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PASSIVE SMOKING DURING PREGNANCY: LONG TERM EFFECTS?

by Judy Makin, B.A., B.Ed., M.A.

A thesis submitted to
the Faculty of Graduate Studies and Research
in partial fulfilment of
the requirements for the degree of
Doctor of Philosophy

Department of Psychology

Carleton University
Ottawa, Ontario
February, 1989

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"Passive Smoking During Pregnancy: Long Term Effects?"

submitted by

Judy Makin

in partial fulfilment of the requirements
for the degree of Doctor of Philosophy

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Abstract

Previous research determined that maternal smoking during pregnancy is associated with negative effects for the child at birth and throughout childhood. This study investigates the long-term consequences of passive smoking during pregnancy.

Ninety-one children between the ages of six and nine years were tested in the domains of speech and language, fine motor, and visual/spatial abilities, lateral dominance, academic achievement, intelligence, and behaviour. Mean scores of three groups (i.e. Nonsmoking, Passive and Active) were compared using multivariate techniques. Children of nonsmoking mothers generally performed better than children in the two smoking groups. The Passive group generally performed in between the Active and Nonsmoking groups.

Using Discriminant Analysis, variables that maximally separated the Active group from the others were motoric, visual/spatial, and behavioural. Active and Passive groups showed decrements on tests of intelligence, language and attention. The Passive group differed from Active in motor and visual/spatial areas; this was interpreted to be a dimension of speed vs. accuracy, with Active demonstrating better motor speed, and Passive showing better attention to detail.

SES, maternal age, education, previous testing, and examiner were controlled using MANCOVA. The Active group could be differentiated after covariate adjustment, whereas such adjustment served to reduce

the differences associated with the Passive group.

Impact of the home environment was assessed using the Moos Family Environment Scales. Path analysis determined that the home environment played an important mediating role for the behavioural outcome associated with both active and passive smoking. Active smoking was also directly associated with increased behaviour problems.

It was concluded that there is evidence of a continuum of long-term smoking effects, with active maternal smoking associated with effects of greater breadth, magnitude and stability than those for passive maternal smoking. Nevertheless, children of women who are passive smokers are also at risk for a pattern of negative developmental outcomes, and implications for public policy and education are discussed.

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I INTRODUCTION

The human organism experiences its greatest period of growth and differentiation between conception and birth. During this prenatal period the developing organism is most vulnerable to environmental influence (Dalby, 1979). Although there are ancient cultural and religious guidelines for maternal diet and behaviour during pregnancy, psychologists have only recently turned their attention to this period of human development.

Environmental agents can produce teratogenic effects that range from severe structural defects noticeable at birth through to much more subtle neurobehavioural deficits that take years to become apparent. The focus of this investigation will be the degree to which prenatal exposure to cigarette smoke is related to specific neurobehavioural sequelae in early childhood. The developmental outcome associated with active maternal smoking will be compared to that of passive exposure to cigarette smoke in the environment during pregnancy.

Prenatal drug use accounts for a small percentage of variance on behavioural outcome measures. Nevertheless, this is an important field of research. Large numbers of women are exposed to environmental tobacco smoke during pregnancy, and any effects on the developing child resulting from this exposure will be of great public concern. For the individual child, such prenatal drug exposure may prevent the realization of his or her full potential.

.1 Active Smoking During Pregnancy

The first questions about cigarettes as a possible health hazard during pregnancy were raised in the 1930's; concern at that time was with the mother's health, not that of the baby. Simpson (1957) made the first observation that cigarette smoking had a negative effect on human infant birthweight. Additional harmful effects have since been found, such as increased frequency of pregnancy complications and perinatal mortality. Long-term effects have emerged in a number of major longitudinal studies in Canada, U.S., Britain and Finland. At school age, offspring of smokers show small but significant decrements in several areas of intellectual, perceptual and academic functioning.

This section describes current smoking patterns of women during pregnancy, and summarizes known effects of maternal smoking during pregnancy on fetal, neonatal and childhood development. Efforts at identifying the mechanism(s) by which smoking produces its effects are examined, and issues concerning interpretation of this research are noted.

A/ Patterns of Maternal Smoking

The most recent information on the smoking behaviour of Canadians is provided in two national surveys conducted in December 1983 and June 1985 by Health and Welfare Canada (1985, 1988). These studies indicate

that the overall prevalence of smoking by adults is declining in all age groups. The latest figures indicate that 30% of the adult population are regular smokers (i.e. smoke every day), compared to 41% in 1970. Males account for the largest share of this decline, with a reduction from 49% in 1970 to 32% in 1985. Although the data for females also followed this downward trend, the rate of change was markedly reduced. The smoking rate among women in general was 32% in 1970, and decreased only to 29% in 1985. Among women of reproductive age, the percentage of regular smokers is even higher. Of women aged 15 to 44 years, 31 to 34% report regular smoking.

Although the number of smokers is declining, many smokers are consuming more cigarettes per day. The proportion of smokers of 1 - 10 cigarettes per day has declined since 1970, whereas the proportion smoking more than 10 cigarettes per day increased. Most importantly for the purposes of this paper, the relative increase in the number of smokers of more than 25 cigarettes per day was 31% for males, and 57% for females. Many smokers appear to have switched to cigarettes with lower tar and nicotine levels, but have compensated by smoking more of them. This is an important finding given evidence that will be presented later indicating that many of the deleterious effects of smoking for the fetus are related to carbon monoxide, which increases directly with the number of cigarettes smoked.

When smoking rates during pregnancy have been measured, it has been found that smokers tend to maintain or slightly reduce their pre-pregnancy smoking levels. Fried et al. (1980) found that 50% of

light smokers and 90% of heavy smokers continued to smoke to some degree once pregnancy was confirmed. In the writer's review of studies of maternal smoking published since 1970, cigarette smoking during pregnancy was reported by 22% to 43% of the women in the samples studied. A questionnaire survey of over 3,000 women who delivered a baby in the Ottawa-Carleton area in 1983 indicated that 37.4% were smokers before pregnancy, and 26% were still smoking at the time of delivery (Stewart and Dunkley, 1985). In comparison with abstention from alcohol and other drugs during pregnancy, which has shown a great increase over the past 20 years, the trend to decreased smoking has been much less dramatic.

In general, smokers differ from non-smokers on a number of demographic and lifestyle variables, many of which are important potential confounds in research into the effects of smoking on child development. Socioeconomic status (SES) shows a negative relationship with smoking (Andrews and McGarry, 1972; Fried, Watkinson, Grant and Knights, 1980; Health and Welfare 1985 and 1988; Kuzma and Kissinger, 1981). This relationship holds true for both components of SES, i.e. income and years of education. Smokers are also more likely to be single than married, and more likely to be divorced, separated or have marital difficulties (Cope et al., 1973; Gritz, 1980). Tobacco use is positively correlated with use of alcohol and caffeine (Istvan and Matazarro, 1984). Smokers have been found to be more extroverted than nonsmokers, more antisocial, more independent, more active, more anxious and more competitive (Dunn, 1973; review by Gritz, 1980).

Smokers are also greater risk takers, and have been found to be 50% more likely to be in traffic accidents and 46% more likely to get tickets for speeding (DiFranza, et al., 1986).

Some researchers have used the above findings to develop a hypothesis that these personality differences arise from a genetic or constitutional predisposition (e.g. Eysenck, 1965, 1973, 1986), and that the deleterious neonatal outcomes associated with smoking are due to the smoker, not the smoke (Yerushalamy, 1972). Although this hypothesis does not currently enjoy scientific credibility, it is nonetheless important to consider whether maternal smoking is part of a particular lifestyle that has implications for the longer term development of children.

The difficulties inherent in the measurement of smoking should be mentioned at this point. There is no common unit of measurement, and different studies have used number of cigarettes per day, number of packs per day, and milligrams of nicotine per day to describe smoking behaviour. There is no agreement as to what consumption levels define "heavy" vs. "light" smoking. The collection of data on smoking during pregnancy has been done at different time periods, including each trimester, once at mid-pregnancy, or retrospectively at the time of delivery. There is also no agreement as to what constitutes the "no smoking" category. Different researchers have interpreted this as: never smoked; smoked previously but quit before pregnancy; or smoked previously but quit early in pregnancy. Consequently, it is very

difficult to compare results across studies.

In addition, reliability problems ensue from the use of self-report measures. There is ongoing research in which urine testing is being used to assess the reliability of cigarette and other drug use among pregnant women. Preliminary findings suggest that awareness that the self-report will be confirmed by urine assay may prompt women to report their use more accurately (Zuckerman, 1985). Nevertheless, in a recent study in which self reports were compared to urinalysis results, no evidence was found of serious under-reporting of smoking by women at the time of delivery (Woodward et al., 1987).

B/ Pregnancy and Neonatal Outcomes

Direct maternal smoking is associated with a variety of problems of reproductive outcome, the first chronological event being the problem of reduced fertility and delayed conception (Baird and Wilcox, 1985; Howe et al., 1985). The incidence of spontaneous abortions is up to two times as great for smokers (Abel, 1980; Fried, 1983; Longo, 1982). This appears to be the result of a toxic effect rather than due to teratogenic damage, as smoking has been found to be associated with an increase in spontaneous abortions of normal karyotypes (Boue, Boue and Lazer, 1975; Witter and King, 1979). During pregnancy, smokers are more likely to experience morning sickness (Kullander and Kallen, 1971). Smokers are also reported to have an increased incidence of

placenta previa, abrupto placenta and other bleeding disorders of pregnancy, and possibly premature rupture of the membranes. These increased risks were found to correlate with the level of maternal smoking during pregnancy and with the number of years the mother smoked prior to pregnancy (Witter and King, 1979; Longo 1982).

In a large-scale study by Meyer, Tonascia and Buck (1975), maternal smoking was found to have a highly significant, dose-related, and independent effect on perinatal mortality. Information from all single births in 10 Ontario hospitals in 1960-1961 (N = 51,490) was analyzed, 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 increase by 20% for smokers of less than 1 pack per day, and the offspring of heavy smokers (one pack or more per day) showed an increased risk of 35%. Meyer et. al.'s results have been corroborated in a number of other studies (reviewed by Landesman-Dwyer and Emmanuel, 1979; Malloy et. al., 1988; Martin, 1982; Witter and King, 1979).

Sudden infant death syndrome (SIDS) is generally considered to be the result of immature or inappropriate respiratory control combined with cardiac arrhythmia. There is some evidence that SIDS is associated with maternal smoking during pregnancy, and that this condition accounts for some of the increased mortality of smokers' offspring (Malloy et. al., 1988; McIntosh, 1984). In a case-control study of 125 SIDS babies, Naeye, Ladis and Drage (1976) found that the

incidence of mothers smoking more than 6 cigarettes per day was almost twice as high in the SIDS babies as in a control group, matched for gestational age, sex, race and SES.

On the average, smokers' babies weigh approximately 200 grams (or 1/2 pound) less than nonsmokers' babies. This finding has been consistently shown by more than 50 studies with data from a total of more than half a million births (reviewed by: Andrews and McGarry, 1972; Butler, Goldstein and Ross, 1972; Cope, Lancaster and Stevens, 1973; Fried and Oxorn, 1980; Longo, 1982; Meyer, 1978; Martin, 1982; U.S. Public Health Service, 1979). This weight reduction is consistent despite the diversity of the populations studied, and is independent of other maternal and infant factors that are known to influence birth weight. Most studies have failed to find a significant correlation between smoking and early birth. The offspring of smokers are therefore "small-for-date" rather than truly premature, and have experienced some degree of intrauterine growth retardation. There is clear evidence that this is a dose-response relationship, with the decrement in birth weight proportional to the number of cigarettes smoked (Longo, 1982; Witter and King, 1979). It is therefore generally accepted that the relationship between maternal smoking and reduced birth weight is one of cause and effect.

The literature generally accepts 2500 grams (approximately 5 1/2 pounds) as the level below which a baby is considered to be at risk due to low birth weight. Smoking mothers have a significantly higher risk

of delivering a baby weighing less than 2500 grams. Following his review of 25 studies in which data was collected on a total of 230,000 births, McIntosh (1984) calculated smokers' average relative risk for delivering a low birth weight baby to be 1.82. There is also evidence of an increase in the proportion of low birth weight babies as the level of maternal smoking increases (Meyer, 1978).

As well as being lighter, smokers' babies are usually of reduced body length, with smaller head and shoulder circumference measurements (Kullander and Kallen, 1971). Several studies have demonstrated that these body measurements are reduced in a direct, dose-response relationship to the level of maternal smoking (U.S. Public Health Service, 1979). Smokers are more likely to have "short-for-date" babies that are symmetrically small, and who maintain this low percentile standing in growth in later infancy. By comparison, infants of low weight gain mothers are relatively long and thin at birth, but after six months they catch up in weight (Miller and Hassanein, 1971). This suggests that maternal smoking leads to an overall retardation of fetal growth that may have long-term consequences.

C/ Infant Behaviour

There has been relatively little research conducted on the behavioural outcomes related to maternal smoking, in part due to the sophisticated observational techniques required to assess subtle

impairments in functioning. One such instrument is the Brazelton Neonatal Assessment Scale, a scale designed to assess an infant's capacity to interact with the environment (Brazelton, 1973). Twenty-seven items are provided to allow for observation of habituation, orientation, reactivity/irritability, motor maturity, and arousal. Saxton (1978) used the Brazelton scale to compare the behaviour of infants whose mothers smoked heavily during pregnancy with a group of controls matched for maternal age, social class and parity. When tested at 4 - 6 days of age, there were no significant differences between the groups on the overall score. Nevertheless, the four items that differentiated between the groups were tests of auditory habituation, responsiveness to a human voice and a bell, and consolability. In addition, the overall impressions recorded by the examiner indicated that smokers' babies tended to be more irritable, showed decreased ability for self-control and a general lack of interest.

Fried also used the Brazelton scale in his Ottawa Prospective Prenatal Study, a longitudinal study concerned with the effects of women's lifestyle habits during pregnancy on the development of their offspring (Fried, et al., 1980). The sample is comprised of volunteers who have entered the project since 1978. These women are typically well-educated and middle class. Drug and alcohol use, smoking data, nutritional, health and demographic information are collected during interviews in each trimester. Approximately 35% of the total sample are smokers. On the Brazelton test, smokers' babies showed increased

tremors and poorer habituation to auditory stimuli. A tendency to show less responsiveness to visual stimuli was also noted, but this was not statistically significant after controlling for potentially confounding factors (Fried and Makin, 1987).

Somewhat different results were found by Jacobson et al. in their use of the Brazelton scale to study the neonatal correlates of prenatal exposure to smoking, caffeine and alcohol. Infants of women who smoked during pregnancy exhibited lower scores on the irritability scale. The zero-order correlation of smoking and the orientation items was significant, but disappeared when the covariates were entered. The reduced irritability effect was attributed to a delayed onset of crying (Jacobson et. al., 1984). Jacobson's sample was tested at 3 to 4 days after birth, whereas Fried's and Saxton's testing occurred between days 3 and 6. When age at testing was controlled for in Fried's analysis, decreased irritability was not found.

Saxton interpreted the auditory orientation and habituation results as representing an impairment in auditory function. Supportive evidence for this conclusion was cited from the work of Beard and Grandstaff (1970) and Zelman (1973), in which exposure to the constituents of cigarette smoke was associated with hearing loss and the ability of adults to discriminate the duration of an auditory signal. Accepting Saxton's interpretation, one might predict later problems in auditory perception and language development for these children.

There is considerable controversy as to whether infant behaviour is predictive of later performance, however. The child development literature suggests that there are continuities in development, but they are not predictable on the basis of infant tests in any simple way. For instance, Rosenblith (1970) found little if any correlation between the auditory responses of 400 neonates with their later performance on pediatric, psychological and neurological tests at 4 months, 1 year and 4 years. There was some evidence, however, that an unusual response or a failure to respond to sound may indicate a neurological dysfunction rather than a hearing impairment. This appears consistent with Fried and Makin's interpretation that the neonatal effects should be considered as evidence of either an immaturity or dysfunction in the infant's central nervous system. This interpretation would be more in line with the view that habituation is a cognitive process requiring integration of sensory and motor responses and therefore at least partially under cortical control (Leader, Baille, Martin and Vermeulen, 1982; Lewis, Goldberg and Campbell, 1969).

Further support for this latter interpretation is provided by a study of the neurological status of infants exposed to cigarette smoke in utero using the Prechtl Neurological Exam, a comprehensive standardized examination of state, elicited reflexes, posture and movement. At 9 and 30 days of age, prenatal cigarette exposure was found to be associated with hypertonicity and greater incidence of fine

tremors and startles (Fried, Watkinson, Dulberg and Dillon, 1987). These results were consistent with an interpretation of increased central nervous system excitation.

Although the direct implications of these behavioural outcomes are uncertain at this time, there may nevertheless be indirect consequences for parent-infant interaction. Difficulty in limiting responses to repetitive and redundant stimuli can cause a baby to become overwhelmed or exhausted, and therefore spend much more time crying and sleeping in order to shut out such stimuli from his/her attention. Such a baby may be perceived as "difficult" and unresponsive by its parent (Fried, 1983), and might thus elicit deficient parenting and be therefore at risk for later developmental delay.

D/ Long-term effects

With a few exceptions, research findings indicate that maternal smoking during pregnancy is related to small but significant decrements in children's physical development and in performance on tests of intellectual ability, perceptual functioning and academic achievement. What follows is a review of these findings, organized to present a picture of what is currently known of the chronological development of children of mothers who smoke from infancy into adolescence.

Many studies have looked at physical measurements in order to determine whether the reduced size found at birth continues into childhood. Results generally indicate that differences in height, weight and head circumference found between children of smokers and non-smokers remain significant throughout early and middle childhood (Davie, Butler and Goldstein, 1972; Dunn, McBurney, Ingram and Hunter, 1976; Fried and O'Connell, 1987; Hardy and Mellits, 1972). By age 16, Fogleman found that there was no significant difference in height for girls, most of whom had reached their adult height. For boys, many of whom were still growing, there remained a significant difference. Fogleman suggested that the effect of smoking on physical growth may therefore be related to a delay in the rate of growth, but not to the eventual size reached at maturity.

Streissguth's Seattle Longitudinal Prospective Study was designed to study the long-term effects of social levels of maternal use of cigarettes, alcohol and caffeine during pregnancy. Their sample of 500 women was selected for follow-up on the basis of cigarettes and alcohol use, so as to include all the heavier drinkers and smokers and a representative sample of light and non-users of alcohol. Streissguth describes this sample as being predominantly white, married and middle class (Streissguth, Barr, Martin and Herman, 1980). Development of the offspring of these mothers has been assessed at various intervals. At 8 months, 462 infants were assessed using the Bayley Scales of Infant Development. A multiple regression analysis was used to evaluate the effects of nicotine after partialling out the effects of alcohol and

caffeine use, gestational age, parity, and maternal education. The effect of nicotine was not statistically significant for either the Mental Development Index nor Psychomotor Development Index scores (Streissguth et al., 1980).

Fried and Watkinson (1988) also analyzed scores on the Bayley Scales with infants at 12 and 24 months in the Ottawa study. At 12 months, smoking (measured as use of nicotine) was significantly related to the Mental Development Index, but not the Psychomotor Index. Smoking was also found to be related to a cluster of items relating to auditory responsiveness on the Bayley Infant Behaviour Record at 12 and 24 months. At 24 months, after partialling out the effects of potentially confounding variables, smoking was not a significant predictor for either Bayley scale. Smoking was, however, significantly related to all six subscales of the HOME test, Caldwell's inventory of home stimulation and maternal involvement. Fried and Watkinson propose that the traditional test of the unique contribution of a drug such as nicotine in a regression analysis is likely too conservative, as it assigns shared variance to the potentially confounding variables. They suggest that although smoking could not be shown to have a significant direct effect on the outcome measures at 24 months, there was evidence of an indirect effect via the home environment.

Hardy and Mellits (1972) reported results from a pediatric neurological examination given to a group of 88 children of smokers compared with a set of matched pairs from non-smokers, matched for

race, maternal age, education, child's sex and date of delivery. At one year of age, no significant difference in the incidence of abnormalities was found. The raw data, however, indicate that 96% of the children of non-smokers were considered normal (i.e. no abnormalities or soft signs present) vs. only 90% of the smokers' children. This was not a statistically significant difference given the small sample size, but it is felt to be, nonetheless, a clinically meaningful finding that should probably have been given more prominence in their report.

Hardy and Mellits also found no significant differences between their smoking and non-smoking pairs at four years of age on tests of intelligence, concept formation and fine and gross motor functioning. This finding is contrary to that of Sexton, Fox and Hebel (1986), in which children of women who quit smoking during pregnancy had significantly higher mean scores on tests of cognitive development than offspring of heavy smokers. As will be discussed later, Hardy and Mellits' study must be interpreted with caution, given their relatively small sample size.

When Streissguth and coworkers used a vigilance task, said to be a measure of sustained attention, four-year-old children whose mothers had smoked during pregnancy showed increased errors of commission and omission. A dose-response effect was found, as was a significant alcohol X nicotine interaction. These children also spent significantly less time oriented to the task (Streissguth, Barr and

Martin, 1984.) These results were recently corroborated with children 4 to 7 years of age, using measures of visual and auditory vigilance and an activity monitor. Smoking during pregnancy was found to be significantly related to poorer visual and auditory attention and to a higher activity level (Kristjansson, personal communication, 1987).

No maternal smoking effects were found by Landesman-Dwyer, Ragozin and Little (1981) using naturalistic observations of four-year olds in their homes using ratings of activity level, attention and compliance. Maternal ratings of their child's temperament, however, revealed that these children were perceived to be more willing to approach strangers and novel situations, and to be more stubborn and show sustained involvement in activities. In addition, smokers' children were rated as displaying more intense negative reactions when upset, annoyed or disciplined.

There are five research reports of the consequences of maternal smoking on children as they reach school age, of which Hardy and Mellits' 1972 study is generally considered to be the pioneering work. At the age of seven years a psychometric test battery was given, including tests of intelligence, perception and academic achievement. No significant mean differences were found between the smoking and non-smoking groups, with the exception of the (WRAT) spelling score, which was significantly lower for the smoking group. Criticisms of this study often mention the small sample size ($N = 88$ pairs), and also that Hardy and Mellits should not have matched their groups for birth

weight, as reduced birth weight is known to be associated with smoking (Donovan, 1973; Dunn et al., 1976).

A British study published in 1972 by Davie, Butler and Goldstein also examined the effects of maternal smoking on children at age seven, as part of the large-scale National Child Development Study. This sample consisted of the cohort of over 17,000 births in England, Scotland and Wales between March 3 to 9, 1958. The net effect of maternal smoking on various outcome measures at age seven was determined using a multiple regression analysis in which smoking was entered after controlling for a number of other confounding variables, such as birth order and sex of infant, birth weight, length of gestation, number of younger siblings in home, and social class. A test of reading achievement showed a significant 4-month retardation for the children of smokers, with no dose effect. In addition, children of women who smoked during pregnancy were more frequently rated as badly adjusted in school.

Dunn et al. (1976, 1977) assessed the effects of maternal smoking as children reached the age of 6 1/2 years. Neurological and encephalographic (EEG) abnormalities were found to be slightly more common among the children of smoking women, although not significantly so. Using a psychometric assessment (similar to that used by Hardy and Mellits), the mean scores of the children of smokers were found to be lower on 45 of 48 comparisons, and significantly lower on 14. Teachers rated more misbehaviour for children of smokers, but this was

significant only for boys. Children of smokers were also found to be less frequently in the grade appropriate to their age, and they were rated as showing poorer overall achievement.

