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**CENTRAL AUDITORY PROCESSING IN SCHOOL-AGE CHILDREN
PRENATALLY EXPOSED TO CIGARETTE SMOKE**

by Joel S. McCartney

Department of Psychology

**A Thesis submitted in fulfillment of the requirements for the Degree of Master of
Arts**

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Central Auditory Processing in School-Age Children Prenatally Exposed
to Cigarette Smoke

submitted by Joel S. McCartney, B.A.

in partial fulfillment of the requirements for
the degree of Master of Arts

Thesis Supervisor

Chair, Department of

Carleton University

May 7, 1993

ABSTRACT

Previous research has revealed that negative effects are associated with prenatal maternal cigarette use. These effects can be both physical and behavioral. In addition, language skill and auditory anomalies have also been related to prenatal exposure to cigarette smoke. These physical and behavioral decrements seem to be long-term.

This study concentrated on one aspect of behavior - central auditory processing (CAP) abilities of children exposed to cigarette smoke in utero. A Screening Test for Auditory Processing Disorders (SCAN) was utilized to examine CAP in a relatively large sample size. SCAN was specifically designed to test children for CAP problems. A male voice presented taped, monosyllable words under three listening conditions: 1) words were low passed filtered at 1000 Hz with filter roll-off of 32 db/octave, 2) words presented with multi-talker speech babble noise, and 3) different words presented to right and left ears simultaneously. The child's task was to repeat the words.

In addition to the SCAN, a variety of prenatal variables, the child's home environment, the mother's personality, prenatal noise exposure, prenatal and postnatal passive smoke exposure and the auditory history of the mother, father and child was considered. In order to rule out other factors apart from maternal cigarette use, the above list of variables were analyzed as potential covariates.

In all three subtests and the SCAN Composite score

(i.e., total of all three subtests) a linear trend was noted between maternal cigarette use and performance on SCAN by the offspring. Univariate (ANOVA) results indicated that a group of children prenatally exposed to heavy amounts of cigarette smoke through the active smoking habits of their mothers' (≥ 16 mg/day of nicotine) scored statistically significantly lower on the Competing Words subtest (different words presented to left and right ears simultaneously) of the SCAN and the SCAN Composite score than the other two maternal smoking groups (i.e., light smokers and controls). This relationship between prenatal cigarette smoke exposure and central auditory processing remained after potential covariates were controlled for in ANCOVA.

At the multivariate level (MANOVA), children's performance on Auditory Figure Ground and Competing Words subtests (Filtered Words was not included in the linear combination of SCAN subtests because it interacted with several covariates) was observed to be significantly, negatively related to maternal cigarette use. After controlling for potential covariates, including the parent's averaged education, prenatal noise exposure and prenatal passive cigarette smoke exposure, the significant relationship of the linear combination of Auditory Figure Ground and Competing Words with maternal cigarette use did not hold.

Passive smoke results indicated that prenatal passive cigarette smoke exposure is negatively related to central auditory processing. Findings suggest that prenatal passive

exposure is more important than postnatal passive exposure in affecting auditory processing. Prenatal passive smoke exposure effects appear to be similar to maternal prenatal exposure to light levels (i.e., less than a pack of cigarettes per day) of cigarette smoke. Potential covariates were not controlled for in the passive smoke analyses due to a lack of relevant data (e.g., home air quality, metabolic rating of each child, etc.) therefore, caution is advised in interpreting the results.

The results of this study imply the importance of considering the risk to the fetus of both smoking and being in the company of smokers during pregnancy. Though subtle, the central auditory processing effects registered in this study can lead to future difficulties in a child's daily life (e.g., academic problems, attention problems) (Keith, 1986). This study has revealed yet another effect that prenatal cigarette smoke exposure may have on the developing CNS, adding to the number of risks already recognized with smoking during pregnancy.

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I INTRODUCTION

General Objective

The Ottawa Prenatal Prospective Study (OPPS) has attempted to discover the effects of prenatal exposure to cigarettes, marijuana and non-alcoholic levels of alcohol. These substances are considered to be several of the most often used non-medicinal drugs by pregnant women and numerous studies have revealed their physical and behavioral teratogenic effects in children at a variety of developmental stages (Fried and Makin, 1987).

The present study represents an adjunct to the OPPS. This research will focus on the prenatal effects of cigarette smoke in children in terms of central auditory processing anomalies. Children prenatally exposed to cigarette smoke have shown a variety of resulting physical and behavioral effects ranging from low birth weight to attention problems. Research into whether the auditory system is affected by prenatal exposure to cigarette smoke in six- to eleven-year-olds has not been undertaken until now.

Central auditory processing (CAP) represents one area of the Central Nervous System (CNS). The CNS is considered to be at risk when exposed to cigarette smoke in utero (e.g. in utero exposure to smoke possibly interferes with CNS development) (Mactutus and Fechter, 1986). Based on a number of reports to be discussed later, aspects of central auditory functioning appear to be vulnerable to prenatal smoke exposure. The aspects of auditory processing that this

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investigation will focus on include 1) resistance to distortion in the acoustic environment, 2) listening skills in noise and 3) children's CAP maturation or development. These areas of CAP seem to be the processes most often affected when a child experiences CAP difficulties (Keith, 1986).

Smoking During Pregnancy: Prevalence

Most North Americans have smoked cigarettes at some point in their lives. Cigarette smoking is of great concern because of its recognized addictive qualities and negative effects on health and because use is often initiated during adolescence (National Household Survey on Drug Abuse: Main Findings, 1990).

In 1990, over 73% of Americans were reported to have smoked cigarettes during their lifetime. Approximately 79% of males and 68% of females in the United States had either smoked in their lives, in the past year and/or in the past month. Of the 68% of females who had ever smoked, 36.3% of 12- to 17-year-olds reported cigarette use in their lifetime, 65% of 18- to 25-year-olds reported cigarette use in their lifetime, 79% of 26- to 34-year-olds reported smoking cigarettes in their lifetime and 70% of those who were 35-years-old or older reported the above as well (National Household Survey on Drug Abuse: Main Findings, 1990).

Importantly, for the purpose of this thesis, 24.2% of American females, 12-years-old or older, reported using cigarettes during the past month. They were considered

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current smokers. Eleven percent of 12- to 17-year-olds, 27.1% of 18- to 25-year-olds, 35.2% of 26- to 34-year-olds and 22% of those aged 35 years or older reported cigarette use in the past month (National Household Survey on Drug Abuse: Main Findings, 1990). It is clear, then, that a significant number of females smoke during the most critical reproductive years (i.e. 18- to 35-years-old).

The prevalence of cigarette use in pregnant North American women is estimated to be 25% (Fried and Makin, 1987). In a Swedish study (Cnattingius, 1989), 2051 pregnant women were interviewed concerning their smoking habits. Six hundred and seventy-three were smoking at the time of conception and only 150 stopped smoking in early pregnancy. Women who started to smoke before 17 years of age were less likely to quit in early pregnancy than women who started to smoke later in life. This sample showed that approximately 25% of pregnant women in Sweden smoke which is similar to the North American finding.

Negative Effects of Prenatal Cigarette Use

When offspring (human and animal) exposed to cigarette smoke in utero are examined, they are more likely to exhibit negative physical and behavioral effects (Abel, 1980). This trend, in terms of prenatal smoke exposure effects, has been supported throughout the literature to a significant degree.

Physical

Among the variety of negative effects that maternal cigarette smoking has been linked to, low birth weight is the

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most predominant. Simpson (1957) was the first investigator to observe this effect. In support of this earlier finding, later research observed similar trends (e.g., Frazier et al., 1961; Kullander & Kallen, 1971; Hardy & Mellits, 1972; Cope et al., 1973; Abel, 1980; Chattingius, 1988). Based on the findings of a number of studies, on the average, smokers' babies weigh approximately 200 grams less than nonsmokers' babies. Investigations have shown this finding to be consistent across a variety of populations. The offspring of smokers are considered "small for date" rather than premature, and have experienced some degree of intrauterine growth retardation. The decrement in birth weight has also been related to the number of cigarettes smoked (i.e. a dose-response relationship) (Longo, 1982). As well, vulnerability of the fetus appears to vary across phases of pregnancy. In a more recent study, Fried and O'Connell (1987) found that in the first and third (more so) trimester maternal use of cigarettes significantly decreased birth weight.

Other negative teratogenic effects of prenatal cigarette use have been described in the literature. Prenatal cigarette use has been related to an increased risk for perinatal mortality (Andrews et al., 1972, Butler & Goldstein, 1972). In an investigation by Meyer et al. (1975), maternal smoking was found to have a highly significant, dose-related, and independent effect on perinatal mortality. Single birth data in 10 hospitals across Ontario in 1960-61 (N = 51 490) was

examined, and the influence of other risk variables, such as age, parity and previous pregnancy history was statistically controlled. The risk of perinatal mortality was found to rise by 20% for smokers of less than one pack per day. The offspring of heavy smokers (one pack or more per day) showed an increased risk of 35%.

In addition to the above, it has been postulated that the prevalence of spontaneous abortions is two times more likely for smokers (Abel, 1980, Longo, 1982). Finally, there is some evidence in the literature that sudden infant death syndrome (SIDS) may be associated with in utero exposure to cigarette smoke (Malloy et al., 1988). These are some of the most often cited negative effects of maternal cigarette smoking during pregnancy.

Behavioral

Research findings into the behavioral effects of prenatal cigarette exposure in children have, with a few exceptions, been fairly consistent. They reveal that in utero exposure to cigarette smoke is related to significant decrements in infant and child behavioral development. A variety of negative behavioral consequences have been observed over the past two decades.

Infant behavioral effects have been noted by various researchers. Prenatal cigarette smoke exposure has been associated with increased central nervous system excitation (Butler & Goldstein, 1973, Fried, 1989). Fried et al. (1987) examined 9- and 30-day-old infants prenatally exposed to

cigarette smoke. The sample studied was found to be representative of a low-risk, middle-class population. In utero cigarette exposure was found to be associated with hypertonicity and a greater incidence of fine tremors and startles.

Older children have also been studied in terms of prenatal cigarette smoke exposure effects. Fried and Watkinson (1990) examined aspects of neurobehavioral development in three- and four-year-old children. The children were administered the Reynell Developmental Language Scales (assessing language comprehension and expression) and the McCarthy Scales of Children's Abilities (assessing verbal, perceptual, quantitative, general cognitive, memory and motor abilities). The researchers observed that children who had been prenatally exposed to cigarette smoke were more likely to evidence poorer language development and lower cognitive scores.

A study by Streissguth et al. (1984) utilized a continuous performance task (CPT) considered to be a measure of sustained attention. The researchers found that four-year-old children who had been exposed to cigarette smoke in utero showed increased errors of commission and omission. As well, a dose response effect was noted and the children spent significantly less time oriented to the task. The findings of Kristjansson et al. (1989) who also studied vigilance performance, support the observations of Streissguth et al. (1984). The sample was composed of four- to seven-year-old

children exposed prenatally to cigarette smoke. A CPT was utilized and errors of commission and omission were recorded. The investigators concluded that children exposed to cigarette smoke in utero were more likely to exhibit impulsivity and poor attention based on the CPT performance of the children.

Similar to the above, several other studies have focused on the effects of prenatal cigarette smoke exposure in school-aged children. These studies have generally found in utero exposure to cigarette smoke to be related to a lowered level of academic performance, poor school adjustment, behavior reflective of hyperactivity and learning difficulties (Davie et al., 1972, Nichols & Chen, 1981, Naeye & Peters, 1984).

A study by Dunn et al. (1977) examined 168 children prenatally exposed to cigarette smoke and 151 controls. Children born to 'smoking mothers' scored lower on psychological tests when compared to a control group. Also, a greater proportion of misbehavior variables were reported by the children's teachers on a behavior rating scale (i.e., Haggerty-Olsen-Wickman Behavior Rating Schedule). In another early study (Denson et al., 1975), researchers found mothers of hyperkinetic children to smoke three times as much as a control group of mothers.

In a more recent study, Sexton et al. (1990) investigated the effects of prenatal cigarette exposure on cognition. The researchers recorded the habits (e.g., smoking, alcohol,

caffeine, pregnancy history, SES, etc.) of 366 pregnant women. At three years of age the children were assessed by the Minnesota Child Development Inventory and the General Cognitive Index of the McCarthy. The Minnesota Child Development Inventory reveals the general development status of a child. It contains four subscales: expressive language, comprehension-conceptual, fine motor development and self-help. It includes descriptions of problem behaviors and symptoms such as immaturity, hyperactivity and language problems and is completed by the mother. The General Cognitive Index possesses verbal, perceptual-performance and quantitative subscales along with measurements for memory and motor skills. The experimenters found the General Cognitive Index and the Minnesota Child Development Inventory scores of the nonsmokers to significantly differ from the scores of the smoking group. Based on the above assessments, children whose mothers quit smoking (before or in the early stages of pregnancy) performed at a significantly higher level than those whose mothers continued to smoke during pregnancy.

In their study of 126 six-year-old children, Fried et al. (1992 I) observed a dose-response association between prenatal cigarette exposure and impulsive behavior. In addition, children's performance on a series of memory tasks was also negatively associated with prenatal cigarette exposure. In a related investigation of five- and six-year-old children (Fried et al., 1992 II), results indicated an association between prenatal cigarette exposure

and lower cognitive and receptive language scores.

In an inquiry by Makin et al. (1991) 91 children between the ages of six and nine years were tested using a comprehensive neuropsychological test battery. Children who had been exposed to cigarette smoke in utero showed lower scores on tests of speech and language skills, intelligence, visual/spatial abilities and on the mother's rating of behavior when compared to nonsmoke exposure and passive smoke exposure (secondary smoke) groups.

Research has also examined the long-term effects of in utero exposure to cigarette smoke. A widely cited prospective investigation by Butler and Goldstein (1973) discovered that at age 11, children prenatally exposed to cigarette smoke were between three to five months behind normal reading levels, mathematics and general ability. In a later longitudinal investigation, Rantakallio (1983) studied 1 819 children of women who smoked during pregnancy and their matched controls (non-smokers). The controls were matched for parity, marital status, maternal age, place of residence and single or multiple birth. At the age of 14, children of smokers evidenced poorer academic performance on school report cards. These findings remained significant even after adjusting for mother's height and age, social class, number of siblings and sex of the child.

Prenatal Cigarette Smoke Exposure and Aspects of Auditory Functioning

Auditory anomalies have been a relatively recent area of

assessment in terms of in utero cigarette smoke exposure. Consequently, a limited number of studies have observed auditory decrements in addition to the above findings.

In a relatively early study by Saxton (1978), the Brazelton Neonatal Behavioral Assessment Scale (BNBAS) was used to compare the behavior of infants whose mothers smoked heavily during pregnancy with a group of controls matched for maternal age, social class and parity. The BNBAS was designed to assess an infant's capacity to interact with the environment and gage the integrity of the CNS (Brazelton, 1973). When tested at four- to six-days-of-age, there were no significant differences between the groups on the overall score. However, there were items that differentiated the groups. These were tests of auditory habituation, responsiveness to a human voice and bell, and consolability.

Picone et al. (1982) examined the effects of diet, weight gain, cigarette smoking and stress on the pregnancy outcomes of 60 women. Based on the BNBAS, maternal smoking was associated with impaired habituation, orientation and regulation of state. More specifically, cigarette smoking adversely affected auditory habituation and orientation and autonomic regulation. A decrease in the ability to orient to the source of a sound was clearly noted. Interestingly, no recovery in auditory responses was observed at two weeks after birth. In a similar study, Jacobson et al. (1984) found a relation between smoking and poor auditory orientation. However, their results only approached significance.

Fried and Makin (1987) examined infant neonatal behavior and its relation to maternal marijuana, cigarette and moderate alcohol use during pregnancy. Two hundred and fifty babies born to healthy mothers were examined three and six days after birth. The BNBAS was administered to the infants on both days. Prenatal nicotine was associated with less irritability, more tremors, less responsiveness to light and sound and poorer auditory habituation. The investigators pointed out that habituation of various responses to repeated stimuli is widely accepted as an indication of nervous system integrity.

In another study (Fried & Watkinson, 1988), motor, mental and language development and home environment were examined in 12- and 24-month-old children prenatally exposed to marijuana, alcohol and cigarette smoke. Infant behavior was assessed in the home using the Bayley Scales of Infant Development (BSID), Reynell Developmental Language Scales (RDLS) (for 24-month-olds) and the Home Observation for Measurement of the Environment (HOME). Important subtests of the of the BSID include Mental Development Index (MDI) (concerning sensory perceptual abilities, early acquisition of object constancy, memory, problem solving, vocalization and the onset of words), Psychomotor Development Index (PDI) (concerning gross and fine motor movement) and the Infant Behavior Record (IBR) (infant attitudes, interests and temperament).

In terms of the IBR, 12- and 24-month-old composite

auditory clusters were not related to either maternal marijuana or alcohol use but were negatively associated with maternal cigarette use. In addition, prenatal cigarette smoke was significantly related to lowered MDI scores.

Kristjansson et al. (1989) found aspects of attentional behavior in four- to seven-year-olds, assessed by auditory and visual vigilance tasks, to be related to prenatal exposure to cigarette smoke. In the auditory task a stimuli was presented to children using a tape-recorder. Children were asked to indicate the presence of the critical stimulus ('house') by tapping a pencil. Eight non-critical stimuli either began with 'h' or rhymed with 'house'. Errors of omission occurred when children failed to respond to the critical stimulus. Errors of commission occurred when children responded to non-critical stimuli. The experimenters found prenatal cigarette use to be significantly related to errors of auditory commission. Errors of visual commission only approached significance. Thus, a differential effect of prenatal cigarette smoke exposure seems to take place in terms of the auditory and visual systems of the CNS. Errors of omission and commission were also related to impulsivity and poor attention.

The above findings constitute the existing research pertaining to auditory anomalies associated with in utero exposure to cigarette smoke. Such studies, though relatively limited in scope, have been consistent in terms of overall results (i.e., maternal smoking during pregnancy is related

to auditory decrements in offspring). However, additional research involving a more in depth analysis is warranted. As well, due to the focus of the cited studies (i.e. research has concentrated on young children and infants), it seems imperative to examine children at later stages in life to see if negative auditory effects are still evident.

In contrast to the general findings, some studies have failed to relate prenatal cigarette smoke to any negative outcomes. Leftkowitz (1981) examined the long-term effects on offspring prenatally subjected to smoke. The study compared the offspring of smokers and non-smokers when the children were 10-years-old. Following tests on physical, intellectual, affective and social functioning, no significant differences were observed. Higher mean scores were actually found for smokers' children on physical growth and intellectual measures. It is important to note that this is the only study to have found such results. A major criticism evident with this report is that no description is given of how the sample was obtained, other than it consisted of 241 children that were involved in a larger investigation on childhood depression. It is possible that the sample could have differed in some significant way from the others.

Similar to the above results, a longitudinal study by Streissguth et al. (1989) failed to find any significant behavioral decrements after examining 421 children exposed to cigarette smoke in utero. However, the non-significant results regarding prenatal cigarette smoke should be

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interpreted with caution as the smoking sample in this study contained a small number of heavy smokers.

Animal Studies

Research with animals has consistently found low birth weight to be related to maternal cigarette smoke (Haworth & Ford, 1972). Slotkin et al. (1986) found maternal nicotine injections to be significantly related to a 25% weight deficit in newborn rats. Brain weights showed a similar reduction. Also, a significant number of rats did not give birth. However, both body and brain weights were essentially the same as the controls by the second postnatal week.

Negative behavioral effects of maternal cigarette smoke have been noted. Mactutus and Fechter (1984) examined the behavioral effects of prenatal exposure to CO in rats. Mild exposure levels were used (150 parts/million). In terms of explicit effects, CO exposure resulted in only minor reductions in birth weights of the rat pups but produced no overt evidence of teratogenesis. However, behavioral evaluation of learning and memory processes in a two-way avoidance task suggested a functional deficit in the central nervous system of the exposed offspring. The researchers related that animal models have revealed that after maternal exposure, CO readily crosses the placenta and decreases fetal O₂. Therefore, they postulated that chronic CO exposure restricted to the prenatal period disrupts the acquisition and retention of a conditioned avoidance response in juvenile rats.

Clearly, there seems to be an assortment of negative behavioral effects related to prenatal cigarette smoke exposure. With a few exceptions, most studies have supported this trend. Thus, there is some evidence that cigarette smoke acts as a behavioral teratogen, related to small but significant neurobehavioral deficits. In addition, central auditory anomalies are among these deficits.

How Can Smoke Affect the Fetus?: Mechanisms and Processes

The primary potentially harmful components of cigarette smoke are considered to be carbon monoxide (CO) (gas phase component) and nicotine (particulate phase component) (Mangan & Golding, 1984). It is hypothesized that these two components adversely affect the development of the CNS.

NICOTINE Nicotine (considered to be the most harmful component of cigarette smoke) is absorbed into the bloodstream through the lungs very rapidly (inhaled tobacco smoke). From the lungs, it travels to the left side of the heart on to the brain and other parts of the body (e.g. fetus). Nicotine becomes converted into two main metabolites: cotinine and nicotine-1'-N-oxide. Cotinine production occurs in the liver, kidneys and lungs. The amount of nicotine-1'-N-oxide excreted in the urine of smokers is about half that of cotinine. The largest proportion of nicotine and its metabolites is eliminated from the body via urine (Mangan & Golding, 1984).

Mosier and Jansons (1972) studied the distribution of nicotine and its metabolites in the rat fetus. Nicotine,

cotinine and other metabolites appeared in the rat fetus within five minutes of IV injection of a tracer amount of labelled nicotine into the mother. Concentration of radioactivity in the fetal plasma exceeded that of maternal plasma from 30 minutes throughout 20 hours of observation. Nicotine readily crossed the placenta and fetal tissues were perfused with higher concentrations of nicotine than maternal tissues within 30 minutes after its entry into the maternal circulation.

Similar to the above study, Slotkin et al. (1986) examined the effects of maternal nicotine injections (3mg/kg, 2/day) on brain development in the rat. The researchers were interested in ornithine decarboxylase (ODC) activity. ODC and the polyamines are known to be major regulators of macromolecule synthesis during replication and differentiation. Each tissue or brain region possesses a characteristic maturational pattern of ODC and perturbation of this pattern typically results in subsequent abnormalities of cell number, size and organization. Based on the results of the study, ODC activity in the nicotine group showed greater abnormalities for fetal brain than for the rest of the organism. Therefore, "brain-sparing" (i.e., effects on nervous system growth are substantially less than those on the rest of the organism) does not seem to take place. The pattern of prolonged elevation of ODC activity is known to be associated with an overall delay in the course of cellular maturation. Profound disturbances of brain maturation (least

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disturbance in midbrain, moderate in cerebral cortex and greatest in cerebellum) were observed in postnatal life.

In a related study by Navarro et al. (1989 I), researchers directly examined the effect of prenatal nicotine on the developing nervous system in rats. Low doses (i.e. relative to the average dose of nicotine used to bring about noticeable affects in rat research) of 2mg of nicotine/kg were administered in order to assess whether an association between prenatal nicotine exposure and central nervous system development did exist without affecting intrauterine and postnatal growth. The investigators observed that all exposed offspring were of normal size. However, the nicotine infusions were found to be related to alterations in [³H] nicotine binding sites, abnormalities of cellular development (assessed by measurements of ornithine decarboxylase activity and deoxyribonucleic acid) and impairment of the development of peripheral noradrenergic projections. After making the appropriate comparisons, neural alterations were virtually equivalent to those obtained previously at higher, growth-suppressing doses. It is important to note, then, that growth impairment alone is insufficient to predict the adverse effects of nicotine on development. As well, adverse nicotine effects can exist regardless of whether growth retardation has occurred.

