Children (blood relative)
Appendix H: Medical Screening Questionnaire (Crawford, unpublished).

1. Have you ever had a concussion where you were completely knocked out in 5 minutes or more?
2. Have you ever had any neurological or cardiovascular medical problems?
3. Have you ever been diagnosed formally as having attention deficit disorder?
4. Have you ever been diagnosed formally as having a learning disorder?
5. Do you presently have any hearing problem that have been formally diagnosed?
Appendix I: Smoking History Questionnaire (Crawford, & Wan, unpublished).

Below are some questions about your use of tobacco.
1. Have you ever smoked tobacco cigarettes? "0" = no, "1" Yes
2. Have you ever smoked cigars? "0" = no, "1" Yes
3. Have you ever smoked pipe? "0" = no, "1" Yes
4. Have you ever chewed tobacco? "0" = no, "1" Yes
5. Presently do you smoke tobacco cigarettes? "0" = no, "1" Yes, but not daily, "2" Yes, daily
6. Presently do you smoke cigars? "0" = no, "1" Yes, but not daily, "2" Yes, daily
7. Presently do you smoke pipe? "0" = no, "1" Yes, but not daily, "2" Yes, daily
8. Presently do you chew tobacco? "0" = no, "1" Yes, but not daily, "2" Yes, daily
9. If appropriate, list any other nicotine-containing substances you now use (e.g., gum, nicotine patch)
10. At what age did you begin smoking tobacco cigarettes? ___ years old. (Leave blank if you never did it or only tried a few times.)
11. At what age did you begin smoking cigars? ___ years old. (Leave blank if you never did it or only tried a few times.)
12. At what age did you begin smoking pipes? ___ years old. (Leave blank if you never did it or only tried a few times.)
13. At what age did you begin chewing tobacco? ___ years old. (Leave blank if you never did it or only tried a few times.)
14. Presently, on the average, how many cigarettes per day do you smoke? ___ cigarettes. (Leave blank if you do not smoke now.)
15. Presently, if you chew, on the average, how many chews per day do you have? ___ chews. (Leave blank if you do not chew now.)
Appendix J: Alcohol Usage Questionnaire (Crawford, unpublished).

We wish to determine if you drink alcohol or not, as it might affect your EEG. "1"=I did not drink alcohol during this time period, "2"=1-2 drinks, "3"=3-4 drinks, "4"=5-6 drinks, "5"=7-8 drinks, "6"=9-12 drinks, "7"=13-16 drinks, "8"=17 or more. (1 drink=12oz beer, 4oz wine, 1-1.5oz hard liquor)

1. Since September, 2003, on the average how many drinks do you have when you drink alcohol?
2. Over the past two weeks, on the average how many drinks per day do you have when you drink alcohol?

"1"=I did not drink alcohol during this time period, "2"=1-2 occasions, "3"=3-4 occasions, "4"=5-6 occasions, "5"=7-8 occasions, "6"=9-12 occasions, "7"=13-16 occasions, "8"=17 or more, "9"=I drank alcohol, but always less than five drinks at one sitting.
3. On how many occasions over the last two weeks did you consume five or more drinks at one sitting?
### Table 1 Compare S1 and S2 for each site in each condition

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