The National Collaborative Perinatal Study was a very large-scale project designed to examine the role of a number of medical, perinatal, and demographic factors as antecedents in the development of later learning and neurological problems in childhood. Naeye and Peters (1984) reported the results from the Boston sub-sample ($N = 9,024$). At seven years of age, children were assessed for attention span, motor activity and achievement in reading, spelling and arithmetic. The inter-correlations between smoking, the 47 other parental and neonatal factors and the outcome measures were examined in order to identify potentially confounding variables. When the independent effects for smoking were analyzed, results showed that children of mothers who smoked throughout pregnancy had slightly lower scores on spelling and reading tests, and were more frequently rated as being hyperactive, with short attention spans.

Naeye and Peters conducted a second set of analyses in an attempt to control for differences in child-raising practices and genetic factors. Intrapair comparisons were made of same-sex siblings whose mothers had smoked in only one of their two pregnancies ($N = 578$ pairs). Results again indicated a statistically significant relationship between smoking during pregnancy and children's development at seven years of age. Quantitatively, however, the

achievement test scores were only 2 to 5% lower and behavioural abnormalities 2% more frequent when the mothers had smoked. This study thus provides strong evidence for an association between smoking during pregnancy and later impairments in behaviour and academic achievement. Although a direct causal relationship was not established, the sibling comparison findings showed that this relationship was independent of many demographic, genetic and home environment influences.

Nicols and Chen (1981) conducted an analysis of the data from the entire National Collaborative Perinatal Project, consisting of information on over 50,000 children. It was not their intention to study maternal smoking per se, but rather to consider smoking as just one of many possible predictors of minimal brain dysfunction in a large unselected population. Three types of minimal brain dysfunction (MBD) were considered: learning difficulties (LD), hyperactive-impulsive (HI), and neurological "soft signs" (NS). Over 300 variables were considered in an attempt to identify important predictors of MBD symptoms.

Children of women with a history of more than 10 years smoking showed a slight increased risk for all three MBD factors. When smoking history was considered in combination with the 32 other demographic and maternal characteristics, it emerged as the most predictive variable for both the HI and NS groups. Smoking more than 20 cigarettes per day during pregnancy also showed an association with increased risk of all three MBD symptoms, the greatest risk being for severe LD. There was

also some evidence of a dose effect, so that an even greater risk was seen with women who smoked more than 40 cigarettes per day. When smoking during pregnancy was considered in combination with 12 other pregnancy and delivery complications variables, it remained a significant predictor for all three MBD factors, again emerging as the "number one" predictor for HI and NS.

This study is unique in the literature in terms of its large sample size, prospective design and its comprehensive analysis of the associations between minimal brain dysfunction and a multitude of possible antecedent factors. Nicols and Chen have provided impressive evidence that maternal smoking is related to MBD, particularly hyperactive-impulsive behaviour.

There have been four studies published to date in which smokers' children have been assessed at older ages. Rantakallio (1983) conducted a longitudinal study of 1,819 children of women who smoked during pregnancy and their matched controls (non-smokers), all born in northern Finland in 1966. Controls were matched for parity, marital status, maternal age, place of residence and single or multiple birth. No difference was found in the incidence of serious mental and physical conditions such as mental retardation, diabetes, etc. At the age of 14, the children of smokers showed poorer academic performance on school report cards. These results remained significant even after adjusting for the mother's height and age, social class (father's occupation), number of siblings, and sex of the child.

Two studies that originated with the National Child Development Study - Butler and Goldstein (1973) and Fogleman (1980) - followed the British sample to the ages of 11 years and 16 years, respectively. Butler and Goldstein found that at age 11, children of women who smoked during pregnancy showed a retardation of 3 to 5 months on tests of general intelligence, reading and mathematics. At the age of 16 years, Fogleman's results also indicated that children of smokers obtained significantly lower scores on tests of reading and mathematics. These results therefore indicate that smoking in pregnancy is associated with slight delays in mental development, and that the gap between children of smokers and non-smokers did not widen between the ages of 7 and 16 years.

The final study is an interesting aberration, in light of the otherwise almost unanimous agreement thus far. Lefkowitz (1981) compared the offspring of smokers and non-smokers when the children were ten years old. No statistically significant differences were found on four classes of variables: physical, intellectual, affective, and personal and social functioning. Indeed, higher mean scores were found for smokers' children on the physical growth and intellectual measures. These findings are puzzling, as this is the only study to have found such results. This was a retrospective study, however, and unreliability could therefore have easily been introduced into the collection of smoking data ten years after the fact. In addition, it is possible that this sample differs in some important way from the

others.. Suspicions are raised towards this possibility because Lefkowitz did not describe how his sample was obtained, only that it consisted of 241 children that were part of a larger study on childhood depression.

Thus with a few exceptions, research findings indicate that maternal smoking during pregnancy is related to small but significant delays in children's physical development and in performance on tests of intellectual ability, perceptual functioning and academic achievement. Thus, there is some evidence of cigarettes acting as a behavioural teratogen, resulting in subtle but generalized neurobehavioural deficits.

E/ Constituents of Cigarette Smoke

The specific etiological agents responsible for the effects discussed in previous sections have yet to be determined. A major difficulty in this regard stems from the fact that cigarette smoke is known to contain more than 3800 compounds (National Research Council, 1986). The two major constituents that have been suspected of causing the harmful effects on the fetus are carbon monoxide and nicotine.

Cigarettes contain up to 2 mg. of nicotine per cigarette, depending on the brand. Nicotine acts by both stimulation and inhibition of the nervous system, depending on the dosage (Abel, 1984).

Nicotine stimulates the release of noradrenaline, and as a result has a vasoconstriction effect on peripheral blood vessels. This produces increased pulse and blood pressure and decreased fetal blood supply. The plasma half-life for nicotine is about 40 minutes, and its main metabolite is cotinine. Decreased intervillous blood flow normalizes 15 minutes after smoking a cigarette (Fried and Oxorn, 1980; Longo, 1982).

Nicotine also readily crosses the placenta to directly affect the fetus. Research with rats has demonstrated that nicotine rapidly accumulates in fetal lung, trachea, adrenals, kidney and brain. Nicotine concentrations in the fetus are persistently higher than in the mother for up to 20 hours after exposure (Martin, 1982) due to the immaturity in the fetal liver and kidney's ability to metabolize and eliminate nicotine. The measured effects on the fetus are increased heart rate and reduced blood supply from the placenta.

Nicotine has been shown to cause an abrupt and significant reduction in the incidence of fetal "breathing movements", which are considered to be important to the development of the fetal respiratory system. This disruption implies a direct effect of smoking on fetal CNS functioning (Longo, 1977; Manning and Feyerabend, 1976). Fetuses of rats exposed to nicotine were found to have brain stem damage (Krous et al., 1981). These effects are hypothesized to be related to the association between maternal smoking and sudden infant death syndrome (SIDS).

Cigarette smoke contains about 4% carbon monoxide by volume (Meyer and Tonascia, 1977), but the amount ingested by the smoker depends on the rate and depth of inhalation. Hemoglobin binds more readily with carbon monoxide than with oxygen, and this combination produces elevated levels of carboxyhemoglobin in the blood of pregnant smokers. Carbon monoxide also impairs the off-loading or transfer of oxygen to tissues. Thus, carbon monoxide acts to reduce the oxygen supply to the fetus. Carbon monoxide also crosses the placenta. Fetal concentrations are much higher than maternal levels and are slower to return to normal (Bureau et al. 1980), due to a slower fetal metabolism and elimination rate and also because fetal hemoglobin shows a stronger affinity to carbon monoxide than found maternally.

Rats prenatally exposed to low concentrations of carbon monoxide showed similar effects as those noted with heavy human maternal smoking, including increased spontaneous abortion, increased perinatal mortality and reduced birth weight. Neurobehavioural testing revealed lower behavioural activity levels; biochemical testing indicated altered central catecholamine activity, a reduction in total brain protein and a dose related deficit in cerebellum growth (Fechter and Annau, 1979; Storm and Fechter, 1985). Cell numbers, but not cell size, were reduced in carcasses and parts of the brain (Haworth, 1973). Prenatal exposure to relatively low levels of carbon monoxide has been shown to be related to differences in amplitudes on visual evoked potentials (Dyer et al., 1979), and to adult learning and memory

deficits (Mactutus and Fechter, 1984, 1985).

There are, of course, many dangers in extrapolating from animal models to the human condition. In this field of research, in particular, the differences in relative maturity at birth must be taken into consideration. The early postnatal period of the rat corresponds to the last trimester of human gestation, so that prenatal exposure of the rat fetus can only be used in understanding first and second trimester effects for humans. A model for third trimester effects would involve postnatal exposure in the rat, which might involve different mechanisms, and thus not be comparable to the human fetus in utero. The animal literature is nevertheless important as the findings suggest that smoking may have an effect on human embryos in the first trimester during organogenesis, and that this effect may be long-term and not reversible postnatally.

F/ Theories re Mechanism

Several hypotheses have been developed in attempts to explain the mechanism by which smoking produces its effects. The explanation that is currently most accepted is the hypoxia hypothesis, whereby cigarette smoking by pregnant women causes a reduction in the supply of oxygen to the fetus, and this in turn is responsible for the growth retardation noted at birth. Nicotine is thought to contribute to this effect via vasoconstriction and decreased blood flow in the utero-placental bed

and perhaps in the umbilical and fetal circulation. This causes a temporary decrease in oxygen exchange and tissue hypoxia. Longo (1982) describes the carbon monoxide contribution to hypoxia as a compounding effect, first by decreasing the oxygen transport capacity of blood and also by lowering the oxygen tension for the transfer of oxygen to the tissues. Nicotine thus rapidly induces hypoxia during the smoking of the cigarette. Carbon monoxide-mediated hypoxia develops slowly, and its effects persist for a longer time than that of nicotine. Longo describes these two substances as working in concert so that regular maternal smoking maintains the fetus in a chronic hypoxic state.

High altitudes have effects on pregnancy very similar to those of maternal smoking. In both instances there is a higher placental ratio, which is the ratio of the weight of the placenta to the baby's birth weight. This ratio increases directly with both higher altitude and with maternal smoking level (Meyer and Tonascia, 1977). Additional similarities include a higher incidence of placenta previa, and reduced birth weight; these effects have been linked to the shortage of oxygen available (Meyer, 1978).

An alternate hypothesis to the hypoxia theory is that smokers have reduced appetites, poorer nutrition, gain less weight, and therefore produce smaller babies. This hypothesis lost credibility after Meyer's analysis of the relationship between smoking, maternal weight gain and birth weight using data from the Ontario Perinatal Mortality Study (Meyer, 1978). This large data base ($N = 51,490$) allowed for the

control of many other factors that influence birth weight and maternal weight gain. Results showed that maternal weight gain distributions were the same for smokers and nonsmokers. Within each level of maternal weight gain, the percentage of neonates weighing less than 2500 grams increased consistently with smoking level. Smoking does not therefore appear to act indirectly through a reduction in nutrition, but rather appears to have a direct and independent effect on fetal growth.

Yerushalamy (1972) proposed the hypothesis that smoking effects are indirect, and are mediated by characteristics of the smoker, not the smoke. Yerushalamy argued that women who become smokers have specific personality and biological characteristics that make them a distinct subgroup predisposed to have infants with lower birth weights. Evidence for this interpretation was provided by his study of birthweight of offspring from women who were smokers, nonsmokers, and "future smokers" (i.e. had not started to smoke during the pregnancy, but subsequently did so). Infants born to future smokers had the same higher incidence of low birthweight as did infants of smokers (Yerushalamy, 1972). Yerushalamy's study has been criticized by most researchers in this area, due to his retrospective design and lack of control for other variables which are known to affect birthweight, such as maternal age, birth order and number of previous pregnancies (Fried, 1983). Additional evidence against the "indirect" theory was provided by the finding that the risks for low birthweight and perinatal mortality for babies of mothers who quit smoking by the fourth month,

were no higher than for babies of nonsmokers (Butler, Goldstein and Ross, 1972). Corroboration of this finding was recently supplied by Jacobson et al. (1984), who found that abstaining from smoking during pregnancy reduces potential effects on birth size.

A Norwegian study provided some support for a genetic influence on smoking habits during pregnancy with the finding that the correlation of smoking habits of monozygotic (MZ) twin pairs was higher than that of dizygotic twins. However, when the birth weights of smoking-discordant twin pairs were studied, offspring of smoking MZ twins weighed 127 g. less than the offspring of matched non-smoking twin pairs (Magnus, Berg, Bjerkedal and Nance, 1985). This result provides further evidence that the effects of smoking are direct, and not dependent on constitutional factors.

Naeye utilized sibling comparisons in which the mother smoked in one pregnancy and not the other. Drawing from the large prospective Collaborative Perinatal Study, he found 578 same-sex sibling pairs, controlling for maternal age, marital status and working outside the home. Although birth order correlated with birth weight, mothers had smaller infants in the pregnancies in which they smoked, regardless of birth order (Naeye, 1978a). When these sibling pairs were followed-up at age 7, significant differences were found on tests of academic achievement and behaviour (Naeye and Peters, 1984).

There is thus considerable support for the hypoxia hypothesis for the mechanism of effect of maternal smoking. Nicotine and carbon monoxide appear to act in concert to reduce blood and oxygen supply to the fetus, and intrauterine growth retardation is the result.

.2 Passive Smoking

The effects of active maternal smoking have been extensively studied over a period of more than 30 years, but the effects of passive exposure to cigarette smoke have not received much attention until relatively recently. This chapter will summarize what is known about the constituents of environmental tobacco smoke and the health consequences of passive smoking on the individual, with particular attention to the effects on the developing child.

The term "passive smoking" is used to refer to the exposure of nonsmokers to tobacco combustion products from the smoking of others (U.S. Department of Health, 1984). In this study "cigarettes" will be used to refer to all tobacco products. Environmental tobacco smoke (ETS) refers to tobacco smoke in the environment, which is derived from exhaled smoke from the smoker and sidestream smoke from the burning end of the cigarette.

A/ Prevalence

The probability of exposure of nonsmokers to environmental tobacco smoke (ETS) is a function of several variables, one of which is the number of active smokers with whom the nonsmoker comes into contact. As mentioned in the previous chapter, 30% of adult Canadians report

regular smoking (Health and Welfare, 1988). This implies that large segments of the Canadian population are exposed to ETS. Although the number of smokers is declining, the total consumption of cigarettes has not declined as rapidly, so that among regular smokers average cigarette consumption has increased from 27 to 30 cigarettes per day from 1955 to 1985. The nonsmoker who has close contact with smokers may be currently exposed to greater amounts of ETS than 30 years ago (National Research Council, 1986).

There is a dearth of large-scale epidemiological studies which would provide data on the extent of passive smoking in the population. The single study reporting prevalence rates for the general (U.S.) population indicates that 86% of nonsmokers are likely to be exposed to tobacco smoke at work or at home or both (Repace and Lowrey, 1983, reported in Collishaw, Kirkbride and Wigle, 1984). Given that Canadians have a higher cigarette consumption rate, this proportion may be higher in Canada, although recent anti-smoking regulations introduced into many businesses may have recently reduced exposure in the workplace.

There are many inherent difficulties in calculating the prevalence of passive exposure. Until recently, studies have relied upon questionnaire data from family members in order to quantify risks for health outcomes such as cancer. Questionnaire data are useful in classification into broad categories (e.g. exposed vs. non-exposed), but misclassification errors can occur due to errors reporting current

and past smoking habits. For instance, current or exsmokers may be wrongly classified as nonsmokers. The unreliability of self-report measures has been quantified in two large longitudinal studies. A 5% error rate was found in both studies for respondents who reported never smoking although they had reported smoking several years earlier (National Research Council, 1986). Another problem occurs when exposure to ETS in another environment is overlooked (e.g. work environment), so that an individual is classified as "non-exposed", whereas they have indeed had considerable exposure.

There is currently no direct measure for dose of exposure to ETS. Quantification of exposure is a highly complex function depending on proximity to the smoker, characteristics of the environment (e.g. size, degree of ventilation), and number and kind of cigarettes smoked. Measurement of long-term exposure also requires a history of hours of exposure per day, over a period of time, and the ages at which these exposures occurred. Intensity over time is sometimes reported as an average exposure to the smoke from x cigarettes per day.

Air monitoring has been used to measure concentrations of ETS constituents in indoor environments. Given that cigarette smoke is comprised of 3800 different compounds, it has been necessary to monitor a few individual constituents as proxies for ETS. Problems that arise with this method are related to the degree to which the marker or tracer substance is unique to tobacco smoke and whether it exists in sufficient quantity to be measured with precision. Personal monitors

are used to measure concentrations of a substance in the immediate breathing zone of an individual, using filter or vapour traps with subsequent laboratory analysis. These monitors assess an individual's total exposure to individual ETS constituents. This procedure apparently shows promise, and has been able to demonstrate that ETS exposure as measured by personal monitors is higher for individuals who report being exposed than for those who report no exposure (National Research Council, 1986).

B/ Constituents of Environmental Tobacco Smoke (ETS)

The major source of ETS is sidestream smoke, which is emitted from the burning end of a cigarette in between puffs. A second source of ETS is exhaled mainstream smoke. Mainstream smoke is filtered twice before it is exhaled - first as it is drawn through the cigarette and secondly by the smoker's own lungs. ETS has different physicochemical characteristics than mainstream smoke inhaled by active smokers. Undiluted sidestream smoke contains the same toxic compounds as in mainstream smoke, but often in much higher concentrations. For instance, nicotine is 2 to 3 times more concentrated, and carbon monoxide is 8 times more concentrated in sidestream smoke (Collishaw, Kirkbride, and Wigle, 1984). These concentrations are diluted in the environment as they mix with the ambient air. Different concentrations of these substances occur depending on the nature of the tobacco product, the degree of inhalation by the smoker, and the degree of

ventilation in the environment. The "age" of the smoke is also a factor, as the chemical composition changes with time.

As mentioned previously, accurate and precise measures of the constituents of ETS in the environment are not yet available. Nevertheless, these chemicals or their metabolites have been detected and measured in samples of blood, urine, and saliva from passive smokers. In theory, such chemicals could be used as biological markers to estimate recent ETS exposure. Two of the major biological markers are nicotine and its metabolite, cotinine, both of which have been found to be sensitive and specific indicators of exposure to tobacco smoke. The mean plasma and urine concentrations of nicotine and cotinine in adult passive smokers range from less than 1% to 10% of the values observed in active smokers (National Research Council, 1986; Pershagen, 1986). These values are difficult to interpret, however, given the reported slower clearance of cotinine in nonsmokers (Sepkovic, Haley and Hoffman, 1986). The extended residence time in the body could conceivably allow for additional harmful effects.

There are conflicting results regarding cotinine concentrations in the amniotic fluid of pregnant passive smokers. Andressen, Ng, Iams and Biachine (1982) screened the amniotic fluids of 24 passive smokers. In comparison with a mean cotinine concentration of 129 ng/ml for active smokers, only 2 of 24 passive smokers had concentrations above 5 ng/ml. This result is in contrast to the finding of Smith, Austen and Rolles (1982) that 4 out of 4 passive smokers had concentrations of 9

ng/ml or more. Additional research is obviously needed in this area, with larger samples, before conclusions can be drawn. Of concern is that neither study reports whether exsmokers were included as passive smokers, a fact that may have influence on the rate of clearance of cotinine, as mentioned above.

Thiocyanate levels have also been studied in pregnant women. No evidence has been found that passive cigarette smoking results in higher maternal or umbilical cord thiocyanate concentrations than found in nonsmokers. This is an important finding as thiocyanate has been shown to have an inverse relationship with birth weight (Hauth et al., 1984).

Carbon monoxide is the major toxic element by weight in ETS (Collishaw, Kirkbride and Wigle, 1984). In a study in which samples of expired air were collected from school children, the samples showed a trend of increasing carbon monoxide levels with the number of close relatives smoking in the home (Gilles, Kristmundsdottir, Wilcox and Pearson, 1986). Carbon monoxide has been shown to play an important role in the effects of active smoking on fetal development. Unfortunately, no studies have thus far attempted to measure the exposure to carbon monoxide by pregnant passive smokers.

The American National Research Committee report concludes, after an exhaustive review of over 200 epidemiologic and experimental studies, that nonsmokers incur considerable health risks from passive smoking (National Research Council, 1986). The latest Surgeon General's report has linked passive smoking with as many as 4700 deaths of nonsmokers a year in the United States and has called for broad and immediate action to restrict smoking in the workplace and public gathering areas (U.S. Department of Health and Human Services, 1986). These reports provide an extensive survey of all available research in this area, and no attempt will be made to duplicate their efforts. What follows is a brief summary of these findings, with particular emphasis on the health of the developing child.

The most common acute effects associated with exposure to ETS are eye, nose and throat irritations, and annoyance with objectionable odour. There is some evidence that certain individuals are "hypersensitive" to the effects of ETS and report allergic-like symptoms as a result of exposure. The effects of long-term exposure have been more difficult to assess in adults, due to the confounding influence of the cumulative exposure to many other environmental and occupational factors. There is some evidence of a minor reduction in pulmonary function in adults exposed to ETS. Further studies are apparently still needed to determine if there are susceptible subgroups (such as asthmatics) who are particularly vulnerable to ETS exposure. Spouses of smokers have been found to have an increased risk of developing lung cancer, but there is no evidence of any increased risk

for any other type of cancer.

There are more than 30 studies of respiratory effects in children, and the majority of the evidence indicates that ETS exposure increases the frequency of respiratory symptoms and infections. Respiratory symptoms such as wheezing and coughing are increased in children of smoking parents. Also, respiratory infections such as pneumonia and bronchitis are significantly increased in children living in homes where parents smoke. An interesting study in this regard is provided by Chen, Li and Wu (1986), who analyzed this relationship for 1,058 children in Shanghai. Smoking was present in 72% of the families, but none of the mothers in this study smoked, thus eliminating the confound of prenatal exposure. Results showed a dose-response relationship for increased risk of severe respiratory illness as the daily cigarette consumption increased.

Parental smoking has been linked with an increased risk of chronic ear infections in children, and a higher incidence of tonsillectomy and adenoidectomy (Kraemer et. al., 1983). Several studies have also examined possible relationships between chronic exposure to ETS and parameters of growth. There is some evidence that children of smokers have reduced growth and development. To date, only one study has attempted to separate the effects of in utero vs. postnatal exposure. After adjusting for social class and other social factors, Rona, Chinn, and Du Ve Florey (1985) found that reduced height was associated with increasing numbers of cigarettes smoked in the

home, regardless of whether the mother smoked during pregnancy and regardless of which parent smoked.

Lest the reader draw the conclusion that the literature is unanimous in finding deleterious effects to the health of passive smokers, the conclusion of Aviado, following his review of the literature, is that "...there is no substantial evidence to support the view that exposure to environmental tobacco smoke presents a significant health hazard to nonsmokers" (Aviado, 1986, p.137). With regard to the association found between parental smoking and childhood respiratory diseases, Aviado argues that this could be due to cross-infection by the parents or to a genetic predisposition to such illnesses in both parents and children. He also suggests that sufficient care has not been taken to control for confounding risk factors such as parental neglect and SES. Although his objections are probably overstated, the suggestions that alternate explanations and adequate control of potential confounds receive adequate attention is probably well-taken.