Another process that maternal nicotine use seems to have an effect on is human intervillous blood flow (HIBF). Lehtovirta and Forss (1978) assessed the acute effect of

smoking on HIBF in 12 healthy women (35 to 40 weeks pregnant, mean age: 25.6 years, 19 to 31). In the experiment, subjects smoked a standard cigarette, inhaling for five minutes. In general, IBF decreased immediately after smoking but returned to presmoking levels within 15 minutes. More importantly, one cigarette caused a decrease in IBF of human placenta in near-term pregnancy. The investigators argued that it was the sympathomimetic effect of nicotine that caused the vasoconstriction of uterine vessels and hence a decrease in IBF. This decrease in IBF, of course, effects blood flow to the fetus.

More recent studies have discovered nicotinic receptor sites in the human fetal brain similar to the adult but at a lower density (Perry et al., 1986, Cairns et al., 1988). The density of these putative nicotinic receptor sites increases with gestational age. Nicotine mimics the role of ACh in the brain by activating these cholinergic nicotinic receptor sites.

Cairns et al. (1988), in addition to making the above observations, postulated that prenatal cigarette exposure may increase the density of nicotinic receptor sites in the fetal brain above and beyond that which is found with natural maturation. This increase due to prenatal nicotine exposure could be a result of the desensitizing properties of nicotine. A study by Hagino and Lee (1985) reported that administration of nicotine to pregnant rats resulted in increased numbers of nicotine binding sites in fetal brains.

However, Wonnacott (1990) points out that research in this area is not unequivocal. In her review of the literature, it seems that, though chronic exposure to nicotine (agonist) paradoxically elicits an increase in numbers of nicotine binding sites (i.e. chronic exposure to an agonist normally results in a decrease in target binding sites [downregulation]), it is not clear whether it produces enhanced, diminished or unchanged responsiveness. A variation of results has been noted across studies.

In an animal study, researchers found that nicotine, acting on its binding sites, suppresses CNS cell replication during critical developmental periods resulting in deficits in the development of central cholinergic pathways and nerve cells (Zahalka et al., 1992; McFarland et al., 1991). Regardless of the equivocal findings in terms of the consequences of nicotine-induced receptor upregulation, it does appear that prenatal nicotine exposure affects the development of central cholinergic systems (Navarro et al., 1989 II).

In addition, cholinergic nicotinic receptor binding sites have been found in outer hair cells of the organ of Corti located in the cochlea (Eybalin and Pujol, 1987). This area represents the 'starting point' of the central auditory system. There are two types of hair cells located in the organ of Corti - inner hair cells (IHC) and outer hair cells (OHC). Both types of hair cells are located on the basilar membrane which stretches the length of the cochlea positioned

throughout the cochlea's midsection (Pickles, 1988). Since the majority of afferent auditory nerve fibers make their synaptic contacts with IHC's, it is suggested that the function of the IHC is to signal the movement of the basilar membrane (e.g. hearing sensitivity).

Sound causes a progressive travelling wave to pass down the basilar membrane of the cochlea. The travelling wave has a sharply tuned peak which is maintained throughout the remainder of the nervous system (Pickles, 1988). The ability of the basilar membrane to become sharply peaked may be due to outer hair cells generating movements in response to auditory stimulation. Therefore, one function of the OHC's appears to be to 'fine tune' the auditory stimulus.

Unlike the visual system that seems to use a variety of neurotransmitters for transduction (Watling, 1981), an important part of the auditory system specifically utilizes ACh - the olivocochlear bundle. This centrifugal pathway sends axons to inner and outer hair cells from the superior olive. The olivocochlear bundle sends small unmyelinated axons from the lateral superior olivary nucleus to the IHC's while OHC's receive larger myelinated axons from the medial superior olivary nucleus. It is hypothesized that stimulation of the olivocochlear bundle interferes with the ability of the OHC's to increase the amplitude and sharpness of tuning of the basilar membrane (Pickles, 1988).

Based on the above evidence, in utero nicotine exposure may affect the normal development of the central auditory

system by interfering with natural central cholinergic cell proliferation. The olivocochlear bundle and OHCs appear to be the most likely areas of the central auditory system to be affected since they are normally driven by ACh.

CARBON MONOXIDE CO from inhaled cigarette smoke effectively reduces the oxygen (O_2) carrying capacity of blood by as much as 15% by combining with hemoglobin to form carboxyhemoglobin (COHb) (Mactutus & Fechter, 1986). Similar findings have been noted earlier (Cole et al., 1972). Hemoglobin has a greater affinity to CO than O_2 and, consequently, reduces the O_2 supply to the fetus due to the mother's O_2 deficient blood. Human fetal blood has a greater concentration of hemoglobin than does maternal blood (therefore a greater O_2 capacity). Since CO binds to hemoglobin more tightly than O_2 by a factor of 210 to 250, the concentration of CO in the bloodstream is greater in the fetus than in the mother (i.e. greater carboxyhemoglobin). As well, washout time for CO from the fetal compartment is significantly longer than is the case for the adult (Mactutus & Fechter, 1986). Mactutus and Fechter (1986) postulated, based on past findings (e.g. Socol et al. 1982), that hypoxia (resulting from increased CO blood concentrations) may be implicated in causing delays/obstructions in behavioral development. Early behavioral development may be adversely affected by conditions that produce either prenatal or neonatal oxygen deprivation.

Ashwal et al. (1981) observed patterns of fetal lamb

regional cerebral blood flow (CBF) to 34 brain regions during and after prolonged hypoxia. Significant fetal regional CBF differences occurred in utero, with brain stem and subcortical flows being greater than flows to other regions of the brain. During prolonged intrauterine hypoxia, total CBF increased about 95%, with evidence of preferential shunting to critical brain stem regions. However, during the posthypoxic recovery period at four, 24 and 48 hours, flows returned to normal, indicating no evidence of a post-ischemic hyperfusion syndrome or impairment of the microcirculation. Nevertheless, hypoxia does adversely effect blood flow to the brain, interrupting its natural pattern.

With reference to the auditory system, the stria vascularis, located in the scala media (one of the three fluid filled canals of the cochlea), would seem to be highly vulnerable to hypoxia (Pickles, 1988). The stria vascularis is an area of high metabolic activity (e.g. 12 times more active than any other cochlear structure). Endolymph and the endocochlear potential (thought to be directly dependent on energy consuming, ion pumping processes in the stria vascularis) are produced by the stria vascularis. The organ of Corti (hair cells located here) is in the scala media, consequently, hair cells are surrounded by endolymph. Stimulation of the stereocilia allows K^+ ions to flow in from the surrounding endolymph driven by the battery of the positive endolymphatic potential and the negative intracellular potential. Intracellular depolarization results

in the release of transmitter causing auditory nerve fibre activation.

Hypoxia causes the endocochlear potential to decay to zero in one to two minutes. Following several minutes, the positive endocochlear potential is replaced by a negative potential that can take several hours to disappear (Pickles, 1988). This could affect normal intracellular depolarization resulting in an abnormal transduction process between hair cells and auditory nerve fibres. To a certain extent, this process could occur in the cochlea of a developing fetus through cigarette smoke exposure, causing reduced oxygen blood levels (due to CO) leading to varying levels of hypoxia.

In summary, the above mechanisms and processes are regarded as having a negative influence on the natural development of the CNS. In turn, this interference conceivably increases the likelihood of behavioral and auditory anomalies in infancy and childhood.

Central Auditory Processing (CAP)

Anatomy

The anatomy of the auditory system is quite complex and is not yet completely understood in terms of its central components. Due to the fact that this investigation does not entirely focus on the components of the auditory system, this study will not attempt to describe the central auditory system's parts and pathways in great detail.

Nerve fibers from the cochlea first synapse in the

cochlear nucleus. From this point, they extend to the superior olivary nucleus then to the inferior colliculus of the midbrain. This all occurs before reaching the medial geniculate nucleus of the thalamus. From the thalamus, nerve fibers extend to the primary auditory receiving area in the temporal lobe of the cortex (Auditory Cortex). In addition to the pathway from the cochlea to the cortex, there are also connections between nuclei on the opposite sides (e.g. cochlear nucleus of right ear sends axons to the superior olivary nucleus and the inferior colliculus of the left ear, and the left and right inferior colliculi communicate with each other) (Goldstein, 1989).

Auditory problems may include hearing loss or tinnitus, to more complex problems such as poor speech discrimination and sound localization, hyperacusis, auditory misperceptions and hallucinations. Sensory neural involvement may be peripheral (inner ear or auditory nerve) or central as the nerve passes through the brain stem, cerebellum, thalamus and cortex (Weisberg & Katz, 1978).

Processing System

The system involved in the processing of the speech signal is even less well understood than the anatomy of the auditory system. The following theory seems to be the most widely held. Following the initial transformation of the speech signal by the peripheral auditory system, acoustic information about spectral structure, fundamental frequency, changes in source function, overall intensity and duration of

the signal, as well as amplitudes onsets and offsets is extracted and coded by the auditory system. These spectral and temporal patterns of the speech signal are assumed to be preserved in sensory memory for a brief period of time during which acoustic feature analysis is carried out. As a result, "speech cues" are provided, described as auditory based representations of the speech signal, subsequently used for phonetic classification and recall if necessary (Pisoni & Luce, 1987).

CAP involves the ability to attend to, discriminate, recognize, or comprehend information presented auditorily. An auditory processing disorder would involve anything that interfered with this ability when the individual possesses normal intelligence and hearing sensitivity. In a normal child, auditory processing proficiency develops in a parallel relationship with language abilities (Keith, 1986).

A Screening Test for Auditory Processing Disorders (SCAN)

SCAN utilizes three imitative tasks, requiring only that the child repeat simple stimulus words immediately after they are presented. Each of the SCAN subtests was selected to describe different auditory processing abilities. The three subtests include Filtered Words, Auditory Figure Ground and Competing Words.

Filtered word tasks are frequently used to identify children with auditory processing difficulties. Research using such tasks indicates that children with CAP disorders have difficulty understanding distorted speech. Auditory

figure ground tasks attempt to check another element of the auditory system - the ability to understand speech in the presence of background noise. Children with CAP difficulties are unable to completely comprehend auditory input under unfavorable listening conditions. Poor performance on such tasks may also indicate a delay in development of the central auditory system. The third subtest, Competing Words, pertains to the ability to understand competing speech signals. It may be used to determine maturation of the auditory system and CAP abilities. Below-average performance may indicate a developmental delay in the maturation of this system (Keith, 1986).

SCAN was standardized by speech/language clinicians who administered the tests to a sample of 1 034 American children between the ages of 3 and 11. All children tested were normally achieving students in regular preschool through fifth grade classrooms. All were verified as having normal peripheral hearing through Pure Tone evaluation and tympanometry (Keith, 1986).

SCAN has been shown to be quite reliable (Keith, 1986). A test-retest reliability check was performed on SCAN with a subset of the original sample used in standardization. Children's performance in the standardization procedure was compared to the performance of the same children six months later. The sample represented grades one to three. If the test is well designed and if the trait being measured is stable, then the relative standing of a student from one

testing to another testing should not change substantially and the correlation between the scores obtained on the two separate occasions should be moderate to high in magnitude. The results of this reliability check gave coefficients in the moderate range (Grade 1: Filtered Words $r=.42$; Auditory Figure Ground, $r=.40$; Competing Words, $r=.73$; Composite Score, $r=.65$; Grade 3: Filtered Words, $r=.22$; Auditory Figure Ground, $r=.41$; Competing Words, $r=.42$; Composite Score, $r=.42$)(Keith, 1986).

It is important to note that many researchers question whether tests designed to measure CAP abilities are valid. This relates to the fact that the definition for a CAP disorder is not accepted by all professionals. Keith (1986) advises that 'central auditory disorder' and other terms attempting to describe the like should be considered "generic" because they relate to a heterogeneous group of individuals (e.g. learning disabled, socially/emotionally disabled, children suffering from language disorders, ADD, family adjustment problems, etc.) who manifest the same behaviors (i.e., difficulty listening to or comprehending auditory information, especially when it is presented under less than optimal acoustic conditions).

In terms of the validity of SCAN, however, it does seem to measure what it was intended to measure. SCAN was concurrently administered with subtests of the Clinical Evaluation of Language Functions (CELF), the Goldman-Fristoe Woodcock Auditory Skills Battery (GFWASB), the

Goldman-Fristoe Woodcock Tests of Auditory Discrimination (GSWTAD) and the Staggered Spondaic Word Test (SSW). The CELF provides diagnostic information on the language processing and production skills of children. The GFWASB is a series of tests designed for the diagnostic assessment of a wide range of auditory skills. The GFWTAD is a screening test of speech-sound discrimination. Finally, the SSW is a measure of central auditory dysfunction (Keith, 1986).

The pattern of coefficients lends support to the validity of SCAN as a measure of auditory processing. In general, the validity verification study seemed to indicate the kinds of relationships with other language measures that SCAN should be expected to show. The results (Table 2) showed positive but low correlations with measures of more general aspects of language and higher correlations with measures assumed to probe the more specific domain of auditory processing (Keith, 1986).

Elements Involved in Identifying CAP Disorders

Keith (1986) defined a central auditory processing disorder as the inability or impaired ability to attend to, discriminate, recognize, or comprehend information presented auditorily even though the person has normal intelligence and hearing sensitivity. These difficulties are more pronounced when listening to low redundancy (distorted) speech, when there are competing sounds, or in poor acoustic environments. CAP disorders are said to be the result of a number of possible causes, including neurological disorders and genetic

traits. Auditory processing disorders are part of a continuum of problems experienced by children in school that are more or less important to children's academic and social achievement and to their general emotional adjustment (Keith, 1985).

Peck et al. (1991) discussed the difficulties and problems surrounding the identification of CAP disorders. They argued that this was due not only to the fact that the entire construct of CAP disorders is not universally accepted but also because of the inefficient tools used to identify CAP disorders. The Staggered Spondaic Word Test and the Willeford Battery have been the traditional methods of testing CAP ability. The Staggered Spondaic Word Test and the Willeford Battery involve the presentation of spoken words and sentences under several conditions (e.g., competing sentences, masked words) in which a person's ability to attenuate to and perceive the auditory signal is tested. Both were normed on small ($N < 100$) and unrepresentative populations which greatly limited the generalizability of the tests. They were designed to be diagnostic tools for the measurement of central auditory processing problems.

The above researchers performed a pilot study in an attempt to identify clinical variables and supportive test data from other disciplines that would consistently correlate with and predict CAP test results. Twenty-four six- to eight-year-old children with normal intelligence and peripheral hearing, and presenting complaints including short

attention spans, hyperactivity, learning difficulties and behavioral problems were examined. They were administered a standard neurodevelopmental, medical, psychological, language processing and educational evaluation. Following this, the Staggered Spondaic Word Test, SCAN and the Competing Sentence Test of the Willeford Battery was given. When all the CAP records from every test were observed only two children had normal CAP scores. Seventeen children received CAP scores that would have been considered abnormal. No significant correlations between CAP scores on the above tests, data from other disciplines or behavioral characteristics, as described by parents and teachers, were discovered.

The authors emphasized that symptoms such as distractibility, not paying attention, school problems (despite normal hearing and IQ) and trouble with listening in noise are common difficulties not stemming from one single etiology. Children with ADD, learning disabilities, behavior problems, emotional disturbances, as well as those with severe psychosocial stressors, may all manifest the above symptoms and obtain low scores on CAP tests (i.e., scores reflecting auditory processing problems). This does not necessarily mean that CNS dysfunction of the auditory processing area has occurred or is occurring in the child. It is important to avoid unnecessary interventions that may not address the actual etiology (i.e., administering a treatment program to deal with CAP dysfunction when the real problem is a learning disability or behavior problem). Therefore,

caution should be exercised in interpreting CAP tests by considering other factors in a child's life that may interfere with normal CAP. Nevertheless, the authors did point out that the SCAN does seem to be more promising than traditional methods. SCAN differs from the two traditional speech audiometry tests in several ways. It is not a diagnostic tool but a screening tool designed to work in conjunction with a variety of assessment methods. Its generalizability is superior, and no detailed, systematic evaluation of the validity and reliability of the Staggered Spondaic Word Test and the Willeford Battery has been carried out to the degree achieved in the evaluation of the SCAN (see previous section on SCAN).

Ballantyne (1990), described two types of hearing tests: pure tone audiometry and speech audiometry. Pure tone audiometry is a quantitative evaluation of hearing for artificial sounds. It was specifically designed to test peripheral hearing since it revealed only limited information about a subjects ability to perceive speech. On the other hand, speech audiometry is a qualitative procedure which measures the ability of the auditory system to analyze, integrate, process and interpret the complex sounds of the human voice. Byllantyne explains that testing children poses a problem because of the lack of standardization of testing procedures, equipment and scoring criteria.

The speech signal possesses extrinsic (phonemic, syntactic and semantic) and intrinsic (binaural integration and fusion)

qualities. The latter is a function of the CNS and involves the correction of possible errors in the identification of the verbal message. Such errors left uncorrected may be due to central disorders such as problems related to the elaboration and decodification of the spoken word (Ballantyne, 1990).

Vulnerability of the Auditory System Over Other Senses

Based on the general belief that the energy metabolism of a tissue is more or less coupled to its functional activity, Kennedy et al. (1975) measured the metabolism rate of the rat's central auditory system with [14C] deoxyglucose while changing the functional state of the animal. Autoradiographs from normal conscious rats, exposed to ambient noise of the laboratory demonstrated a "high optical density" in regions corresponding to structures mediating hearing. When a rat was exposed to sound, an increased metabolic rate of [14C] deoxyglucose was measured. The auditory cortex, medial geniculate nucleus, inferior colliculus, lateral lemniscus and superior olive were the most metabolically active areas noted.

Following this experiment, the researchers blocked the ears of the rats (external auditory canals occluded with wax) and measured the metabolic activity of the central auditory system in a quiet, soundproof room. A decrease metabolic rate of the tracer was observed. In fact, the central auditory areas could barely be distinguished from the adjacent grey matter. The results of this study were consistent with

neuroanatomical knowledge of the central organization of the auditory system.

Of all the sensory systems that influence behavior, clearly the auditory and visual systems seem to play the most influential role. We rely more often and, in most cases, to a greater degree on our sense of hearing and sight than on taste, touch, smell and balance to decide the appropriate behavioral response. That is, most individuals depend more on their hearing and eyesight in order to function in their day-to-day lives.

The auditory system would seem to be more at risk than the visual system when prenatally exposed to cigarette smoke based on the order of sensory system development which allows the auditory system to be metabolically active for a longer period of time during pregnancy (Hofer, 1981). The first evidence of auditory function was obtained by evoked electrical response at 21 weeks and in the visual system at 26 weeks. All parts of the auditory apparatus are present and morphologically mature at six months, while photoreceptor development in the retina is not complete until birth. Consequently, the auditory system would seem more likely to be metabolically active sooner and, therefore, for a longer period of time during pregnancy than the visual system since it is able to respond to stimulation (external and internal to the intrauterine environment) sooner than the visual system. Also, since sound can more readily reach the fetus from outside the uterus than visual stimulation can, in utero

metabolic activity of the auditory system would more likely be greater than the visual system.

From the above, one can argue that prenatal cigarette smoking may place the central auditory system of the fetus at risk. By way of relatively early metabolic processes that are, therefore, active for a longer time during pregnancy, the developing auditory system could be interfered with because of "toxic" levels of CO and nicotine in the mothers blood (blood that feeds the developing fetus) due to hemoglobin's higher affinity for CO rather than O₂. Similar concepts have been proposed by a other investigators (Denson et al., 1975).

Passive Smoke Exposure

The role of passive smoke will be examined based on past research that has related second-hand smoke to negative outcomes in children of mothers passively exposed to smoke (Martin and Bracken, 1986; Makin et al., 1991). Since this study is focused on the effects of prenatal exposure to cigarette smoke, it must be determined whether (if any) auditory effects are related to in utero exposure to maternal cigarette smoke or are a result of prenatal passive smoke exposure or are associated to both modes of cigarette exposure.

Martin and Bracken (1986) studied the effects of passive exposure to cigarette smoke on birth weight. After examining approximately 970 infants born to mothers who reported passive exposure to cigarette smoke and controlling for

confounding factors, the investigators observed a significant mean reduction in birth weight (30 grams) for babies delivered at term. The magnitude of effect for passive smoking was found to be considerably smaller than that due to active smoking.

A study by Makin et al. (1991) examined 91 children between the ages of six- and nine-years-old using a comprehensive neuropsychological test battery. The sample was composed of three groups: 1) children of active smokers during pregnancy, 2) children of passive smokers during pregnancy and 3) children of non-smokers during pregnancy. Based on the results, the researchers observed that the consequences of passive smoking were, in some respects, similar to maternal smoking during pregnancy but smaller in magnitude. The performance of children of passive smokers was found, in most areas, to be between that of the active smoking and the non-smoking groups. The investigators concluded that there is a continuum of long-term smoking effects and that, although active maternal smoking during pregnancy is associated with effects of greater breadth and magnitude than passive maternal smoking, children of passive smokers are also at risk for a pattern of negative developmental outcomes.

In addition to the above findings, postnatal exposure to cigarette smoke will also be considered. Studies have related this type of smoke exposure to low birth weight, middle ear effusion and impairment of lung function (Jarvis et al.,

1992).

Strachan et al. (1989) studied the effects (i.e., incidence of auditory problems) of postnatal cigarette smoke exposure in children. The researchers observed that children postnatally exposed to second-hand cigarette smoke were more likely to develop middle ear effusions. Similar to these observations, Kraemer et al. (1983) included postnatal second-hand cigarette smoke exposure in a list of risk factors related to the development of persistent middle-ear effusions.

An investigation looking into the behavioral effects of prenatal and postnatal exposure to cigarette smoke in children found that both types of exposure were implicated in the development of behavior problems (Weitzman et al., 1992). Some of the problem behaviors included hyperactivity, immaturity and conduct disorder. The results of the above studies reveal the importance of accounting for prenatal and postnatal second-hand smoke.

Current Investigation

This review of the literature has provided a summary of the research concerning the effects of prenatal cigarette use as well as a description of the mechanics and processes of the central auditory system. The present study will attempt to observe the relationship, if any, between in utero cigarette exposure and CAP. The primary tool in this investigation will be SCAN.

This particular facet of behavioral teratology has only

been examined in an indirect fashion in infants and young children. The earlier findings (Picone et al., 1982, Fried & Makin, 1987, Fried & Watkinson, 1988, Kristjansson et al., 1989) combined with the prevalence of prenatal cigarette use, lends impetus to the research addressed here. That is, previous findings have pertained to newborns and young children. The present study will examine older children in order to study whether auditory processing anomalies observed during the newborn stage continue through to more advanced stages of childhood. This study will attempt to observe if CAP deficiencies are more likely to exist in older children prenatally exposed to cigarette smoke as was evident in earlier studies with younger children. In effect, the present inquiry will attempt to corroborate and extend past findings.

II METHOD

The present study is a part of the Ottawa Prenatal Prospective Study (OPPS) (briefly described on pg. 1). Longitudinal research, although essential for the investigation of the consequences of prenatal drug use, has been criticized for a variety of reasons.