Indeed there are several methodological issues involved in studying causal relationships for outcomes with low relative risks. Pershagen (1986) suggests several sources of systematic error to which such studies are sensitive. Sample selection bias can occur as these studies are restricted to subjects for whom exposure data is known. As mentioned earlier, an overestimation of risk associated with passive smoking could happen if some smokers or exsmokers reported themselves

as nonsmokers, and if this was more common among spouses of smokers than spouses of nonsmokers. A bias could be introduced in case-control studies if the interviewer is not blind to the smoking status of the subjects. Systematic errors also occur when confounding factors such as exposure to other sources of ETS constituents (such as outdoor air pollution) have not been adequately controlled. In addition, random misclassification in the exposure categories as a result of failure to consider all sources of exposure, would reduce any association found for passive smoking, as would a small sample size.

For these reasons, the risk estimates for health outcomes may be invalid to some degree, and it is thus difficult to evaluate these findings. Further epidemiological research will be required, with emphasis given to the development of more precise and accurate exposure information.

D/ Passive Smoking During Pregnancy

Three studies reporting data for passive exposure during pregnancy indicate that about 34 to 50% of nonsmoking pregnant women are passive smokers (Karakostov, 1985; Martin and Bracken, 1986; Smith, Austin and Rolles, 1982). The effects on the fetus of passive smoking during pregnancy are not as well-documented as are the effects of active smoking. Some studies have approached this question indirectly by evaluating outcomes associated with paternal smoking. Results until

recently have been equivocal, and the associations that were found were sometimes dismissed as biologically nonsensical.

In nine studies, paternal smoking was found to be related to reduced birthweight and reduced height, higher perinatal mortality, and a higher incidence of miscarriages and malformations (Borlee et al., 1978; Gibel and Blumberg, 1973; Karakostov, 1985; Koo, Ho and Rylander, 1988; MacMahon et al., 1966; Mau and Netter, 1974; Rubin et al., 1986; Schwartz-Bichenbach et al., 1987; Yerushalamy, 1972). In three other studies, however, no neonatal effects were attributed to paternal smoking (Holmberg and Numinen, 1980; Hughes et al., 1982; Underwood et al., 1967). None of these studies attempted to measure the mother's actual exposure to her husband's smoke during pregnancy, nor did they consider exposure in the work environment. Thus, paternal smoking served as a proxy measure for passive smoking, and this poses a problem in interpreting these findings.

The single study of the long-term consequences of paternal smoking was done in Finland by Rantakallio (1983). A sample of more than 1700 children of smokers and their matched controls were followed from birth to 14 years of age. As mentioned previously, Rantakallio found that maternal smoking was related to decreased height and poorer performance at school. Using a regression analysis, family background factors (eg. social class, place of residence, number of children in family) and paternal smoking were entered as possible explanatory variables. Maternal smoking was found to be less important than many

other family background characteristics in terms of accounting for variance at 14 years. However, paternal smoking showed about as strong an association with mental and physical development as did maternal smoking, after controlling for other background factors. Given the high correlation found between paternal and maternal smoking, comparison of regression coefficients to determine relative importance is probably not a wise statistical procedure (Pedhauzer, 1982). In addition, as Rantakallio observes, it is impossible to determine whether prenatal or postnatal exposures (or both) are the cause of the deleterious effects. Nevertheless, this study provides some evidence that the mother's passive exposure to her husband's smoking is related to physical and mental outcomes that are similar to those found for active maternal smoking during pregnancy.

Martin and Bracken (1986) have recently published a prospective study in which passive smoking was defined as exposure to someone else's cigarette smoke for at least two hours per day, either at home or at work, during pregnancy. With a sample size of 3,881, approximately 25% of the women had not smoked (actively) during pregnancy, but reported passive exposure. After adjusting for a number of confounding factors, passive smoking was found to double the risk of delivering a baby of low birth weight. Although the mean reduction in birthweight was not significant for the sample as a whole, when only the babies delivered at term were considered the mean decrease in birthweight was significantly reduced by 30 grams. The magnitude of effect for passive smoking was found to be considerably smaller than

that due to active smoking. The adjusted odds ratio for delivering a low birth weight baby was 2.17 for passive smoking, in comparison with 3.54 for active smoking. The reduction in mean birth weight for passive smokers was 30 g., vs. 137 g. for active smokers. No additive effect was found for passive exposure for the active smokers, and passive smoking showed no effect on the length of gestation.

Martin and Bracken's study provides convincing evidence that passive smoking during pregnancy may be related to similar neonatal outcomes as active smoking. This study is the first to define passive smoking and to consider exposure from sources other than from paternal smoking. Measurement issues remain problematic, however, and some degree of misclassification is still a possibility in this study. In addition, it appears that ex-smokers who did not smoke during pregnancy were considered to be non-smokers. This may have introduced bias into the Results, as smoking history is also a predictor of birthweight. Nevertheless, it appears evident that ETS exposure increases the risk that an infant will be born weighing less than 2500 grams. It also appears that passive smoking appears to exert its effect primarily through growth retardation in term newborns.

.3 Objectives of Current Research Study

The major issue addressed in the current research study is whether the long-term consequences of passive smoking during pregnancy parallel those of active smoking. Environmental tobacco smoke (ETS) consists of the same toxic constituents as mainstream smoke, although relative proportions of these constituents differ. It was therefore predicted that children of women passively exposed to ETS during pregnancy would demonstrate a similar profile of abilities to that of children of active smokers. It was also predicted that the magnitude of differences between passive smoking and non-smoking groups would be smaller than the differences found between active smoking and non-smoking groups.

Demographic characteristics such as income and level of education have been found to correlate with smoking. Family socioeconomic status has been found to act as a moderating variable, mitigating the direct relationship between smoking and behavioural outcomes in long-term studies of active smoking. It was predicted that passive smoking would also show a negative correlation with demographic factors, and that control for these potential confounds would reduce the magnitude of the relationship between passive smoking and behavioural outcomes.

Home environment is also an important predictor of behavioural outcome at school age, and the relationship between home environment

and smoking status was addressed. Smoking has been shown to have significant effects on the child's health and development during infancy and early childhood, and it is possible that smoking status is associated with differences on measures of family environment. It was also predicted that family environment would act as a mediating variable in the direct relationship between smoking and behaviour.

II METHOD

.1 Design

A three-group design was employed in this study. The three groups will be referred to as Active Smoking, Passive Smoking and Nonsmoking. The Active Smoking group was defined as children of mothers who smoked any quantity of cigarettes at any time during their pregnancy. The Passive Smoking group was comprised of children of nonsmoking women who were passively exposed to cigarette smoke during pregnancy. The Nonsmoking group was defined as children of mothers who were neither active nor passive smokers during pregnancy.

The subjects in this study were a subsample drawn from the Ottawa Prenatal Prospective Study (OPPS), an ongoing longitudinal study of the development of children born to mothers who used "soft" drugs (i.e. marihuana, cigarettes, and alcohol) during pregnancy. Researchers in child development have identified several problems associated with the longitudinal design, and these will be addressed as they pertain to this study.

Sample loss occurs to some degree in longitudinal studies and this often undermines the representativeness of the sample. In the OPPS, there has been a retention rate of 98%, aside from families who moved

out of the Ottawa-Carleton region (32%), indicating that sample loss is not a significant problem for this study. A key factor in this high retention rate has been the advantage of maintaining staff continuity. The principal investigator and the two examiners have remained consistent over the past 8 years. This has undoubtedly increased the motivation for parents (and children) to return for follow-up.

Parents who are willing to make a long-term commitment to a longitudinal study tend to be cooperative and well-educated, and thus are not necessarily representative of the population (Bayley, 1974). An analysis of demographic characteristics of the OPPS sample nonetheless found that it was representative of the Ottawa region in terms of family income and maternal age at the time of delivery (Fried, et al. 1980; Fried, Innes and Barnes, 1984).

The advantages that occur with the utilization of a middle-class sample, are, first, that such a homogenous group eliminates many sources of extraneous variance (Little, Asker, Sampson, Renwick, 1986). There is evidence that prenatal drug use interacts with other risk factors, so if effects can be found in a middle class sample, one can presume the findings are a conservative estimate of the effects for the population as a whole. Children from middle-class families would also be considered at low risk for developmental problems and, unlike many other prenatal drug use subjects, these low-risk children do not demonstrate generally depressed cognitive functioning. Thus, subtle differences between smoking-exposed and not-exposed groups could be

expected to be more easily determined without the interference of a generalized deficit.

Longitudinal studies are sometimes criticized as being atheoretical and lacking in direction or focus. This view is warranted to some degree with this study, given its exploratory and descriptive nature. The field of behavioural teratology is a relatively recent field of endeavour, however, and explanatory theories have not yet been developed that would allow for the testing of specific experimental hypotheses. At this early stage of inquiry, the focus must be on the development of a crude map of the territory, before a more detailed map can be developed and tested.

Controlling for confounding variables is a problematic issue for longitudinal studies. Other prenatal determinants of behaviour, such as maternal age, education, other drug use, and family income, can confound the relationship between smoking and later behavioural outcomes. These variables were therefore included as potential confounds and were statistically controlled as covariates. Postnatal determinants of behaviour, such as home environment could be both a confounding variable and an indirect consequence of smoking. For instance, if smoking exposure produces an unresponsive infant, this could in turn affect the parent-child relationship. It is the current view that such postnatal characteristics should not be treated as covariates, but as intervening variables, representing the environmental condition through which smoking effects are manifested

(Sampson, Streissguth, Barr and Bookstein, 1986). A causal path analysis was therefore utilized, whereby family environment was examined as a mediating variable.

.2 Sample

Since 1978, the OPPS has collected data on approximately 700 pregnant women in the Ottawa area. Mothers-to-be were informed of the study by their doctors or by notices in the waiting rooms of prenatal clinics in the four largest Ottawa hospitals. The study was described as an investigation of the influence of prenatal lifestyle habits on the developing fetus. Upon volunteering to participate, each subject was interviewed once during each of the remaining trimesters in her pregnancy.

The OPPS sample was compared with Statistics Canada information for the Ottawa population as a whole. The average age of the subjects and the family incomes were found to be representative. The one dimension that distinguished the sample was the extent of formal education, with just over 50% of the participating mothers reporting post-secondary education, in comparison with 25% of women in the general population (Fried et al., 1980).

During the initial interview, information was collected regarding demographic variables, mother's and father's health history, previous

obstetrical history, and a 24-hour nutrition recall. Information on past and current drug use was collected during each interview, with questions regarding smoking, use of marihuana, and use of alcohol. The use of other drugs was also recorded. Women who reported use of "hard" drugs such as opium were retained in the sample, but excluded from statistical analysis.

The average number of cigarettes smoked per day was recorded and subjects were categorized as smokers if they smoked any cigarettes at any time during their pregnancy. Smoking was reported by 29% of the women in the year before pregnancy, and by 24% during pregnancy (Fried, Inness and Barnes, 1984). Marihuana use was calculated in terms of the average number of "joints" (i.e. marihuana cigarettes) per week during pregnancy. If hashish use was reported, an equivalent number of joints per week was calculated. Alcohol consumption was determined using a quantity-frequency formula, and is reported as average ounces of absolute alcohol per day.

During each interview, women were also asked the following question regarding passive smoking: "Are you regularly exposed to a smoke-filled environment?" . Thirty-five percent of the non-smokers responded "Yes" to this question in at least one interview during their pregnancy.

From the original 700 women, a cohort of approximately 210 were followed, including all marihuana users, all heavier social drinkers,

and all regular cigarette users. In addition, 50 non-drug users were selected as controls. Over time, attrition and budget restraints reduced the total follow-up sample to approximately 105 children. Women who were passive smokers tended not to be chosen for follow-up, as this was not a factor of interest at the time, and it was considered an undesirable confound in the study of the other drug effects.

For the purpose of this study, a subsample was drawn from the (larger) OPPS sample. A total of 54 children had already been tested on the OPPS neuropsychological battery as part of their regular follow-up testings, and all but 2 of these children were included in the sample for this study. (One family had moved out of the country; one child refused any further testing.) Additional children were recruited from the list of mothers who had volunteered to participate in the OPPS and for whom prenatal questionnaire data were available, but who had not been recently followed. A copy of the letter to mothers requesting their participation is provided in Appendix C. Of 60 letters mailed, 18 families had moved or could not be contacted by phone, and 3 refused to participate. A total of 39 "new" children were recruited for this study, making a total sample of 91 children in all. The follow-up status of the children was recorded as "regular" or "new" to indicate whether the child had been regularly followed in the OPPS study. The group sizes are reported in Table 1, along with other characteristics of the sample. The children were ages 6 to 9 years, and an attempt was made to match the groups for age and sex.

TABLE 1: DEMOGRAPHIC CHARACTERISTICS OF TOTAL SAMPLE AND GROUPS
A: Continuous Variables

	Total Sample (n=91)		Nonsmoking (n=23)		Passive (n=35)		Active (n=33)		Anova F/Sig.
	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	
Maternal Smoking (mg. nicotine/day)	5.07	10.49	0.0	--	0.0	--	13.98	13.46	31.17/.000
Maternal Marijuana Use (joints/week)	0.62	2.11	0.55	1.47	0.38	2.11	0.92	2.46	0.54/.58
Maternal Alcohol Use (oz. AA/day)	0.23	0.44	0.24	0.39	0.19	0.28	0.26	0.58	0.23/.79
Current Maternal Age (years)	38.85	4.34	38.74	3.65	37.37	3.57	34.97	4.86	6.18/.003
Maternal Education (years)	15.03	2.23	16.22	2.58	15.51	1.85	13.70	2.35	9.94/.0001
Father's Occupation Rating*	56.97	12.25	65.70	8.16	57.71	13.04	48.98	14.00	11.84/.000
Mother's Occupation Rating*	53.06	11.41	60.70	7.49	55.08	10.22	46.69	14.04	7.65/.001
Child's Position in Family	1.87	0.85	2.08	0.73	1.65	0.84	1.94	0.93	1.96/.148
Family Income Category**	6.06	1.17	6.57	0.95	6.32	1.01	5.42	1.44	7.87/.001
SES***	0.00	1.00	0.56	0.70	0.18	0.78	-0.59	1.10	12.37/.000
Passive Exposure of Child (years)	3.15	2.90	1.26	2.65	2.14	3.20	5.55	2.73	18.23/.000
Child's Chronological Age (months)	92.19	10.34	91.83	11.32	92.80	10.29	91.79	9.98	0.09/.907

* Occupational rating determined using Boyd's (1986) Index

** Family Income Categories: 1 = < \$10,000; 2 = \$10,000 to \$14,999; 3 = \$15,000 to \$19,999; 4 = \$20,000 to \$29,999;

5 = \$30,000 to \$39,999; 6 = \$40,000 to \$49,999; 7 = \$50,000 + .

*** See text for explanation of derived score.

TABLE 1 (con'd): Demographic Characteristics of Total Sample and Groups
B: Discrete Variables

	Total Sample (n=91)		Nonsmoking (n=23)		Passive (n=35)		Active (n=33)		Chi Square sig.
	N	%	N	%	N	%	N	%	
Sex: Boys	46	50.5	9	39.1	19	54.3	18	54.5	1.61/.448
Girls	45	49.5	14	60.9	16	45.7	15	45.5	
History of Illness, accidents: Yes	21	23.1	4	17.4	9	25.7	8	24.2	.58/.748
No	70	76.9	19	82.6	26	74.3	25	75.8	
School Program:									.85/.652
English/Regular	42	46.2	9	39.1	18	51.4	15	45.5	
Fr. Immersion	49	53.8	14	60.9	17	48.6	18	54.5	
Mother Working Outside home: Yes	68	74.7	14	60.9	29	82.9	25	75.8	3.58/.167
No	23	25.7	9	39.1	6	17.1	8	24.2	
Divorce/Separation:									4.30/.12
Yes	11	12.1	0	0.0	6	17.1	5	15.2	
No	80	87.9	23	100.0	29	82.9	28	84.8	
Problems in School:									2.29/.318
Yes	15	16.5	3	13.0	4	11.4	8	24.2	
No	76	83.5	20	87.0	31	88.6	25	75.8	
Examiner: #1	66	72.5	16	69.6	30	85.7	20	60.6	5.51/.064
#2	25	27.5	7	30.4	5	14.3	13	39.4	
Follow-up Status:									37.16/.0002
Regular	52	57.1	19	82.6	6	17.1	27	81.8	
New	39	42.9	4	17.4	29	82.9	6	18.2	
Child's Postnatal Passive Exposure to Smoke: Yes	52	57.1	7	30.4	13	37.1	32	97.0	33.79/.0000
No	39	42.9	16	69.6	22	62.9	1	3.0	
Maternal Education:									16.88/.0002
High School or less	38	41.8	5	21.7	10	28.6	23	69.7	
Post Secondary	53	58.2	18	78.3	25	71.4	10	30.3	

[Insert Tables 1A and 1B here.]

.3 Measures

Data on smoking exposure and other drug use was collected via maternal interviews during each trimester of pregnancy. Despite the potential shortcomings of self-report measures, no practical alternative was available for gathering this information. Several factors are relevant to a discussion of the reliability and validity of this method. During each interview, the questions pertaining to drug use for each trimester were repeated, thus allowing for assessing the test-retest reliability of the self report measure. The consistency of information was very high, with fewer than 8% inconsistently reporting their smoking habits. Whenever a difference was noted, the higher value was used. Reliability was also enhanced by the fact that the mother-to-be was required to recall this information over a relatively short period of time (i.e. three months). The validity of the information is enhanced by the fact that all women were volunteers who were assured that the information would be held in confidence. Finally, in order to establish a relaxed rapport, the interviews were conducted at the woman's home and the same female interviewer followed the woman through her pregnancy.

The behavioural outcome measures provided a comprehensive developmental assessment, including tests from a standard

neuropsychological battery, plus tests of language development, academic achievement, and behavioural ratings. Measures were chosen that were sensitive to potential smoking effects, using the results from the preliminary analysis as guidelines. Satisfactory reliability and validity ratings were important criteria for test selection, in addition to the practical issues of time and resources required for administration. Measures were also chosen to represent a range of functioning which would have clinical relevance in characterizing the child's developmental status. A list of the tests used as outcome measures can be found in Appendix B.

A preliminary statistical analysis was conducted using the children already tested on the OPPS battery. (A list of the tests used in the OPPS battery is provided in Appendix 2.) The scores of 26 children of smokers were compared with those of a group of 21 children of nonsmokers. Using a stepwise discriminant function analysis, it was determined that a pattern of scores for the smoking group could be significantly differentiated from that of the nonsmoking group. A correct classification rate of 87% was obtained, with a canonical correlation of .73 ($p = .0008$).

Using the stepwise procedure, a reduced set of variables was identified that maximized the separation between groups. The primary variables that distinguished between groups were the Sound Blending test and the Conduct Problems scale on the Conners Questionnaire. Children of smokers scored more poorly on both measures. Also

contributing to the discrimination between groups were scores on the Peabody Picture Vocabulary Test, the Impulsivity/Hyperactivity scale on the Conners, the McCarthy Memory scale, Developmental Drawings test, Tactual Performance test, and the General Cognitive Index of the McCarthy Scales of Children's Abilities. Results of this preliminary analysis are provided in Table 1.

[Insert Table 2 here.]

The results of this preliminary analysis were used to determine which tests from the OPPS would be retained and which areas of functioning required more detailed testing. Tests retained from the OPPS neuropsychological battery were: Sound Blending, Conners Parent Questionnaire, Developmental Drawings, Lateral Dominance, and the Peabody Picture Vocabulary Test. These tests were all found to be sensitive to active smoking effects in the preliminary analysis. The Wide Range Achievement Test was also retained, as it has been found to show smoking effects in previous research studies.

Although the McCarthy Scales emerged as important discriminators in the preliminary analysis, the mean scores of both groups were noted to be about 1.8 standard deviations above the population norm. This test had been used from ages 3 to 5 years in the yearly follow-up testings, however, which meant that these children had been tested using this instrument at least three times previously. The inflated scores were therefore believed to be largely due to test familiarity.

Table 2: Results of Preliminary Analysis
of OPPS Test Battery

a Test	Structure coefficient (Discrim. Function)	Smoking group mean standard score	Non-smoking mean standard score	T-test/ signif.
Sound Blending	.34	.01	.60	2.44*
Conners - Conduct Prob.	-.32	.51	.09	-1.78+
Peabody Pict. Vocabulary	.26	115.1	121.5	1.89+
Conners - Impulsive/Hyp.	-.21	.20	-.08	-.93
McCarthy - Memory	.21	59.1	61.3	1.06
Developmental Drawings	.21	-1.03	-.29	1.81+
TPT - Memory	-.20	.45	.23	-1.00
McCarthy - Gen.Cog.Index	.20	124.8	130.8	1.43
WRAT - Spelling	.18	88.1	92.6	1.28
TPT - Location	.17	.32	.66	1.19
Pegboard - Speed	-.16	.94	.79	-1.11
Conners - Psychosomatic	-.13	.62	.27	-.95
Progress. Holes - errors	-.08	.60	.40	-.56
Boston Speech	.05	1.69	1.88	.34
Mazes - Speed	.04	-.05	.19	.30
Aphasia Screening	-.03	.43	.39	-.21

a all tests contributing to the
discriminant function and those
with high structure coefficients

* $p \leq .05$
+ $p \leq .10$

In addition, ceiling effects were noted for children above 6 years. It was therefore decided that the Wechsler Intelligence Scale for Children - Revised (WISC-R) would be substituted as the test for overall intellectual ability.

The WISC-R (Wechsler, 1974) is one of the most widely used tests of general intellectual abilities, and its reliability and validity are well-established. The Full Scale score is still one of the best predictors of school achievement (Kaufman, 1979) and the WISC-R profile is used by school psychologists in the identification of learning disabilities. Rather than using the Verbal and Performance subscale scores, the three-factor scores developed by Kaufman (1979) were used, i.e. Verbal Conceptualization, Perceptual Organization and Freedom from Distractability. This latter scale has been found to be sensitive to attentional and information processing problems in children. The subtest loading patterns for the three factor scores are as follows:

Verbal Conceptualization

Information

Similarities

Vocabulary

Comprehension

Perceptual Organization

Picture Completion

Picture Arrangement

Block Design

Object Assembly

Distractability

Arithmetic

Digit Span

Coding

The Test of Language Development, Primary (revised) edition (TOLD), by Newcomer and Hammill (1982) assesses a child's comprehension and expression of language. The test is considered to be a well-documented and well-researched instrument, with adequate evidence of reliability and validity (Allen, 1985). Seven subtests are provided, but only four were used to evaluate speech articulation and oral syntax.

Qualitative analysis of the performance of the smoking group vs. the nonsmoking group on the Developmental Drawings test and other tests requiring fine motor expression (i.e. Aphasia drawings, McCarthy drawings, WRAT Spelling) revealed many examples of spatial orientation and directionality errors, such as reversals and inversions. Two tests were added to investigate this phenomenon in more detail. The Jordan Left-Right Reversal Test (Jordan, 1980) is concerned with the the directionality perception of letters, words and numbers. The psychometric characteristics of this instrument are considered to be adequate for research purposes (Poplin, 1985). The far-point copying subtest of the Slingerland Screening Tests (Slingerland, 1970) was chosen to allow for a qualitative analysis of how any underlying perceptual-motor weaknesses would be evidenced on a task representative of real-life academic demands. Although there are no norms available, reliability and validity of the Slingerland tests has been considered acceptable (Fujiki, 1985).

The Draw-a-Man test was also used as a measure of visual-motor ability. The Goodenough-Harris scoring system was used (Harris, 1963). This test is a standard clinical tool used with school-aged children. Preliminary analysis of the McCarthy Draw-a-Child subtest suggested that smokers' children drawings contained less detail and noticeable shifting of body parts. It was felt that this deserved further investigation using an instrument with detailed scoring and normative data.