One criticism of prospective investigations deals with sample loss which occurs to some degree with this type of research. This has the potential of diminishing the representativeness of the sample. However, the OPPS has had a retention rate of approximately 98% since its inception in 1979, not including families who have moved away and subject loss due to budgetary cut backs. Fortunately, subjects who could not be followed were found to be no different from those subjects who were studied with respect to prenatal drug habits and a variety of health and demographic characteristics (e.g., diet, SES). This suggests that sample loss is not a significant problem for this study. Staff continuity has been an important element in keeping this high retention rate.

In addition to the above, problems with the original selection of the sample may arise. Subjects who are willing to make a long-term commitment to a prospective study are not necessarily representative of the population (Bayley, 1974). They tend to be cooperative and well-educated. However, an examination of the demographic characteristics of the OPPS sample found that it was representative of the Ottawa region

in terms of family income and maternal age at the time of delivery, but not in terms of education (Fried et al., 1980; Fried et al., 1984).

Another problem evident in longitudinal studies is the control of confounding variables. Maternal age, education, other drug use and income represent other prenatal determinants of behavior that can confound the relationship between prenatal cigarette smoke exposure and later behavioral outcomes. Also, postnatal determinants of behavior, such as the home environment, might be potentially confounding or, as a consequence of smoking, may act as mediating variables. For example, if prenatal smoke exposure produces an unresponsive infant, this may in turn affect the parent-child relationship which in turn would affect the child's behavior. Consequently, the role of both covariates and mediating variables have been considered in the analysis.

Subjects

Participants were selected from the larger OPPS pool of subjects (698 women) who had already been involved in a number of previous inquiries. The OPPS has collected data since 1978. Pregnant women were informed of the study by their doctors or by notices in the waiting rooms of the prenatal clinics in four of the largest Ottawa hospitals. The study was described as an investigation of the influence of prenatal lifestyle habits on the developing fetus (Fried et al., 1980).

In order to obtain the necessary information, interviews

were conducted during each trimester of pregnancy. Multiple interviews during pregnancy enabled a reliability monitor on the self-report. In each of the interviews data were collected on caffeine, cigarette, alcohol and marijuana consumption and the use of any other drugs (medicinal or non-medicinal) for each trimester of pregnancy was recorded. Cigarette use was calculated by establishing a nicotine score through multiplying the number of cigarettes smoked on average per day by the nicotine content of the specific brand.

In addition, information concerning the degree and pattern of maternal drug use was gathered, along with details about socioeconomic status (SES) (i.e., income and education), the home environment (HOME, Appendix I) mother's weight, mother's weight gain during pregnancy, father's and mother's age, health (present and prior to pregnancy), obstetrical history of previous pregnancies and father's medical history. A 24 hr dietary history was taken (e.g. protein and calorie intake) and, at birth, data were collected including gestation length, gender, type of birth, birth weight and medication used during labor.

From a list of 303 participants on file in the OPPS with children between the ages of 6- and 11-years-old (SCAN is applicable for up to 11-years, 11-months and 30-days) 157 were known to be unavailable. The majority of the 157 had moved away, a few had withdrawn and some had not been followed for various reasons (e.g., budgetary). Of the 146

remaining subjects, a six percent attrition rate was expected (i.e. the typical rate found during recruitment in OPPS investigations). Therefore, approximately 132 subjects were expected to participate in the present study. The number of subjects that actually participated was 111. Of the 35 subjects who did not participate, 26 had either moved or were unlocatable, 8 did not wish to participate (e.g. summer vacations, viewed as too time consuming, etc.) and one subject's primary language was French (all subjects had to have English as their primary language in order to participate so that language did not become an issue in the study). In terms of smoking categories, 41 nonsmokers, 44 light smokers and 26 heavy smokers (≥ 16 mg/day nicotine) made up the total sample that was followed. One subject from the heavy smoking category was unable to complete the SCAN thus reducing that category to 25 and the overall case total to 110. The smoking habits of the 35 mothers of subjects who were not tested did not differ in terms of their distribution from the sample that was tested - 18 nonsmokers, 9 light smokers and 9 heavy smokers.

Procedure

Using the list of 146 possible participants, potential subjects were contacted by phone (Appendix B). If the person wished to be involved in the study an appointment was made. The entire testing procedure took place at the participant's home. Three separate informed consents were developed (Appendix C, D and E). One applied to the parents (two

copies, leaving one with the parents with appropriate phone numbers for questions and debriefing purposes), another to children eight-years-of-age and older and the third applied to the younger children in the sample. The children's informed consents were read by the children themselves and were age-appropriate. All consent forms were signed in the participants home prior to testing. The experimenter was blind to the participants prenatal and postnatal smoking habits.

The child was tested in the most quiet room in the house. The first test involved a brief pure tone audiometry examination. This was advised by Keith (1986) to ensure that the children to be tested all had hearing within a normal range. If a child was unable to detect a tone ≤ 25 dBHL at either 1000, 2000, 4000 or 500 Hz, it was recorded and testing continued. The Pure Tone audiometer (Appendix M) was calibrated to ANSI-G9 standards at 80 dB (see Table 1) as advised by Wilber (1978). The audiometer was tested with a Sound Level Meter/type 2209 on March 5, 1992.

The procedure for testing subjects was adapted from Anderson (1978). Tones were presented in one ear at a time. The first tone was set at 1000 Hz and presentations were made at 40, 30 and 25 dB. Two presentations at the same signal level were always made to rule out guessing. The same procedure followed for 2000, 4000 and 500 Hz in that order. The pure tone test was used in order to identify any subject that may have had an outstanding hearing problem that could

have interfered with his/her performance on the SCAN and was not used as a hearing test. Therefore, the exact range of children's hearing was not measured.

Following the pure tone test, the SCAN was administered using a Sony Walkman Sports WM-AF54 radio cassette player with Radio Shack Realistic Nova 38 headphones. In order to reduce the amount of ambient noise heard by the child, a pair of Audiomate Peltor P7 auraldomes were placed over the outputting headphones. As well, in order to reduce distraction and increase concentration, a blindfold was used to cover the eyes of the child. This decision was based on pilot studies which noted increased errors as the child gazed at various objects in the room.

The SCAN (A Screening Test for Auditory Processing Disorders) (Appendix A) is applicable for children between the ages of 3- to 11-years, 11 months and 30 days. It is used as a screening tool in the detection of central auditory processing disorders and takes approximately 30 minutes to administer. The child is asked to simply repeat (monosyllable words) what has been heard over stereo headphones under three conditions (filtered words, auditory figure ground and competing words). Incorrectly repeating a word or failing to repeat a word constitutes an error.

The words recorded for the Filtered Words subtest were low-passed filtered at 1000 Hz with filtered roll-off of 32 dB/octave so that high frequency sounds were removed. The filters used were 3 UREI model 565 dip filters connected in

series (Keith, 1986).

For the Auditory Figure Ground subtest, the words were presented at eight decibels above the background noise (party conversation). No single speaker and no background message are identifiable in the babble background noise. Also, the background tape underwent peak compression to eliminate acoustic peaks that naturally occur during spontaneous speech (Keith, 1986).

In the Competing Words subtest all words were measured for duration, matched for equal duration and controlled for semantic content. The word pairs were presented simultaneously on the stereo channels, with one word in one ear, the other word in the opposite ear. The listener was given two tasks. For the first task, the listener had to repeat pairs giving the word heard in the right ear first. In the second task, the listener was asked to repeat word pairs giving the word heard in the left ear first (Keith, 1986).

All SCAN tasks are imitative with the overall purpose being to screen for auditory processing problems and not for difficulties in understanding vocabulary. Consequently, the presented list of words on the tape are easily recognizable and familiar to children (Keith, 1986).

Keith (1986) has suggested some environmental requirements when administering SCAN which were followed with this study. Testing was conducted in a quiet room removed from distractions or disruptions. The test was given in a face-to-face situation that allowed the examiner to utilize

visual clues in interpreting oral responses. Having the child wear a blindfold removed the possibility of distraction that the child may have experienced by viewing the examiner score his/her performance.

Subsequent to the administration of the SCAN, a behavior checklist was completed by the experimenter which assessed the child's behavior during the administration of SCAN (Appendix F). This checklist was used as a subjective screening tool. It aided the experimenter in deciding what tests results may have been invalid due to a child's behavior during the SCAN and, consequently, would be excluded from analysis.

As the child was being tested, parents were asked to complete four short questionnaires - HOME Environment Questionnaire (Appendix I), Neo Five-Factor Inventory (Appendix J), Parent Questionnaire (Appendix K) and the Conners' Parent Rating Scale (short form) (Appendix L). The HOME Environment Questionnaire (HEQ) consists of short, declarative statements about specific, objectively verifiable aspects of children's home, neighbourhood and school environments. Statements are answered "True" or "False" with respect to the child (Sines, 1983). Two types of HEQ's were designed - one for single parent families (1-R) and the other for two parent families (2-R).

The socioeconomic status (SES) item of the HEQ was not analyzed in this study. The SES variable of the HEQ is defined as activities, interests and educational levels that

are associated with academic and intellectual pursuits. This definition seemed somewhat ambiguous and made it difficult to understand the relationship between this possible covariate and the outcomes as well as the relationship between it and prenatal cigarette use. Instead of using this item, an income and an education (averaged education of both parents) item were used for SES.

The NEO Five-Factor Inventory (Costa & McCrae, 1991) consists of 60 statements for the parent to answer. It was designed to measure the views, attitudes, affect, etc. of the respondent in order to calculate a personality rating. Parents responded according to their opinions of themselves.

The Parent Questionnaire was designed specifically for the OPPS lab in order to obtain sample characteristic information on such variables as school and health history of the child, family characteristics (e.g. current income range, occupation, etc.) along with the current cigarette and marijuana habits of the parents. This questionnaire consists of 15 questions.

Finally, parents were asked to complete the Conners' Parent Rating Scale (short form) (Conners, 1990). This rating scale, consisting of 48 items, was designed to characterize patterns of child behavior. It is a brief instrument, evaluating the behavior of a child based on the views/opinions of the parent and is applicable for children from three- to seventeen-years-old. The respondent chooses from among four possible choices when answering: not at all,

just a little, pretty much, very much.

Following the completion of the questionnaires and testing of the child, a brief interview was carried out with the mother or father of the child tested. A short auditory health questionnaire was given in order to obtain the subjects' auditory history (Auditory History Questionnaire, Appendix G). The Auditory History Questionnaire attempted to record the history of hearing problems that may have occurred in a family (i.e., pertaining to the child being studied and the respective parents). This questionnaire was designed to be administered by the experimenter.

In order to measure the amount of passive smoke a child was exposed to the Passive Smoke Exposure Questionnaire (Appendix H) was administered to the mother or father (mother in 95% of cases). The Passive Smoke Exposure Questionnaire inquires into the level of smoke exposure experienced by the child throughout his/her life up until the day of testing. It is an estimate based on a typical week of passive smoke exposure for each year of the child's life and accounts for any changes in the smoking pattern of individuals who have regularly exposed the child to smoke. In terms of passive smoke exposure, the questionnaire addresses two issues: 1) typical level of exposure over time and 2) the timing and level of child's most recent exposure. The level of exposure is expressed in the number of hours a child is in a room or a car where an individual(s) is/are smoking.

The Passive Smoke Exposure Questionnaire also

approximated the amount of passive smoke the mother was exposed to during pregnancy, excluding her own smoke if she happened to smoke cigarettes. Finally, a brief home environment measure was taken dealing with air ventilation and air exchange. Due to the complexity of the questionnaire, it was administered and recorded by the experimenter through questioning of the parent.

As well, a urine sample was obtained from the child to detect the level of cotinine present. Cotinine is a metabolite of cigarette smoke and is widely used as a biochemical marker for passive smoke exposure (Greenberg et al., 1991). The urine sample was obtained upon entering the subject's house after the informed consents were signed, and was placed in an ice cooler until it could be refrigerated. All samples were obtained in the privacy of the individual's bathroom while the test administrator prepared the equipment for administering SCAN in a designated room of the house. If needed, children were aided in the bathroom by their parents.

When the testing session was over (lasting no longer than 1.5 hours) the urine sample was frozen at the experimenter's home freezer and then brought to a laboratory (Ottawa Civic Hospital) where the urine analysis took place. Cotinine lasts approximately three days in the body. Therefore, this part of the procedure dealt exclusively with recent smoke exposure. Individuals examining the urine at the Ottawa Civic Hospital were blind to the subjects' postnatal smoke exposure environment.

The method employed for detecting cotinine in the urine samples was Fluorescence Polarization Immunoassay (FPIA), marketed by Abbot diagnostics for their TDx instrument. Abbot TDx is generally regarded as the gold standard for therapeutic drug monitoring and toxicology screening in most North American clinical laboratories.

The assay uses a competitive binding immunoassay methodology to allow fluorescene-labelled cotinine to compete with endogenous cotinine in the patient sample for binding sites in the anti-body molecule. The assay is completely automated and designed to measure urine cotinine concentrations in the range of 50 - 400 ng/mL. The Abbott TDx Cotinine Assay was modified to allow preanalysis concentration of samples on a Sep Pak. A Sep Pak is a plastic extraction device that is filled with resin. As the urine is passed through this device it cleanses and purifies the samples by removing unnecessary (i.e., for the particular analysis) particles and substances.

In order to test the sensitivity of the TDx method, urine samples from nonsmokers were spiked with cotinine to yield concentrations of 0, 1, 2, 5, 10, 20, 50, 100, 500 ng/mL. These samples were run straight on the TDx kit (after dilution of the standard curve to give values between 0 and 500 ng/mL). Ten mL from each sample were also concentrated on a Sep Pak and reconstituted in a 200 uL of non-smoker urine or saline. This resulted in a net concentration of 50X. Thus, for a urine sample containing 5 ng/mL, after concentration

the measured value would be 250 ng/mL which is well within the assay range of the TDx kit. Based on pilot study, preconcentrated samples gave values which were within 90% of the expected value. The between run precision (i.e., coefficient of variability) of the TDx Cotinine Assay with the preconcentration modification is approximately 8% at a urine cotinine concentration of 50 ug/L. The apparatus for measuring cotinine was located at the biochemical laboratory of the Ottawa Civic Hospital.

The subjects' urine samples were stored in the hospital freezer at -70 degrees celsius until analysis. Samples were extracted in batches of 7 patient samples with blank quality control samples and positive quality control samples (i.e., spiked). After the samples were thawed, an aliquot of each sample was sent for creatinine analysis. Cotinine was divided by the creatinine concentration to adjust the data to reflect the concentration of the urine sample. Research has shown that adjusting cotinine levels for creatinine allows for the best separation of a group of nonsmokers from passive smokers by controlling for dilution effects (Wall et al. 1988, Greenburg et al., 1991).

Testing for cotinine in the urine was done for two reasons. It is an objective measure of recent passive smoke exposure and cotinine results can be compared to the recent exposure score for the Passive Smoke Exposure Questionnaire to determine a level of correspondence between self-report on the questionnaire and cotinine results. That is, there should

be a moderate to high positive correlation between hours of exposure reported and the amount of cotinine measured if parents are in fact accurately reporting their child's level of recent smoke exposure (Knight et al., 1989).

Second-hand smoke was considered in this study to see if it was associated with children's performance on the SCAN. The Passive Smoke Exposure Questionnaire addressed the issue of the history of exposure as well as recent exposure (i.e. typical week and the last three days of exposure). Measuring for cotinine strictly pertained to the last three days of passive smoke exposure.

Method of Analysis

Setting alpha at .05 and using the arithmetic sample mean of 36.7 (summed across prenatal cigarette use groups and divided by the number of groups), with 80% power an effect size of approximately .3 of a standard deviation can be detected (Cohen, 1977). This estimate was calculated using $k-1 = 2$ groups.

For analytical purposes, smoking was categorized into three groups: nonsmoking (controls), light and heavy (≥ 16 mg nicotine/day, approximately a minimum of one package of average strength). Smoking has traditionally been grouped in this manner both throughout the history of the OPPS and by other research groups (Naeye and Peters, 1984). More importantly, categorization of maternal smoking was carried out because of the non-normal distribution in the sample that has been noted in the past. Creating groups of the sample

also helped to ease the interpretation of the results.

All analyses were one-tailed. This decision was based on evidence that has consistently shown prenatal cigarette smoke exposure to be related to negative outcomes rather than positive ones (discussed throughout the introduction). No study has observed enhanced performance on any measure in children exposed to cigarette smoke in utero. Therefore, based on what has been observed in the past, enhanced performance on the SCAN in children prenatally exposed to cigarette smoke was not expected.

Multivariate analysis of variance was carried out on data obtained from the SCAN with an alpha set at .05. The three levels of the SCAN, which constitute three separate elements of auditory processing (Keith, 1986), were used as dependent variables and categorized maternal smoking was used as the independent variable. This analysis helped to reduce Type I errors by examining simultaneously differences among groups on multiple dependent variables as apposed to calculating all possible zero order correlations or squared multiple correlations (Pedhazur, 1982). Prior to multivariate analyses, data was evaluated using univariate descriptive statistics to check for accuracy of input, outliers and normality of distribution.

Since this study is a quasi-experimental design, random assignment to groups was not possible. As a result, there was a risk that observed differences would represent only the preexisting differences in history and background. This

concern was addressed by the statistical control of variables that were found to be related to both the outcome measures and prenatal cigarette use.

An analysis of variance (ANOVA) was carried out on data obtained from the SCAN Composite (dependent variable) with an alpha set at .05. Categorized maternal smoking was the independent variable of interest. An analysis of covariance was then carried out in order to control for the effect of variables that were found to be related to both the dependent and independent variables of interest.

A total of 22 potentially confounding and mediating variables were analyzed in terms of their relationship with prenatal smoke exposure and the SCAN by performing Pearson product-moment correlations. The variables considered as potential covariates or mediators were family income, averaged education (mother's and father's combined), mother's age, mother's weight, mother's weight gain during pregnancy, mother's personality rating, parent rating of child's behavior, prenatal alcohol, marijuana and caffeine use, prenatal protein and calorie intake, parity, birth weight, the home environment, divorce/separation, whether the child received remedial help in school, gender, mother's history of hearing problems, father's history of hearing problems, child's history of hearing problems, mother's rating of noise exposure during pregnancy and passive smoke exposure. The original 22 variables were chosen as possible confounds based on the literature and recommendations by Keith (1985).

Variables were selected from the above 22 potential covariates on the basis of a relationship with both prenatal smoking status and each of the three subtests of the SCAN and the SCAN Composite score at an alpha level of .10 or lower.

The potential confounds that were selected based on the above procedure were entered as covariates in a multivariate analysis of covariance (MANCOVA). This tested whether measures that were shown to be associated with group differences remained significant after statistically controlling for confounds. Potential mediating variables were analyzed individually and not integrated into the main analysis.

RESULTS

Characteristics of the Sample

Data were used from a total of 110 subjects who participated in the study. The children ranged in age from 6- to 11-years-old. The sample included 62 males and 48 females. Based on prenatal cigarette use, the sample was composed of 41 nonsmokers, 44 light smokers (<16mg/day) and 25 heavy smokers ($\geq$ 16mg/day). Of the children who were studied, the average income of households during pregnancy was \$31833.00 / year. The average education of both of the children's parents combined was equivalent to some university education (i.e., one to three years without the completion of an undergraduate degree). The average intake of nutrients by the sample of mothers during pregnancy was 128% of the recommended daily allowance. Only eight mothers used illicit

drugs during the first trimester (e.g., dexidrine, LSD, cocaine). Most of these mothers stopped using after realizing they were pregnant. One mother used cocaine once during the second trimester. Illicit drugs were not used during the third trimester by any of the participants. Finally, one mother used tranquilizers (Diazepam) during the first trimester. Characteristics of the sample are displayed in Table 3. Based on these findings, the sample seemed to be representative of a middle class, low-risk population.

Preliminary Findings

Mean standard score results and standard deviations (std) for each of the three subtests of the SCAN (Filtered Words, Auditory Figure Ground and Competing Words) and the SCAN Composite (the total of the three subtests) based on the three categories of prenatal cigarette exposure are displayed in Table 4. Differences between the three cigarette groups on each subtest outcome are displayed in Figure 1 and the SCAN Composite mean standard scores can be seen in Figure 2. There is a negative linear trend between each of the outcomes of the SCAN and cigarette smoking levels. What is also interesting is that the heavy smoking category seems to follow a separate pattern from the other two groups in terms of standard score means across subtests (Figure 1). This pattern shows standard score means to be highest for the Filtered Words subtest and lowest for the Competing Words subtest. For light smokers and controls, the standard score means are highest also for Filtered Words but lowest for

Fig.1: SCAN Subtest Standard Score Means By Prenatal Cigarette Use Categories

Fig.2 SCAN Composite Standard Score Means By Prenatal Cigarette Use Categories

Auditory Figure Ground.

A one-way analysis of variance was carried out for each of the three SCAN subtests and the SCAN Composite based on the three cigarette smoking levels. Mean standard scores for the heavy, light and no-use prenatal cigarette use categories respectively were as follows: Filtered Words (10.72, 2.1 std; 11.4, 2.3 std; 11.41, 2.29), Auditory Figure Ground (9.56, 1.78 std; 9.86, 1.89 std; 10.29, 2.55 std), Competing Words (8.68, 1.91 std; 10.31, 2.52 std; 10.46, 2.51 std), SCAN Composite (96, 9.5; 103, 12.22 std; 104, 11.95 std). Though each subtest and the composite score showed a linear relationship with cigarette smoking levels, only Competing Words and the SCAN Composite standard score means differed significantly across cigarette smoking levels with alpha set at .05 (Filtered Words, $F(2,107)=.923$, $p=.2$; Auditory Figure Ground, $F(2,107)=.977$, $p=.19$; Competing Words, $F(2,107)=4.95$, $p=.0045$; SCAN Composite, $F(2,107)=4.14$, $p=.009$).

Following the above analysis, means were contrasted using the Scheffe technique in order to discern what means were significantly different from each other with alpha set at .05. With regard to Competing Words, the heavy prenatal cigarette use category ($\geq 16\text{mg/day}$) was significantly different from both the control (no prenatal cigarette use) and light prenatal cigarette use categories. In terms of the SCAN Composite, the heavy prenatal cigarette use category significantly differed from only the control group.

Control Variables

Potential covariates were chosen based either on an observed association ($p \leq .10$) between both the dependent (Competing Words subtest of the SCAN) and independent (prenatal cigarette use) variables of interest or on a theoretical basis. The Filtered Words and Auditory Figure Ground subtests were not used because they were not found to be significantly related to prenatal cigarette use at the univariate level.

Parent-reported postnatal passive exposure to cigarette smoke along with the cotinine/creatinine results were not found to be associated ($p \leq .10$) with the outcomes of the SCAN. Based on the history of passive smoke exposure items of the Passive Smoke Exposure Questionnaire, an item that was related to the outcomes was child's typical week of passive smoke exposure from the primary caretaker (95% mothers) at ages one-, two-, three-, four-, and six-years. These variables were not used as covariates because of their high association with prenatal cigarette use (i.e., issue of multicollinearity).

Prenatal marijuana use and prenatal alcohol use were used as covariates even though they did not fit the statistical criteria since both have been shown to be related to behavioral outcomes in children (Fried and Makin, 1987, Fried and Watkinson, 1988). Prenatal marijuana use was measured by estimating the number of joints, on average, smoked per week. Prenatal alcohol use was measured by

estimating the amount of absolute alcohol, on average, consumed per day. Mean prenatal marijuana use and alcohol use for the heavy, light and no-use prenatal cigarette use categories respectively were as follows: Marijuana (1.93, 5.21 std, 2.17, 5 std, 2.87, 8.76 std), Alcohol (.198, .26 std, .153, .18 std, .17, .31 std).

The hours of passive cigarette smoke exposure a child experienced during a typical week was also used as a possible covariate, even though it failed to meet the selection criteria, because studies have shown passive smoke exposure to be related to an increase in behavioral problems (Makin et al., 1991, Weitzman et al., 1992). Means for this variable for the heavy, light and no-use cigarette use categories respectively were as follows: 36.16, 29.3 std, 25.41, 24.72 std, 12.28, 19.63 std).

Potential covariates that did meet the statistical selection criteria included mother's and father's averaged education (Competing Words: $r=.22$, $p=.01$; SCAN Composite: $r=.17$, $p=.041$; prenatal cigarette use: $r=-.42$, $p<.001$), the prenatal passive smoke exposure item of the Passive Smoke Exposure Questionnaire (Competing Words: $r=-.17$, $p=.037$; SCAN Composite: $r=-.16$, $p=.045$; prenatal cigarette use: $r=.37$, $p<.001$) and the exposure to excessive noise during pregnancy item of the Auditory History Questionnaire (Competing Words: $r=-.19$, $p=.025$; SCAN Composite: $r=-.14$, $p=.071$; prenatal cigarette use: $r=.19$, $p=.024$).

Means for the above three potential covariates for the

heavy, light and no-use cigarette use categories respectively were as follows: parents' averaged education (2, .74 std, 2.67, .67 std, 2.9, .75 std), prenatal passive smoke (36.6, 30.3 std, 25.4, 24.7 std, 12.52, 20 std), prenatal excessive noise exposure (.24, .43 std, .2, .41 std, .07, .26 std).

Main Analysis

Since the analysis of variance for the SCAN Composite score across prenatal cigarette use categories was statistically significant, an analysis of covariance was carried out with the SCAN Composite score using SPSS-PC ANCOVA. Again, the independent variable was prenatal cigarette use (heavy, light and no use). Covariates were averaged education of both parents, prenatal passive smoke exposure, prenatal exposure to excessive noise, prenatal alcohol and marijuana use and the amount of passive cigarette smoke a child was exposed to during a typical week.

Results of evaluation of the assumptions of normality of sampling distributions, linearity, homogeneity of variance, homogeneity of regression and reliability of covariates were satisfactory. Presence of outliers led to adjustment of two of the covariates. Two cases from the prenatal passive cigarette smoke exposure variable were replaced with dummy values. Value 107 hours/week was replaced by 96 hours/week and value 105 hours/week was replaced by 95 hours/week. Three cases from the child's typical week of passive cigarette smoke exposure were replaced with dummy values. Values 53, 52 and 50 hours/week were replaced by value 49 hours/week. All

values were chosen as the next one hour increment from the highest case value that was not an outlier. This method of dealing with outliers was used in order to include these cases in the analysis.

After adjustment of covariates, the SCAN Composite score varied significantly with prenatal cigarette use categories, with $F(2,101)=2.63$, $p=.039$. On average, children from the heavy prenatal cigarette smoking group scored lower on the SCAN Composite when compared to the light and no smoking groups. Adjusted mean standard scores for the heavy, light and no-use prenatal cigarette use categories retained a linear pattern for the SCAN Composite (96.2, 102.9, 103.9).

A multivariate analysis of variance was performed on three dependent variables: Filtered Words subtest, Auditory Figure Ground subtest and Competing Words subtest of the SCAN. The independent variable was prenatal cigarette use (heavy, light and no use).

SPSS-PC MANOVA was used for the analysis with the sequential adjustment for nonorthogonality. Results of evaluation of assumptions of normality, sample sizes, linearity, outliers, homogeneity of variance-covariance matrices and multicollinearity were satisfied. With the use of Wilks' criterion, the combined dependent variables were not significantly affected by prenatal cigarette use, $F(6, 210)= 1.73$, $p=.057$.

In order to assess whether the above results would hold after potential confounds were entered, an analysis of

covariance was performed on the three independent variables. Adjustment was made for six covariates: the averaged education of both parents, prenatal passive cigarette smoke exposure, prenatal exposure to excessive noise, prenatal marijuana and alcohol use and the amount of passive cigarette smoke a child was exposed to during a typical week. The independent variable remained the same.

The analysis was terminated upon discovering that the assumption of homogeneity of regression for MANCOVA was not satisfied. Within the subtest Filtered Words, prenatal cigarette use interacted with passive cigarette exposure during pregnancy $F(2, 104)=5.59$, $p=.005$ and marijuana use during pregnancy, $F(2, 104)=3.2$, $p=.045$. This problem was addressed by dropping Filtered Words from the main analysis and examining the two interactions separately. The interactions were examined by plotting the cell means for each interaction and comparing prenatal cigarette use groups. The interactions were not further analyzed because no potentiating effects were noted and the trends represented by the three prenatal cigarette use groups did not reflect any coherent pattern. As well, due to the small cell sizes (i.e., a number of cells < 5) the comparisons were unreliable.

The corrected MANOVA, then, consisted of two dependent variables: Auditory Figure Ground and Competing Words. The independent variable remained the same. All of the previously mentioned MANOVA assumptions were satisfied. With the use of Wilks' criterion the combined dependent variables (i.e.,

Auditory Figure Ground and Competing Words) were significantly affected by prenatal cigarette use, $F(4,212)=2.6$, $p=.019$.

Following this observation, a MANCOVA was carried out. The covariates remained the same as in the first attempted MANCOVA. Presence of outliers led to adjustment of two of the covariates. Two cases from the prenatal passive cigarette smoke exposure variable and three cases from the child's typical week of passive cigarette smoke exposure were replaced with the same dummy values as before (i.e., ANCOVA with SCAN Composite). Assumptions of normality, homogeneity of variance-covariance matrices, reliability of covariates, linearity and multicollinearity were satisfactory.

With the use of Wilks' criterion, the combined dependent variables were not significantly related to the combined covariates. However, when the three covariates that were entered into the analysis based on theoretical grounds and not statistical criteria (prenatal marijuana use, prenatal alcohol use and exposure to passive cigarette smoke during a typical week) were removed, the combined dependent variables were significantly related to the two combined covariates and thus had to be adjusted for in subsequent analyses.

After adjustment for covariates was carried out the combined dependent variables were not significantly related to prenatal cigarette use, $F(4, 200)=1.53$, $p=.098$. At the univariate level, Competing Words subtest maintained its significant relationship with prenatal cigarette use

following the adjustment for covariates, $F(2, 101) = 2.5$, $p = .047$. Adjusted mean standard scores for the heavy, light and no-use prenatal cigarette use categories did not retain a linear pattern for Competing Words (8.89, 10.30, 10.27). A summary of the steps to the multivariate main analysis is depicted in Table 6.

Passive Smoking

COTININE/CREATININE Only one subject refused to give a urine sample thus reducing the number of recorded cotinine cases to 109 from a possible 110. All samples were tested and none was lost. Overall, the cotinine/creatinine results showed that, in general, this sample was not exposed to high levels of passive smoke compared to other studies (Perkins, 1992). For example, the sample mean for cotinine/creatinine in this study was .91 ug/mmol while results in a study by Wall et al. (1988) observed a mean of 6.3 ug/mmol cotinine/creatinine ratio in subjects who were passively exposed to cigarette smoke.

Pearson product-moment correlations were performed on the cotinine/creatinine ratio with two items of the Passive Smoke Questionnaire: 1. the 'parent-reported past three days of smoke exposure of the child in hours' and 2. the time interval between the last recalled smoke exposure period of the child and the time the urine sample was collected. In terms of the first item, cotinine/creatinine concentrations in the children's urine increased significantly as parent-reported passive exposure to cigarette smoke increased

($r=.71$, $p<.001$). Similarly, based on the second item, cotinine/creatinine concentrations in the children's urine increased significantly as parent-reported time intervals decreased ($r=-.63$, $p<.001$).

In order to determine the importance of considering outside sources of passive smoke exposure (i.e., smokers coming in contact with a subject who did not live at the subject's residence), data pertaining to this on the Passive Smoke Exposure Questionnaire was dropped from the child's past three days of passive smoke exposure item. Therefore, only parental and other residential sources of passive smoke exposure (i.e., other inhabitants of subject's home) made up the child's past three days of passive smoke exposure variable. When this modified variable was correlated with the cotinine/creatinine results a decrease in the strength of the relationship between the two variables was noted, $r=.67$. Therefore, consideration of outside sources of passive cigarette smoke does seem to account for some of the variance in the cotinine/creatinine findings.

An analysis of the effect of passive smoke exposure in children who were not directly prenatally exposed (i.e., mother's active smoking) to cigarette smoke ($n=41$) in utero was carried out in order to isolate passive smoke exposure from direct prenatal smoke exposure. Three main groups were found to exist in our sample: 1. children exposed to passive smoke prenatally (father smoked in pregnant, nonsmoking wife's presence) and postnatally ($n=9$), 2. children exposed

to passive smoke postnatally only ($n=10$), and 3. children having no passive cigarette smoke exposure either prenatally or postnatally ($n=20$). A small group ($n=2$) remained (i.e., exposure to passive smoke by father prenatally but not postnatally) and was not used in the analysis because of its small size.

Mean standard scores for the prenatal/postnatal, postnatal only and no cigarette smoke exposure groups respectively were as follows: Filtered Words (10.11, 3.4 std; 12.6, 1.77 std; 11.3, 1.72 std), Auditory Figure Ground (10.44, 3.1 std; 10, 2.7 std; 10.35, 2.3 std), Competing Words (9.5, 2 std; 10.6, 2.4 std; 10.75, 2.57 std), SCAN Composite (101.5, 11.6 std; 103.7, 10.7 std; 105, 12.24 std). Based on the groups' SCAN subtest mean standard scores and the SCAN Composite score, only Filtered Words was significantly associated with passive smoke categories (Filtered Words: $F(2,36)=2.99$, $p=.031$; Auditory Figure Ground: $F(2,36)=.08$, $p=.46$; Competing Words: $F(2,36)=.79$, $p=.23$; SCAN Composite: $F(2,36)=.9$, $p=.21$).

Following the above analysis, means were contrasted using the Scheffe technique in order to discern what means were significantly different from each other with alpha set at .05. With regard to Filtered Words, the prenatal/postnatal passive smoke exposure category was significantly different from only the no cigarette smoke exposure category. On average, children from the post and prenatal passive cigarette smoke exposure group scored lower on Filtered Words

than the other two groups.

The 'father's smoking habits during the mother's pregnancy' variable was used in the above analysis as a measure of prenatal passive smoke exposure. It was used because of the small time interval between the father actually smoking and recall of the event (i.e., this information was recorded during the mother's pregnancy) thereby reducing memory error. However, due to the limitations of using the father's prenatal smoking habits for estimating passive smoke exposure of the pregnant mother (i.e., other sources are ignored), a second analysis was performed. It was similar to the above, but used the prenatal passive smoke exposure item of the Passive Smoke Exposure Questionnaire in place of the father's prenatal smoking habits. It asked the mother to estimate, in hours, how much she had been passively exposed to cigarette smoke at work and at home during pregnancy. Therefore, this estimate included the father's smoking habits as well as other sources.

In terms of this item, three main groups were found to exist in the sample, though differing in size and definition from the above: 1. children exposed to passive smoke prenatally and postnatally (n=16), 2. children exposed to passive smoke prenatally only (n=7) and 3. children not exposed to passive smoke at all (n=15). A small group (n=3) remained (i.e. children exposed to passive smoke postnatally only) and was not used in the analysis because of its small size.

Mean standard scores for the prenatal/postnatal, prenatal only and no passive cigarette smoke exposure groups respectively were as follows: Filtered Words (11.12, 3 std; 12.86, .9 std; 10.73, 1.5 std), Auditory Figure Ground (10.37, 3 std; 10.14, 2.27 std; 10.47, 2.4 std), Competing Words (10, 2 std; 9.14, 2.9 std; 11.5, 2.4 std), SCAN Composite (102, 13.6 std; 102.14, 11.11 std; 107, 10.5 std). Based on the groups' SCAN subtest mean standard scores and the SCAN Composite score, only Competing Words was significantly associated with passive smoke categories (Filtered Words: $F(2,35)=2.2$, $p=.063$; Auditory Figure Ground: $F(2,35)=.04$, $p=.485$; Competing Words: $F(2,35)=2.86$, $p=.035$; SCAN Composite: $F(2,35)=.77$, $p=.24$).

Following the above analysis, means were contrasted using the Scheffe technique in order to discern what means were significantly different from each other with alpha set at .05. With regard to Competing Words, the prenatal only smoke exposure category was significantly different from only the no passive cigarette smoke exposure category. In contrast to the former analysis, this latter analysis observed that, on average, children from the prenatal passive cigarette smoke exposure group scored significantly lower on Competing Words than the other two groups. However, similar to both analyses is the observation that prenatal passive cigarette smoke exposure, in general, seems to be associated with nonsignificant lower scores on SCAN outcomes. SCAN subtest mean standard scores and standard deviations for the latter

analysis are displayed in Table 5 and depicted in Figure 3 and 4.

Following the above, the control group was then divided into two groups that were compared; one group being made up of the first two groups in the above analysis ($n=26$) vs the third group in the above analysis. Therefore, a group that received some type of second-hand smoke was compared to the "pure control" group (i.e. no passive smoke).

Mean standard scores for the prenatal and/or postnatal and no passive cigarette smoke exposure groups respectively were as follows: Filtered Words (11.81, 2.58 std; 10.73, 1.5 std), Auditory Figure Ground (10.19, 2.67 std; 10.47, 2.4 std), Competing Words (9.84, 2.4 std; 11.53, 2.38 std), SCAN Composite (102.3, 12.58 std; 107, 10.5 std). A statistically significant difference was observed between the two groups for Competing Words only (Filtered Words: $t(1,38.92) = -1.67$, $p=.051$; Auditory Figure Ground: $t(1,39) = .33$, $p=.37$; Competing Words: $t(1,39) = 2.17$, $p=.018$; SCAN Composite: $t(1,39) = 1.21$, $p=.12$). On average, children postnatally and/or prenatally passively exposed to cigarette smoke scored significantly lower on Competing Words than children who were not passively exposed to cigarette smoke.

A second comparison was made by categorizing the control group into a group that was exposed to passive smoke postnatally only ($n=22$) and another that did not receive any passive cigarette smoke postnatally ($n=19$) regardless of the amount of prenatal passive smoke exposure in both groups.

Fig.3 SCAN Subtest Standard Score Means By Passive Smoking Groups For Controls

Fig.4 SCAN Composite Standard Score Means
By Passive Smoke Categories for Controls

Mean standard scores for the postnatal only and no postnatal passive cigarette smoke exposure groups respectively were as follows: Filtered Words (11.41, 1.68 std; 11.42, 2.9 std), Auditory Figure Ground (10.36, 2.32 std; 10.21, 2.86 std), Competing Words (10.77, 2.74 std; 10.1, 2.23 std), SCAN Composite (105.45, 10.7 std; 102.37, 13.36 std). No statistically significant differences were observed between the two groups for any of the subtests (Filtered Words: $t(1,27.99) = -.02$, $p = .49$; Auditory Figure Ground: $t(1,39) = .19$, $p = .43$; Competing Words: $t(1,39) = .85$, $p = .2$; SCAN Composite: $t(1,39) = .82$, $p = .21$).

A final comparison was made by partitioning the control group into a group that was prenatally exposed to passive smoke ($n=23$) and a group that was not ($n=18$) regardless of the amount of postnatal passive smoke exposure in both groups. Mean standard scores for the prenatal only and no prenatal passive cigarette smoke exposure groups respectively were as follows: Filtered Words (11.65, 2.7 std; 11.11, 1.68 std), Auditory Figure Ground (10.3, 2.79 std; 10.28, 2.3 std), Competing Words (9.78, 2.3 std; 11.33, 2.5 std), SCAN Composite (102, 12.64 std; 106.5, 10.83 std). A statistically significant difference was observed between the two groups for Competing Words only (Filtered Words: $t(1,39) = -.75$, $p = .23$; Auditory Figure Ground: $t(1,39) = -.03$, $p = .49$; Competing Words: $t(1,39) = 2.04$, $p = .023$; SCAN Composite: $t(1,39) = 1.22$, $p = .12$). On average, children prenatally exposed to passive cigarette smoke scored lower on Competing

Words than did children who were not prenatally passively exposed. It is interesting to note that, unlike the previous comparison, in this comparison (i.e. defined by levels of prenatal passive smoke exposure) a statistically significant difference is observed with Competing Words.

Research has observed that the effects of prenatal exposure to light levels (less than a pack a day) of cigarette smoke are similar to passive cigarette smoke exposure in children (Makin et al., 1991). Consequently, multiple t-tests were performed to observe whether the light smoking group (n=44) of the prenatal cigarette use categories was similar to the prenatal and/or postnatal passive smoke exposure group (n=26) of the control category.

Mean scores on a number of important sample characteristics for the light smoking group and the prenatal and/or postnatal passive smoke exposure group respectively were as follows: parents' averaged education (2.65, .67 std; 2.8, .81 std), child's auditory history (.79, 1.4 std; .5, 1.2 std), prenatal marijuana use (2.38, 5.9 std; 4.33, 10.76 std), prenatal alcohol use (.15, .18 std; .22, .37 std). The groups were found to be similar to each other: parents' averaged education, $t(1,68) = -.89$, $p = .376$; child's auditory history, $t(1,68) = .89$, $p = .379$; prenatal marijuana use, $t(1,68) = -.69$, $p = .492$; prenatal alcohol use, $t(1,37.34) = -.79$, $p = .433$.

SCAN subtest and Composite standard score means for the light smoking group and the prenatal and/or postnatal passive

smoke exposure group respectively were as follows: Filtered Words (11.4, 2.3 std; 11.81, 2.58 std), Auditory Figure Ground (9.86, 1.89 std; 10.19, 2.67 std), Competing Words (10.31, 2.52 std; 9.85, 2.4 std), SCAN Composite (103, 12.22 std; 102.3, 12.58 std). The groups were, again, found to be similar to each other: Filtered Words, $t(1,68) = -.67$, $p = .506$; Auditory Figure Ground, $t(1,39.94) = -.55$, $p = .584$ (separate variance estimate); Competing Words, $t(1,68) = .77$, $p = .445$; SCAN Composite, $t(1,68) = .23$, $p = .82$. Based on the above results, children exposed to light levels of cigarette smoke in utero (i.e. mother smoked during pregnancy) do not significantly differ from children who were exposed to passive cigarette smoke prenatally and/or postnatally and whose mothers did not actively smoke during pregnancy.

The prenatal and/or postnatal passive cigarette smoke exposure group ($n=26$) was then compared to the "pure control" group ($n=15$) (no passive cigarette smoke exposure) to see if they differed on any important sample characteristics (recall that these groups significantly differed on Competing Words). Mean scores for the respective groups were as follows: parents' averaged education (2.81, .81 std; 3.17, .59 std), child's auditory history (.5, 1.21 std; .07, .07 std), prenatal marijuana use (3.22, 6.4 std; .33, 1.29 std), prenatal alcohol use (.22, .37 std; .08, .119 std). Both groups were similar on all variables except prenatal marijuana use: parents' averaged education, $t(1,39) = 1.5$, $p = .14$; child's auditory history, $t(1,39) = -.81$, $p = .38$;

prenatal marijuana use, $t(1,35.51)=-2.28$, $p=.03$; prenatal alcohol use, $t(1,37.52)=-1.91$, $p=.064$.

Potential Mediating Variables

One potential covariate that met the statistical criteria but was not used in the main analysis was whether the child had received remedial help in school (yes/no answer to question) (Competing Words: $r=-.25$, $p=.01$; SCAN Composite: $r=-.164$, $p=.06$; prenatal cigarette use: $r=.186$, $p=.04$). Means for this variable for heavy, light and no-use cigarette use categories respectively were .38, .5 std, .18, .39 std, .14, .35 std. This potential confound was not used in the overall analysis because it was viewed as a possible mediating variable that, if used, could cause overcontrolling in the analysis. Consequently, a one-way analysis of variance was carried out to examine the extent to which standard score means on Competing Words (the only subtest remedial help was significantly related to) and the SCAN Composite score differed across the variable's two categories (i.e., received remedial help, yes/no).

Mean standard scores for the 'yes' and 'no' groups respectively were as follows: Competing Words (8.53, 2.4 std; 10.03, 2.44 std), SCAN Composite (96.37, 12.67 std; 101.17, 11.78 std). The results indicated that the two categories significantly differed only in the case of Competing Words (Competing Words: $F(1,87)=5.66$, $p=.01$; SCAN Composite: $F(1,87)=2.4$, $p=.06$). When used as a confound in an analysis of covariance (ANCOVA) with Competing Words by prenatal

cigarette use, the 'sought remedial help' variable only slightly decreased the statistical significance of the difference between the standard score means of Competing Words across prenatal cigarette use categories, $F(2,85)=3.9$, $p=.012$. This observed change in statistical significance could also have been due to the reduction in sample size since 22 cases were missing the 'sought remedial help' variable.

Another variable that could have been used as a potential covariate based on theoretical grounds but was not because of its possible mediating effects was the child's auditory health history (Appendix G question 3). Means for this variable for the heavy, light and no-use cigarette use categories respectively were .28, .46 std, .25, .44, .12, .33 std. A number of studies have noted that postnatal exposure to passive cigarette smoke in children increases the likelihood of middle-ear problems (Strachan et al., 1989; Kraemer et al., 1983). As well, when the auditory history of the child was controlled for (ANCOVA) with Competing Words by passive smoking categories in the control group (prenatal and postnatal passive smoke exposure vs no exposure) the significance of the relationship between these two variables was reduced by the covariate to $F(1,39)=3.25$, $p=.04$, from $F(1,39)=2.17$, $p=.018$. Consequently, this variable was analyzed in a similar manner to the 'sought remedial help' variable in order to examine to what extent, if any, the standard score means for Competing Words and the SCAN

Composite score differed across auditory health history groups (i.e., an auditory problem group vs a no auditory problem group).

Mean standard scores for the 'problems' and 'no problems' child auditory history groups respectively were as follows: Competing Words (8.65, 2.29 std; 10.36, 2.41 std), SCAN Composite (95.13, 10.96 std; 103.55, 11.55 std). The results indicated that the two means did significantly differ (Competing Words: $F(1,108)=9.24$, $p=.0015$; SCAN Composite: $F(1,108)=9.87$, $p=.001$). This variable was modified into two categories from five due to the very small group sizes that were found (five possible totals of variable: 0: $n=87$, 1: $n=2$, 2: $n=1$, 3: $n=16$, 4: $n=4$). When used as a confound in an analysis of covariance (ANCOVA) with Competing Words by prenatal cigarette use and the SCAN Composite score by prenatal cigarette use, the 'auditory history of the child' variable only slightly decreased the significance of the difference between the standard score means of Competing Words, $F(2,106)=4.65$, $p=.006$, and SCAN Composite, $F(2,106)=3.52$, $p=.017$, across prenatal cigarette use categories.