The Reading Understanding subtest from the Kaufman Assessment Battery for Children (Kaufman and Kaufman, 1983) was included to provide information on reading comprehension, as the WRAT Reading test only measures reading recognition. The Test D'Orthographe Pour Eleves d'Immersion (Schnarr, unpublished) was also given to those students enrolled in a French Immersion program in school. This test addressed the question as to whether any differences in spelling are also evident in French, which is the language of instruction for these students. This test is an experimental edition, recently standardized on a sample of students in the Ottawa-Carleton region.

The instrument chosen to assess family environment was the Family Environment Scale (FES), by Moos and Moos (1986). This 90-item scale was answered by the mother in a true-false manner. The FES purports to assess social environmental characteristics of families along ten dimensions. (See Appendix B for a list of the ten dimensions plus

sample items.) This test has good psychometric properties and is considered one of the best measures available for assessing families (Busch-Rossnagel, 1985). The Cohesion, Expressiveness and Intellectual-Cultural Orientation subscales have been shown to be significantly correlated with cognitive ability in young children (Gottfried and Gottfried, 1984; Wilson and Matheny, 1983). A study of FES ratings by mothers of children with behaviour problems found these families were rated as less cohesive, less expressive and more in conflict when compared with controls (McGee, Silva and Williams, 1984).

An examiner rating scale was also used to rate the behaviour of the children during the testing session in terms of attention to task, amount of motor activity, performance rate, manual dexterity and degree of cooperation (adapted from Atwell, Orpet and Meyers, 1967). A short questionnaire was completed by the child's mother in order to gain information about the child's school history, family demographic information, and smoking habits of the parents. These two forms are provided in Appendices D and E.

.4 Procedures

The 37 "new" children were tested with the complete assessment battery listed in Appendix B. The 52 "regular" follow-up children were tested only on those tests added to the OPPS battery. All children

were tested by examiners who were blind as to the mother's prenatal drug use. The examiners were the author (Examiner 1) and the main examiner from the OPPS (Examiner 2).

Subjects were tested in their own homes or in a laboratory at Carleton University, depending on convenience for the family and availability of transportation. Time required was 2 1/2 to 3 hours for the short battery, and 3 1/2 to 4 hours for the complete battery. Breaks for drinks, bathroom and physical activity were provided according to the child's needs. The order of the tests was kept constant for all subjects. Standardized procedures for administration and scoring were followed (Knights and Norwood, 1980).

.5 Statistical Procedures

Data were analyzed using group comparisons. The three groups represented children of Nonsmoking, Passive Smoking and Active Smoking mothers. Multivariate analysis was chosen in order to minimize Type I error and to take into account the intercorrelation among the predictor variables. Prior to analysis, data were evaluated using univariate descriptive statistics to check for accuracy of input, outliers, and normality of distribution. Data were also evaluated in terms of assumptions for multivariate analysis, namely those of missing data and homogeneity of variance-covariance matrices.

One subject was dropped from multivariate analysis due to missing data on 14 of the tests. The child refused to speak to the examiner during the testing session, but was otherwise reasonably attentive and cooperative. (Note: This child has a history of elective mutism, and has previously refused to speak to examiners during regular testing follow-up sessions.) This child was in the Active Smoking group; this omission reduced the Active group size to 32, and the total N to 90. On 10 of the remaining cases, data were missing for a total of 29 data points across 23 of the "dependent" and "independent" variables. (N's for each variable are reported in Appendix III.) The amount of missing data was minute when compared with the total data collected, representing 0.4% of the total data set. In order that information on these 10 subjects not be lost via listwise deletion, missing values were recoded as mean scores. This conservative treatment of missing data creates no distortion in the central tendency of variables (Tabachnick and Fidell, 1983).

Equality of group covariance matrices was tested using Box M's test. The nonsignificant result ($F(132,15792) = 1.03, p = .379$) indicates the covariance matrices are approximately equal, thus meeting one of the important assumptions for multivariate analysis.

SPSSx Discriminant Function analysis was used to determine whether the three groups could be accurately classified using performance on this test battery. Using the stepwise method of entry, variables entered individually, according to their discriminating power. At each

step, previously entered variables were examined for removal. This procedure resulted in a reduced set of variables that, in combination, could optimally predict group membership. Given the low N to variable ratio, this reduction in the number of variables proved to be valuable with this data set.

The stability of the discriminant functions was assessed using a cross-validation procedure. The data were analyzed a second time, randomly selecting two thirds of the sample to obtain a discriminant function to use for the remaining third. The resulting classification tables were compared for shrinkage.

A total of 19 potentially confounding variables were examined for their relationship with smoking using the Chi Square test for dichotomous variables and Analysis of Variance (ANOVA) for continuous variables. Variables were selected on the basis of a relationship with smoking status at an alpha level .10 (Jacobson et. al., 1984). Variables selected as potentially confounding were entered as covariates in a multivariate analysis of covariance (MANCOVA) to determine whether measures that were associated with group differences remained significant after statistically controlling for confounds.

The SPSSx MANOVA program provides a variety of test statistics for evaluating multivariate differences. Pillai's criteria was chosen as it is recommended as the most robust criterion with a small sample, unequal N's, and mild violations of the assumptions (Tabachnick and

Fidell, 1983; Norusis, 1985).

A path analysis was used to determine whether the Moos Family Environment Scale (FES) would act as a mediating variable in the relationship between smoking and behavioural outcome. Group membership representing smoking status was entered via two dummy-coded vectors into a regression analysis, with two other independent variables: maternal education (MATED), and socioeconomic status (SES). Smoking, SES and MATED were considered to be exogenous variables, and no attempt was made to explain their intercorrelation. Dependent or outcome measures were those variables that emerged as significantly related to smoking, from the Discriminant and MANCOVA analyses. The causal path model is depicted in Figure 1.

[Insert Figure 1 here.]

The path analysis procedure was used selectively with a few representative dependent variables in order to illustrate its effectiveness as a means of studying the direct and indirect effects of prenatal smoking exposure. The advantage of this approach over traditional multiple regression is that it allows the researcher to investigate the total effect of a variable, rather than just the unique or direct effect. Given that the unique effect of a prenatal variable is expected to be very small by early childhood, it is important to explore the developmental pattern of how the longterm effects occur, as well as answering the question of the magnitude of effects. In addition to the statistical benefits, this analytical approach probably

Figure I: Causal path diagram

better represents the real world where developmental outcome is influenced by an interaction between early and later environmental factors. Given the state of knowledge in this area of research, however, the causal model suggested is very rudimentary, and there are a number of important variables omitted. For this reason, the model was not overidentified for the purpose of testing its efficiency in reproducing the correlation matrix.

III RESULTS

Descriptive statistics for independent and dependent measures are reported for the total sample and for the three groups. The multivariate analysis was conducted first with all three groups in order to determine the dimensions along which the groups were maximally different. Data from the Passive Smoking and Active Smoking groups were compared separately with the Nonsmoking group to determine if the important discriminating variables were similar or different. The Active and Passive smoking groups were then compared directly. A few variables had to be excluded from the multivariate analysis, and were analyzed individually. Finally, a path analysis procedure was used with selected dependent variables in order to explore the potential mediating role of the home environment. The Results section is therefore organized as follows:

- .1 Description of the sample
- .2 Three-group analysis
- .3 Active smoking and Nonsmoking groups
- .4 Passive smoking and Nonsmoking groups
- .5 Active smoking and Passive smoking groups
- .6 Other variables
- .7 Path Analysis
- .8 Summary

.1 Description of Sample

This sample of 91 subjects maintains the "middle class" character of the larger OPPS sample. On the questionnaire given to the mothers during the current testing session the majority reported post-secondary education (58%) and the mean family income was between \$40,000 and \$49,000. Occupations for father and mother received overall mean ratings of 56.97 and 53.06, respectively, using the Boyd scale (Boyd, 1986). These mean values correspond to such occupations as office manager, architectural technician, elementary school teacher, financial manager.

Demographic characteristics are reported for the entire sample and are broken down by group in Tables 1A and 1B (on pages 52 and 53). A significant downward trend was noted for the Passive and Active Smoking groups for family income, parental occupation(s), maternal age and maternal education. No differences were found between the groups in the incidence of divorce or in the numbers of mothers working outside the home. Prenatal use of marijuana and alcohol also did not vary significantly across groups.

The measure of smoking exposure used in the OPPS is mg of nicotine consumed per day. This is calculated by multiplying the number of cigarettes smoked by the nicotine content of the brand specified. The mean amount of nicotine consumed by mothers who reported active smoking during pregnancy was 13.98 mg, when averaged over the 3 trimesters. This would have corresponded to slightly less than a pack of cigarettes

70

per day. (One pack of regular cigarettes on average had 16 mg of nicotine.) The range reported was from .2 to 60 mg, or from less than 1 cigarette to more than 3 packs per day.

In the Passive Smoking group, only 54% reported that their husbands smoked during the prenatal interview. This suggests that for the remaining 46%, the maternal passive exposure to smoke reported by this group occurred outside the home, likely in a work setting.

The results of the questionnaire given to the mothers during the current testing session show no significant differences across groups for the child's chronological age, sex, school program, position in the family, or history of illness. Although not statistically significant, it was notable that learning problems in school were recorded twice as frequently for the Active Smoking group (24%) as for the Nonsmoking and Passive groups (13% and 11%).

Mothers were also asked whether the children had been regularly exposed to second hand smoke since birth. Data in Tables 1A and 1B indicate that children in all three groups were exposed to smoke in the postnatal environment. The percentage of children so exposed was 30% and 37% for the Nonsmoking and Passive Smoking groups, whereas 97% of the children in the Active Smoking group were exposed to smoke postnatally. Information was also gathered regarding the duration in years since birth for such exposure, and again the trend is noted for significantly greater exposure for the children in the Passive and Active Smoking groups.

The examiner was noted during each testing session, as two examiners were used to conduct the testing of the children. Groups differed significantly in the ratio of examiners, with more children in the Passive group tested by Examiner 1. Also recorded was whether the child was in the regular follow-up sample in the OPPS or whether the child had been recalled from the original OPPS cohort just for this testing session. A smaller proportion of children in the Passive group had been seen for regular follow-up testing sessions. These two variables (Examiner and Follow-up Status) plus the other significant demographic variables were included as covariates in later MANCOVA analyses.

Figures 2A to 2F illustrate the profile of mean test scores on all variables collected in the test battery. The tests have been grouped into 6 logical clusters: Speech and Language, Intellectual, Motor and Visual/Spatial, Academic Achievement, Behavioural, and Growth.

[Insert Figures 2A to 2E here.]

It was hypothesized that the Passive Smoking group would show a similar profile of abilities as the Active Smoking group, but that the magnitude of differences between Passive and Nonsmoking groups would be smaller than the Active and Nonsmoking groups. The group profiles were generally in the expected configuration in the speech and language, intellectual, and behavioural domains. On tests in these areas, the Active Smoking group obtained the lowest or poorest scores, with the

Figure 2A: Group means on Speech and Language tests

Figure 2B: Group means on Intellectual and Motor tests

Figure 2C: Group means on Visual/Spatial and Academic Achievement tests

Figure 2D: Group Means on Behaviour Scales

Figure 2E: Group scores on growth measures

standard error

Passive group in between the Active and Nonsmoking groups. This pattern held true for the following variables within these three clusters: Sound Blending, Syntax, Articulation, WISC-R Verbal Conceptualization and Freedom from Distractability, and all the Conners' behaviour problems scales with the exception of Anxiety. (Note: The Conners' scales are usually scored in the reverse direction, so that a higher score indicates more behaviour problems. The scores were reversed in sign in Figure 2F to aid comparison across tests. The original direction was used, however, in the statistical analysis.)

On tests of academic achievement, the expected profile held true for Reading Understanding and Arithmetic, but the Passive group surpassed the Nonsmoking group slightly on the Reading (decoding) and Spelling tests. On all academic achievement tests the Active Smoking group scored the lowest.

A very different pattern emerged on tests of fine motor speed. On the Pegboard test the children of Active smokers outperformed the others. A return to the predicted pattern was noted on the Developmental Drawings and Draw a Man tests, but on the Jordan Reversals test, the Passive group scored the highest, followed by the Active, then Nonsmoking groups.

Table 3 indicates that there was no significant difference in the frequency of distribution of right vs. left dominance across groups. It is interesting to note that the Nonsmoking group showed the highest proportion of left-handers, followed by the Passive group, and then the

Active group. Within the sample as a whole, 14% were left-handed, but almost 22% of the Nonsmoking group were left-handed, representing double the expected frequency based on a population rate of approximately 10% for left-handedness (Liebert, Poulos and Strauss, 1974).

[Insert Table 3 here.]

The Nonsmoking group were taller and heavier than both smoking groups, but the expected profile occurred only for weight, as the Active group were lighter than the Passive group but slightly taller. Unexpectedly, the Active group had the largest head circumference measurements, followed by the Passive group and then the Nonsmoking groups. This reversal of the predicted profile may be due, in part, to the fact that the only available normative data for head circumference were the mean and ± 2 standard deviations. More gradations in classification may have resulted in a slightly different pattern of results.

Refer to Appendix F for the correlation matrix of the dependent variables.

.2 Three-Group Analysis

A/ Preliminary Analysis

A total of 52 test scores and behaviour ratings were collected on

Table 3: Hand Dominance Results by Group

	Group						Total	
	Nonsmoking		Passive		Active			
	N	%	N	%	N	%	N	%
Right	18	78.3	30	85.7	30	90.9	78	85.7
Left	5	21.7	5	14.3	3	9.1	13	14.3
Total	23	25.3	35	38.5	33	36.3	91	100.0

Chi-square (2) = 1.77, $p = .413$

each subject as potential outcome measures. Scores on the Spelling test for French Immersion students ("Test d'Orthographe Pour Eleves d'Immersion") were omitted entirely from further analysis, as there was an excess of missing data, and the data available did not appear to be valid. Scores were available for only 28 children, i.e. those who had completed at least grade one in a French Immersion program in school. When these scores were compared with those on the WRAT and the Reading Understanding tests, they appeared unexplainably low. In addition, it was found children tested late in the summer scored considerably below grade level, whereas scores in the winter were higher. This was probably because norms were available only for the end of the school year. With these concerns about the validity and reliability of scores, and given the experimental nature of this instrument, it was decided that it would be omitted from further analysis. (Note: Scores are included in Appendix H.)

Five additional variables were omitted from the multivariate analysis, but were analyzed separately using univariate methods. The Kauffman Test of Reading Understanding was attempted with all children, but standard scores were calculated for only 76 (out of 91) children, as norms are only provided for 7.0 years and up. The Slingerland copying test has no norms available, and was scored using somewhat subjective criteria developed by this researcher. Scores were not felt to be sufficiently reliable to qualify for entry into multivariate analysis. The three growth measurements (Height, Weight and Head Circumference) were also omitted as they did not constitute the same

kind of behavioural data. These five variables were analyzed separately using univariate methods.

In order to improve the N to variable ratio, summary or total scores were also used whenever available, leaving 23 variables remaining for multivariate analysis. These variables are listed below:

Speech and Language

- Sound Blending
- Peabody Vocab.(PPVT)
- Syntax Quotient (TOLD)
- Articulation

Motor

- Pegboard (dominant and nondominant)

Visual/Spatial

- Developmental Drawings

Lateral Dominance

- Handedness

- Draw a Man

- Reversals

Academic Achievement

- Reading
- Spelling
- Arithmetic

Intelligence (WISC-R factor scores)

- Verbal Conceptualization
- Perceptual Organization
- Freedom from Distractability

Behaviour

- Examiner Ratings
- Conners scales (Conduct, Learning, Psychosomatic, Impulsive/Hyperactive, Anxiety, Hyperactivity)

Mean scores on these 23 variables are provided in Table 4.

[Insert Table 4 here.]

Table 4: Means and Standard Deviations for total sample and three groups on
Dependent Variables selected for Multivariate Analyses

Variable Name	Std. Score Mean	Total Sample S.D.	Valid N	Non-smoking Group Mean (n=23)	S.D.	Passive Smoking Mean (n=35)	S.D.	Active Smoking Mean (n=33)	S.D.	Univariate F/Signif.
Pegboard - Dominant Hand	1.06	0.64	88	1.11	0.62	0.73	0.64	1.35	0.49	9.61**
Pegboard - Non-dominant Hand	.57	0.47	88	0.48	0.39	0.50	0.49	0.69	0.50	1.78
WRAT - Reading (decoding)	94.26	15.11	91	99.48	18.70	100.89	14.57	94.63	12.41	1.57
WRAT - Spelling	93.28	13.52	91	93.52	12.67	94.57	13.83	91.73	14.01	0.38
WRAT - Arithmetic	99.56	10.87	91	100.09	13.88	99.74	9.74	99.00	9.92	0.07
Sound Blending	0.21	0.91	91	0.54	1.00	0.18	0.97	0.02	0.74	2.29
Peabody Picture Vocabulary	115.63	11.07	91	120.83	11.98	111.14	10.96	116.76	8.69	6.23**
Developmental Drawings	-0.28	1.22	91	-0.07	1.31	-0.26	1.03	-0.44	1.36	0.63
Draw a Man	103.79	13.52	90	106.82	15.81	107.69	11.41	97.63	12.00	6.06**
T.O.L.D. Syntax Quotient	112.09	11.79	90	117.61	12.16	112.11	10.98	108.09	11.09	4.72**
Articulation	10.89	2.27	90	11.61	1.67	11.05	1.97	10.19	2.75	2.91+
Reversals	42.52	21.62	91	38.65	25.22	46.71	18.99	40.76	21.47	1.13
WISC-R Verbal Concept.	13.38	2.20	90	14.26	2.45	13.24	2.09	12.91	2.00	2.74+
WISC-R Perceptual Org.	13.02	1.95	91	13.77	2.01	12.62	1.61	12.92	2.13	2.60+
WISC-R Freedom from Dist.	11.49	2.11	90	12.42	2.10	11.18	1.94	11.14	2.15	3.20*
Examiner Behav. Rating (total)	20.38	3.35	90	20.74	2.78	19.91	3.33	20.63	3.76	0.55
Conners - Conduct Problems	0.13	0.91	91	-0.04	0.74	-0.01	1.02	0.41	0.84	2.42+
Conners - Learning Problems	-0.07	1.08	91	-0.36	1.25	0.00	1.13	0.05	0.87	1.14
Conners - Psychosomatic	0.37	1.22	91	0.20	0.88	0.31	1.28	0.56	1.37	0.67
Conners - Impulsive/Hyper.	-0.09	1.07	91	-0.28	1.07	-0.24	1.15	0.22	0.94	2.09
Conners - Anxiety	0.18	0.94	91	-0.06	0.68	0.32	1.21	0.19	0.75	1.14
Conners - Hyperactivity	-0.15	1.01	91	-0.44	1.11	-0.15	1.11	0.07	0.78	1.75

** $P < .01$; * $p < .05$; + $p < .10$

B/ Three-group Discriminant Function Analysis

The results of the stepwise discriminant function analysis indicate that prenatal smoking status can be predicted using performance on this test battery. Two discriminant functions were calculated, with a combined Chi square (22) = 63.56, p .0001. After removal of the first function, there was still significant discriminating power (Chi square (10) = 24.99, p = .005). As shown in Table 5 the canonical correlation of the first function was .61, representing the correlation between the discriminant score on the first function (or canonical variate) and the groups. Thus the first discriminant function and the grouping variable shared 37% variance. Similarly, the squared canonical correlation for the second function indicates 26% shared variance. The group centroids indicate that the first function maximally separates the Active Smoking group from the Nonsmoking and Passive groups. The second function, on the other hand, separates the Nonsmoking group from the Passives and Actives.

[Insert Table 5 here.]

With the stepwise method of entry, variables enter the discriminant function based on the significance of their independent contribution in explaining group variance. If predictor variables are correlated, some may not have high standardized coefficients and some may not enter the function at all. In this study, because of correlations among the test variables, standardized discriminant coefficients were not considered as a valid representation of the relative importance of variables. Instead, structure coefficients were

TABLE 5: Discriminant Analysis Results for Total Sample (Three Groups)
(N = 90)

	<u>1st function</u>	<u>2nd function</u>	
Chi-squared (22)	63.56	24.99	
Significance	.0000	.005	
Canonical Correlation	.61	.51	
% Variance	37%	26%	

Variable	Structure Coefficient		Univariate F/signif.
	1st function	2nd function	
Pegboard (dominant)	.61		11.03**
Draw a Man	-.47		5.69**
Pegboard (nondominant)	.37		3.70*
Conners - Hyperactivity	.32		1.71
Conners - Conduct Problems	.30		2.52+
Conners - Impulsivity/Hyp.	.30		2.05
Articulation	-.28		2.91+
WRAT - Reading	-.26		1.73
Peabody Vocabulary		.57	6.12**
WISC-R Distractability		.44	3.20*
Syntax	-.30	.41	4.72*
WISC-R Perceptual Org.		.40	2.57+
WISC-R Verbal Concept		.37	2.74+
WRAT - Arithmetic		.02	0.01

	Group Centroids	
	1st function	2nd function
Nonsmoking group	-.31	.97
Passive group	-.71	-.51
Active group	1.00	-.14

** $p \leq .01$

* $p \leq .05$

+ $p \leq .10$

used as an index of their relative importance. Structure coefficients represent the correlations between the discriminant function and the predictor variables (whether in the function or not). In this three-group analysis, the dimensions were rotated using the varimax option in order to facilitate interpretation. Rotation serves to maximize the variance of loadings across variables within functions, so that variables load more clearly on only one function.

The loading matrix of structure coefficients for the first function indicates that the primary variables that distinguish the Active Smoking group from the other two groups are motoric, visual/spatial, and behavioural. Variables with meaningful structure coefficients (i.e. .30, as suggested in Pedhazur, 1982) were Pegboard (dominant and nondominant), Draw a Man, and three of the Conners' behaviour scales: Hyperactivity, Conduct Problems, and Impulsivity. Also contributing, although not significantly, were Articulation and Reading. With the exception of the Pegboard tests, the Active group scored more poorly than the other groups.

The second function maximally separates the Nonsmoking group from the Passive and Active groups. The primary variables responsible are measures of language, intelligence and attention. Variables with meaningful structure coefficients were the Peabody Picture Vocabulary Test, Syntax, and the three WISC-R factor scores: Verbal Conceptualization, Perceptual Organization, and Freedom from Distractability. On all of these variables, the Active smoking group scored more poorly than the Nonsmoking group. On the Syntax, Verbal

Conceptualization and Distractability scales the Passive group scored in between Active and Nonsmoking, whereas on the Peabody Vocabulary Test and the Perceptual Organization scale the Passive group scored below the other groups.

Table 5 presents the 11 variables that entered into the discriminant functions plus the 3 additional variables with high structure coefficients. Included in this table are the univariate F ratios indicating which variables would show significant group differences, if tested individually. (Note that the level of significance has not been adjusted for the number of comparisons.)

Using both functions, 69% of the subjects were correctly classified into their three groups. Classification results indicate that the Active Smoking group was classified the best, with 75% correct, followed by the Passive group with 69% correct. The greatest source of error was in misclassifying Nonsmoking subjects as belonging to the Passive group, with 26% incorrect. (See Appendix K for classification table.)

The cross-validation procedure yielded some reduction in the classification rates. The shrinkage noted was from 69 to 52% correct. This latter figure is nevertheless still above that which would be obtained by chance alone (i.e. 33% correct for three groups).

In summary, the discriminant analysis using three groups provided two significant functions. The variables that maximally separated the

 

Active group were motoric, visual/spatial, and behavioural. The variables primarily responsible for distinguishing the Nonsmoking group from the two smoking groups were measures of intelligence, language and attention.

C/ Three-group MANCOVA

Nineteen variables were considered as potential confounds. Potential experimental confounds considered were age and sex of the child, examiner effects (two examiners conducted the testing), and follow-up status (i.e. whether the child was in the regular follow-up sample in the OPPS, or was recalled especially for this testing session). Demographic and background variables considered included: maternal age, other drug use (marijuana and alcohol), maternal education, parental occupation, family income, divorce, mother working outside the home, school program, child's position in the family, and history of illness.

A new variable - "SES" - was created to represent the sum of information regarding parental occupation and family income. SES, standing for socioeconomic status, was calculated by taking the sum of the higher score from Father's Occupation or Mother's Occupation, plus the Family Income level. All scores were standardized (using z transformation), so that occupation and income contributed equally to the new variable. Creation of this variable relieved the problem of missing data on the occupation measures due to single parent households or mothers not working outside the home. It also acted to reduce the

number of variables, and given the high correlations between income and occupation (see correlation matrix, Appendix F), to reduce the likelihood of multicollinearity between demographic measures. In addition, missing data on Mother's and Fathers's Occupation were (dummy) coded to produce two new variables representing information on divorce and mother at home/working.

The following five variables were associated with smoking status (using the $p .1$ criterion) and were selected as covariates: Examiner, Follow-up Status, Maternal Education, Maternal Age, and SES. (See Tables 1A and 1B.) These variables represented both experimental and demographic controls. Postnatal passive exposure to smoke was eligible for treatment as a covariate ($F = 18.23$, $p .001$), but due to insufficient variance in the Active smoking group, it was not included.

A multivariate analysis of covariance (MANCOVA) was performed to ascertain the predictive power of the test scores after adjustment for the five covariates. The 14 predictor variables from Table 6 that entered or were highly correlated with the discriminant function were entered as dependent variables. Box M's multivariate test of the homogeneity of variance-covariance matrices was re-run for this reduced set of variables, with a resulting $F(380, 15373) = 1.13$, $p = .038$. This significant result implies that the data do not meet an important assumption for MANCOVA. However, this test is notoriously sensitive to departures from normality (Norusis, 1985; Tabachnick and Fidell, 1983). The level of significance, therefore, indicates only a mild violation.

As mentioned earlier, Pillai's F was chosen as the criterion for statistical inference, as it is considered more robust in the presence of mild violations to the assumptions.

The results of the MANCOVA are reported in Table 6. The homogeneity of regression assumption was met, with no significant group by covariate interaction ($F(140,501) = 1.16, p = .126$). The combined dependent variables were not significantly related to the combined covariates ($F(70,365) = 1.16, p = .189$). The first canonical variate of the covariates explained only 8.1% of the variance in the combined dependent variables.

[Insert Table 6 here.]

The multivariate test for group differences, after statistical control for covariates showed only borderline significance ($F(28,140) = 1.45, p = .083$). The covariates, although not showing a statistically significant relationship with the dependent variables, nonetheless provided sufficient adjustment to the mean scores such that group differences were reduced. After adjustment, the groups account for 25% variance on the first canonical variate, and 20% variance on the second function. (By comparison, these values were 37% and 26% on the discriminant analysis.)

A stepdown analysis of the relative importance of dependent variables was not possible in this instance, as there were no theoretical grounds for a priority ordering of entry. Variables were

Table 6: MANCOVA Results for total Sample (N = 90)

Variable	Structure Coeff.		Univariate F(2,87)signif.
	1st canon. variate	2nd canon. variate	
Articulation	0.55	0.06	4.33*
Pegboard			
(non dominant)	-0.40	0.19	2.59+
Syntax	0.38	0.38	3.41*
Conduct Problems	-0.35	0.08	1.82
Draw a Man	0.32	-0.22	1.88
Pegboard (dominant)	-0.32	0.31	2.40+
Hyperactivity	-0.25	-0.23	1.44
Impulsivity	-0.24	-0.14	0.99
Peabody Vocabulary	0.21	0.42	2.35+
WISC-R Verbal Conc.	0.18	0.44	2.42+
WRAT Reading	0.11	0.26	0.86
WISC-R Distract.	0.09	0.47	2.29
WRAT Arithmetic	-0.03	0.00	0.01
WISC-R Perc. Org.	-0.01	0.38	1.44

* $p \leq .05$ + $p \leq .10$

assessed instead using the loading matrix of correlations between variables and the canonical variates. The loading matrices for both canonical variates are included in Table 6 in order to allow comparisons between the functions in the Discriminant analysis and the MANCOVA, even though the second function does not meet the .05 level of statistical significance ($p = .23$).

Examination of the structure coefficients for the first function indicates that after adjustment for potential confounds, Articulation was the best discriminator between groups, followed by Pegboard, Syntax, Conduct Problems, and Draw a Man. The second function was a primarily a combination of the WISC-R factor scores (Verbal Conceptualization, Perceptual Organization and Distractability), Peabody Vocabulary, and again, Syntax. Variables evidenced a similar loading pattern in both the MANCOVA and the Discriminant analyses. The two dimensions that best discriminated between the groups thus showed stability even after adjustment for differences on covariates.

Univariate F values taken from analyses of covariance (ANCOVA) indicate significant differences across groups would remain for Articulation and Syntax, if they were to be analyzed individually.

In summary, the results of the three-group MANCOVA indicate that the discriminating power of scores on this test battery is somewhat reduced when statistical adjustment is made for differences on potential confounds. A comparison of these results with those of the

Discriminant analysis reported earlier (in Table 5) reveals that the variables on the first discriminant function generally maintained significant group differences after adjustment for covariates, whereas variables on the second function, particularly the intellectual measures, did not remain significant after covariates were entered. It would therefore appear that the Active Smoking group can still be differentiated from the other groups after covariate adjustment, whereas the variables differentiating the Nonsmoking group from the two smoking groups are more related to differences on confounding factors.

.3 Comparison of Active Smoking and Nonsmoking groups

A/ Active vs. Nonsmoking Discriminant Analysis

This analysis was conducted to explore further the nature of the dimensions that distinguished the Active smoking group from the Nonsmoking group. With the Passive group eliminated from consideration the available N was 55. Using the same 23 dependent variables, a highly significant discriminant function was calculated, with a $X^2(8) = 30.06$, $p = .0002$. The canonical correlation of .68 indicates 46% shared variance between the groups and the variables in the discriminant function. Table 7 presents the eight variables that entered stepwise into the function, plus variables with high structure coefficients. Variables with meaningful structure coefficients (.30) were Syntax, Verbal Conceptualization, Pegboard (nondominant), Draw a Man, Distractability, Articulation, Sound Blending, and Conduct Problems. The univariate F's indicate that the differences between

these two groups would be significant for all these variables, if each were to be tested individually.

[Insert Table 7 here.]

A comparison with the important variables from the three-group analysis reported earlier indicates that many of the variables from both the first and second functions that distinguished the three groups are included on this function (i.e. Syntax, Articulation, Verbal Conceptualization, Distractability, Pegboard, Draw a Man, and Conduct Problems). Not included on this dimension were scores on Hyperactivity, Impulsivity, Peabody Picture Vocabulary, and Perceptual Organization.

The correct classification rate using this function was 87%, with both groups showing approximately the same rate. Using the cross validation procedure, no shrinkage was found. In fact, the classification rate was almost unchanged, thus lending support for the stability of this function. (See Appendix L.)

In summary, the important factors in this dimension were poorer scores for the Active group on measures of speech and language ability, verbal intelligence, attention, behaviour problems, and better performance on a test of fine motor dexterity.

TABLE 7: Discriminant Analysis Results for Active
Smoking and Nonsmoking Groups (n = 55)

• Chi - Squared (8)	30.06
Significance	.0002
Cannonical Correlation	.68
% Variance	46.2

Variable	Structure Coeff.	Univariate F/signif.
TOLD - Syntax	.45	9.09**
WISC-R Verbal Concept.	.42	5.04*
Pegboard (nondominant)	-.37	6.29*
Draw-A-Man	.36	5.68*
WISC-R Distractability	.33	4.68*
Articulation	.33	4.83*
Sound Blending	.31	4.37*
Conners - Conduct	-.31	4.36*
WRAT - Arithmetic	.02	0.02

The same five demographic and experimental variables were used as covariates in a MANCOVA with the nine dependent variables from the discriminant function. Again, the Passive group was eliminated from the analysis, so that group comparisons were between the Active and Nonsmoking groups only.

Initially, there was a significant Group by Covariate interaction ($F(45,195) = 1.51, p = .03$), indicating that the relationship between the covariates and the dependent variables in one group was not the same as the relationship in the other group. The homogeneity of regression condition was therefore not met, because the amount of adjustment provided by the covariates would differ for different groups.

As is suggested by Tabachnick and Fidell (1983), an attempt was made to determine which covariate was responsible for this interaction effect. This was achieved by repeating the analysis five times, each time eliminating one covariate. Using this process of elimination, it was determined that SES was the variable responsible. A "blocking strategy" was then employed whereby scores on SES were divided into two levels (i.e. low and high), and SES was re-entered into the MANCOVA as a second factor, along with smoking group. This allowed a straightforward interpretation of the main effect of the smoking factor, plus allowed for the direct investigation of the smoking by SES interaction.

On this revised MANCOVA, the four remaining covariates showed no significant relationship with the combined dependent variables ($F(36,168) = .90, p = .639$). The main effect for the SES factor was not statistically significant ($F(9,39) = .46, p = .90$). The SES by group interaction was also not significant ($F(9,39) = 1.47, p = .192$), a not unexpected finding, given that SES had been converted from a continuous variable to a discrete factor. Although not statistically significant, the interesting nature of this interaction is illustrated in Figures 3A to 3C. On the Syntax, Articulation and Draw a Man tests the scores of the Nonsmoking group increased with increased SES, but the scores of the Active group were lower with higher SES. The difference between the groups was therefore greater at the higher SES level.

[Insert Figures 3A to 3C here.]

After adjusting for potential confounds, SES, and SES by group interaction, the main effect of active smoking was still significant ($F(9,39) = 2.32, p = .034$). The squared canonical coefficient indicates that 35% of the variability in the (adjusted) dependent variables is attributable to group differences.

The loading matrix of correlations between dependent variables and the linear function that best separated the groups indicates that all but Arithmetic contributed to the function. (See Table 8.) The most important variables (i.e. those with structure coefficients $.30$) were Conduct Problems, Sound Blending, Syntax, Articulation, Verbal Conceptualization, and Draw a Man. Of these, Conduct Problems and

Figure 3: Smoking by SES interaction for Syntax, Articulation and Draw a Man tests

A/ Syntax

B/ Articulation

C/ Draw a Man

Sound Blending would continue to demonstrate significant group differences if tested individually using a one-way analysis of covariance test.

[Insert Table 8 here.]

These results thus indicate that the children of active smokers scored more poorly than children of nonsmokers on tests of speech and language ability, verbal intelligence and visual-spatial skills, and were rated by their mothers as having more conduct problems, even after adjustment was made for demographic differences.

.4 Comparison of Passive and Nonsmoking Groups

A/ Passive vs. Nonsmoking Discriminant Analysis

A stepwise discriminant analysis was performed using the same 23 variables (in Table 4) as predictors of membership in the Nonsmoking and Passive Smoking groups. This time the Active group was eliminated from the analysis, resulting in a total N of 58. The results, reported in Table 9, indicate statistically significant discrimination between the two groups, with a $\chi^2(9) = 28.15$, $p = .0009$. The canonical correlation of .65 indicates the discriminant function accounts for 42% of the between group variability.

[Insert Table 9 here.]

TABLE 8: MANCOVA Results with Active Smoking and
Nonsmoking Groups

Variable Name	Structure Coefficient	Univariate F(1,47)
Conners - Conduct Problems	-.57	8.20**
Sound Blending	.43	4.72**
Syntax	.37	3.49+
Articulation	.35	3.14+
WISC-R Verbal Conceptualization	.31	2.38
Draw a Man	.30	2.31
WISC-R Freedom from Distract.	.28	2.04
Pegboard (nondominant)	-.24	1.48
WRAT - Arithmetic	.06	0.10

** $p \leq .01$

* $p \leq .05$

+ $p \leq .10$

TABLE 9: Discriminant Analysis Results for Passive Smoking
and Nonsmoking Groups (N = 58)

Chi-squared (22)	28.15
Significance	.0009
Canonical Correlation	.649
% Variance shared	42.1%

Variable	Structure Coefficient	Univariate F/signif.
Peabody Vocabulary	.50	10.06**
WISC-R Perceptual Organization	.38	5.84*
WISC-R Distractability	.36	5.26*
TOLD Syntax	.28	3.19+
Reversals	-.22	1.93
Articulation	.17	1.22
Examiner Behaviour Rating	.15	0.97
Conners - Psychosom.	-.05	0.12
WRAT - Arithmetic	.02	0.01

Using this function, 83% of the subjects were correctly classified into their respective groups, with slightly more members of the Passive group correctly classified (86%) than the Nonsmoking members (78%). The results of the crossvalidation procedure suggest that moderate shrinkage would occur, as the correct classification rate was reduced from 81% to 63%. (See Appendix M.) This latter rate is still well above chance, however, and indicates the function is relatively stable (Fletcher, Rice and Roy, 1978).

A total of 9 variables entered stepwise into the function. Examination of the loading matrix of structure coefficients reveals that only three variables had high correlations with the function, namely Peabody Picture Vocabulary, Perceptual Organization, and Distractability. Univariate group comparisons for these three variables would reach statistical significance if analyzed individually. Also contributing to the function, but not significantly, were scores on Syntax and Hyperactivity.

On the dimension separating the Passive and Nonsmoking groups, therefore, were measures of language, intelligence and attention. When these variables are compared with those reported in the two previous discriminant functions (i.e. all three groups, and Active vs. Nonsmoking), it becomes apparent that these variables also appeared as important on the second function in the three-group analysis. It will be recalled that this second function acted to separate the Nonsmoking group from the two smoking groups. The only significant variable shared between the Passive vs. Nonsmoking and the Active vs. Nonsmoking

functions is Distractability. The Syntax variable showed some relationship on both functions, as well.

B/ Passive vs. Nonsmoking MANCOVA

The variables from the discriminant function were entered into a MANCOVA analysis to see if the differences between Nonsmoking and Passive groups would remain significant after adjusting for potential confounds. The combined covariates were not related to the dependent variables ($F(45,235) = .99, p = .490$). No significant Covariate by Group interaction was found ($F(45,210) = .90, p = .654$).

The multivariate test for group differences, after adjusting for covariates, was statistically significant ($F(9,43) = 2.50, p = .021$). The canonical correlation of .59 indicates 34% of the variability in test scores is explained by differences between the groups. Examination of the structure matrix in Table 10 reveals that none of the variables show a meaningful (i.e. .30) correlation with the linear combination. Nor would any of the variables show a statistically significant univariate F ratio, if group differences were tested on each variable individually. Thus, a statistically significant multivariate effect was found, even though individual variables show no significant group differences, after adjustment for potential confounds.

[Insert Table 10 here.]

TABLE 10: MANCOVA Results for Passive Smoking
and Nonsmoking Groups

Variable	Structure Coeff.	Univariate F(1,51)/signif.
Reversals	-.29	2.31
Syntax	.28	2.06
Peabody Vocabulary	.27	1.92
Examiner Behaviour Rating	.24	1.52
WISC-R Distractability	.22	1.32
Conners - Psychosomatic	-.22	1.28
WISC-R Perceptual Org.	.19	1.00
WRAT - Arithmetic	-.17	0.74
Articulation	.15	0.58

** $p \leq .01$

* $p \leq .05$

+ $p \leq .10$

The results thus far support the second hypothesis, namely that statistical control for demographic confounds would act to reduce the magnitude of the relationship between passive smoking and behavioural outcome. The fact that this did not occur to the same extent on the Active Smoking vs. Nonsmoking analysis suggests a greater magnitude and stability of effects associated with active smoking. The remaining question to be answered, then, is on what dimensions are the Active and Passive smoking groups alike and/or different?

.5 Comparison of Active and Passive Smoking groups

A/ Passive vs Active Discriminant Function Analysis

A stepwise discriminant analysis using the same 23 predictor variables reveals these two smoking groups can be statistically distinguished ($\chi^2(13) = 52.67, p = .0000$). Using this function, 88% of the subjects were correctly classified. The square of the canonical correlation coefficient indicates that 59% of the total variance is attributable to differences between groups. Results are reported in Table 11. The important variables in this discriminant function were the Pegboard (dominant hand) and Draw a Man tests. The children in the Passive group scored lower on the Pegboard test, but higher on the Draw a Man.

[Insert Table 11 here.]

TABLE 11: Discriminant Analysis Results for Passive and Active Smoking Groups (N = 67)

Chi-squared (13)	52.67
Significance	.0000
Canonical Variance	.77
% Variance Shared	59%

Variable	Structure Coefficient	Univariate F/signif.
Pegboard (dominant)	.49	23.05**
Draw a Man	-.35	11.77**
Peabody Vocabulary	.23	4.89*
WRAT - Reading	-.20	3.95+
Conners - Conduct Problems	.19	3.51+
Conners - Impulsivity	.18	3.13+
Articulation	-.15	2.24
Examiner Behaviour Rating	.11	1.08
Conners - Hyperactivity	.09	0.85
WISC-R Perceptual Organization	.09	0.69
Conners - Psychosomatic	.08	0.65
Dominance	-.06	0.37
Conners - Learning	.03	0.06

The crossvalidation results indicated a reduction in correct classification from 92% to 65%. (See Appendix N.) This represents a moderately large shrinkage rate, but the correct classification rate is still above chance, and the function appears to be relatively stable (Fletcher, Rice and Roy, 1978).

In summary, when the two smoking groups are analyzed, the dimension along which they differ is primarily in the motor and visual/spatial domains. It appears to be a dimension of speed vs. accuracy, with the Active group demonstrating better motor speed, and the Passive group showing relatively better attention to detail.

B/ Passive vs Active MANCOVA

A MANCOVA analysis was done with the 13 variables in Table 11 entered as predictors, along with the same five covariates used previously. The results indicate that there are still significant differences between the Passive and Active Smoking groups, after statistical adjustment for differences on control variables ($F(13,48) = 3.12, p = .002$). The covariates did not show a significant overall relationship with the combined outcome variables ($F(65,260) = 1.25, p = .118$), and there was no significant interaction between covariates and groups ($F(65,235) = 1.05, p = .384$). The canonical variate of the covariates explained less than 10% of the variance in the dependent variables.

After covariate adjustment, the variables showing meaningful correlation with the canonical variate were the Examiner Behaviour

ratings and scores on Draw a Man. (Refer to Table 12.) The correlation of the Pegboard scores was also very close to the .3 level. The Passive group scored lower on the Pegboard test and Examiner ratings, but higher on Draw a Man.

[Insert Table 12 here.]

Given the importance of the Examiner Behavioural ratings, this total score was further analyzed into its 6 components, i.e. Attention, Motor Activity, Rate of Performance, Manual Dexterity, Cooperation, and Effort. T - test comparisons indicate that these ratings were significantly different for the two groups on Rate of Performance ($t(87) = 2.14, p = .035$) and Manual Dexterity ($t(87) = 2.34, p = .022$). (See Table 13.) After adjustment for (the usual) covariates, these two ratings continued to show significant differences (Rate: $F(60) = 12.02, p = .001$; Dexterity: $F(60) = 17.55, p = .000$). On both variables, adjustment for covariates acted to further separate the groups. In addition, Cooperation also showed significant group differences, after adjustment for covariates ($F(60) = 4.69, p = .034$). The active smoking group scored higher on all three variables, and was therefore rated as showing better manual dexterity, faster performance rate and better cooperation. Indeed, the Active group scored higher, although not statistically so, on the remaining ratings as well.

[Insert Tables 13 and 14 here.]

TABLE 12: MANCOVA Results for Passsive and Active Smoking Groups
(N = 67)

Variable	Structure Coefficient	Univariate F(1,60)/signif.
Examiner Behaviour Rating	.42	8.86**
Draw a Man	-.32	5.04*
Pegboard (dominant)	.29	4.40*
Articulation	-.21	2.33
WISC-R Perceptual Organization	.20	1.95
Dominance	-.17	1.41
Conners - Conduct	.16	1.26
WRAT - Reading	.13	0.92
Conners - Psychosomatic	.08	0.39
Conners - Learning	.07	0.27
Peabody Vocabulary	.07	0.25
Conners - Impulsivity	.02	0.02
Conners - Hyperactivity	.00	0.00

** p < .01; * p < .05; + p < .10

TABLE 13: Means, T-test and ANCOVA Results for Examiner Behaviour Ratings Subtests (N = 67)

Subtest	Non Smoking (n = 23)		Passive Smoking (n = 35)		Active Smoking (n = 33)		Passive vs. Active Smoking	
	Mean	S.D.	Mean	S.D.	Mean	S.D.	t-test/sig. (1,64)	ANCOVA F/sigif.
Attention	3.61	0.66	3.54	0.92	3.41	0.95	-0.60	0.44
Motor Activity	2.78	0.85	3.29	1.20	3.19	1.03	-0.36	0.39
Performance Rate	3.17	0.94	2.74	1.01	3.25	0.95	2.12*	12.02**
Manual Dexterity	3.04	0.77	2.97	0.62	3.38	0.75	2.39*	17.55**
Cooperation	4.26	0.62	3.80	1.02	3.97	0.82	0.75	4.69*
Effort	3.87	0.55	3.57	0.98	3.47	0.76	-0.48	1.07

TABLE 14: ANOVA Results for Pegboard Errors for Passive and Active Smoking Groups

	Non Smoking [@] (n = 22)		Passive Smoking (n = 32)		Active Smoking (n = 33)		Passive vs. Active Smoking	
	Mean	S.D.	Mean	S.D.	Mean	S.D.	t-test	signif.
Pegboard (dominant) errors	1.00	1.00	0.50	0.72	0.82	1.01	1.46	.149
Pegboard (nondom.) errors	1.18	1.18	0.84	0.88	1.00	1.32	0.56	.577
Total Errors	2.18	1.65	1.34	1.31	1.82	2.07	1.11	.273

[@] Note that Non Smoking group data is included for comparison purposes, but t-tests and ANCOVA F ratios refer to contrasts between Passive and Active groups.

** p < .01

* p < .05

+ P < .10

The Pegboard test was further examined in terms of the accuracy of performance. The Pegboard (dominant and nondominant) scores reported thus far reflected fine motor speed and dexterity. Also noted during the testing session were the number of "errors" made (i.e. dropping the pegs). These errors are relatively rare with this age group, and preliminary analysis during the pilot stage of this research found that the conversion to standard scores provided highly suspect variability. Using the raw scores representing the mean number of errors per group, the Passive group was found to make fewer errors than the Active group, with both the dominant and nondominant hands. Interestingly, the Nonsmoking group made the most errors. Analysis of variance showed that these group differences did not reach statistical significance. (See Table 14.)

The Active group was therefore faster, but less accurate, on tests of fine motor dexterity. The Passive group showed better scores on the Draw a Man test, which is not timed, and on which attention to detail is important. The Active group also scored higher on Examiner ratings for Cooperation, Rate of Performance and Manual Dexterity.

.6 Other Variables

As was explained earlier, five variables were omitted from the multivariate analysis. The results of the univariate analysis of these variables are reported here.