III DISCUSSION

Based on the linear combination of Auditory Figure Ground and Competing Words subtests of the SCAN (MANOVA), prenatal cigarette smoke exposure seems to negatively affect auditory processing. At the univariate level (ANOVA), differences were detected in auditory processing across prenatal cigarette exposure groups with Competing Words. Based on the present sample, children exposed to heavy quantities of cigarette smoke (≥ 16 mg of nicotine/day) in utero showed decreased performance in dichotic listening abilities compared to children exposed to light levels or no smoke at all. The Filtered Words and Auditory Figure Ground subtests of SCAN did not differentiate among any of the maternal prenatal smoking groups. Finally, prenatal cigarette smoke exposure was also negatively related to lower SCAN Composite scores. Again, children prenatally exposed to heavy levels of cigarette smoke were more likely to have lower scores on the SCAN Composite.

In further univariate analyses (ANCOVA), prenatal smoke exposure remained negatively associated with auditory processing based on the Competing Words subtest and SCAN Composite even after covariates were controlled for. The results of this study seem to support the literature on the effects of prenatal cigarette smoke exposure on auditory processing in children (Saxton, 1978; Picone, 1982; Fried and Makin, 1987; Kristjansson et al., 1989). However, after potential covariates were controlled for at the multivariate

level (MANCOVA), the statistically significant relationship between prenatal cigarette smoke exposure and auditory processing did not hold.

One reason why the ANCOVA with the SCAN Composite score and the MANCOVA gave different statistical results could be because each analysis asked a different question. The Composite score is a simple addition of the three subtest scores, giving a general idea of a child's overall performance on SCAN. The MANCOVA looked at the linear combination (unlike simple addition) of Auditory Figure Ground and Competing Words.

Apart from the fact that Filtered Words was removed from the analysis, the MANCOVA approach accounted for each remaining subtest individually by analyzing the differences between the three prenatal cigarette use categories on each of the SCAN subtests simultaneously. Therefore, the ANCOVA with the SCAN Composite asked whether the three prenatal cigarette use categories differed according to their overall SCAN performance; whereas, the MANCOVA asked whether the three prenatal cigarette use categories differed on each of the SCAN subtests.

Further, Competing Words, the only subtest to statistically significantly differentiate between the three prenatal cigarette use groups, contributes more to the SCAN Composite score when the three subtests are added (i.e., 100 items are totalled for a Competing Words score while only 40 items are totalled for each of the other subtest scores).

Therefore, Competing Words is represented in the SCAN Composite more so than the other two subtests. In contrast, the MANCOVA analyzes the subtests standard scores, giving each subtest equal weight in the analysis. Based on the above arguments, then, the ANCOVA with the SCAN Composite score should give different statistical results from the MANCOVA.

As mentioned before, the SCAN Composite score reflects a child's overall central auditory processing performance. Children exposed to cigarette smoke in utero in this study were more likely to have lower SCAN Composite scores. This possibly reflects a subtle deficit in central auditory processing and/or language abilities existing in children from the heavy smoking category (Keith, 1986).

In order to assess the validity of SCAN, researchers compared it to the Clinical Evaluation of Language Functions (CELF), the Goldman-Fristoe Woodcock Auditory Skills Battery (GFWASB), the Goldman-Fristoe Woodcock Test of Auditory Discrimination (GFWTAD) and the Staggered Spondaic Word Test (SSW) (Keith, 1986). A low score on CELF suggests poor language processing and production skills in children. Lowered performance on GFWASB may indicate difficulties with memory for sound sequence, sound-symbol association and sound mimicry. Poor performance on the GFWTAD implies difficulties with speech-sound discrimination. Finally, a low score on SSW indicates general central auditory dysfunction.

By comparing SCAN to the above general measures of language and auditory processing skills, SCAN was considered

to be a valid test of these skills (Keith, 1986). Therefore, in relation to other better known auditory tasks, a SCAN lower score suggests language difficulties, reduced auditory discrimination abilities and abnormal maturation of the auditory system.

Not only does Competing Words seem to be the most important in differentiating across prenatal smoking categories, it was also the subtest that differentiated between groups constructed from the main control category of prenatal cigarette use, when the role of prenatal and postnatal passive smoke exposure was analyzed.

Five analyses were carried out in order to observe the role of passive exposure to cigarette smoke (pre and/or postnatal) using the control group of the main sample to isolate passive smoke exposure from direct, prenatal smoke exposure due to the active smoking habits of the mother. The first analysis made comparisons between a prenatal and postnatal passive smoke exposure group, a postnatal only passive smoke exposure group and a no passive (pre or postnatal) smoke exposure group. This analysis used the father's prenatal smoking habits as an indicator of prenatal passive smoke exposure. The father's prenatal smoking habits were used because of the small time interval between the father actually smoking and recall of the event (i.e., this information was recorded during the mother's pregnancy) thereby reducing recall error.

The second analysis made comparisons between a prenatal

and postnatal passive smoke exposure group, a prenatal only passive smoke exposure group and a no passive smoke exposure group. This analysis, along with the following ones, used the prenatal passive smoke exposure item of the Passive Smoke Exposure Questionnaire instead of only the father's prenatal smoking habits in order to include other sources of passive cigarette smoke exposure (e.g., at work, friends, relatives, etc.). The third analysis compared a pre and/or postnatal passive smoke exposure group with a no passive smoke exposure group. The fourth analysis compared a postnatal only passive smoke exposure group with a no postnatal passive smoke exposure group regardless of prenatal passive smoke exposure. Finally, the fifth analysis compared a prenatal only passive smoke exposure group with a no prenatal passive smoke exposure group regardless of postnatal passive smoke exposure.

Based on the above analyses, passive smoke exposure seems to affect a child's performance on SCAN. Subjects exposed to prenatal and/or postnatal passive cigarette smoke scored significantly lower on Competing Words than subjects who were not exposed to any cigarette smoke. Further, prenatal passive smoke exposure seems to be more important in affecting the auditory processing system than postnatal exposure. In all of the statistically significant differences noted between passive smoking groups, prenatal passive smoke exposure was associated with the lowest scores on SCAN outcomes. Postnatal passive smoke exposure was only noted to be associated with

lower scores on SCAN when it was combined with prenatal smoke exposure.

Consistent with the above trend, the present study observed that prenatal passive exposure to cigarette smoke significantly contributed to removing the effect of maternal prenatal cigarette smoke exposure at the multivariate level, while postnatal passive smoke exposure failed to even meet the statistical criteria for potential covariates. This is additional evidence to suggest that prenatal passive cigarette smoke exposure is more important in contributing to auditory processing anomalies than postnatal passive exposure.

This implies that the developing auditory system is more vulnerable at the fetal stage than at the newborn and beyond stage of life. It is possible that normal fetal auditory system development requires a very set, supportive environment. During the fetal stage, mechanisms may not be developed enough to protect the auditory system from putatively harmful agents that have penetrated into the fetus' system. As well, due to the greater degree of change going on at the fetal stage of life (compared to postnatal life stages), the fetus' natural auditory system development may be more easily modified or altered by potentially damaging agents (e.g., nicotine, CO). A more static system may not be as easily affected. The above observations add to the growing amount of evidence that portends prenatal exposure to toxic agents to be related to interfering with

Resolution test chart showing patterns of vertical and horizontal lines with numerical labels: 1.0, 1.1, 1.25, 1.4, 1.6, 1.8, 2.0, 2.2, 2.5, 2.8, 3.0, 3.2, 3.6, 4.0, 4.5.

MICROCOPY RESOLUTION TEST CHART
NATIONAL BUREAU OF STANDARDS
STANDARD REFERENCE MATERIAL 1010a
(ANSI and ISO TEST CHART No. 2)

the natural auditory system development of the fetus (Dietrich et al., 1992; Weitzman et al., 1992; Sexton, 1990; Kristjansson et al., 1989).

Studies that cite the possible role of postnatal passive cigarette smoke exposure in affecting behavior (Weitzman et al., 1992) and contributing to the development of auditory problems (Strachan et al., 1989; Kraemer et al., 1983) may not be "telling the whole story". Based on the results of this study, it seems at least as important, if not more important, to account for prenatal passive cigarette smoke exposure as well. However, these results ought to be interpreted conservatively due to the small sample sizes that existed in the various groups. As well, because relevant data could not be obtained due to resource limitations (e.g., home air quality, metabolism of each child, etc.), further analyses controlling for potential covariates were not carried out.

Competing Words was the only SCAN item to differentiate between passive smoke exposure categories in three of the four statistically significant analyses: i) pre and postnatal passive smoke exposure compared to both prenatal only and no passive exposure, ii) pre and/or postnatal compared to no passive smoke exposure, iii) prenatal compared to no prenatal smoke exposure. In the remaining statistically significant analysis (pre and postnatal passive smoke exposure compared to postnatal only and no passive smoke exposure), Filtered Words was the only SCAN item to detect differences among

passive smoke exposure categories. This was the only case in which a SCAN item other than Competing Words was statistically significantly related to passive or direct, prenatal smoking categories.

In all cases in which Competing Words has been significantly related to smoke exposure categories, the lowest scoring group's mean was below the normed subtest standard score mean for Competing Words (i.e., 10). The case in which Filtered Words was significantly related to passive smoke exposure groups, the lowest scoring group's (i.e., prenatal and postnatal passive smoke exposure group) mean (i.e., 10.11) was above the normed subtest standard score mean for Filtered Words (i.e., 10). Since no group in this analysis was performing below average, a passive smoke exposure effect would not seem to be an appropriate explanation. This may suggest that the significant finding noted with Filtered Words was spurious.

A prominent pattern noted with the light prenatal cigarette use category was that the mean standard scores for all three SCAN subtests were more similar to the mean standard scores for the no-use than the heavy prenatal cigarette use category. This suggests that prenatal exposure to light levels of cigarette smoke (<16mg/day of nicotine) contributes only marginally, if at all, to auditory processing problems. Significant auditory problems may only develop through heavy (≥ 16 mg/day of nicotine) prenatal cigarette smoke exposure.

A threshold may exist in terms of observing auditory processing anomalies by way of prenatal smoke exposure. Below a certain level of prenatal exposure to nicotine per day, the developing central auditory system may not be affected and/or is able to defend against any changes in natural development. Once this threshold is surpassed, any mechanisms (in the mother and/or fetus) that may have existed to ensure natural development and protect against any aberrations become overwhelmed and the auditory system's natural development is interfered with.

It is interesting to note that based on the percentage correct for normative scores of the SCAN subtests, children, in general, find the three subtests to vary according to difficulty. From ages six to eleven, the mean percentage correct for each subtest is: Filtered Words, 83%; Auditory Figure Ground, 81%; Competing Words, 78%. Competing Words requires the listener to perform two tasks at once. A subject must listen to a word spoken in one ear and, at the same time, listen to a different word spoken in the other. The other two subtests demand the use of only one ear at a time.

Based upon the above observations, it may be theorized that since Competing Words seems to be the most difficult task of the SCAN subtests, it can detect subtle differences between the smoke exposure categories and poses the greatest problem for children exposed to heavy amounts of cigarette smoke in utero who may already be at a disadvantage. Further, the maturational development of the auditory system in these

children may be delayed. Children prenatally exposed to cigarette smoke, in general, seem to have no difficulty with Filtered Words and Auditory Figure Ground subtests possibly because their auditory systems are mature enough to perform at normal standards. However, Competing Words may require a more mature auditory system than the other two subtests, based on its difficulty level, potentially causing these children to perform below normal on Competing Words.

The following theories are put forth in an attempt to explain the negative effects of prenatal cigarette smoke exposure on central auditory processing noted in this study. Competing Words subtest is a dichotic listening task designed to assess the auditory maturation or developmental level of children. Keith (1986) theorizes that it may probe the integrity of the auditory pathways of the CNS. Therefore, one theory that may explain how the auditory pathways are affected by prenatal cigarette smoke exposure is based on possible effects prenatal nicotine exposure has on the prenatal development of auditory system cells and pathways.

Recent studies have found nicotinic binding sites on the outer hair cells of the organ of Corti in the cochlea (Eybalin and Pujol, 1987). In addition, the olivocochlear bundle (centrifugal pathway sending axons to inner and outer hair cells from the superior olive) is known to utilize acetylcholine (ACh) as one of its neurotransmitters (Pickles, 1988). Since nicotine is a cholinomimetic, it has been argued that maternal prenatal cigarette smoke exposure may affect

the natural development of the olivocochlear bundle and the outer hair cells by prematurely eliciting receptor stimulation resulting in premature onset of cell differentiation at the expense of replication (Slotkin, 1989). This would lead to deficits in cell number and normal patterns of synaptogenesis and synaptic activity.

The above hypothesis pertaining to the effects of fetal stimulation of nicotinic receptors in the auditory system has been supported by a number of studies. In an experiment by Slotkin et al. (1987), the effects of 16 days of prenatal nicotine exposure in rats were examined. The researchers observed that cholinergic nerve cell replication had decreased while cell differentiation had increased in the brains of rats prenatally exposed to nicotine. This process eventually led to deficits in cell numbers. Other studies observed similar outcomes (McFarland et al., 1991). Zahalka et al. (1992) observed parallel results in rats prenatally exposed to nicotine at doses that did not cause overt signs of maternal, fetal or neonatal toxicity or growth impairment.

Another hypothesis is based on the role of carbon monoxide (CO) (present in cigarette smoke) and how it affects the functioning of the stria vascularis. The stria vascularis is located in the scala media which is one of the three fluid chambers of the cochlea and is responsible for producing endolymph (the type of fluid filling this particular chamber) and the positive endocochlear potential (Bridgeman, 1988).

Prenatal cigarette smoke exposure may affect the

development of the stria vascularis, which is 12 times more metabolically active than any other cochlear structure (Pickles, 1988), through anoxia. Anoxia would occur due to blood hemoglobin's higher affinity for CO than oxygen. Since metabolic activity requires increased oxygen levels, anoxia (i.e., state of decreased oxygen levels) would most likely interrupt systems or structures that have the greatest demand for oxygen, thus making the stria vascularis particularly vulnerable. This would affect the endocochlear potential which, in turn, would affect normal ion flow from the endolymph into the stereocilia (extensions of the hair cells that are surrounded by endolymph) which would finally result in an abnormal transduction process following intracellular depolarization. Again, the type of affect that would result in terms of auditory processing is speculative.

Keith (1986) describes the Filtered Words subtest as one that assesses children's ability to understand distorted speech. The stimulus is filtered in such a manner that some of the acoustic spectrum is absent, requiring the child to "fill in" missing information in order to comprehend the word.

One reason that the present study did not observe any statistically significant results with Filtered Words may have been due to the design (e.g., difficulty level) of the subtest. It may not have been sensitive enough to detect differences between the three smoking groups. This may have been the case because a dose response relationship was noted

with Filtered Words. Across the three prenatal smoke exposure categories, scores on Filtered Words decreased as prenatal exposure to cigarette smoke increased. This linear relationship may imply that the ability to understand distorted speech in children prenatally exposed to cigarette smoke is subtly impaired, but not to the extent to which this particular test could reveal statistically significant differences. This potential subtle impairment is consistent with the hypothesis previously set forth pertaining to how prenatal cigarette smoke exposure could affect the auditory system (i.e., "stria vascularis" argument, p. 85-86).

Another possible reason for not detecting a prenatal cigarette smoke exposure affect with Filtered Words deals with power. This study was estimated to have 80% power to detect an effect size of .3 of a standard deviation. Therefore, an important negative effect between prenatal cigarette smoke exposure and auditory processing may have occurred with Filtered Words but was not detected due to the design of this investigation (e.g., insufficient power).

Keith (1986) describes the Auditory Figure Ground subtest as one designed to assess children's ability to listen or perform academically when competing environmental noises are present. In this subtest, Children must ignore background conversation (speech babble) and attend to the word being spoken in order to correctly repeat the word.

Though not statistically significant, a dose response relationship was observed between prenatal cigarette smoke

exposure and performance on Auditory Figure Ground. One explanation as to why no statistically significant differences were observed between prenatal cigarette use categories based on Auditory Figure Ground scores, could have been due to the design of the subtest (e.g., difficulty level). Auditory Figure Ground may not have been sensitive enough to distinguish between the prenatal cigarette use categories.

A subtle deficit in the ability to listen when competing environmental noises are present may actually exist in children who were exposed to cigarette smoke in utero, but not to the extent to which this subtest could reveal statistically significant differences. As well, similar to an explanation given above for Filtered Words, insufficient power in this study to detect effect sizes smaller than .3 of a standard deviation could have contributed to not detecting a prenatal cigarette smoke exposure affect with Auditory Figure Ground. This potential subtle impairment in the ability to listen when competing environmental noises are present could be explained based on the two hypotheses previously set forth pertaining to how prenatal cigarette smoke exposure could affect the auditory system (i.e., "nicotinic receptor" and "stria vascularis" arguments, pp. 84-86).

However, as already noted, the Filtered Words and Auditory Figure Ground subtests did not detect any statistically significant differences between the three smoking groups.

Therefore, it may be argued that, based on the present study's findings, prenatal cigarette smoke exposure does not affect children's ability to perform these two auditory processing tasks.

Potential Covariates

When potential covariates were controlled for at the multivariate level (linear combination of Auditory Figure Ground and Competing Words), the statistically significant difference observed between the prenatal smoking categories was removed. The averaged education of both parents, prenatal passive cigarette smoke exposure (previously discussed) and prenatal excessive noise exposure all appear to significantly contribute to the differences noted between the maternal prenatal cigarette smoke exposure groups. Children who scored lower on the SCAN subtests were more likely to have parent's with lower education and mothers who were exposed to passive cigarette smoke and excessive noise during pregnancy.

Lower education has consistently been related to the heavy smoking group throughout OPPS investigations (Fried and Watkinson, 1990; Makin et al., 1991). What type of conclusions can be drawn from this trend, in terms of the present study's focus, is uncertain, since auditory processing is a CNS function and its vulnerability to the parents' level of education is speculative. Even though the experimenter felt that all the subjects understood the test instructions well enough to properly perform the SCAN, there still may have been a degree of misunderstanding or

uncertainty in some children that even the children were unaware of. This misunderstanding or uncertainty, possibly related to a lower I.Q. which is often associated with individuals from families of limited education, could have contributed to lower SCAN scores.

The mother's exposure to excessive noise during pregnancy was also a statistically significant covariate. The data for this variable was obtained from one question in the Auditory History Questionnaire. The mother of the child was asked whether she had been regularly exposed to excessive amounts of noise (e.g., loud music, machinery, etc.) during pregnancy. However, an affirmative answer to this question is difficult to interpret in terms of the fetus' auditory system development.

Stimulation of the fetus' auditory system comes primarily from internal sources (e.g., mother's heartbeat) (Bernard, 1993). The children in this study were most likely not directly stimulated by external noise in utero due to the fact that sound entering the fetal compartment from outside of the mother is attenuated by up to 95 dB (e.g., approximately the level of sound made by a train at full speed) (Bernard, 1993). This attenuation of sound is thought to come about because of the fluid in the uterus.

Since the fetus is therefore unlikely to be stimulated to any great degree by sound from the external environment, one possible explanation as to how a pregnant mother's exposure to excessive noise may affect a fetus' auditory system is

based on the mother's physiological responsiveness to external noise. The mother's exposure to excessive noise could conceivably have created a subtly, louder internal environment by increasing the mother's physiological activity (e.g., varying heart rate) until adaptation of the mother's system to the sound occurred. This change in the mother's physiological activity could then have stimulated the fetus' auditory system more so, or in a different manner (e.g., variation in the pattern of stimulation) than in fetus' of mother's not exposed to excessive noise.

Considering the above hypothesis (i.e., mother's prenatal exposure to excessive noise indirectly affects fetus' auditory system), the findings from the mother's exposure to excessive noise during pregnancy variable support an argument set forth in the introduction that describes the auditory system to be more vulnerable for developing negative effects than other sensory systems. This potential vulnerability is founded on the idea that the longer a system is metabolically active during pregnancy and is utilizing contaminated blood (e.g., CO, nicotine) its natural development will be interfered with more so than a system that becomes metabolically active later on.

The hypothesis is based on studies that have observed the auditory system to respond to stimuli earlier and, therefore, for a longer period of time during pregnancy than the visual system (Hofer, 1981) and the fact that auditory stimuli can more readily stimulate the fetus than visual stimuli can.

Therefore, it is argued that, more than any other system, the natural prenatal development of the auditory system could be interfered with through prenatal exposure to cigarette smoke.

This type of exposure would increase the amount of contaminated blood (CO and nicotine) feeding the metabolically active auditory system, decreasing the amount of oxygen (due to hemoglobin's higher affinity for CO) that is needed to sustain metabolic activity and potential development. A mother's exposure to excessive noise during pregnancy could conceivably increase her physiological activity (e.g., varied heart rate) which could, in turn, increase, or vary, the metabolic activity of a fetus' auditory system. This would also increase the amount of CO going to the auditory system of the fetus, potentially interfering with the auditory processing system's natural development (previously discussed in more detail, p. 85-86).

Increased levels of nicotine in the blood supply of the fetus' auditory system, by way of increased metabolic activity, could also interfere with the natural development of the auditory system. Due to nicotine's cholinomimetic properties, nicotine has been observed to stimulate the auditory system's nicotinic receptors (previously discussed in more detail, pp. 84-85). This has been observed to bring about a premature onset of cell differentiation and a decrease in cell number (Slotkin, 1989).

Passive Smoke

In general, urinary cotinine/creatinine levels in children

were noted to be low (Perkins, 1992). Consequently, it was concluded that no children in the study were actively smoking (at least within the past three days of the test). As well, the low levels of cotinine/creatinine observed in children's urine implied that, on average, children who were passively exposed to cigarette smoke were exposed to low levels of smoke. The mean level of urine cotinine/creatinine in childrer exposed to passive cigarette smoke was .91 ug/mmol with a range of 0 to 3.54. In a study by Wall et al. (1988), the mean level of urine cotinine/creatinine in subjects exposed to passive cigarette smoke was 6.3 ug/mmol with a range of 0 to 17.2.

One likely reason why the urine cotinine/creatinine results in this study differed so much from the results of the Wall et al. (1988) study could be because of the differences in the type of subjects used in each study. The present study tested children while Wall et al. (1988) looked at adults. Adults, more so than children, engage in activities in locations where passive smoke exposure is inevitable (e.g., night clubs, pool halls, parties, office, etc.). The only passive smoke that children come in contact with is usually in-home exposure of limited amounts compared to adult sources of passive smoke.

Wall et al. (1988) argued that although considerable intersubject overlap occurred (also occurring in the present study), adjustment of urine cotinine levels for urine creatinine concentration afforded the best separation of a

group of nonsmokers from passive smokers. Researchers agree that substantial numbers of supposedly unexposed nonsmokers have detectable levels of cotinine in their urine but that levels in the urine of passive smokers tend to be higher (Wall et al., 1988; Jarvis et al., 1984).

The levels of cotinine/creatinine observed among those children exposed to passive cigarette smoke, in the present study, were also lower than other reports in the literature pertaining to school-age children (Casale et al., 1991; Jarvis et al., 1992). The difference in cotinine/creatinine levels in this study compared to others could be due to the differences between research methodologies. Some studies failed to adjust urine cotinine levels for urine creatinine concentrations while other studies looked at cotinine concentrations in fluids other than urine (e.g., saliva). However, the low levels of cotinine/creatinine noted in this study are probably not due to the above since the urine cotinine (unadjusted for creatinine) levels in this study were greater than the saliva cotinine levels noted in Jarvis et al. (1992). Due to physiological processes, saliva cotinine levels are usually lower than urine cotinine levels within a subject (Perkins, 1992).

Cotinine detection study methodologies vary in other respects as well. Researchers use different biochemical methods (e.g., radioimmunoassay vs gas chromatography) to measure for cotinine which sometimes give different results (Biber et al., 1987). As well, few investigations use similar

criteria for defining a passive smoking group. No study looking at urine cotinine was found to have the same type of subjects (school-age children), definition of passive smoke group and biochemical method of detecting cotinine as the present study.

Besides the variability in methodologies across studies, other reasons for the potentially low levels of urine cotinine/creatinine observed in the present study relate to the season during which testing took place. Jarvis et al. (1992) observed that cotinine levels were statistically significantly higher in children who were tested during the winter than in children who were tested during the spring. In the present study, children were tested during the latter part of spring and throughout the summer. For a majority of families, the norm was to keep windows and doors open during the day and into the night to enjoy the good weather. Consequently, indoor/outdoor air exchange was quite high, most likely reducing the amount of smoke in the house. As well, due to the season in which testing occurred, most children spent very little time indoors, thereby reducing the amount of indoor contact they may have had with a smoker. Had the season been winter or fall, higher urinary cotinine/creatinine levels may have been recorded.

Another interesting observation that may have led to low levels of cotinine/creatinine in children's urine was the negative view most children held toward smoking. Many parents told the experimenter that their children protested whenever

they attempted to smoke in front of their child. Consequently, either the child or the parent would leave the room.

Based on the correlation results between the cotinine/creatinine findings and the two relevant items of the Passive Smoke Exposure Questionnaire (i.e., 1. past three day [from day questionnaire was administered] total hours of child's passive cigarette smoke exposure and 2. the time interval between the last recalled smoke exposure period of the child and the time the urine sample was collected), the questionnaire seems to be a good estimate of passive smoke exposure. The results showed that urinary cotinine/creatinine concentrations increased significantly as parent-reported child exposure increased. These findings support studies using similar analyses (Knight et al., 1989). Based on these results, parent-report, in this study, seems to be reliable to a significant extent.

It is interesting to note that of the two items from the Passive Smoke Exposure Questionnaire, the child's three days of cigarette smoke exposure was more highly related to the cotinine/creatinine results than the time interval between smoke exposure and producing the urine sample. One possible reason for this may be due to the fact that the former item deals with hours of exposure to passive cigarette smoke while the latter does not deal with a quantity (i.e., total hours of cigarette smoke exposure) but, rather, deals with an estimated time interval. Therefore, the latter item cannot

serve as a surrogate for levels of cotinine/creatinine in the urine whereas the former item can.

Unfortunately, the Passive Smoke Exposure Questionnaire did not account for all of the variance in the cotinine/creatinine results ($r=.71$). The missing variance may be due to the inherent weaknesses in the questionnaire. Though shown to be quite reliable in this study, parent-report or self-report is prone to bias and errors of recall. For example, the parents' estimate of their child's level of cigarette smoke exposure from sources outside of the home would likely be subject to the most inaccuracies since their knowledge of their child's activities outside of the home would, naturally, not be as great as inside of the home. A three day log of a child's activities, personal contacts and level of cigarette smoke exposure completed by the parents during each day could have improved recall accuracy. Methods used to detect cotinine/creatinine in urine are also not perfect. Wall et al. (1988) points out that one reason for this may be intersubject variance in nicotine metabolism.

An important contribution made by the Passive Smoke Exposure Questionnaire is its consideration of outside sources (i.e., smokers who do not live at the child's residence). In an analysis to measure the importance of outside sources of passive smoke, only parental and other residential sources were correlated with the cotinine/creatinine results. This decreased the correlation between the cotinine/creatinine results and the child's past

three days of passive smoke exposure from $r=.71$ to $r=.67$. Therefore, it does seem meaningful to consider outside sources of passive smoke exposure.

Another important contribution to the analysis of passive smoke exposure made by the questionnaire deals with the type of research question being asked. The questionnaire, unlike biochemical measures, estimates a child's amount of passive cigarette smoke exposure (hours) during a typical week. As well, the questionnaire estimates the child's history of passive smoke exposure from birth to present. Biochemical measures, in general, can estimate levels of passive smoke exposure for only a very limited time span (e.g., three days in this study).

Based on the above discussion, then, future studies interested in estimating levels of passive smoke exposure not only in terms of recent exposure but in terms of a subject's history of exposure as well, should incorporate both questionnaires and biochemical measures. Also, using both methods of estimating passive smoke exposure provides a reliability check on self-report. Other studies have described the need to use biochemical measures in addition to questionnaires (Cook et al., 1993).

Makin et al. (1989) examined 91 children between the ages of six and nine years with a comprehensive neuropsychological test battery. Children prenatally exposed to cigarette smoke through the passive smoking habits of their mothers (as determined by a questionnaire) were more likely to score

lower than children who were not prenatally exposed to cigarette smoke on tests of speech and language skills, intelligence, visual/spatial abilities and on the mother's rating of behavior. The researchers argued that the effects were similar to low levels of prenatal cigarette smoke exposure through the active smoking habits of the mother (<16mg/day of nicotine).

Comparable to the above study, it would seem that the auditory processing effects of passive exposure to cigarette smoke noted in the present study were similar to the auditory processing effects of prenatal exposure to light levels of maternal cigarette smoke. This conclusion is based on observations that found the light prenatal cigarette use category (<16mg/day of nicotine) and the passive smoke exposure category of the controls to be similar on all four outcomes (i.e., three subtests and SCAN Composite). These two groups were also found to be similar on such sample characteristics as the averaged education of both parents, child's auditory history and prenatal marijuana and alcohol use.

The passive smoke exposure group was also found to be similar to the "pure" control (no type of cigarette smoke exposure) group on all of the above sample characteristics except for prenatal marijuana use. Mother's of children from the passive smoke exposure group were more likely to use marijuana during pregnancy than mother's from the "pure" control group.

The difference noted between the latter two groups on Competing Words, then, may have been due to prenatal marijuana use and not passive smoke exposure. However, since prenatal marijuana use did not satisfy the statistical criteria for covariates in the main analysis, this difference between the two groups may not be important in terms of contributing to auditory processing anomalies. No studies have related prenatal marijuana exposure to auditory processing problems in offspring. These observations imply, then, that the effects of passive cigarette smoke exposure prenatally and/or postnatally are similar to the effects of light, in utero maternal cigarette smoke exposure.

Potential Mediating Variables

Children were more likely to score lower on the SCAN subtests if they had received remedial help during school. This variable was not controlled for in the main analysis because of its potential mediating effects. Other studies have related prenatal cigarette smoke exposure to poor cognitive abilities and receptive language problems in children (Fried, 1992). The observation that prenatal cigarette smoke exposure is related to receptive language problems implies that an auditory component may be contributing to lowered cognitive skills.

Further to the above, Keith (1985) points out that children with auditory processing problems commonly exhibit academic difficulties as well which are, in many cases, related to an auditory processing disorder. Since the words

presented in SCAN are simple (i.e., monosyllabic) and very familiar to children (Keith, 1986), it is more likely that these children scored lower on the SCAN subtests due to actual auditory processing problems than to learning difficulties. A strong cognitive component does not exist within SCAN. Therefore, this would imply that children, in this study, who were prenatally exposed to cigarette smoke and have received remedial help in school have diminished auditory processing abilities and that these diminished auditory processing abilities did not arise from academic problems.

Another variable that was considered to have potential mediating effects was the child's history of auditory problems. Subjects who scored lower on the SCAN subtests were more likely to have had auditory problems during their lifetime. These included such auditory complications as frequent ear aches to otitis media.

While also including neurological disorders and genetic traits, Keith (1985) described early and frequent otitis media with effusion as one of a number of possible causes of auditory processing disorders. Given this and the findings in the present study, this implies that children who scored lower on SCAN, had a history of auditory complications and were prenatally exposed to cigarette smoke possessed an auditory processing deficit. In the case of these children, the auditory processing deficit was most likely mediated by postnatal auditory complications which could have come about

because of prenatal smoke exposure.

Research Limitations and Recommendations

The present investigation is a quasi-experimental design and, as such, is subject to a number of limitations. One of the most important weaknesses evident in this inquiry is the variance that occurred in the testing environment from house to house. It would have been ideal to have tested each child in the same room, away from distraction and ambient noise. However, due to a lack of resources and the design of the experiment, this was not possible. For example, since urine samples were required, having the experimenter come to the home was intended to make the procedure more comfortable for the child. As well, having the experimenter come to the subject's home allowed an appointment to be made at the participant's convenience. These measures were taken in order to make the children and parents in the study feel at ease with the whole procedure.

Although compared to almost all published reports, the present study considered an extremely large number of background variables, an additional limitation to this investigation is the inability of quasi-experimental designs to consider all potential covariates that actually exist within and between each subject. However, by considering the role of potential covariates that were chosen in this study, based on the literature and previous OPPS investigations, it was intended that the most important ones were taken into account. Other potential covariates were not taken into

account because they were either decided to be unimportant (e.g., child's I.Q.) or available resources did not allow for their measurement (e.g., children's metabolic rates, home air quality). A complete account of all possible covariates was, of course, beyond the means of this study.

Finally, one more limitation evident with this study pertains to statistical power which was previously discussed with reference to why Auditory Figure Ground and Filtered Words did not differentiate between the three prenatal cigarette use categories. The heavy prenatal cigarette use category was, inevitably (i.e., more non-smokers than smokers in the community), the smallest ($n=25$) of the three groups and contributed to limiting the study's statistical power. Increasing this groups size would obviously have strengthened the power of the present study.

Before subjects were actually tested for this study the experimenter performed ten practice sessions with volunteer families who were friends of a colleague of the OPPS lab. Practice sessions are generally recommended to allow the experimenter to become familiar with testing procedures and equipment function. More importantly, since children were being tested, it allowed the experimenter a chance to prepare for any problems that may have been unavoidable come the actual testing of subjects for the study.

For example, the experimenter noted, during the practice sessions, that since the testing was done in the home, the amount of visual stimulation could not be controlled causing

children (especially those under the age of nine) to become somewhat distracted by objects that entered their field of vision while performing the SCAN subtests. Some children attempted to touch and fondle the objects (e.g., coffee table leg, foot stool, house pet). To avoid having to examine the testing room of each home visited and possibly offending or inconveniencing parents, a blindfold was placed over the child's eyes. This greatly reduced distraction in further testing episodes. In addition, it increased most children's interest in participation by adding a certain level of excitement. Not being able to see for 25 minutes while listening to a tape was intriguing for many children.

Conclusion

Based on the above discussion and research findings, maternal prenatal cigarette smoke exposure appears to be associated with auditory processing in school-age children after controlling for potential covariates. As revealed by the SCAN Composite score, prenatal exposure to heavy levels of cigarette smoke (i.e., ≥ 16 mg/day of nicotine) seems to contribute to deficits in children's central auditory processing abilities. More specifically, as revealed by the Competing Words subtest, these auditory processing deficits seem to be related to problems with dichotic listening. These findings are in support of the literature (Saxton, 1978; Picone, 1982; Fried and Makin, 1987; Kristjansson et al., 1989). In addition, linear trends were noted between prenatal cigarette use categories and all three SCAN subtests as well

as the SCAN Composite.

Finally, prenatal passive cigarette smoke exposure seems to be negatively related to auditory processing, as well. In this study, postnatal passive smoke exposure did not differentiate among passive smoke exposure groups which may imply the importance of considering prenatal passive smoke exposure in future studies investigating the effects of postnatal passive smoke exposure and/or prenatal smoke exposure via the mother's active smoking habits on the auditory system.

Both the prenatal cigarette exposure and passive cigarette smoke exposure analyses have the prenatal aspect in common. The results of this study imply the importance of prenatal exposure to cigarette smoke in affecting central auditory processing. Though the effects of prenatal passive exposure do not seem to be as marked as prenatal "active" exposure to cigarette smoke, prenatal passive cigarette smoke exposure may be comparable to light levels (less than a pack of cigarettes a day) of prenatal "active" exposure. Therefore, based on the results of this study, not only is it important not to smoke during pregnancy if a mother is concerned about decreasing her future child's risk for developing auditory processing deficits, but it is important for the mother to not be exposed to passive cigarette smoke during pregnancy, as well.

In addition to parents' concerns about subtle decreases in auditory processing abilities in their children, there is the

further importance of considering the implications of such decreases. Keith (1986) includes poor receptive and expressive language, academic problems and attention problems in a list of variables connected to deficits in central auditory processing. It is interesting to note that along with subtle auditory processing deficits, children prenatally exposed to cigarette smoke, in this study, were more likely to have received remedial help in school.

However, since mean standard scores for the light prenatal cigarette use category were so similar to the no-use category for all three SCAN subtests, it is difficult to discern whether such a level of prenatal smoke exposure (<16mg/day of nicotine) influences the development of the central auditory system. Based on the present results, it seems doubtful.

It is important to note that the sample in this study was representative of a middle-class population and is, consequently, generally considered to be low-risk compared to lower-class populations. This may have contributed to the statistically non-significant findings observed in the MANCOVA. Similarly, the type of sample studied could have contributed to the lack of difference observed between prenatal cigarette smoking categories with Filtered Words and Auditory Figure Ground subtests. A supportive environment (more common in middle-class than lower-class) could potentially counteract any subtle auditory processing problems a child may be having. Likewise, the statistically significant effects of prenatal cigarette smoke exposure on

central auditory processing that were observed may be 111
enhanced in a lower-class population due to the lack of a
supportive environment.

It is important to keep in mind that the effect of any
single variable may not be a meaningful way to explain
auditory processing problems. Keith (1985) explains that
auditory processing problems are considered to result from a
variety of possible causes. Being subtle and multi-causal,
the potential effects of maternal prenatal cigarette smoke
exposure are extremely difficult to detect but may,
nevertheless, be one variable among many that inhibits a
child from reaching his/her potential.

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TABLE 1

Calibration Results for the Pure Tone Audiometer

Frequency (Hz)	Ideal (dB)	Actual (dB)
500	80 dB + 11.5 = 91.5	92
750	" + 8 = 88	91
1000	" + 7 = 87	89
1500	" + 6.5 = 86.5	88
2000	" + 9 = 89	89
3000	" + 10 = 92	92
4000	" + 9.5 = 89.5	92

*Results are acceptable within ± 3 dB of the ideal values (ANSI-69 threshold values).

TABLE 2

Partial Correlations between SCAN Raw Scores and Scores from Subtests of the CELF (Clinical Evaluation of Language Functions), GFWASB (the Goldman-Fristoe Woodcock Auditory Skills Battery), GFWTAD (the Goldman-Fristoe Woodcock Test of Auditory Discrimination) and the SSW (Staggered Spondaic Word Test) Administered to a Sample in Grades 1-3

Other Measures of Language and Auditory Development	SCAN			
	Filtered Words	Auditory Fig.-Grd.	Competing Words	Composite
CELF				
(n=29; df=26)				
Linguistic Concepts	.189	.120	.257	.264
Relationships and Ambiguities	.121	-.189	.221	.156
Oral Directions	.362	-.104	.357	.337
Spoken Paragraphs	.371	-.173	.367	.403*
Word Associations	.239	.101	.258	.273
Model Sentences	.279	.072	.426*	.401*
GFWASB				
(n=31; df=28)				
Memory for Sequence	.325	.248	.301	.358
Sound-Symbol Association	.368*	.287	.330	.399*
Sound Mimicry	.322	.268	.345	.394*
GFWTAD				
(n=31; df=28)				
Quiet	.001	-.041	-.044	-.040
Noise	.227	.227	.305	.331
SSW				
(n=31; df=28)				
Right Noncompeting	.086	-.108	-.012	.070
Right Competing	-.407*	-.343	-.572**	-.598**
Left Competing	-.365*	-.341	-.220	-.328
Left Noncompeting	.221	.268	.039	.142

* $p < .05$

** $p < .01$

(adapted from Keith, 1986)

TABLE 3
CHARACTERISTICS OF THE SAMPLE

	2	1	0
Prenatal Nicotine Exposure: Heavy		Light	None
(mg of nicotine /day)	$\geq 16\text{mg/day}$	$>0<16\text{mg/day}$	0mg/day
Sample Size:	25	44	41
Average Annual Income:	\$28 488	\$32 378	\$33 337
*Average Education of Both Parents:	2	2.67	2.9
Age in Months:	119	111	106

Females: n=48

Males: n=62

- * 2 represents the completion of high school
- 2.67 represents some years in university or college
- 2.9 represents the completion of a university or college program

Table 4 SCAN SUBTEST AND COMPOSITE MEAN STANDARD SCORES ACROSS
 PRENATAL NICOTINE USE CATEGORIES

FILTERED WORDS SS	PRENATAL NICOTINE USE			Total
	0	1	2	
Mean	11.41	11.4	10.72	11.25
Standard Deviation (std)	2.29std	2.3std	2.10std	2.23std
Cases	41	44	25	110
.....				
AUDITORY FIGURE GROUND SS	PRENATAL NICOTINE USE			Total
	0	1	2	
Mean	10.29	9.86	9.56	9.95
Standard Deviation (std)	2.55std	1.89std	1.78std	2.14std
Cases	41	44	25	110
.....				
COMPETING WORDS SS	PRENATAL NICOTINE USE			Total
	0	1	2	
Mean	10.46	10.31	8.68	10
Standard Deviation (std)	2.51std	2.52std	1.91std	2.48std
Cases	41	44	25	110
.....				
SCAN COMPOSITE SS	PRENATAL NICOTINE USE			Total
	0	1	2	
Mean	104.02	103	96	101.79
Standard Deviation (std)	11.95std	12.22std	9.5std	11.9std
Cases	41	44	25	110
.....				
0: NO SMOKE				
1: LIGHT SMOKE				
2: HEAVY SMOKE				

Table 5

SCAN SUBTEST MEAN STANDARD SCORES ACROSS
PASSIVE SMOKING GROUPS FOR CONTROLS

Filtered Words	1	2	3
Mean:	10.73	12.86	11.12
Standard Deviation (std):	1.53std	.9std	3std
Group Size:	15	7	16
.....			
Auditory Figure Ground	1	2	3
Mean:	10.47	10.14	10.37
Standard Deviation (std):	2.41std	2.27std	3std
Group Size:	15	7	16
.....			
Competing Words	1	2	3
Mean:	11.53	9.14	10.06
Standard Deviation (std):	2.38std	2.9std	2.08std
Group Size:	15	7	16
.....			
SCAN Composite	1	2	3
Mean:	107	102.14	102
Standard Deviation (std):	10.5	11.11	13.6
Group Size:	15	7	16
.....			

1: no passive smoke exposure

2: only prenatal passive smoke exposure

3: prenatal and postnatal passive smoke exposure

Table 6

A Screening Test for Auditory Processing Disorders

Robert W. Keith, Ph.D.

Name/I.D. No. _____ Date of Testing _____
 School _____ Date of Birth _____
 City _____ State _____
 Age _____ Grade _____ Sex _____ Chronological Age _____
 Examiner _____ Position _____
 Is English the child's primary language? Yes _____ No _____

Pure Tone Results

	500 Hz	1000 Hz	2000 Hz	4000 Hz
RE				
LE				

Tympanogram Results

	Pressure	Shape	Compliance
RE			
LE			

Middle ear pressure reported in mm H₂O or daPa.
 Tympanogram shape reported as peaked, rounded or flat.
 Compliance reported in cc equivalent volume.

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SCORING SUMMARY

Age _____	Raw Score	Normed Score	SS Confidence Range
Filtered Words			to
Auditory Figure Ground			to
Competing Words			to
SCAN Composite			to

STANDARD SCORE PROFILE

		SCAN Composite	Subtests				
			FW	AFG	CW		
+3 SD	145	.	.	.	.	19	+3 SD
+2 SD	130	.	.	.	.	16	+2 SD
+1 SD	115	.	.	.	.	13	+1 SD
MEAN	100	.	.	.	.	10	MEAN
-1 SD	85	.	.	.	.	7	-1 SD
-2 SD	70	.	.	.	.	4	-2 SD
-3 SD	55	.	.	.	.	1	-3 SD

ANALYSIS OF SUBTEST DIFFERENCES				COMPETING WORDS EAR ADVANTAGE			
Mean Subtest Score (from SSN)	FW	AFG	CW	Right-Ear-First Task		Left-Ear-First Task	
Difference from Mean (Subtest SSN Mean)				RE Correct		RE Correct	
Difference Required for Significance				LE Correct		LE Correct	
Difference Significant at What Level				Ear Advantage		Ear Advantage	
				REA	LEA	REA	LEA
				Prevalence	%	Prevalence	%

* Use the table below to find the differences required for significance when comparing each SCAN Subtest Standard Score with the Mean Standard Score. Consider an obtained difference Not Significant (NS) if smaller than value in table.

AGE	Filtered Words		Auditory Figure Ground		Competing Words	
	p .05	.01	.05	.01	.05	.01
3	1.9	2.4	1.7	2.1	1.4	1.7
4	2.9	3.6	2.2	2.8	1.8	2.2
5	3.2	3.9	3.5	4.2	2.4	2.9
6	2.9	3.6	2.8	3.5	2.2	2.7
7	3.0	3.7	3.2	3.9	2.3	2.8
8	3.1	3.9	3.3	4.0	2.5	3.1
9	3.2	4.0	3.5	4.2	2.6	3.2

Subtest 1 Filtered Words

IMPORTANT: Make certain that headphones are placed over the correct ears.

DIRECTIONS: Words are presented to the right ear first. Circle the + for words repeated correctly. Circle the - for words repeated incorrectly. *Optional:* Mark a line through words omitted and write substitutions on the line beside the word.

RIGHT EAR				LEFT EAR			
a. that	+	-	_____	c. man	+	-	_____
b. white	+	-	_____	d. room	+	-	_____
<hr/>							
1. had	+	-	_____	21. those	+	-	_____
2. did	+	-	_____	22. grew	+	-	_____
3. need	+	-	_____	23. air	+	-	_____
4. own	+	-	_____	24. mouth	+	-	_____
5. leave	+	-	_____	25. low	+	-	_____
6. you	+	-	_____	26. great	+	-	_____
7. on	+	-	_____	27. such	+	-	_____
8. may	+	-	_____	28. hot	+	-	_____
9. find	+	-	_____	29. wide	+	-	_____
10. if	+	-	_____	30. duck	+	-	_____
11. yes	+	-	_____	31. card	+	-	_____
12. while	+	-	_____	32. way	+	-	_____
13. most	+	-	_____	33. put	+	-	_____
14. bad	+	-	_____	34. five	+	-	_____
15. true	+	-	_____	35. box	+	-	_____
16. no	+	-	_____	36. ride	+	-	_____
17. ship	+	-	_____	37. hit	+	-	_____
18. lay	+	-	_____	38. is	+	-	_____
19. them	+	-	_____	39. sing	+	-	_____
20. wait	+	-	_____	40. tree	+	-	_____

TOTAL Right Ear

TOTAL Left Ear

Subtest 2 Auditory Figure Ground

IMPORTANT: Make certain that headphones are placed over the correct ears.

DIRECTIONS: Words are presented to the right ear first. Circle the + for words repeated correctly. Circle the - for words repeated incorrectly. *Optional:* Mark a line through words omitted and write substitutions on the line beside the word.