A/ Reading Understanding

This is a test of silent reading comprehension, as opposed to the WRAT Reading test which requires oral decoding of single words. This test was not included in the multivariate analysis due to the preponderance of missing data. The group means are reported in Table 15, and are in the predicted orientation (i.e. Nonsmoking group performed the best, followed by Passive and then Active Smoking). The ANOVA results showed no significant group differences ($F(2,73) = 0.41$, $p = .663$). All pairs contrasts were calculated and the results also showed no significant differences between groups.

[Insert Tables 15 and 16 here.]

B/ Slingerland Copying Test

This is a copying task whereby a short passage is copied from a wall chart onto a sheet of lined paper. The test manual provides suggestions for scoring, but norms for conversion to standard scores are not available. A scoring system was therefore developed by this author so that the scores represent the number of errors. The types of errors scored are listed in Appendix I. Group means are reported in Table 16.

Grade in school was felt to be an important confounding factor for performance on this test. Test scores were therefore entered into an ANCOVA with Grade in School as a covariate. These results show that, as predicted, Grade acted as an important covariate, providing

TABLE 15: Means and ANOVA Results for Reading Understanding

Group	Mean	S.D.
Nonsmoking	111.53	18.88
Passive Smoking	109.90	17.06
Active Smoking	107.07	15.46
Total Sample	109.30	16.86

ANOVA $F(2,73) = 0.41$; $p = .663$

TABLE 16: Means, ANCOVA and MANCOVA for Slingerland Copying Test Scores (adjusted for grade in school)

Group	Error Score Mean	S.D.	Adjusted Mean
Nonsmoking	3.50	4.31	3.41
Passive Smoking	7.20	4.93	7.52
Active Smoking	6.39	5.86	6.12
Total Sample	6.00	5.16	5.86

ANCOVA $F(2,86) = 4.38$; $p = .015$

MANCOVA $F(2,81) = 1.68$; $p = .193$

significant adjustment to the scores. Group differences on the adjusted scores were statistically significant ($F(2,86) = 4.38, p = .015$).

The profile of mean scores is such that children in the Nonsmoking group made the fewest errors, and the Passive group the most errors, with Active Smoking in between. An interesting contrast can be drawn with the accuracy scores on the Pegboard test reported in section .5, in which the Passive group was the most accurate and the Nonsmoking group the least accurate. It must be remembered that the errors made on Pegboard reflect motor dexterity and perhaps impulsivity, whereas this Copying test required higher level cognitive processing.

To determine whether these group differences would remain significant after adjusting for potential demographic and experimental confounds, the same five covariates were entered, along with Grade, into a MANCOVA analysis. After adjustment for covariates, the group effect was no longer statistically significant. The covariates responsible for significant adjustment were Grade and Maternal Age.

C/ Growth Measurements

The measurements for Height and Weight were converted to age percentile scores. Head circumference measurements were converted to standard (z) scores. It will be recalled that the profile of group means found in Figure 2E indicated that the Nonsmoking group was taller and heavier, on average, than both smoking groups. The Passive group weighed slightly more, on average, than the Active group, whereas the

Active group was slightly taller. The Nonsmoking group showed a smaller head circumference than both smoking groups.

Cases were grouped above and below the 50th percentile, and the distributions are presented in Table 17. A Chi-square analysis of the frequency distribution showed that there were no statistically significant differences between groups.

[Insert Table 17 here.]

.7 Path Analysis

Path analysis is a useful analytic tool to assess the relative importance of the direct effect of prenatal smoking exposure vs. the indirect effect as mediated by the home environment. This analytic method was used selectively with several dependent variables, chosen as important indices of smoking effects, and representative of the different domains of abilities tapped with this battery. The dependent variables chosen were: Articulation, Syntax, Conduct Problems, Pegboard, Draw a Man, and Verbal Conceptualization.

Preliminary analysis determined that the individual FES subscales did not correlate significantly with the dependent measures. (See correlation matrix, Appendix G.) Analysis of variance also revealed that scores on only one subscale (Intellectual-Cultural Orientation) were found to show significant group differences. (See Table 18.) In

Table 17: Chi-square Analysis of Growth Measures

A/ WEIGHT

	GROUP						Total	
	Nonsmoking		Passive		Active			
	N	%	N	%	N	%	N	%
< 50th percentile	4	18.2	6	20.0	7	21.2	17	20.0
≥ 50th percentile	18	81.8	24	80.0	26	78.8	68	80.0
Total	22		30		33		85	

Chi-square (2) = 0.08; p = .963

B/ HEIGHT

	GROUP						Total	
	Nonsmoking		Passive		Active			
	N	%	N	%	N	%	N	%
< 50th percentile	3	13.6	5	15.2	5	15.2	13	14.8
≥ 50th percentile	19	86.4	28	84.8	28	84.8	75	85.2
Total	22		33		33		88	

Chi-square (2) = 0.03; p = .985

C/ HEAD CIRCUMFERENCE

	GROUP						Total	
	Nonsmoking		Passive		Active			
	N	%	N	%	N	%	N	%
< 50th percentile	3	13.6	4	12.5	4	12.1	11	12.6
≥ 50th percentile	19	86.4	28	87.5	29	87.9	76	87.4
Total	22		32		33		87	

Chi-square (2) = 0.03; p = .986

order to retain the FES as a viable measure of the potential mediating influence of the home environment, a factor analysis was conducted to determine whether factor scores could be utilized instead of the individual FES subscales. Using SPSSx Factor with varimax rotation five factors were calculated, and their loading matrices are reported in Table 19. The first factor, FES1, was found to account for 22% of the variance in the FES scale, and received high loadings from 7 of the 10 subscales. Three of these subscales, Cohesion, Intellectual-Cultural Orientation, and Expressiveness have been found in previous studies to predict cognitive ability and behaviour problems in young children (Gottfried and Gottfried, 1984; McGee, Silva and Williams, 1984; Wilson and Matheny, 1983). Scores on FES1 were significantly different across groups, whereas scores on the other factors showed no such relationship with smoking. FES1, therefore, was used as the measure of home environment in the path analysis.

[Insert Tables 18 and 19 here.]

The three smoking exposure groups were dummy coded into two vectors - S1 and S2. The coding scheme operated as follows: Nonsmoking members were assigned 0 on both vectors, representing the control group. Passive members were assigned 1 on S1, and 0 on S2. Active members were assigned 0 on S1 and 1 on S2. Thus the vector S1 represented Passive group membership, and S2 represented Active group membership.

Table 18 : Group Means on Family Environment Scale

FES subscale	Total Sample		Nonsmoking		Passive Group		Active Group		ANOVA F (2,87)/ signif.
	mean	s.d.	mean	s.d.	mean	s.d.	mean	s.d.	
Cohesion	56.09	11.94	60.43	7.50	55.15	12.45	54.03	13.38	2.18
Expressiveness	55.33	11.26	57.22	13.47	53.44	10.53	55.97	10.33	0.85
Conflict	52.57	11.23	50.87	12.81	51.12	10.20	55.24	10.91	1.50
Independence	48.82	10.45	46.52	10.49	50.82	9.17	48.36	11.54	1.22
Achievement	47.77	11.42	48.22	10.67	47.15	11.95	48.09	11.68	0.08
Orientation									
Intel-Cult.	57.04	10.85	62.74	7.53	57.44	10.97	52.67	11.01	6.62**
Orientation									
Active-Rec.	54.94	9.88	57.17	8.17	54.06	9.29	54.30	11.48	0.79
Orientation									
Moral Relg.	51.01	10.60	52.91	10.61	48.88	11.44	51.88	9.61	1.17
Emphasis									
Organization	51.08	11.33	53.26	10.32	52.29	11.57	48.30	11.52	1.62
Control	52.34	10.86	50.39	11.51	53.79	11.29	52.21	10.01	0.67
FES1	0.00	1.00	0.43	0.87	0.00	0.76	-0.30	1.21	3.83*

** p < .01

* p < .05

+ p < .10

TABLE 19: Factor Loading Matrix for Family Environment Scale

FACTOR MATRIX:

<u>Variable</u>	<u>Factor 1</u>	<u>Factor 2</u>	<u>Factor 3</u>	<u>Factor 4</u>	<u>Factor 5</u>
Cohesion	.78	.20	-.04	-.20	.12
Intellectual Cult. Orient.	.59	.21	-.21	.51	.00
Control	-.04	.69	.37	-.05	-.31
Achievement Orient.	-.41	.65	-.08	.26	.13
Moral Religous. Emph.	.06	.57	.14	-.36	.55
Organization	.53	.53	-.19	-.02	-.17
Independence	.12	-.15	.84	.00	.21
Conflict	-.57	.24	.12	.58	.13
Expressiveness	.49	-.27	.04	.43	.51
Active Rec. Orient.	.43	-.03	.41	.26	-.47

FACTOR	EIGEN VALUE	% of VARIANCE	CUM. %
1	2.18	21.8	21.8
2	1.74	17.4	39.2
3	1.13	11.3	50.6
4	1.09	10.9	61.4
5	1.01	10.1	71.5

Path coefficients represent the proportion of variance in the dependent variable for which an independent variable is uniquely responsible, with all other independent variables partialled out (Achenbach, 1978). As all variables were standardized, the path coefficients in Figures 4A to 4F represent the Beta weights in the regression equations. Only those coefficients .10 are reported (Kerlinger and Pedhazur, 1973). All variables were standardized using Z-transformations to aid interpretation of the path coefficients.

The first stage of the path analysis consisted of a regression of Home Environment (FES1) onto the independent variables. SES and Maternal Education were therefore entered (as a block) into a multiple regression, followed by the smoking group variables (S1 and S2, also entered as a block), with Home Environment as the dependent variable. A significant multiple correlation was obtained ($R=.35$, $F(4,86) = 3.09$, $p = .02$). Maternal education was the best predictor of Home Environment, followed by the smoking variables, with SES making a minor contribution. This first stage was common for all outcome variables, and it is noted that the results of the first step are identical on all path diagrams.

[Insert Figures 4A to 4F here.]

At the next stage the different outcome variables were regressed individually as dependent variables with Home Environment, SES, Maternal Education and smoking status as predictors. Each regression was done hierarchically, tracing the causal path backwards. Thus, Home

Figure 4: Path diagrams with path coefficients

A/ Articulation

$R = .36$; $R^2 = 12.6\%$
 $F(5,85) = 2.46$; $p = .040$

number (1,2,3) indicates order of entry of variables into final regression equation

a path coefficient (β)

** $p \leq .01$; * $p \leq .05$; + $p \leq .10$

B/ Syntax

$R = .33$; $R^2 = 10.6\%$
 $F(5,85) = 2.03$; $p = .083$

Figure 4 (continued): Path diagrams with path coefficients

C/ WISC-R Verbal Conceptualization

$R = .29$; $R^2 = 8.1\%$
 $F(5,85) = 1.50$; $p = .197$

D/ Pegboard (Dominant)

$R = .44$; $R^2 = 19.0\%$
 $F(5,85) = 3.99$; $p = .003$

** $p \leq .01$; * $p \leq .05$; + $p \leq .10$

Figure 4 (continued): Path diagrams with path coefficients

E/ Draw a Man

$R = .39$; $R^2 = 15.0\%$
 $F(5,85) = 3.00$; $p = .015$

F/ Conduct Problems

$R = .35$; $R^2 = 12.1\%$
 $F(5,85) = 2.34$; $p = .049$

** $p \leq .01$; * $p \leq .05$; + $p \leq .10$

Environment, Maternal Education and SES entered first, as a block, followed by the smoking variables entered last, also as a block. The relative magnitudes of the path coefficients varied considerably with the different outcome measures.

With the Articulation test as outcome, the predictors accounted for a significant proportion of variance ($R=.36$, $F(5,85) = 2.46$, $p = .040$). Home Environment showed no relationship with Articulation. Maternal Education showed a small direct effect. The direction and significance of the coefficient for SES indicated it has acted as a suppressor variable in this regression. Inspection of the correlation matrix reveals a negligible zero-order correlation, whereas the Beta coefficient is relatively large and in the reversed direction. SES has apparently suppressed error variance in another predictor, possibly smoking, in order to enhance the multiple correlation.

The smoking variable S2, representing Active Smoking, was the best predictor of Articulation, with a path coefficient of $-.44$. The other smoking variable S1, representing Passive Smoking, showed a less significant relationship with Articulation (Beta $= -.17$). The two smoking variables together accounted for a significant change in R^2 , from 3.3 to 12.6 (F change $= 4.54$, $p = .01$). The path diagram illustrates that smoking has significant negative effects on Articulation, with no indirect effects mediated by the home environment.

The path diagram for Syntax reveals a similar picture for the direct effects of smoking. The first stage regression results with Home Environment and the predictors are the same as reported above. When Syntax was regressed hierarchically onto all predictors, a borderline significant multiple correlation was the result ($R = .33$, $F(5,85) = 2.03$, $p = .083$). Only S2, representing active smoking made a significant contribution to this prediction, however, with a B of $-.33$. The passive smoking variable showed a minor effect, also in a negative direction ($B = -.21$). Home environment and the other demographic variables showed neither direct nor indirect causal paths with Syntax.

With the WISC-R Verbal Conceptualization factor score, the overall regression was not significant ($F(5,85) = 1.50$, $p = .197$). Nevertheless, Passive and Active Smoking showed equal, but not significant, path coefficients. No indirect effects of smoking were found, as Home Environment showed no relationship with this outcome. Maternal Education emerged as a minor predictor. With this outcome variable, it appears that other factors must be entered into the causal model before interpretation can be made of causal relationships.

The Pegboard results, on the other hand, reveal a highly significant overall regression ($F(5,85) = 3.99$, $p = .003$). The direct effects of Active and Passive Smoking are in reverse directions, with Passive Smoking significantly related to lower Pegboard scores, and Active Smoking associated with higher scores. Home environment and SES achieved negligible predictive power, and Maternal Education again

played a minor role.

The regression results for Draw a Man were also highly significant ($F(5,85) = 3.00, p = .015$). Both SES and Active Smoking emerged as predictors. Active smoking was related to lower scores, whereas SES showed a positive relationship. Passive Smoking showed no direct relationship with the outcome, and neither did the other independent and mediating factors. This finding is interesting, in light of the importance of Draw a Man as one of the best discriminators between Active and Passive groups. This may suggest that the family environment may not play a mediating role with this outcome, but that socioeconomic differences are a primary factor.

When the Conners Conduct Problems score was used as dependent variable in the regression analysis, a very different causal path relationship emerged. A significant overall regression was obtained ($F(5,85) = 2.34, p = .049$), and Home Environment was the major predictor ($B = .23$). Neither SES nor Maternal Education made a significant unique contribution, although this time Maternal Education appears to have acted as a suppressor variable. The active smoking variable also showed a small but nonsignificant direct effect, whereas the passive variable showed no direct relationship to this behavioural outcome. These results suggest that higher scores on the Home Environment measure are significantly associated with fewer conduct problems in children. Home environment appears to play a mediating role in the negative relationship between prenatal smoking exposure and conduct problems.

Using this model, the total effect of Active Smoking on Conduct Problems can be estimated by adding the direct and indirect effects, as illustrated below:

$$\begin{aligned}\text{Total effect} &= \text{Direct Effect} + \text{Indirect Effect} \\ \text{Total effect (Active)} &= .15 + (-.23)(-.28) \\ &= .15 + .06 \\ &= .21\end{aligned}$$

Similarly, the indirect effect of Passive Smoking on behaviour can be calculated as follows:

$$\begin{aligned}\text{Indirect effect (Passive)} &= (-.20)(-.23) \\ &= .05\end{aligned}$$

This example serves to illustrate the importance of considering patterns of shared variance, rather than just unique effects, when assessing the relative importance of a predictor variable.

In summary, the Home Environment factor score played an important mediating role solely in the behavioural area. Active Smoking showed both direct and indirect effects with Conduct Problems. Although no direct effect for Passive Smoking emerged with behaviour, an indirect path was calculated whereby its influence was mediated by Home Environment.

In the speech and language domain, Active Smoking showed a significant direct effect on performance on the Articulation and Syntax tests. Scores on tests of intelligence (Verbal Conceptualization), fine motor dexterity (Pegboard), and visual/spatial ability (Draw a

Man) showed direct, but not significant, effects of Active Smoking. The direct effects of Passive Smoking were significant for fine motor dexterity, and in the opposite direction to the influence of Active Smoking. Passive Smoking also showed direct predictive power, although not statistically significant, with tests of speech and language and intellectual abilities.

.8 Summary of Results

Scores on 23 tests in a neuropsychological test battery were used as predictors of membership in three groups. The groups consisted of school-aged children of women who had been nonsmokers, passive smokers, and active smokers during pregnancy. The sample was comprised of 91 children, 1 of whom was dropped from analysis due to excess missing data.

Using Discriminant Function Analysis, two highly significant discriminant functions were calculated. The first function maximally separated the Active Smoking group from the other two groups. Important variables on this function were tests of fine motor dexterity (Pegboard), visual/spatial ability (Draw a Man) and three of Conners' behaviour scales: Conduct Problems, Impulsive/Hyperactive, and Hyperactivity. The second function discriminated the Nonsmoking group from the two smoking groups. This dimension consisted of scores on tests of speech and language abilities (Syntax, Peabody Vocabulary), and the 3 WISC-R factor scores: Verbal Conceptualization, Perceptual Organization and Distractability. Using MANCOVA to control for potential confounding factors, the results were of borderline statistical significance. Two functions again were calculated and they showed a similar loading structure to the Discriminant functions. Variables that maintained the most significant group differences after adjustment for covariates were tests in the speech and language domain

(Articulation and Syntax).

When the Active group was compared with the Nonsmoking group using Discriminant analysis, highly significant group differences were found. Even after adjusting for possible confounds using MANCOVA, variables showed highly significant discriminating power in the domains of speech and language (Sound Blending, Syntax, Articulation), intelligence (Verbal Conceptualization), visual/spatial ability (Draw a Man), and behaviour (Conduct Problems). On each of these measures the Active group performed more poorly than did the Nonsmoking group.

Comparing the Passive and Nonsmoking groups using Discriminant and MANCOVA analyses, the results were also statistically significant. Important variables were tests of speech and language (Peabody Vocabulary) and intellectual abilities (Perceptual Organization and Distractability), and the Passive group showed poorer performance in these areas than did the Nonsmoking group. Control for covariates acted to reduce the magnitude of group differences, however.

A comparison of the two smoking groups (Passive and Active) found that they could be best discriminated by scores on tests of fine motor dexterity and visual/spatial ability (i.e. Pegboard and Draw a Man). After adjustment for potential confounds it was also found that the Active group was rated more favourably by the examiner on scales of Cooperation, Rate of performance and Manual dexterity.

The Path analysis results largely confirmed the above pattern, but added the potential mediating influence of the home environment to the analysis. Factor scores on a measure of home environment were found to act as a mediating factor only for behaviour (Conduct Problems). The relationship between Passive Smoking and behaviour was indirect, and mediated by the home environment. Active Smoking showed both a direct and indirect effect with behaviour.

The percent of variance accounted for by the two prenatal smoking variables was significant for Articulation (9.3%, $p = .01$), Pegboard (15%, $p = .0007$), and Draw a Man (6.3%, $p = .05$). Otherwise, the variance accounted for by Passive and Active smoking exposure was 2 to 5% of total variance.

IV DISCUSSION

In this chapter, the results will be reviewed with respect to the stated research hypotheses. Smoking effects will be compared and contrasted with findings of previous researchers, and tentative explanations will be suggested for results that are inconsistent. Experience gained as a result of this research study will be translated into suggestions for future research, and implications for public policy and intervention will be discussed.

.1 Review of Hypotheses

Three hypotheses were made at the beginning of this research study. The main prediction was that the the long-term consequences of passive smoking during pregnancy would be similar to those of active smoking, but that the magnitude of differences between Passive and Nonsmoking groups would be smaller than differences between Active and Nonsmoking groups. The results of the discriminant function analyses showed that the Passive group could be statistically differentiated from the Nonsmoking group, and that the important tests in this discrimination were in many cases the same tests that were found to discriminate between the Active and Nonsmoking groups. Of particular note were scores on tests of intellectual and speech and language abilities, which were depressed in both the Passive and Active smoking groups. Examination of the profile of mean scores of the three groups

on the test battery confirmed that the effects of active smoking during pregnancy were generally greater in terms of breadth and magnitude.

The second hypothesis was concerned with the association between passive smoking and demographic factors. It was predicted that control for socioeconomic factors would reduce the magnitude of the relationship between passive smoking and developmental outcome. Results confirmed that mothers in the Passive Smoking group were younger, had less education, lower occupation ratings, and lower family income when compared to the Nonsmoking group. When these and other experimental potential confounds were statistically controlled using a multivariate analysis of covariance the three-group multivariate test was at a borderline level of significance ($p = .083$). These results are reported and discussed, however, because smoking is one factor in a lifestyle that can be shown to influence child development. The "true" relationship of prenatal smoking exposure and long-term outcome likely lies somewhere between the correlation in which no confounding variables are considered and the unique contribution of smoking after the variance attributed to covariates has been removed (Fried and Watkinson, 1988).

On the two-group covariance analyses, the Passive group could be differentiated from the Nonsmoking group, but differences on individual tests no longer made an important contribution. In contrast, when the same potential confounds were considered in the analysis of covariance for Active Smoking and Nonsmoking groups, the differences between

groups was maintained at both a multivariate and univariate level. Variables that showed the greatest stability in maintaining group differences after covariate adjustment were Articulation, Syntax and Pegboard.

Finally, a prediction was made with regard to the potential mediating role of the home environment in influencing the long-term outcome of prenatal smoking. When Conners' Conduct Problems ratings were analyzed it was found that the home environment served as an important mediating variable for both the Passive and Active Smoking groups, but that Active Smoking was also directly associated with more behaviour problems. The home environment was not found to be an important variable in the prediction of developmental outcome in the other domains, i.e. intellectual, speech and language, motor, and visual/spatial.

.2 Comparison of Results with Previous Literature

The significant results found on tests of speech and language abilities are consistent with findings of previous research with infants and younger children. In infancy, for instance, smoking has been found to be related to scores on tests of auditory orientation and habituation, responsiveness to sound and the delayed onset of crying (Jacobson et al., 1984; Picone et al., 1982; Saxton, 1978). Within the Ottawa Prenatal study, current results are consistent with previously

published findings with auditory tests in infancy (Fried and Makin, 1987) and at 12 and 24 months of age (Fried and Watkinson, 1988). If one accepts the assumption of a continuity between infant auditory functioning and later language development, it appears that the effects of prenatal exposure to cigarette smoke show long-term consistency. Smoking during pregnancy is related to reduced auditory responsiveness at birth, poorer development of auditory perceptual skills during infancy, and eventually a mild delay in oral language and speech articulation skills at school age.

Children in both smoking groups were rated on all scales in the Conners' Parent Questionnaire as showing more behaviour problems than the Nonsmoking group. None of the scales showed a statistically significant difference (at the .05 level of significance) across the three groups, but the Conduct Problems scale showed a significant difference between the Active and Nonsmoking groups. The increased incidence of conduct problems associated with prenatal smoking is consistent with those of Dunn et al., 1977, and Davie, Butler and Goldstein, 1972, who found that children of smokers were rated as showing more misbehaviour and poorer adjustment in school. Once again a continuity of effect can be traced back to Saxton's (1978) finding that examiners rated the babies of (active) smokers as showing decreased self-control and lower consolability.