RIGHT EAR				LEFT EAR			
a. race	+	-	_____	c. his	+	-	_____
b. cat	+	-	_____	d. turn	+	-	_____
<hr/>							
1. all	+	-	_____	21. sheep	+	-	_____
2. back	+	-	_____	22. loud	+	-	_____
3. end	+	-	_____	23. hurt	+	-	_____
4. take	+	-	_____	24. pass	+	-	_____
5. coat	+	-	_____	25. bee	+	-	_____
6. me	+	-	_____	26. drop	+	-	_____
7. gray	+	-	_____	27. quick	+	-	_____
8. case	+	-	_____	28. nest	+	-	_____
9. thick	+	-	_____	29. thank	+	-	_____
10. sell	+	-	_____	30. sled	+	-	_____
11. next	+	-	_____	31. frog	+	-	_____
12. got	+	-	_____	32. park	+	-	_____
13. path	+	-	_____	33. neck	+	-	_____
14. bag	+	-	_____	34. bus	+	-	_____
15. day	+	-	_____	35. shop	+	-	_____
16. feet	+	-	_____	36. key	+	-	_____
17. rain	+	-	_____	37. fat	+	-	_____
18. fair	+	-	_____	38. shoe	+	-	_____
19. waste	+	-	_____	39. tall	+	-	_____
20. ball	+	-	_____	40. feed	+	-	_____

TOTAL Right Ear

TOTAL Left Ear

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Subtest 3 Competing Words

IMPORTANT: Make certain that headphones are placed over the correct ears.

DIRECTIONS: Circle the + for words repeated correctly. Circle the - for words omitted or repeated incorrectly. Credit is given for words accurately repeated even if only one word of a pair is given or if both words are repeated in reverse order.

RIGHT-EAR-FIRST TASK

a. low + - smile + -
b. else + - bad + -

LEFT-EAR-FIRST TASK

c. is + - put + -
d. true + - great + -

1. drop + -	hen + -	1. turn + -	did + -
2. shoe + -	fat + -	2. cake + -	pig + -
3. waste + -	cage + -	3. most + -	ball + -
4. room + -	leave + -	4. end + -	new + -
5. need + -	case + -	5. bus + -	fair + -
6. if + -	take + -	6. pass + -	seed + -
7. may + -	them + -	7. fall + -	card + -
8. feed + -	path + -	8. fresh + -	bar + -
9. five + -	while + -	9. laugh + -	dress + -
10. large + -	find + -	10. park + -	suit + -
11. feet + -	thank + -	11. lay + -	rain + -
12. dog + -	thick + -	12. ride + -	gray + -
13. dark + -	hot + -	13. fire + -	you + -
14. face + -	long + -	14. name + -	bank + -
15. show + -	clown + -	15. fish + -	door + -
16. race + -	home + -	16. year + -	tie + -
17. bag + -	day + -	17. hide + -	knee + -
18. sell + -	fly + -	18. shake + -	car + -
19. white + -	get + -	19. joy + -	pond + -
20. dad + -	on + -	20. wide + -	use + -
21. are + -	cow + -	21. yes + -	as + -
22. few + -	men + -	22. mouth + -	grew + -
23. tree + -	box + -	23. back + -	cat + -
24. that + -	bee + -	24. air + -	camp + -
25. had + -	key + -	25. duck + -	ship + -

Right Ear

Right Ear

Right
Ear
Total

Left Ear

Left Ear

Left
Ear
Total

Subtest Total (Right Total + Left Total)

1. Hello, may I speak to ----- (potential subject's mother) please?
2. Hi, my name is Joel McCartney and I'm working with Peter Fried in the Ottawa Prenatal Prospective Study. Do you recall the study?
3. Confirmation and/or brief description by caller.
4. Do you have a moment to talk? (yes - continue; no - best time to call back)
5. The present study that I'm involved in is looking at the effects of prenatal nicotine exposure on the central auditory processing system of children. I'm phoning you to ask if you and your child would like to participate in this new aspect of the OPPS?
6. Response (if no, go to 7).
7. Alright then, thank you for your time. (END)
8. The entire assessment of your child(ren) would be done in your home at your own convenience. The procedure involves a brief pure tone hearing test and a central auditory processing test which would last no more than 40 minutes altogether. No discomfort will be experienced. Due to the nature of the two tests a quiet room would be required away from distraction. In addition to the tests, I would be bringing two brief questionnaires for you to answer concerning the amount of passive smoke your child(ren) is/are exposed to and their auditory health. As well, I need you to assist your child, if necessary, in obtaining two very small urine samples in order to analyze the metabolite cotinine which can reveal how much cigarette smoke a person is exposed to.
9. Response (if yes, go to 11).
10. Alright then, thank you for your time. (END)
11. Could you try to make sure that your child(ren) do(es) not go to the bathroom for at least 1.5 hours before I arrive.
12. Could you also think of the most quiet room in your home that would be best for the assessment, please.
13. What is your exact address?

14. Child(ren)'s name(s): Age:
15. Make appointment:
16. My phone number: (h) 828-6728, (o) 788-2600 Ext. 2677
Peter Fried: 788-2600 Ext. 2676

INFORMED CONSENT (parent)

I acknowledge that I have been informed about the nature of the study to be conducted by Joel McCartney, a Masters graduate student, under the direction of Peter Fried, Ph.D.. I understand that the purpose of this study is to examine central auditory processing in children prenatally exposed to a variety of environmental factors including cigarette smoke. Two tests will be given to my child(ren): 1) a standard pure tone hearing test and 2) a central auditory hearing test. This latter test is called the SCAN (a screening test for auditory processing disorders). It is made up of three subtests in which one syllable words are presented to my child(ren) through earphones under three different conditions: 1) part of the speech sound removed, 2) background noise and 3) competing words (i.e. different words presented to each ear at the same time). My child(ren)'s task is to repeat the words as best they can. As well, I will participate with the experimenter to complete two questionnaires: 1) inquiring into the level of passive smoke my child(ren) is/are exposed to and 2) my child(ren)'s auditory health. I also understand that I will assist my child(ren), if necessary, in obtaining two very small urine samples in order that the experimenter may analyze for a cigarette smoke metabolite (i.e., cotinine). All testing will

take place in my own home.

• No foreseeable risk or discomfort will be experienced by the child. My child(ren) will be tested for only approximately 40 minutes. The entire procedure will take approximately 1.5 hours.

As in the past, all information will be treated in an entirely confidential manner. Feedback will be provided orally by Peter Fried.

If I have any questions pertaining to the research I can contact Peter Fried (director) 788-2600 ext. 2676, L. Paquet (Chair of the Ethics Committee) 788-2600 ext. 2689 or W. Jones (Chair of the Department) 788-2600 ext. 2648 at the Department of Psychology, Carleton University, Ottawa.

I understand the procedures to be followed. My child's participation is totally voluntary and I am willing to participate in this research knowing that, as in the past, I and my child am free to withdraw from the study at any time without penalty.

Signed _____ (parent or guardian)

Print Name _____

Date _____

Witness _____

Appendix E

INFORMED CONSENT (child)

I have been told about the study that I am participating in. The experimenter's name is Joel McCartney. He is a university student and his teacher is Peter Fried, the "boss" of the study I'm participating in. I understand that this study is examining an aspect of hearing. First, I will be listening to tones and telling Joel if I hear them or not. Then I will be listening to a man say words that I will try to repeat aloud. These words will be hard to hear. I realize that these two "games" will last about 40 minutes altogether. Also, Joel will be collecting two very small urine samples from me. If I need help doing this, I can ask my mother or father.

I know that I can stop during the "games" anytime I feel I have to. If I decide I don't want to participate, I can end the game. No one will be upset because it's up to me if I want to do it or not.

If I have any questions I can ask Joel or my mother or father.

Signed_____

Print Name_____

Date_____

Witness_____

Appendix D

INFORMED CONSENT (older child)

I have been informed about the study (Ottawa Prenatal Prospective Study) I am participating in. The experimenter, Joel McCartney, is a Master's student at Carleton University. The director of the study is Peter Fried. The part of the investigation that I am involved in is concerned with auditory processing (an aspect of hearing). A pure tone hearing test will be the first section of the assessment that I will participate in. Then I will listen to spoken words on a "walkman" that will be difficult to hear. I will attempt to repeat these words the best I can. I understand that these two sections of the study will last approximately 40 minutes in total. The other part of the study involves urine collection. I will do this in the bathroom using a bottle given to me.

I understand that my participation in this study is entirely voluntary. I can stop anytime I wish to. No one will pressure me to continue.

If I have any questions I can ask Joel or my mother or father.

Signed_____

Date_____

Print Name_____

Witness_____

Appendix F

OBSERVED TEST BEHAVIOUR (SCAN)

Child's Name: _____ I.D. #: _____ Date: _____

1. Comprehension (CB):

low 1 2 3 high

1 = instructions had to be repeated more than three times

2 = instructions were repeated two or three times

3 = instructions were repeated once or not at all

2. Cooperativeness (CVB):

1 2 3

1 = little willingness to participate

2 = was willing to participate

3 = very willing and helpful

3. Attentiveness (AB):

1 2 3

1 = quite distracted and restless

2 = somewhat distracted

3 = not distracted

4. Enthusiasm (EB):

1 2 3

1 = seemed disinterested

2 = seemed fairly interested

3 = was eager and interested

5. Concentration (CTB):

1 2 3

1 = difficulty focusing on task

2 = some difficulty focusing on task

3 = little difficulty focusing on task

6. Enjoyment (EJB):

1 2 3

1 = did not seem pleasurable

2 = somewhat pleasurable and gratifying

3 = very satisfying and pleasurable

7. Length of test:.....Time (LTB) =

poor 1 2 3 good

1 = ≥ 35 min.

2 = ≥ 25 min. < 35 min.

3 = < 25 min.

8. Frustration level (FB):

poor 1 2 3 good

1 = seemed thwarted and disappointed

2 = somewhat baffled

3 = completed test with ease

9. Test condition (TCB):

poor 1 2 3 ideal

1 = disruptions (e.g. noisy, distractions)

2 = adequate (1 interruption/quiet)

3 = optimal (no interruptions/very quiet)

10. Response latency (RLB):

slow 1 2 3 fast

1 = some answers were made during a following command

2 = answers were voiced just in time

3 = answers were voiced with time to spare

Behaviour Total (BEHSCAN) =

Comments:

Appendix G

AUDITORY HISTORY QUESTIONNAIRE

Child's name: _____ I.D. #: _____ Date: _____

A. During your pregnancy with _____, were you exposed to excessive levels of noise (e.g. loud music, machinery, etc.) on a regular basis?

1,0 (yes/no)

☐

1. Have you ever had a hearing problem that required a hearing aid or any other such device?

1,0 (yes/no) (If no, go to 2)

1,0 a. Was the hearing problem diagnosed?
(yes/no)

1,0 b. Does it still persist?
(yes/no)

☐

= Mother's auditory total (var. #)

2. Has the father of your child ever had a hearing problem that required a hearing aid or any other such device?

1,0 (yes/no) (If no, go to 3)

1,0 a. Was the hearing problem diagnosed?
(yes/no)

1,0 b. Does it still persist?
(yes/no)

☐

= Father's auditory total (var. #)

3. Has your child ever had a hearing problem?

1,0 (yes/no)
(If no, end)

If yes,

1,0 a. Was the hearing problem diagnosed?
(yes/no)

1,0 b. Did it require treatment?
(yes/no)

1,0 c. Does it still persist?
(yes/no)

1,0 d. Is it presently a problem?
(yes/no)

☐

= Child's auditory total: questions 3, 4, 5, 6 (CAUDIT)

☐

= Composite auditory total: all questions (TAUDIT)

- i. If yes, what was it? (middle ear infection, effusion, other)
If other, please specify, _____.
- ii. How many infections?_____.
- iii. Age of your child at onset of the hearing problem?_____.
- iv. For how many years did/has it persist(ed)?_____.

Comments:

Appendix H

PASSIVE SMOKE EXPOSURE QUESTIONNAIRE

Name: _____ I.D.#: _____ Date: _____

(Please answer questions only with respect to indoor exposure.)

Part A:

1. How confident in your memory are you in recalling your smoking habits of the past week?
not at all 1 2 3 4 5 very confident
2. How confident in your memory are you in recalling your smoking habits since the birth of your child?
not at all 1 2 3 4 5 very confident
3. Do you smoke? (yes/no)
(If yes, go to 6)
4. Have you ever smoked? (yes/no)
(If no, go to 13)
5. Was your child living while you were a smoker?
(If no, go to 13)
(if yes, skip to 8 then 11)
6. Last week (excluding weekends), how many hours did you smoke in the presence (e.g. child in same room) of your child?

Mon. Tue. Wed. Thu. Fri. Total

--	--	--	--	--	--

7. Was last week a typical week? (yes/no)
(If yes, go to 9)
8. How many hours do/did you smoke during a typical week in the presence (e.g. child in same room) of your child? _____
9. During the past weekend, how many hours did you smoke in the presence (e.g. child in same room) of your child?

Sat. Sun. Total

--	--	--

10. Was the past weekend a typical weekend?
(If yes, go to 12)
11. During a typical weekend, how many hours do/did you smoke in the presence (e.g. child in same room) of your child? _____

12. Since your pregnancy with this child, how has your pattern of smoking changed? (percentage estimates)

Age	LESS	SAME	MORE
1	-	-	-
2	-	-	-
3	-	-	-
4	-	-	-
5	-	-	-
6	-	-	-
7	-	-	-
8	-	-	-
9	-	-	-
10	-	-	-
11	-	-	-

13. How confident in your memory are you in recalling the smoking habits of others when they are around your child?
not at all 1 2 3 4 5 very confident

14. How many smoke (excluding yourself) in the house and (have) live(d) there as well? _____
(If none, go to 22)

15. Last week (excluding weekends), how many hours did they smoke in the presence (e.g. child in same room) of your child? _____

Mon. Tue. Wed. Thu. Fri. Total

<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>
----------------------	----------------------	----------------------	----------------------	----------------------	----------------------

16. Was last week a typical week? (yes/no)
(If yes, go to 18)

17. During a typical week (excluding weekends), how many hours does/did they smoke in the presence (e.g. child in same room) of your child? _____

18. During the past weekend, how many hours did they smoke in the presence (e.g. child in same room) of your child? _____

Sat. Sun. Total

<input type="text"/>	<input type="text"/>	<input type="text"/>
----------------------	----------------------	----------------------

19. Was this a typical weekend? (yes/no)
(If yes, go to 21)

20. During a typical weekend, how often do/did they smoke in the presence (e.g. child in same room) of your child?-----

21. How has this pattern of smoke exposure changed throughout your child's life? (percentage estimates)

Age	LESS	SAME	MORE
1	-	-	-
2	-	-	-
3	-	-	-
4	-	-	-
5	-	-	-
6	-	-	-
7	-	-	-
8	-	-	-
9	-	-	-
10	-	-	-
11	-	-	-

22. Is/has your child (been) exposed to cigarette smoke by any regularly seen individuals who do/did not live in your home (e.g. relatives, babysitter, carpool/transportation)? (yes/no)
(If no, go to 30)

23. Last week (excluding weekends), how many hours did the person smoke in the presence (e.g. child in same room) of your child?-----

Mon. Tue. Wed. Thu. Fri. Total

<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>
----------------------	----------------------	----------------------	----------------------	----------------------	----------------------

24. Was last week a 'typical week'? (yes/no)
(If yes, go to 26)

25. During a typical week, how many hours does/did the person smoke in the presence (e.g. child in same room) of your child?-----

26. During the past weekend, how many hours did the person smoke in the presence (e.g. child in same room) of your child? _____

Sat. Sun. Total

--	--	--

27. Was the past weekend a typical weekend? (yes/no)
(If yes, go to 29)
28. During a typical weekend, how many hours does/did the person smoke in the presence (e.g. child in same room) of your child? _____
29. How has this pattern of smoke exposure changed throughout your child's life? (percentage estimates)

Age	LESS	SAME	MORE
1	-	-	-
2	-	-	-
3	-	-	-
4	-	-	-
5	-	-	-
6	-	-	-
7	-	-	-
8	-	-	-
9	-	-	-
10	-	-	-
11	-	-	-

30. When, to your knowledge, was your child exposed to smoke last?
_____ = _____ (hours between exposure and urine sample)

= Part A1 Total (hours of typical exposure = CPASSY)

= Part A2 Total (hours of last 7 days exposure = PRESENTY)

= Part A3 Total (hours of exposure during past three days = COTX)

Part B: Passive Smoke During Pregnancy

1. How confident in your memory are you in recalling the the amount of passive smoke (i.e. second hand) you were exposed to during a typical week when you were pregnant?

not at all 1 2 3 4 5 very confident

2. Did you live or work with anyone who smoked while you were pregnant?

(yes/no)

(If no, end)

3. Did this person (these people) smoke in your presence?

(yes/no)

(If no, end)

4. During a typical week, how many hours would you be exposed to smoke?

i. Home:-----

ii. Work:-----

5. During a typical weekend, how many hours would you be exposed to smoke?

i. Home:-----

ii. Work:-----

= Part B Total (PPASSX)

Part C: Ventilation

1. Is your home equipped with central air? (yes/no)

2. Does the air circulate/exchange, or does it remain in the room undisturbed?

1 for windows open

1 for fan

1 for smoke 'absorber' (e.g. vacuum ashtray)

= total

= ventilation total (VPASSX)

Comments:

HOME ENVIRONMENT QUESTIONNAIRE

Form 2-R
(HEQ-2R)

Jacob O. Sines, Ph.D.

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INSTRUCTIONS

Whenever possible, this questionnaire is to be answered by the mother of the child named below. This questionnaire consists of statements about your home, family and neighborhood. Read each statement carefully. If it accurately describes your home or family or neighborhood, put an X through the "T" (for True) to the left of that statement. If the statement does not accurately describe your home or family or neighborhood, put an X through the "F" (for False) to the left of that statement. Please answer all the questions either True or False.

In many of the statements there is a blank space. Simply imagine your child's name in the blank space. The word "house" is used in some of the statements. If you live in an apartment, imagine the word "apartment" for "house." Do not discuss the answers to the questionnaire while you are completing it. Please complete the questionnaire by yourself.

Date _____

Child: Name: _____

Age: _____ Sex: _____ Grade in School: _____

V: _____ P: _____ FS: _____ ID No: _____

Respondent: Name: _____

Relation to Child: _____

T	_____	_____	_____	_____	_____	_____	_____	_____	_____
Raw	_____	_____	_____	_____	_____	_____	_____	_____	_____
	Ach	A-E	A-H	A-T	Sup	Ch	Aff	Sep	Soc SS

- T F 1. We have an air conditioner.
- T F 2. Our house has a fireplace.
- T F 3. ____ has a desk of his/her own.
- T F 4. Many of the things our family does are centered around ____.
- T F 5. We have an encyclopedia.
- T F 6. There is a student center at ____'s school.
- T F 7. We don't believe in rewarding a child for doing things he's/she's supposed to do.
- T F 8. I jog or exercise strenuously several times a week.
- T F 9. Other children tease ____.
- T F 10. Other children don't seem to like ____.
- T F 11. Many of our neighbors criticize ____.
- T F 12. ____ has no very close friends.
- T F 13. There are many friendly children in ____'s class.
- T F 14. Children come over and ask if ____ can come out and play.
- T F 15. We live in a friendly neighborhood.
- T F 16. ____ is often invited to parties or celebrations.
- T F 17. Most of ____'s friends are sociable.
- T F 18. ____ has lots of friends.
- T F 19. I sometimes lose my temper with ____.
- T F 20. I would describe myself as a person with a quick temper.
- T F 21. I tend to get impatient with ____.
- T F 22. I am often depressed.

- T F 23. I'm easily upset.
- T F 24. I cry when something upsets me.
- T F 25. Sometimes I am too busy to be consistent in the discipline I use.
- T F 26. My work tires me out a lot, so I don't have much energy to do other things.
- T F 27. ____ and I don't talk to each other very much.
- T F 28. I don't have much time to play with ____.
- T F 29. My home responsibilities take up all my time.
- T F 30. My moods change fast.
- T F 31. Dirt upsets me a great deal.
- T F 32. My feelings are easily hurt.
- T F 33. A certain time is set aside for ____ to do homework.
- T F 34. We have two or more pieces of playground equipment in our yard.
- T F 35. I like to plan my children's activities.
- T F 36. When ____ signs up for an activity (lessons, team, etc.) I feel that he/she should complete the full program even though he/she finds that he/she dislikes the activity.
- T F 37. We have urged ____ to attend summer camp.
- T F 38. Most of ____'s friends get books from the public library.
- T F 39. I do not allow ____ to watch certain TV shows.
- T F 40. I try to avoid arguments.
- T F 41. We have very high standards for everyone in our family.

- T F 42. We have moved within the last two years.
- T F 43. During ____'s lifetime I have been divorced.
- T F 44. There are mainly apartments in our neighborhood.
- T F 45. Discipline in our family is left up to me.
- T F 46. There is a school near our house.
- T F 47. I support my children alone.
- T F 48. We have lived in our home more than six years.
- T F 49. We live in a quiet neighborhood.
- T F 50. We live on or very near a busline.
- T F 51. I make most of the important decisions in our family.
- T F 52. I have been married more than once.
- T F 53. Other people say that I'm too lenient with ____.
- T F 54. During ____'s lifetime, my husband and I have tried marital separation.
- T F 55. ____'s father is not living with the family.
- T F 56. My husband is unreliable.
- T F 57. ____ lives with a step-mother or step-father.
- T F 58. My husband is a good provider.
- T F 59. My husband makes the important decisions around our house.
- T F 60. My husband has had a problem with the police.
- T F 61. Our family gets along very well.
- T F 62. We are a pretty easygoing family.
- T F 63. Our family often does things together.
- T F 64. Almost everyone in our family agrees on how to do things.
- T F 65. Some people in our family are picked on more than others.
- T F 66. There are lots of arguments in our family.
- T F 67. Our home sometimes feels crowded.
- T F 68. On weekends, our family often does things together such as short trips, going shopping, going to the zoo, etc.
- T F 69. Sometimes things get so bad at home I want to leave.
- T F 70. Outside of birthdays and holidays, ____ is rarely given gifts or presents.
- T F 71. My husband helps ____ make many important decisions.
- T F 72. My husband and I have arguments about how to raise ____.
- T F 73. My husband often finds time to play with ____.
- T F 74. My husband and I have considered separating.
- T F 75. My husband occasionally helps ____ with his/her homework.
- T F 76. My husband is impatient with ____.
- T F 77. My husband's moods change very quickly.
- T F 78. My husband is a nervous man.
- T F 79. My husband and I need to deal with our own problems before we can take care of ____'s.
- T F 80. Within the last two years, my husband and I seem to be having more disagreements than we used to.
- T F 81. My husband often loses his temper.
- T F 82. My husband is a quiet man.
- T F 83. My husband has a level disposition.
- T F 84. My husband and I agree on when ____ needs to be disciplined.
- T F 85. My husband and I don't argue very much.

- T F 86. I have had quite a bit of surgery.
- T F 87. Within the last two years, _____ has been in the hospital.
- T F 88. One or more of _____'s friends have moved recently.
- T F 89. I have been hospitalized for emotional problems.
- T F 90. We have regular meal times.
- T F 91. I wouldn't want my husband to take a job where he had to travel and be away from our family.
- T F 92. My husband's work or recreational activities often force him to be away from home in the evening.
- T F 93. My husband's working hours have changed within the last two years.
- T F 94. My husband has changed jobs within the last two years.
- T F 95. We often have friends over to our house to play cards, talk, etc.
- T F 96. There are a lot of young families in our neighborhood.
- T F 97. We often have relatives or friends visit our house.
- T F 98. My husband enjoys being around people.
- T F 99. We have begun to make plans for _____'s college education.
- T F 100. I had to have special training for my job.
- T F 101. We have a piano.
- T F 102. I have read one book or more in the last month.
- T F 103. Within the last several years, I have taken an adult education course or a university extension course.
- T F 104. We have a typewriter.
- T F 105. I belong to a social, civic, political, study, literary, or art club.
- T F 106. I got good grades in school.
- T F 107. _____ went to a nursery school when he/she was younger.
- T F 108. I have had some college education.
- T F 109. I read a lot.
- T F 110. I am active in community affairs.
- T F 111. At least one member of our family is active in political organizations.
- T F 112. Our home has more than one hundred books (excluding children's books).
- T F 113. Most people in our family are too busy to spend much time reading.
- T F 114. I can't get very interested in activities outside the home.
- T F 115. Our family is active in community affairs.
- T F 116. I restrict the amount of TV that _____ watches.
- T F 117. My husband works in an unskilled job.
- T F 118. My husband has had some college education.
- T F 119. Either or both my husband and I play golf or tennis.
- T F 120. My husband has completed high school.
- T F 121. My husband belongs to a social, civic, political, study, literary, or art club.
- T F 122. My husband has read one book or more in the last month.
- T F 123. Within the last several years, my husband has taken an adult education or a university extension course.

HOME ENVIRONMENT QUESTIONNAIRE

Form 1-R
(HEQ-IR)

Jacob O. Sines, Ph.D.

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INSTRUCTIONS

Whenever possible, this questionnaire is to be answered by the mother of the child named below. This questionnaire consists of statements about your home, family and neighborhood. Read each statement carefully. If it accurately describes your home or family or neighborhood, put an X through the "T" (for True) to the left of that statement. If the statement does not accurately describe your home or family or neighborhood, put an X through the "F" (for False) to the left of that statement. Please answer all the questions either True or False.

In many of the statements there is a blank space. Simply imagine your child's name in the blank space. The word "house" is used in some of the statements. If you live in an apartment, imagine the word "apartment" for "house." Do not discuss the answers to the questionnaire while you are completing it. Please complete the questionnaire by yourself.

Date _____

Child: Name: _____

Age: _____ Sex: _____ Grade in School: _____

V: _____ P: _____ FS: _____ ID No: _____

Respondent: Name: _____

Relation to Child: _____

T	_____	_____	_____	_____	_____	_____	_____	_____	_____	_____
Raw	Ach	A-E	A-H	A-T	Sup	Ch	Aff	Sep	Soc	SS

- T F 1. We have an air conditioner.
- T F 2. Our house has a fireplace.
- T F 3. ____ has a desk of his/her own.
- T F 4. Many of the things our family does are centered around ____.
- T F 5. We have an encyclopedia.
- T F 6. There is a student center at ____'s school.
- T F 7. We don't believe in rewarding a child for doing things he's/she's supposed to do.
- T F 8. I jog or exercise strenuously several times a week.
- T F 9. Other children tease ____.
- T F 10. Other children don't seem to like ____.
- T F 11. Many of our neighbors criticize ____.
- T F 12. ____ has no very close friends.
- T F 13. There are many friendly children in ____'s class.
- T F 14. Children come over and ask if ____ can come out and play.
- T F 15. We live in a friendly neighborhood.
- T F 16. ____ is often invited to parties or celebrations.
- T F 17. Most of ____'s friends are sociable.
- T F 18. ____ has lots of friends.
- T F 19. I sometimes lose my temper with ____.
- T F 20. I would describe myself as a person with a quick temper.
- T F 21. I tend to get impatient with ____.
- T F 22. I am often depressed.
- T F 23. I'm easily upset.
- T F 24. I cry when something upsets me.
- T F 25. Sometimes I am too busy to be consistent in the discipline I use.
- T F 26. My work tires me out a lot, so I don't have much energy to do other things.
- T F 27. ____ and I don't talk to each other very much.
- T F 28. I don't have much time to play with ____.
- T F 29. My home responsibilities take up all my time.
- T F 30. My moods change fast.
- T F 31. Dirt upsets me a great deal.
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- T F 33. A certain time is set aside for ____ to do homework.
- T F 34. We have two or more pieces of playground equipment in our yard.
- T F 35. I like to plan my children's activities.
- T F 36. When ____ signs up for an activity (lessons, team, etc.) I feel that he/she should complete the full program even though he/she finds that he/she dislikes the activity.
- T F 37. I have urged ____ to attend summer camp.
- T F 38. Most of ____'s friends get books from the public library.
- T F 39. I do not allow ____ to watch certain TV shows.
- T F 40. I try to avoid arguments.
- T F 41. We have very high standards for everyone in our family.
- T F 42. We have moved within the last two years.
- T F 43. During ____'s lifetime I have been divorced.
- T F 44. There are mainly apartments in our neighborhood.
- T F 45. Discipline in our family is left up to me.
- T F 46. There is a school near our house.
- T F 47. I support my children alone.
- T F 48. We have lived in our home more than six years.
- T F 49. We live in a quiet neighborhood.
- T F 50. We live on or very near a busline.
- T F 51. I make most of the important decisions in our family.
- T F 52. I have been married more than once.
- T F 53. Other people say that I'm too lenient.
- T F 54. During ____'s lifetime, my husband and I have tried marital separation.
- T F 55. ____'s father is not living with the family.
- T F 56. Our family gets along very well.
- T F 57. We are a pretty easygoing family.
- T F 58. Our family often does things together.
- T F 59. Almost everyone in our family agrees on how to do things.
- T F 60. Some people in our family are picked on more than others.
- T F 61. There are lots of arguments in our family.
- T F 62. Our home sometimes feels crowded.
- T F 63. On weekends, our family often does things together such as short trips, going shopping, going to the zoo, etc.
- T F 64. Sometimes things get so bad at home I want to leave.
- T F 65. Outside of birthdays and holidays, ____ is rarely given gifts or presents.

- T F 66. I have had quite a bit of surgery.
- T F 67. Within the last two years, ____ has been in the hospital.
- T F 68. One or more of ____'s friends have moved recently.
- T F 69. I have been hospitalized for emotional problems.
- T F 70. We have regular meal times.
- T F 71. We often have friends over to our house to play cards, talk, etc.
- T F 72. There are a lot of young families in our neighborhood.
- T F 73. We often have relatives or friends visit our house.
- T F 74. We have begun to make plans for ____'s college education.
- T F 75. I had to have special training for my job.
- T F 76. We have a piano.
- T F 77. I have read one book or more in the last month.
- T F 78. Within the last several years, I have taken an adult education course or a university extension course.

- T F 79. We have a typewriter.
- T F 80. I belong to a social, civic, political, study, literary, or art club.
- T F 81. I got good grades in school.
- T F 82. ____ went to a nursery school when he/she was younger.
- T F 83. I have had some college education.
- T F 84. I read a lot.
- T F 85. I am active in community affairs.
- T F 86. At least one member of our family is active in political organizations.
- T F 87. Our home has more than one hundred books (excluding children's books).
- T F 88. Most people in our family are too busy to spend much time reading.
- T F 89. I can't get very interested in activities outside the home.
- T F 90. Our family is active in community affairs.
- T F 91. I restrict the amount of TV that ____ watches.

NEO

Five-Factor Inventory

Form S

Paul T. Costa, Jr., Ph.D., and Robert R. McCrae, Ph.D.

Instructions

Write only where indicated in this booklet. Carefully read all of the instructions before beginning. This questionnaire contains 60 statements. Read each statement carefully. For each statement fill in the circle with the response that best represents your opinion. Make sure that your answer is in the correct box.

Fill in **(SD)** if you *strongly disagree* or the statement is definitely false.

Fill in **(D)** if you *disagree* or the statement is mostly false.

Fill in **(N)** if you are *neutral* on the statement, you cannot decide, or the statement is about equally true and false.

Fill in **(A)** if you *agree* or the statement is mostly true.

Fill in **(SA)** if you *strongly agree* or the statement is definitely true.

For example, if you *strongly disagree* or believe that a statement is definitely false, you would fill in the **(SD)** for that statement.

Example

☒ (SD) ☐ (D) ☐ (N) ☐ (A) ☐ (SA)

Fill in only one response for each statement. Respond to all of the statements, making sure that you fill in the correct response. **DO NOT ERASE!** If you need to change an answer, make an "X" through the incorrect response and then fill in the correct response.

Note that the responses are numbered in rows. Before responding to the statements, turn to the inside of the booklet and enter your name, age, and sex and the date.

PAR Psychological Assessment Resources, Inc.

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98765432

Reorder #1452-TB

Printed in the U.S.A.

1. I am not a worrier.
2. I like to have a lot of people around me.
3. I don't like to waste my time daydreaming.
4. I try to be courteous to everyone I meet.
5. I keep my belongings clean and neat.
6. I often feel inferior to others.
7. I laugh easily.
8. Once I find the right way to do something, I stick to it.
9. I often get into arguments with my family and co-workers.
10. I'm pretty good about pacing myself so as to get things done on time.
11. When I'm under a great deal of stress, sometimes I feel like I'm going to pieces.
12. I don't consider myself especially "light-hearted."
13. I am intrigued by the patterns I find in art and nature.
14. Some people think I'm selfish and egotistical.
15. I am not a very methodical person.
16. I rarely feel lonely or blue.
17. I really enjoy talking to people.
18. I believe letting students hear controversial speakers can only confuse and mislead them.
19. I would rather cooperate with others than compete with them.
20. I try to perform all the tasks assigned to me conscientiously.
21. I often feel tense and jittery.
22. I like to be where the action is.
23. Poetry has little or no effect on me.
24. I tend to be cynical and skeptical of others' intentions.
25. I have a clear set of goals and work toward them in an orderly fashion.
26. Sometimes I feel completely worthless.
27. I usually prefer to do things alone.
28. I often try new and foreign foods.
29. I believe that most people will take advantage of you if you let them.
30. I waste a lot of time before settling down to work.
31. I rarely feel fearful or anxious.
32. I often feel as if I'm bursting with energy.
33. I seldom notice the moods or feelings that different environments produce.
34. Most people I know like me.
35. I work hard to accomplish my goals.
36. I often get angry at the way people treat me.
37. I am a cheerful, high-spirited person.
38. I believe we should look to our religious authorities for decisions on moral issues.
39. Some people think of me as cold and calculating.
40. When I make a commitment, I can always be counted on to follow through.

41. Too often, when things go wrong, I get discouraged and feel like giving up.
42. I am not a cheerful optimist.
43. Sometimes when I am reading poetry or looking at a work of art, I feel a chill or wave of excitement.
44. I'm hard-headed and tough-minded in my attitudes.
45. Sometimes I'm not as dependable or reliable as I should be.
46. I am seldom sad or depressed.
47. My life is fast-paced.
48. I have little interest in speculating on the nature of the universe or the human condition.
49. I generally try to be thoughtful and considerate.
50. I am a productive person who always gets the job done.
51. I often feel helpless and want someone else to solve my problems.
52. I am a very active person.
53. I have a lot of intellectual curiosity.
54. If I don't like people, I let them know it.
55. I never seem to be able to get organized.
56. At times I have been so ashamed I just wanted to hide.
57. I would rather go my own way than be a leader of others.
58. I often enjoy playing with theories or abstract ideas.
59. If necessary, I am willing to manipulate people to get what I want.
60. I strive for excellence in everything I do.

Enter your responses here—remember to enter responses across the rows.
 SD = Strongly Disagree; D = Disagree; N = Neutral; A = Agree; SA = Strongly Agree

1 SD D N A SA	2 SD D N A SA	3 SD D N A SA	4 SD D N A SA	5 SD D N A SA
6 SD D N A SA	7 SD D N A SA	8 SD D N A SA	9 SD D N A SA	10 SD D N A SA
11 SD D N A SA	12 SD D N A SA	13 SD D N A SA	14 SD D N A SA	15 SD D N A SA
16 SD D N A SA	17 SD D N A SA	18 SD D N A SA	19 SD D N A SA	20 SD D N A SA
21 SD D N A SA	22 SD D N A SA	23 SD D N A SA	24 SD D N A SA	25 SD D N A SA
26 SD D N A SA	27 SD D N A SA	28 SD D N A SA	29 SD D N A SA	30 SD D N A SA
31 SD D N A SA	32 SD D N A SA	33 SD D N A SA	34 SD D N A SA	35 SD D N A SA
36 SD D N A SA	37 SD D N A SA	38 SD D N A SA	39 SD D N A SA	40 SD D N A SA
41 SD D N A SA	42 SD D N A SA	43 SD D N A SA	44 SD D N A SA	45 SD D N A SA
46 SD D N A SA	47 SD D N A SA	48 SD D N A SA	49 SD D N A SA	50 SD D N A SA
51 SD D N A SA	52 SD D N A SA	53 SD D N A SA	54 SD D N A SA	55 SD D N A SA
56 SD D N A SA	57 SD D N A SA	58 SD D N A SA	59 SD D N A SA	60 SD D N A SA

Have you responded to all of the statements? _____ Yes _____ No

Have you entered your responses in the correct boxes? _____ Yes _____ No

Have you responded accurately and honestly? _____ Yes _____ No

PARENT QUESTIONNAIRE

Child's ID# _____

Date _____

1. Child's name _____
2. Child's position in family (circle) (oldest) 1 2 3 4 5 6 (youngest)
3. Current grade in school (circle) K 1 2 3 4 5
4. School history -- please check if appropriate:
 - ☐ Has switched from French Immersion to English program.
(if so state when _____)
 - ☐ Has been referred for learning problems.
(results _____)
 - ☐ Has received remedial help. Grade(s) _____;
Subjects _____
 - ☐ Has repeated a grade. (if so, which grade _____)
5. How many days has your child missed school due to sickness during this year? _____
- 6a. Current cigarette habits of mother of child:
 - ☐ regular smoker (average # cigarettes per day _____)
 - ☐ former smoker (year quit smoking 19__)
 - ☐ never smoked
- 6b. Current cigarette habits of father of child:
 - ☐ regular smoker (average # cigarettes per day _____)
 - ☐ former smoker (year quit smoking 19__)
 - ☐ never smoked
- 7a. Current marijuana habits of mother of child:
 - ☐ regular user (average # joints per ^{week}~~day~~ _____)
 - ☐ former user (year quit 19__)
 - ☐ never used
- 7b. Current marijuana habits of father of child:
 - ☐ regular user (average # joints per ^{week}~~day~~ _____)
 - ☐ former user (year quit 19__)
 - ☐ never used

APPENDIX L

CONNERS

Parent's Questionnaire

Name of Child _____

Date _____

Please answer all questions. Beside each item below, indicate the degree of the problem by a check mark (✓)

	Not at all	Just a little	Pretty much	Very much
1. Picks at things (nails, fingers, hair, clothing).				
2. Sassy to grown-ups.				
3. Problems with making or keeping friends.				
4. Excitable, impulsive.				
5. Wants to run things.				
6. Sucks or chews (thumb; clothing; blankets).				
7. Cries easily or often.				
8. Carries a chip on his shoulder.				
9. Daydreams.				
10. Difficulty in learning.				
11. Restless in the "equilibrium" sense.				
12. Fearful (of new situations; new people or places; going to school).				
13. Restless, always up and on the go.				
14. Destructive.				
15. Tells lies or stories that aren't true.				
16. Shy.				
17. Gets into more trouble than others same age.				
18. Speaks differently from others same age (baby talk; stuttering; hard to understand).				
19. Denies mistakes or blames others.				
20. Quarrelsome.				
21. Pouts and sulks.				
22. Steals.				
23. Disobedient or obeys but resentfully.				
24. Worries more than others (about being alone; illness or death).				
25. Fails to finish things.				
26. Feelings easily hurt.				
27. Bullies others.				
28. Unable to stop a repetitive activity.				
29. Cruel.				
30. Childish or immature (wants help he shouldn't need; clings; needs constant reassurance).				
31. Distractibility or attention span a problem.				
32. Headaches.				
33. Mood changes quickly and drastically.				
34. Doesn't like or doesn't follow rules or restrictions.				
35. Fights constantly.				
36. Doesn't get along well with brothers or sisters.				
37. Easily frustrated in efforts.				
38. Disturbs other children.				
39. Basically an unhappy child.				
40. Problems with eating (poor appetite; up between bites).				
41. Stomach aches.				
42. Problems with sleep (can't fall asleep; up too early; up in the night).				
43. Other aches and pains.				
44. Vomiting or nausea.				
45. Feels chested in family circle.				
46. Boasts and brags.				
47. Lets self be pushed around.				
48. Bowel problems (frequently loose; irregular habits; constipation).

APPENDIX N
Cotinine and Cotinine/Creatinine Levels For Each Subject

Subject Number	Cotinine (ug/L)	Cotinine/Creatinine (ug/mmol)
562	.3	.13
712	1.1	.09
782	4.5	.68
783	3.8	1.24
922	.5	.05
1552	.1	.01
2142	1.2	.12
2272	.2	.01
2372	.8	.09
2561	.4	.03
2582	4.8	.29
2671	8.7	1.01
2771	4.1	.27
2881	.1	.01
2882	1.4	.07
2931	13.1	1.32
2932	8.8	1.36
3081	.2	.02
3381	10.0	1.19
3421	.7	.06
3461	5.2	.43
3462	4.6	1.00
3541	1.7	.18
3542	2.5	.19
3601	4.5	.41
3801	1.3	.12
3881	.0	.00
3882	.2	.01
3891	2.4	.13
3911	1.1	.07
3912	.3	.03
4011	.1	.01
4012	.2	.03
4071	.0	.00
4331	1.2	.14
4391	.7	.09
4421	5.8	.49
4451	3.4	.15
4511	.7	.08
4631	1.8	.20
4632	3.4	.18
4661	1.5	.10
4771	1.1	.06
4891	.1	.01
5101	.2	.01
5241	1.5	.29
5242	2.6	.21

Subject Number	Cotinine (ug/L)	Cotinine/Creatinine (ug/mmol)
5271	.0	.00
5351	11.0	.80
5352	4.1	1.14
5421	1.1	.08
5531	3.1	.32
5532	2.3	.30
5691	.7	.33
5692	.0	.00
5721	1.5	.63
5881	.4	.02
5882	.3	.02
5931	10.7	.78
5941	1.1	.08
5981	.0	.00
6001	.4	.03
6011	.3	.04
6031	.0	.00
6041	.6	.07
6051	3.6	.30
6052	4.4	.44
6071	1.8	.28
6072	3.2	.43
6081	.2	.05
6171	.4	.04
6191	.5	.03
6271	.4	.06
6331	1.1	.08
6361	12.0	2.45
6381	.0	.00
6391	.2	.03
6401	.8	.08
6411	.	.
6431	14.8	1.24
6451	8.8	.92
6481	13.9	1.36
6491	11.6	1.07
6521	11.2	.84
6531	2.7	.33
6541	.3	.03
6551	10.3	1.41
6561	10.3	.79
6571	1.7	.23
6581	9.4	1.14
6582	5.2	1.06
6591	16.9	2.09
6601	9.1	1.26
6611	12.1	2.11
6621	1.8	1.01
6631	3.8	.50
6661	1.1	.21

Subject Number	Cotinine (ug/L)	Cotinine/Creatinine (ug/mmol)
6671	3.6	.28
6681	4.8	.57
6701	1.1	.28
6711	2.4	.29
6741	4.0	.62
6751	14.4	1.27
6761	6.7	1.39
6771	5.1	.47
6772	5.5	.72
6781	9.5	3.54
6791	.3	.03
6801	20.3	2.26
6811	.1	.04

N = 110

APPENDIX O

SCAN Standard Scores and Prenatal Cigarette Use Category For
Each Subject

Subject	FW	AFG	CW	SC	Prenatal Cigarette Use Category
562	14	11	12	115	0
712	13	10	10	108	1
782	14	13	12	118	1
783	14	10	10	106	1
922	12	11	7	95	0
1552	12	8	7	89	0
2142	10	14	13	86	0
2272	9	11	3	65	1
2372	10	10	13	108	0
2561	11	11	12	108	1
2582	9	12	12	112	1
2671	13	10	9	101	1
2771	12	12	10	105	2
2881	10	9	11	101	0
2882	11	10	10	103	0
2931	13	12	13	121	1
2932	6	9	6	79	2
3081	11	10	11	106	0
3381	9	9	8	92	2
3421	12	14	12	116	0
3461	12	11	13	116	1
3462	11	8	9	96	1
3541	13	8	10	101	2
3542	14	8	10	102	1
3601	14	7	13	109	1
3801	11	8	10	99	0
3881	13	8	8	95	0
3882	14	8	10	103	0
3891	14	12	12	118	2
3911	11	11	9	100	1
3912	9	10	11	102	1
4011	10	10	8	94	1
4012	11	14	13	118	0
4071	11	15	8	100	0
4331	9	8	11	99	0
4391	12	9	15	114	1
4421	9	7	5	75	1
4451	13	12	10	108	0
4511	14	13	7	100	0
4631	8	7	6	84	2
4632	11	9	14	114	1
4661	13	8	7	92	0
4771	13	11	11	109	0
4891	12	10	10	105	0
5101	9	10	6	86	1

Subject	FW	AFG	CW	SC	Prenatal Cigarette Use Category
5241	13	8	11	106	1
5242	14	10	11	109	1
5271	14	9	6	90	0
5351	11	10	7	93	2
5352	11	11	10	103	2
5421	12	13	15	125	0
5531	14	7	8	94	1
5532	7	8	8	87	1
5691	13	9	11	107	0
5692	9	8	12	102	0
5721	10	11	6	88	2
5881	14	10	11	108	1
5882	10	6	11	95	0
5931	11	9	12	106	0
5941	12	9	14	115	0
5981	9	8	8	89	0
6001	14	12	14	125	0
6011	13	13	14	123	1
6031	10	7	9	91	0
6041	9	10	10	98	1
6051	13	6	10	97	1
6052	13	10	11	110	2
6071	11	10	9	97	2
6072	10	12	8	95	1
6081	11	14	15	125	0
6171	10	11	14	115	0
6191	15	7	8	92	0
6271	9	6	7	84	1
6331	12	11	12	112	1
6361	13	7	9	97	2
6381	9	11	10	100	0
6391	13	11	10	106	0
6401	11	11	11	105	1
6411	13	11	12	110	1
6431	13	11	10	104	2
6451	12	8	8	93	2
6481	11	11	10	104	1
6491	11	7	12	102	2
6521	9	11	8	92	2
6531	4	11	9	86	0
6541	9	6	5	77	2
6551	5	5	5	69	0
6561	3	11	7	83	1
6571	9	8	6	84	2
6581	10	7	10	97	1
6582	13	9	14	118	1
6591	12	12	10	107	1
6601	11	9	9	100	1
6611	13	8	9	99	1
6621	11	9	11	106	2

Subject	FW	AFG	CW	SC	Prenatal Cigarette Use Category
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6631	14	15	11	118	0
6661	12	14	11	112	0
6671	9	9	8	90	2
6681	11	12	9	102	0
6701	9	10	9	94	2
6711	13	11	12	112	1
6741	14	10	9	100	2
6751	9	10	9	95	2
6761	14	11	11	110	1
6771	9	9	9	96	1
6772	13	10	13	114	1
6781	10	14	13	117	1
6791	11	11	9	96	2
6801	11	13	9	100	2
6311	14	9	14	118	0

N = 110

FW: Filtered Words

AFG: Auditory Figure Ground

CW: Competing Words

SC: SCAN Composite

0: no smoke

1: light smoke

2: heavy smoke (≥ 16 mg/day of nicotine)

END

1 9-0 7-9 4

FIN