The Active group were rated as having the most behaviour problems on all Conners' subscales and the Passive group was rated in the middle

except for Anxiety, on which the Passive group was rated as showing slightly more anxiety symptoms. On the Examiner rating scale, however, the three groups showed variable standings. The Nonsmoking group was rated as showing the best attention, cooperation and effort, but the Active group was rated as showing the fastest performance rate and the best motor dexterity. The Passive group showed the least motor activity and achieved a slightly lower overall Examiner rating than did the Active group. One reason for the discrepancies between rating scale scores could be that the Examiner was rating the child's behaviour during a relatively short testing session during in which the tasks expected of the child were highly structured, whereas the child's mother rated the child's typical behaviour. Given the personality characteristics of smokers (reported earlier), one might also ask whether it is the child who has more problems or the mother who has less patience or tolerance. Future researchers would therefore be wise to include a teacher rating scale to provide an additional source of information of day-to-day behavioural functioning.

The Active Smoking group outperformed the other groups on the Pegboard test of fine motor skill. This test requires the child to place keyhole-shaped metal pegs into rows of holes. The task is done first with the child's dominant hand, and then repeated with the nondominant hand. The results showed that the superior speed of the Active group was greatest for the dominant hand, suggesting a ceiling effect for speed was reached when the task became more difficult. When errors were also considered (e.g. dropping a peg), it became clear that

the Passive group performed the task more slowly, but also made the fewest errors.

The Active group thus performed more quickly on fine motor tasks and were rated as showing superior speed and manual dexterity. They were also rated by their mothers as showing more impulsive and hyperactive behaviour. Again, if infant motor responses are assumed to be predictive of later motor development, this motor and behavioural pattern could be seen to be a long-term outcome of the finding that at birth babies of smokers show increased hypertonicity, with more tremors and startles (Fried and Makin, 1987; Fried et al., 1987).

A surprising result was found in hand preference. It was predicted that the smoking groups would show an increased incidence of left-handedness, but the results show that a greater percentage of children in the Active and Passive groups used their right hand for handwriting. The differences were small and not statistically significant, however. The fact that no significant effect was found is perhaps realistic if one accepts the hypothesis that maternal smoking will be associated with very subtle changes in neuropsychological performance, and not with the more dramatic deficits that would be required to alter brain laterality organization.

Scores on the WISC-R test of intellectual abilities frequently loaded onto the discriminant functions in the multivariate analyses, and group differences on these variables were significant when tested

at a univariate level. The present study did not find statistically significant group differences after potential confounds were included in the analysis. This is consistent with Dunn et al., 1977, who followed children of smokers and nonsmokers in a similar prospective study, and found that I.Q. measures were strongly correlated with social class, and were not significantly linked with smoking status of the mother. Hardy and Mellits (1972) also found no significant intellectual differences between smoking and nonsmoking groups.

Other researchers, however, have found a significant association between maternal smoking and lower scores on intellectual tests, after controlling for social background factors (e.g. Davie, Butler and Goldstein, 1972; Naeye and Peters, 1984; Sexton, Fox and Hebel, 1986). There are several possible explanations for these variable findings. The children in Sexton, Fox and Hebel's study were only 3 years of age. It is possible that if an intellectual deficit has occurred as a result of prenatal smoking exposure, it could be proportionately more pronounced at a younger age and reduced in magnitude in the course of development. This would be particularly likely with a middle-class, well-educated sample such as the OPPS, in which parents would be expected to provide supportive and enriched environments for their children. Indeed, in a recently published OPPS report of intellectual development of smokers' children at 12 and 24 months of age, a significant negative association was found between prenatal nicotine use and 12-month Bayley scores, although by 24 months this relationship had attenuated (Fried and Watkinson, 1988). The most significant

predictor of 24-month intellectual performance were scores on a measure of home environment; this was interpreted as indicating that as the child gets older, home and family environmental factors play an increasingly greater role in determining the child's performance.

An alternate explanation for the above findings can be found by examining the differences in sample size between studies that found significant differences and those that did not. Studies in the former category reported sample sizes of 17,000 and 9,000, respectively, whereas the latter studies all had samples of less than 275. Effects of small magnitude require a large sample to demonstrate statistical significance. Power calculations for the current study determined that a group size of 25 to 30 would provide power of 70% to 80% with an effect of small magnitude and an alpha level of .05. (Note: Groups sizes were 23, 35, 33 for Nonsmoking, Passive and Active groups, respectively.) This also meant, of course, that there was a 20% to 30% chance of not detecting a subtle but significant effect. Future studies should therefore consider the value of a larger group size in order to reduce the likelihood of missing an important effect.

In contrast with most previous research, no significant effect was found for academic achievement in this study. Examination of the raw data reveals that the Active Smoking group scored below the other two groups on tests of Reading and Spelling, so the lack of statistical significance could again be due to the small sample size of this study in contrast with the samples cited in the larger American and British

studies (Davie, Butler and Goldstein, 1972; Naeye and Peters, 1984).

The WRAT-R (Wide Range Achievement Test - Revised) measure of academic achievement tests the child's basic decoding, spelling dictation and arithmetic computation skills. This test was used in one previous study (i.e. Naeye and Peters, 1984), but their results are reported as regression coefficients, so a direct comparison of raw data is not possible. The British studies (Davie, Butler and Goldstein, 1972; and Butler and Goldstein, 1973) used the "Southgate" test of reading - a test unknown in North America. It could be that this test measures reading comprehension rather than just decoding, in which case the comparable measure from this test battery would be the test of Reading Understanding. Scores on this variable could not be included in the multivariate analysis due to excess missing data, but it is important to note that a trend was found for the smoking groups to score below the nonsmoking group. A criticism that could be directed to all studies in this area, present study included, would be that these test instruments assess only very basic skills, and do not address the development of problem-solving and written language skills. If an effect were to be found in these important areas, it would likely prove to be more serious for the child's future educational success than a lag in basic skills.

A related factor in the discussion of the results of academic achievement tests is the fact that many of the children in this study (54%) were enrolled in Early French Immersion programs in school and

therefore have received instruction in the basic academic skills in French. The academic testing, however, was done in English, and it is known that Early French Immersion students lag behind in English skills during the primary grades but show parity with students in the regular (English) program by grades 4 to 5 (Genessee, 1983). This would also explain why the sample as a whole scored only at mid-Average levels (when compared with age norms) on academic tests, although they were well above Average on tests of intellectual ability. Limited instruction in English academic skills may have also resulted in a ceiling effect whereby the more capable children could not yet demonstrate their superior academic abilities. It is possible that if this same sample were tested in two to three years time, the differences between the groups might be magnified.

Aside from scores on academic tests, however, mothers reported "problems in school" for 24% of the Active Smoking group, in comparison with only 11% and 13% for the Passive and Nonsmoking groups, respectively. Although not statistically significant, this finding is of considerable clinical interest; it suggests that many children of smokers have achieved basic academic skills at a level almost commensurate with their age peers in spite of experiencing problems in school, and perhaps as a result of remedial assistance. The increased incidence of school problems is probably also related to the delay in speech and language skills mentioned above, as language-related learning problems are, in the author's experience in school psychology, the most common cause for concern in the primary grades in school.

The profile of scores of the two smoking groups on the total test battery shows indications of underlying attentional problems. The lowered scores on the Freedom from Distractability factor on the WISC-R is an important indicator in this regard, and higher ratings on Conners' "Impulsivity/Hyperactivity" scale and "Hyperactivity" index support this interpretation. The idea that prenatal smoking exposure may be related to attentional problems would be consistent with the findings of Streissguth et al., 1984 and Kristjansson (personal communication), who found that children of smokers showed increased errors on a sustained attention task. The association between smoking and hyperactivity has also been reported in several previous studies (Denson and Nanson, 1975; Naeye and Peters, 1984; Nicols and Chen, 1981).

If this interpretation is valid, there remain the discrepancies between the two smoking groups on tests of motor and visual/spatial skills. The Active Smoking group generally scored better than the Passive group on timed tests of motor coordination, but poorer on untimed tests requiring attention to detail. One explanation is that this reflects a differential effect for passive versus active smoking with respect to a common underlying attentional deficit. Children in the Active group could have been exposed to a greater dose effect and so developed a more impulsive coping style of performing with speed at the cost of accuracy on tests. Behaviourally, this group also shows more signs of impulsivity, as evidenced by their higher ratings on the

Impulsivity/Hyperactivity scale. Children in the Passive group, on the other hand, would have received a lower dose effect, and would be able to compensate by slowing down output in order to attend to detail. The mediating influence of the home environment could also have aided the Passive group to a greater extent, perhaps by encouraging more efficient compensatory strategies.

Future studies could investigate this hypothesis in more detail by including tests that have been found to be particularly sensitive to attentional deficits, such as Kagan's Matching Familiar Figures Test (Aaron, 1981). More detailed observations should be recorded for attention span, distractability and impulsivity, as well as examiner notes regarding compensatory strategies spontaneously used by the child during the testing session. As previously mentioned, a behavioural measure completed by the child's teacher would also be valuable in this regard.

Although the results overall confirmed the expectation that the Nonsmoking group would outperform the two smoking groups, with the Passive group in between, scores on a few tests and subtests did not follow this pattern. Most notable in this regard were scores on the Peabody Picture Vocabulary Test, on which the Nonsmoking group scored the highest, followed by the Active Smoking group and then the Passive group. This test is considered to be a measure of receptive language, as it requires the child to point to one of four pictures on a page, one of which illustrates a word that is said to the child. The

performance demands for this test were such that an anxious response style would hinder optimal performance (i.e. reluctance to guess if unsure). It is interesting to note that children in the Passive group rated highest (in a negative sense) on the Anxiety index on the Conners scale.

It is unlikely that the unusual pattern of results is solely due to the task parameters of the Peabody test in particular. Subtests in the Test of Language Development (TOLD) also used pictures and pointing to assess receptive language, and on these measures the expected profile emerged (i.e. first the Nonsmoking group, followed by the Passive and then the Active groups.) The most likely explanation is the fact that this test is included in the OPPS battery for 4 and 5 year olds. The majority of children in the Nonsmoking and Active Smoking groups were regular follow-up subjects in the OPPS sample, and therefore had previous exposure to this test, whereas the majority of the Passive group were taking this test for the first time. This was also the case for the Pegboard test, and it is possible that the relatively superior performance of the Active and Nonsmoking groups on this test was also due to a practise effect.

There are several alternate explanations for the inconsistent group profiles on a few of the tests in the test battery. For instance, it is established that sidestream smoke differs qualitatively from mainstream smoke. Higher levels of some constituents in sidestream smoke (e.g. carbon monoxide, nicotine) may result in passive

smokers inhaling more of these contaminants than the amount inhaled by active smokers. This could result in a reversal in the expected dose response, with the Passive group showing a greater effect in some areas. Remmer (1987) recently developed an argument to explain the same or even higher risk for malformation and perinatal death associated with paternal smoking in comparison with maternal smoking during pregnancy. He maintains that active smoking of mainstream smoke promotes the induction of placental enzymes, which serve a protective role for the fetus. Many of the harmful constituents in cigarette smoke are converted to inactive metabolites by these placental enzymes in the active smoker. Sidestream smoke lacks the compounds that induce this enzyme production. This results in the otherwise implausible observation that sidestream smoke passively inhaled by the mother during pregnancy might be even more harmful to the fetus than active smoking, because the toxic compounds are not detoxified in the placenta.

Another explanation for the variation in group profiles would be that the Passive group findings may be related to another unknown occupational hazard during the prenatal period, since more mothers in the Passive group worked outside the home (Martin and Bracken, 1986).

Future researchers addressing the issue of long-term effects of passive smoking during pregnancy would be wise to consider pre-pregnancy smoking habits when selecting their nonsmoking and passive smoking groups, as it is possible that a former smoker will have smoked early in the pregnancy, perhaps before the pregnancy is confirmed, and still report no active smoking during pregnancy. In addition, the finding by Nicols and Chen (1981) that pre-pregnancy smoking was a significant predictor of later learning problems also suggests that this may be an important confounding factor. Unfortunately, due to limited sample availability, this factor could not be controlled in the present study. Lack of control, however, means that it will be less likely that a "smoking" effect will be found, as the other groups will be contaminated by this factor. A better controlled study may therefore find even more significant differences between groups.

An issue that should be addressed in future studies is the role of postnatal passive exposure of the child to cigarette smoke in the home environment. To assess whether prenatal smoking (active or passive) affects children's development independently of postnatal smoking it will be necessary to identify mothers who change their smoking habits after giving birth. It will therefore be important to have a large enough total sample size so that each subgroup would be large enough to allow for reliable comparisons. This was not possible in the current study, because of the 33 children in the Active Smoking group, only 1 was not also exposed to passive smoking postnatally.

Another interesting factor that could be investigated is that of sex differences. Animal models of prenatal nicotine exposure have shown that males show more significant effects than females in terms of reduced birth weight, altered behaviour and neurophysiological changes (Peters, 1984; Peters and Tang, 1982). A recent human study has also shown that the negative effect of maternal smoking on birth weight is more pronounced among males than females (Wertelecki, Hoff and Zansky, 1987). The only long term studies that have considered sex as a factor have been Fogelman (1980) in his examination of growth at age 16 years, and Dunn et al. (1977), who analyzed the results of teacher behaviour ratings for boys and girls separately. Both studies found poorer results for males. It would be of interest to see if this pattern holds true for performance across a test battery.

Replication of these findings with a larger, more representative sample is desirable. The difficulties of maintaining such a sample in a longitudinal prospective study have been discussed previously. An alternative method would be to use a reliable source of prenatal information, such as doctor's records during prenatal examinations, and to follow-up as large a cohort as possible from several large prenatal clinics and obstetrical practices. This method would have the disadvantage of being somewhat retrospective in nature, but would probably allow for a broader socioeconomic background in the sample.

Statistical issues are very difficult to resolve in this area of research. For instance, the issue of statistical versus clinical significance of findings can pose serious problems to the interpretation of findings. The effects found in this study, as in all other related studies, are very small; prenatal smoking accounts for a very small percentage of variance in long-term development. The value of this research, however, lies not in the level of statistical significance of the findings, but rather in the pattern of results that can be replicated across different samples. A small effect size should therefore not in itself be used as reason to discount the relevance of this field for child development. So many factors that negatively influence development are beyond the control of the average citizen, but smoking (both active and passive) is one factor that can be avoided.

The use of covariance procedures to statistically control for potential confounds such as socioeconomic status is controversial. Pedhazur warns that analysis of covariance should not be used to "equate" intact groups in quasi-experimental research (Pedhazur, 1982). Unfortunately, although the problems associated with analysis of covariance procedures are well known, there is yet no accepted statistical remedy when the researcher is working with preexisting groups. Given ethical considerations, it is of course impossible to assign women to treatment groups, so research in this area must work within these constraints.

In a related field of research, that of the neuropsychological effects of lead exposure in children, Winneke and Kraemer (1984) addressed this issue by adopting an interaction model whereby the degree of effect was contrasted between children from socially disadvantaged families and middle-class families. Their results showed that the degree of association between performance deficits and lead exposure was much stronger in socially disadvantaged children. This procedure was similar to the blocking strategy employed in the current study, whereby the sample was divided into two groups, representing high and low SES. Results indicated that, contrary to expectation, children of smokers in the low SES category scored better on several measures than smokers' children in the high SES category. It is important to note, however, that there were only 7 subjects in the high SES category for the Active Smoking group, and so this small number of subjects may not be representative. Given that this analysis was unplanned and post-hoc, rather than drawing conclusions, these results were included to aid further research in the field.

The path analysis procedure has not typically been used in this field, but it has nevertheless demonstrated its usefulness in decomposing relationships between prenatal and postnatal factors. This procedure allows the investigator to go beyond the main effect model of development, whereby a prenatal insult is expected to produce a long-term deficit irrespective of environmental circumstances (Sameroff and Chandler, 1975). Rather than trying to separate the influence of prenatal and postnatal factors, it allows for an analysis of the

interaction between prenatal and environmental factors in predicting developmental outcome.

The limitation of this approach, however, is that it implies a static environmental influence, in that it does not allow for an interplay between the child and the environment. Adopting Sameroff's "transactional" model of development, it is clear that multiple analyses of the reciprocal process of interaction across time are required. This complex pattern of relationships between the environment and development demands more sophisticated methods of analysis. Using data collected within a longitudinal study, perhaps the LISREL (Linear Structural Relations) procedures for path analysis could be used at several points in the child's development. LISREL allows for reciprocal causation within a recursive model of development. (See Sorbom and Joreskog, 1981). This type of analysis would help to integrate knowledge from teratology with the child development literature, and would address the very interesting question of how prenatal and environmental influences transact to influence development.

In order for this approach to be successful, however, a better measure of the home environment is needed. The Moos Family Environment Scale was chosen as the best available, but it proved to have several limitations. It was not popular among the mothers in this study, and many questioned its validity, given their difficulty with the true/false format of response. A Likert scale (e.g. 1 to 5) would be

preferable. Many of the items were also not appropriate for families with small children. Following from the above discussion, the items should also attempt to assess the degree of compensating aspects of parenting that are occurring in the home. Ideally, the inclusion of a naturalistic observation component would also allow for direct observation of parent - child interaction. An improved home environment measure may also eventually prove useful in the investigation of optimal strategies for intervention.

.4 Implications

Before one considers the implications of these results, one must remember the limitations of this study. The sample used in this study is small and largely middle class, and was obtained from volunteers rather than random selection. Generalization to the population as a whole must therefore be made with caution. On the other hand, since significant results were obtained with this generally high-functioning, low-risk sample, it is possible that the effects of prenatal smoking exposure for children from lower status families would be even greater.

The results of this study suggest that the long-term outcome of both passive and active smoking during pregnancy is that exposed children will suffer mild lags in their cognitive and behavioural development, and as a result may not reach their full potential. The effects of prenatal smoking are subtle in nature, and there is no

evidence of a relationship with significant learning disabilities or serious academic failure. As a result, such effects may go undetected in the child's early years. Early childhood educators would therefore be advised to be aware of these findings and to include questions regarding such prenatal exposure in their intake procedures. If exposed children begin to show lags in speech and language development, they should be referred to a Speech Pathologist as soon as possible, as these results indicate that lags in speech and language will probably not disappear with time.

At school-age, when such a child is identified as having learning problems, and when psychoeducational assessments determine that this is related to weaknesses in attention and speech and language skills, the remedial approach to be employed should consider the fact the underlying deficit may stem from the prenatal period and therefore may not respond to the usual stimulation therapy. These children may respond better to a remedial approach that capitalizes on their relative strengths (i.e. visual perception, reasoning, motor speed, etc.). These children may also benefit from the teaching of more efficient strategies for learning, in order to better compensate for underlying weaknesses.

The implications for public policy are clear. Women must be educated regarding the risks of both active and passive smoking during pregnancy. The long-term risks of active smoking have been known for some time, but these results are the first to indicate that similar,

although reduced, risks are also associated with exposure to second-hand smoke during pregnancy. This should add fuel to the current debate regarding "No Smoking" policies in the workplace. The impact of these results will also, hopefully, be to emphasize a sharing of responsibility for the prenatal environment of unborn children. Rather than placing the total burden of such responsibility solely on pregnant mothers, such results can be used to pressure expectant fathers and coworkers to change their smoking habits as well.

The overall results, then, of this study indicate that prenatal exposure to environmental tobacco smoke leads to negative effects which, with very few exceptions, tend in the same direction as those from active maternal smoking. There is now ample evidence of the negative psychological and health-related effects of both passive and active exposure to tobacco smoke, whether pre- or post-natal. The key task for social scientists will be finding the most effective means to communicate the results of such research to the designers of public policy, and to support the development of more effective smoking cessation programs.

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Appendix A: OPPS (Ottawa Prospective Prenatal Study)
Neuropsychological Test Battery

SENSORY TESTS:

Tactile, Auditory and Visual Perception tests
Sensory recognition tests, including Finger Recognition,
Finger-tip Writing and Tactile Form Recognition

MOTOR TESTS:

Lateral Dominance
Finger and Foot Tapping
Maze Test
Progressive Holes Test
Pegboard Test

VISUAL-SPATIAL TESTS:

Developmental Drawings
Target Test

SPEECH AND LANGUAGE TESTS:

Aphasia Screening Test
Fluency Test
Boston Speech Test
Sound Blending
Sentence Memory
Peabody Picture Vocabulary Test

ACADEMIC ACHIEVEMENT:

Wide Range Achievement Test - Revised

PSYCHOMOTOR:

Tactual Performance Test (TPT)

ABSTRACT REASONING:

Category Test

INTELLIGENCE:

McCarthy Scales of Children's
Abilities

BEHAVIOUR:

Conners Parent Questionnaire

ATTENTION:

Gordon Response Inhibition
Gordon Vigilance

Appendix B: Test battery used in current research study

Starred (*) tests are the "new" tests added by the author to the tests retained from the OPPS battery. These tests were given as part of the complete battery, below, and also comprised the "short battery" required for those 52 subjects already tested by the OPPS project.

MOTOR TESTS:

Lateral Dominance examination determines the child's preferred and non-preferred hand, foot and eye.

Pegboard Test is a measure of eye-hand coordination. The child is asked to place keyhole-shaped metal pegs into rows of matching holes. Speed and accuracy are recorded.

VISUAL-SPATIAL TESTS:

Developmental Drawings requires the child to copy eleven stimulus figures. Each figure is drawn twice and the better of the two is scored to criteria for form, size, orientation, distortion.

* Jordan Left-Right Reversal Test is an untimed measure of directionality perception involving letter and number reversals. Raw scores are derived from errors in identifying reversed letters (from an array of 27 upper case letters, 11 of which are reversed) and numerals (5 of 14 reversed).

* Far-Point Copying subtest from the Slingerland Screening Tests for Identifying Children with Specific Language Disability. This untimed test requires the child to copy a paragraph from a chart posted on a wall. There are no norms for this test, and performance is analyzed for quality and type of error (e.g. correctness of letter formation, spelling errors, spacing).

* Draw-a-Man Test. The child is requested to draw a picture of a man. There is no time limit. The drawing is later scored for conceptual maturity using objective criteria for number, position and detail of body parts, clothing, line quality, etc.

SPEECH AND LANGUAGE TESTS:

Sound Blending involves the blending of sounds into words. Sound components of words are presented on an audio tape (e.g. "p - en - c - il"). The child names the word formed by blending the sounds together.

Peabody Picture Vocabulary is a measure of receptive vocabulary. A word is said to the child, who then points to the correct of four pictures on a page.

Test of Language Development - Primary (T.O.L.D.) Four subtests will be used, the first three of which sum to provide a factor score on language syntax:

Grammatical Understanding assesses the child's ability to comprehend the syntactic elements of sentences. The child selects from three pictures the one that most accurately represents the stimulus sentence. (E.g. In response to the stimulus "He had ridden", the child must select from pictures of a boy mounting a horse, a boy riding a horse, and a boy walking away from a horse.)

Grammatical Completion uses a cloze technique to assess the child's ability to understand and use common morphological forms, such as plurals, possessives and past tense. The examiner reads an unfinished sentence and the child supplies the missing word. (E.g. "Joey likes to play. Right now he is _____".)

Sentence Imitation measures the child's ability to imitate grammatically correct sentences. (E.g. In response to the stimulus, "Although she won't play with him, he likes her", the child must repeat the sentence verbatim.)

Word Articulation measures the child's ability to correctly produce speech sounds. Stimulus pictures and questions are used to elicit the spontaneous articulation of common words that contain key speech sounds. (E.g. "scissors").

ACADEMIC ACHIEVEMENT TESTS:

Wide Range Achievement Test - Revised provides subtests for Reading (word recognition), Spelling and Arithmetic. Items are provided from pre-school through to college level.

* Kaufman - Reading Comprehension test presents the child with sentences in which a gestural response is requested. The child is asked to read each sentence (silently) and to "do what the words say". (E.g. "Peel a banana in your hand.")

* Test d'Orthographe Pour Eleves d'Immersion evaluates the written spelling skills of students in early french immersion programs at school.

INTELLIGENCE:

* Wechsler Intelligence Scale for Children - Revised (WISC-R) is a test of general intelligence. Eleven different subtests will be given. Four tests of verbal comprehension include measures of general knowledge, verbal abstract reasoning, oral vocabulary and practical judgement. Four tests of perceptual organization are timed and involve the use of concrete materials such as pictures, blocks and puzzles. Tests sensitive to attention and distractibility include tests of mental arithmetic computation, short-term auditory memory and fine motor speed on a paper-pencil task.

BEHAVIOUR:

Conners Parent Questionnaire is a behavioural symptom checklist, completed by the child's mother. Factor scores in Conduct Problems, Anxiety, Impulsivity-Hyperactivity, Learning Problems, and Psychosomatic Problems are derived by summing item weights.

HOME ENVIRONMENT:

* Family Environment Scale measures the social environmental characteristics of families. The 90 items are naturalistic statements about families and are answered in a true-false manner. (E.g. "We spend most evenings and weekends at home.") The items load onto ten subscales or dimensions:

1. Cohesion - the degree of commitment, help and support family members provide for one another

2. Expressiveness - the degree of encouragement to act openly and to express feelings directly

3. Conflict - the amount of openly expressed anger, aggression and conflict

4. Independence - the extent to which family members are assertive, self-sufficient and make their own decisions

5. Achievement Orientation - the extent to which activities (such as school and work) are cast into an achievement-oriented or competitive framework

6. Intellectual-Cultural Orientation - interest in political, social, intellectual and cultural activities

7. Active-Recreational Orientation - participation in social and recreational activities

8. Moral-Religious Emphasis - emphasis on ethical and religious issues and values

9. Organization - importance of organization and structure in planning activities and responsibilities

10. Control - the extent to which set rules and procedures are used to run family life

Carleton University
Ottawa, Canada K1S 5B6

September 22, 1987

Dear

As you know, Dr. Peter Fried's Ottawa Prenatal Prospective Study has been following the development of children since 1978. Mothers in this study volunteered to participate during their pregnancy, at which time interviews were conducted and questions asked regarding a variety of lifestyle factors. You may not have been contacted lately for follow-up because staffing and budget constrictions have required that we limit the number of children being followed on a regular basis. I am writing at this time to explain my follow-up testing of children in order to solicit your participation in this aspect of the study.

I will be seeing children in Dr. Fried's study for a developmental assessment, including tests of perceptual and language abilities, academic skills and reasoning. These tests are designed to be attractive and interesting to children, and they typically enjoy the testing session. Mothers will receive verbal feedback regarding their child's performance. The testing session will last for about 3 1/2 hours, with time allowed for snacks and breaks when necessary. In addition, I will be asking mothers to complete a short questionnaire and two rating scales.

The testing session can be scheduled during the day, after school, or on the weekend, and will be either at Carleton University or at your home, whatever is most convenient for you.

The importance of such research in furthering our understanding of factors influencing child development cannot be overstated. Your continued interest and cooperation is greatly appreciated.

You will be contacted by phone in the near future to discuss the participation of your child in this study. If you have any questions in the meantime, please call myself (Carleton U. 564-5549; home - 820-5284) or Dr. Fried (564-5697).

Sincerely,

Judy Makin, B.A., B.Ed., M.A.
Doctoral Candidate,
Department of Psychology.

Appendix D: Test Behaviour Observation Guide

A. Attention, Distractability

- ☐ almost impossible to get and hold
- ☐ easily distracted
- ☐ moderately attentive
- ☐ relatively undisturbed by external stimuli
- ☐ oblivious to external stimuli

B. Amount of Motor Activity

- ☐ almost motionless
- ☐ infrequent movement
- ☐ average movement
- ☐ frequent movement
- ☐ extremely active

C. Performance Rate

- ☐ extremely slow
- ☐ slow
- ☐ average speed
- ☐ rapid
- ☐ extremely rapid

D. Manual Dexterity

- ☐ extremely awkward
- ☐ awkward
- ☐ average
- ☐ skillful
- ☐ extremely skillful

E. Cooperation

- ☐ negativistic, uncooperative
- ☐ somewhat uncooperative
- ☐ generally good
- ☐ cooperates readily
- ☐ cooperates enthusiastically

F. Effort Displayed

- ☐ lackadaisical, indifferent
- ☐ works perfunctorily
- ☐ strives for success
- ☐ works diligently
- ☐ expends maximum effort

PARENT QUESTIONNAIRE

date _____

1. Child's Name _____
2. Child's position in family (circle) 1 2 3 4 5 6
3. Grade in school as of Sept., 1987 (circle) K 1 2 3 4 5
4. Present program in school (check)
 - ☐ English (regular)
 - ☐ French Immersion
 - ☐ Bilingual (half day Eng., half Fr.)
 - ☐ Special Education class
5. School history - Please check below if appropriate:
 - ☐ Has switched from French Immersion to English program.
(If so, state when _____)
 - ☐ Has been referred for testing re learning problems.
(Results _____)
 - ☐ Has received remedial help. Grade(s) ____; Subjects _____
 - ☐ Has repeated a grade. (If so, which grade ____)
6. How many days has your child missed from school due to sickness during this school year? _____
7. Current smoking habits of mother of child:
 - ☐ regular smoker (Average # cigarettes per day ____)
 - ☐ former smoker (Year quit smoking 19____)
 - ☐ never smoked
8. Current smoking habits of father of child:
 - ☐ regular smoker (Average # cigarettes/day ____)
 - ☐ former smoker (Year quit smoking 19____)
 - ☐ never smoked

9. Since birth, has your child ever been regularly exposed to second hand smoke (i.e. for at least two hours a day)? Please indicate yes or no for each year since your child's birth:

Age of child	yes (exposed)	no (not exposed)
0 to 1 year		
1 to 2		
2 to 3		
3 to 4		
4 to 5		
5 to 6		
6 to 7		
7 to 8		
8 to 9		

10. Has your child had any major illnesses/accidents requiring hospitalization? ____ If yes, please describe: _____

11. Circle the highest level of education attained by the child's mother:

High School 9 10 11 12 13 College or University 1 2 3 4
Post Graduate M.A. PhD.

12. Occupation of father _____
Occupation of mother _____

13. Annual family income (Check appropriate category) -

☐ under \$10,000	☐ \$30,000 to 39,999
☐ \$10,000 to 14,999	☐ \$40,000 to 49,999
☐ \$15,000 to 19,999	☐ \$50,000 and over
☐ \$20,000 to 29,999	

14. Have there been any recent events in your child's life that could affect his/her performance on the tests? (e.g. death or seperation in the family) ____ If yes, please explain _____

Appendix F: Correlation matrix for dependent and independent variables

Key to variable names:

Dependent Variables

PEGD	Pegboard (dominant)
PEGN	Pegboard (nondominant)
READ	Reading (WRAT)
SPELL	Spelling (WRAT)
ARITH	Arithmetic
SOUND	Sound Blending
PPVT	Peabody Picture Vocabulary
DRAW	Developmental Drawings
MAN	Draw a Man
SYNQ	Syntax Quotient (TOLD)
ARTIC	Articulation
REV	Reversals
VERB	Verbal Conceptualization)
PERC	Perceptual Organization) WISC-R
DIST	Distractability)
BEHAV	Examiner Behaviour Ratings (Total)
COND	Conduct Problems)
LEARN	Learning Problems)
PSYCH	Psychosomatic) Conners Parent
IMP	Impulsivity) Questionnaire
ANX	Anxiety)
HYPER	Hyperactivity)

Independent/ Control Variables

YRED	Maternal Education
FOCC	Father's Occupation
MOCC	Mother's Occupation
INC	Family Income
ZSES	Socioeconomic status
MATAGE	Maternal Age
SL	Follow-up status
EX	Examiner

Appendix F: Correlation matrix for dependent and independent variables

--- PEARSON CORRELATION COEFFICIENTS ---

	PEGD	PEGI	READ	SPELL	ARITH	SOUND	PPVT	DRAG	HAN	STWQ
PEGD	1.0000**									
PEGI	.5660**	1.0000**								
READ	-.0605	.1700	1.0000**							
SPELL	.0377	.2152	.7977**	1.0000**						
ARITH	.0378	.2152	.7977**	.4664**	1.0000**					
SOUND	.1037	.1523	.4598**	.4664**	.0503	1.0000**				
PPVT	.2509*	.0718	.2346	.1806	.1278	.2562*	1.0000**			
DRAG	.0556	.2324	.1313	.2180	.1278	.1613	.0768	1.0000**		
HAN	-.1491	.0064	.1078	.1700	.3311**	.0086	-.1786	.3040*	1.0000**	
STWQ	-.0433	.0464	.3695**	.3077*	.3351**	.2507*	.3331**	.2146	.1562	1.0000**
ARITH	.0096	-.0095	.0640	.0804	-.0560	.0423	.0954	-.0686	.0569	.1711
RIV	.1193	.1875	.2210	.2132	.1790	.0508	-.1367	.1396	-.0512	-.0259
VERB	-.0357	-.0020	.4200**	.3874**	.3413**	.2842*	.4110**	.1092	.1260	.5563**
PLNC	.2273	.2693*	.2587*	.2585*	.3546**	.1426	.1891	.2899*	.2058	.4070**
DIST	.0998	.1325	.4536**	.4683**	.5003**	.0597	.1715	.3035*	.2704*	.3311**
DEHAV	.1480	.3236**	.3539**	.4451**	.4156**	.1175	.0812	.2893*	.1299	.2456
COND	-.0005	.0157	.1137	-.1336	-.1336	-.1912	-.0437	-.2286*	-.1895	-.2897*
LEARN	-.1166	-.0784	-.0516	-.0620	-.1842	-.0679	-.0623	-.2344*	-.1134	-.2603*
PSYCH	-.0712	.0139	-.1170	-.1176	-.0414	-.0105	.0346	.1027	.1332	.1713
IMP	.1555	.0811	-.0668	-.1034	-.1695	-.0563	.0923	-.3137*	-.3333**	-.1985
ANX	-.1115	.0587	.0359	.0895	-.0924	-.0661	.1605	.1102	.0790	-.0764
HYP	.0333	.0674	-.0322	-.1148	-.1773	.0156	.0437	-.2627*	-.2628*	-.2710*
MAIE	-.0907	-.0163	.0911	-.0364	-.1054	-.0272	.0538	-.0349	.0300	-.0570
YRED	-.1908	-.0767	.1717	.0699	.0862	.0515	.1092	.0878	.1648	.1852
FOCC	.0624	.0504	.1620	.1799	.0975	.2328	.0413	.1183	.3366*	.2153
MOCC	-.1470	-.0091	.1050	.1042	.2041	-.0310	-.0152	.1904	.2577	.1529
I-IC	-.1855	-.1374	.1422	.0932	.1304	.0431	-.0145	.0471	.2093	.2181
ZSES	-.1213	-.0595	.1611	.1215	.1198	.0433	.0305	.0894	.2859*	.2207
MATAGE	-.1803	-.1904	.0408	-.0173	-.1019	-.0300	.0809	-.0703	.1623	.1093
SL	-.4291**	-.0120	.3413**	.2630*	.0188	-.0610	-.2853*	-.0356	.1622	.1921
EX	.0593	.0380	.0138	-.0181	.0273	.0044	.1819	-.1342	-.1176	.0704

* - SIGNIF. LE .01

** - SIGNIF. LE .001

(1-TAILED)

--- PEARSON CORRELATION COEFFICIENTS ---

	REHAV	COND	LEARN	PSYCH	IMP	ARTIC	REV	VERB	PERC	DIST
PEGO	.1480	-.0005	-.1166	-.0712	.1555	.0096	.1193	-.0357	.2273	.0998
PEGN	.2336**	.0157	-.0734	.0139	.0811	-.0095	.1075	-.0020	.2693*	.1826
READ	.3339**	-.1137	-.0516	-.1170	-.0663	.0640	.2210	.4200**	.2587*	.4536**
SPELL	.4451**	-.1426	-.0620	-.1176	-.1034	.0304	.2132	.3874**	.2583*	.4683**
ARITH	.4156**	-.1336	-.1342	.0414	-.1695	-.0560	.1790	.3413**	.3546**	.5083**
SOUND	.1175	-.1912	-.0679	-.0105	-.0563	.0423	.0508	.2842*	.1426	.0597
PRVT	.0312	-.0467	-.0623	.0346	.0923	.0954	-.1367	.4110**	.1891	.1715
DRAW	.2893*	-.3286**	-.2544*	.1027	-.3137*	-.0806	.1396	.1092	.2099*	.3035*
MAN	-.1296	-.1395	-.1134	.1332	.3333**	.0369	-.0512	.1260	.2058	.2784*
SINQ	.2456	-.2397*	-.2603*	-.1713	-.1985	.1711	-.0259	.5563**	.4070**	.3311**
ARTIC	.0404	-.0261	.0975	-.1663	.1270	1.0000**	.0814	.0192	.0287	-.1085
REV	.2394*	-.0844	-.2216	-.0906	-.0229	.0314	1.0000**	.0663	.0146	.0248
VERB	.2664**	-.3613**	-.3075*	-.0971	-.2559*	.0192	.0663	1.0000**	.3581**	.4555**
PERC	.3433**	-.2927*	-.3258**	-.0212	-.1429	.0287	.0146	.3581**	1.0000**	.4209**
DIST	.3654**	-.2353	-.3320**	.0332	-.3347**	-.0287	.0248	.4555**	.4209**	1.0000**
REHAV	1.0000**	-.1714	-.2463*	-.0543	-.2407	.0404	.2894*	.2664*	.3438**	.3694**
COND	-.1714	1.0000**	.5349**	.2720*	.5391**	-.0261	-.0844	-.3613**	-.2927*	-.2350
LEARN	-.2463*	.5349**	1.0000**	.1041	.5560**	.0375	-.2213	-.3076*	-.3258**	-.3320**
PSYCH	-.0343	.2720*	.1041	1.0000**	.0779	-.1663	-.0906	-.0371	-.0212	.0332
IMP	-.2407	.5391**	.5560**	.0779	1.0000**	.1270	-.0229	.2559*	.1429	-.3347**
ARTIC	-.0404	.2894*	.2664*	.2754*	.1023	.0328	.0002	-.2704*	-.0337	-.0372
REV	.0814	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
VERB	.0663	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
PERC	.3581**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**
DIST	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**
REHAV	1.0000**	-.1714	-.2463*	-.0543	-.2407	.0404	.2894*	.2664*	.3438**	.3694**
COND	-.1714	1.0000**	.5349**	.2720*	.5391**	-.0261	-.0844	-.3613**	-.2927*	-.2350
LEARN	-.2463*	.5349**	1.0000**	.1041	.5560**	.0375	-.2213	-.3076*	-.3258**	-.3320**
PSYCH	-.0343	.2720*	.1041	1.0000**	.0779	-.1663	-.0906	-.0371	-.0212	.0332
IMP	-.2407	.5391**	.5560**	.0779	1.0000**	.1270	-.0229	.2559*	.1429	-.3347**
ARTIC	-.0404	.2894*	.2664*	.2754*	.1023	.0328	.0002	-.2704*	-.0337	-.0372
REV	.0814	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
VERB	.0663	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
PERC	.3581**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**
DIST	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**
REHAV	1.0000**	-.1714	-.2463*	-.0543	-.2407	.0404	.2894*	.2664*	.3438**	.3694**
COND	-.1714	1.0000**	.5349**	.2720*	.5391**	-.0261	-.0844	-.3613**	-.2927*	-.2350
LEARN	-.2463*	.5349**	1.0000**	.1041	.5560**	.0375	-.2213	-.3076*	-.3258**	-.3320**
PSYCH	-.0343	.2720*	.1041	1.0000**	.0779	-.1663	-.0906	-.0371	-.0212	.0332
IMP	-.2407	.5391**	.5560**	.0779	1.0000**	.1270	-.0229	.2559*	.1429	-.3347**
ARTIC	-.0404	.2894*	.2664*	.2754*	.1023	.0328	.0002	-.2704*	-.0337	-.0372
REV	.0814	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
VERB	.0663	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
PERC	.3581**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**
DIST	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**
REHAV	1.0000**	-.1714	-.2463*	-.0543	-.2407	.0404	.2894*	.2664*	.3438**	.3694**
COND	-.1714	1.0000**	.5349**	.2720*	.5391**	-.0261	-.0844	-.3613**	-.2927*	-.2350
LEARN	-.2463*	.5349**	1.0000**	.1041	.5560**	.0375	-.2213	-.3076*	-.3258**	-.3320**
PSYCH	-.0343	.2720*	.1041	1.0000**	.0779	-.1663	-.0906	-.0371	-.0212	.0332
IMP	-.2407	.5391**	.5560**	.0779	1.0000**	.1270	-.0229	.2559*	.1429	-.3347**
ARTIC	-.0404	.2894*	.2664*	.2754*	.1023	.0328	.0002	-.2704*	-.0337	-.0372
REV	.0814	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
VERB	.0663	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
PERC	.3581**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**
DIST	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**
REHAV	1.0000**	-.1714	-.2463*	-.0543	-.2407	.0404	.2894*	.2664*	.3438**	.3694**
COND	-.1714	1.0000**	.5349**	.2720*	.5391**	-.0261	-.0844	-.3613**	-.2927*	-.2350
LEARN	-.2463*	.5349**	1.0000**	.1041	.5560**	.0375	-.2213	-.3076*	-.3258**	-.3320**
PSYCH	-.0343	.2720*	.1041	1.0000**	.0779	-.1663	-.0906	-.0371	-.0212	.0332
IMP	-.2407	.5391**	.5560**	.0779	1.0000**	.1270	-.0229	.2559*	.1429	-.3347**
ARTIC	-.0404	.2894*	.2664*	.2754*	.1023	.0328	.0002	-.2704*	-.0337	-.0372
REV	.0814	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
VERB	.0663	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
PERC	.3581**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**
DIST	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**
REHAV	1.0000**	-.1714	-.2463*	-.0543	-.2407	.0404	.2894*	.2664*	.3438**	.3694**
COND	-.1714	1.0000**	.5349**	.2720*	.5391**	-.0261	-.0844	-.3613**	-.2927*	-.2350
LEARN	-.2463*	.5349**	1.0000**	.1041	.5560**	.0375	-.2213	-.3076*	-.3258**	-.3320**
PSYCH	-.0343	.2720*	.1041	1.0000**	.0779	-.1663	-.0906	-.0371	-.0212	.0332
IMP	-.2407	.5391**	.5560**	.0779	1.0000**	.1270	-.0229	.2559*	.1429	-.3347**
ARTIC	-.0404	.2894*	.2664*	.2754*	.1023	.0328	.0002	-.2704*	-.0337	-.0372
REV	.0814	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
VERB	.0663	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
PERC	.3581**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**
DIST	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**
REHAV	1.0000**	-.1714	-.2463*	-.0543	-.2407	.0404	.2894*	.2664*	.3438**	.3694**
COND	-.1714	1.0000**	.5349**	.2720*	.5391**	-.0261	-.0844	-.3613**	-.2927*	-.2350
LEARN	-.2463*	.5349**	1.0000**	.1041	.5560**	.0375	-.2213	-.3076*	-.3258**	-.3320**
PSYCH	-.0343	.2720*	.1041	1.0000**	.0779	-.1663	-.0906	-.0371	-.0212	.0332
IMP	-.2407	.5391**	.5560**	.0779	1.0000**	.1270	-.0229	.2559*	.1429	-.3347**
ARTIC	-.0404	.2894*	.2664*	.2754*	.1023	.0328	.0002	-.2704*	-.0337	-.0372
REV	.0814	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
VERB	.0663	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
PERC	.3581**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**
DIST	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**
REHAV	1.0000**	-.1714	-.2463*	-.0543	-.2407	.0404	.2894*	.2664*	.3438**	.3694**
COND	-.1714	1.0000**	.5349**	.2720*	.5391**	-.0261	-.0844	-.3613**	-.2927*	-.2350
LEARN	-.2463*	.5349**	1.0000**	.1041	.5560**	.0375	-.2213	-.3076*	-.3258**	-.3320**
PSYCH	-.0343	.2720*	.1041	1.0000**	.0779	-.1663	-.0906	-.0371	-.0212	.0332
IMP	-.2407	.5391**	.5560**	.0779	1.0000**	.1270	-.0229	.2559*	.1429	-.3347**
ARTIC	-.0404	.2894*	.2664*	.2754*	.1023	.0328	.0002	-.2704*	-.0337	-.0372
REV	.0814	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
VERB	.0663	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
PERC	.3581**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**
DIST	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**
REHAV	1.0000**	-.1714	-.2463*	-.0543	-.2407	.0404	.2894*	.2664*	.3438**	.3694**
COND	-.1714	1.0000**	.5349**	.2720*	.5391**	-.0261	-.0844	-.3613**	-.2927*	-.2350
LEARN	-.2463*	.5349**	1.0000**	.1041	.5560**	.0375	-.2213	-.3076*	-.3258**	-.3320**
PSYCH	-.0343	.2720*	.1041	1.0000**	.0779	-.1663	-.0906	-.0371	-.0212	.0332
IMP	-.2407	.5391**	.5560**	.0779	1.0000**	.1270	-.0229	.2559*	.1429	-.3347**
ARTIC	-.0404	.2894*	.2664*	.2754*	.1023	.0328	.0002	-.2704*	-.0337	-.0372
REV	.0814	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
VERB	.0663	.2894*	.2664*	.3027*	.8349**	.0731	-.1017	-.3261**	-.2378	-.3687**
PERC	.3581**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**
DIST	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**	.4209**	.4555**
REHAV	1.0000**	-.1714	-.2463*	-.0543	-.2407	.0404	.2894*	.2664*	.3438**	.3694**
COND	-.1714	1.0000**	.5349**	.2720*	.5391**	-.0261	-.0844	-.3613**	-.2927*	-.2350
LEARN	-.2463*	.5349**	1.0000**	.1041	.5560**	.0375	-.2213	-.3076*	-.3258**	-.3320**
PSYCH	-.0343	.2720*	.1041	1.0000**	.0779	-.1663	-.0906	-.0371	-.0212	.0332
IMP	-.2407	.5391**	.5560**	.0779	1.0000**	.1270	-.0229	.2559*	.1429	

PEARSON CORRELATION COEFFICIENTS

	ANX	HYPER	NAME	YRED	FOCC	MOCC	INC	ZSES	MATAGE	SL	EX
PECO	-.1115	.0333	-.0907	-.1908	.0624	-.1470	-.1055	-.1213	-.1803	-.4291**	.0593
PEGY	.0587	.0674	-.0163	-.0767	.0904	-.0091	-.1374	-.0699	-.1904	-.0120	.0880
REA	.0359	-.0822	.0911	.1717	.1620	.1030	.1422	.1611	.0408	.3410**	.0138
SPELL	.0895	-.1148	-.0364	.0699	.1799	.1042	.0932	.1213	-.0170	.2630*	-.0181
ARITH	-.0024	.1773	-.1054	.0862	.0975	.2041	.1304	.1198	-.1019	.0188	.0273
SOUND	-.0661	.0156	-.0272	.0515	.2328	-.0310	-.0431	.0433	-.0300	-.0610	.0044
PPVT	-.1605	.0437	.0538	.1092	.0413	-.0132	-.0145	-.0305	-.0809	-.2833*	.1819
DRUH	.1102	-.2627*	-.0349	.0870	.1183	.1904	.0471	.0894	-.0703	-.0356	-.1342
NAV	.0790	-.2626*	.0300	.1648	.3366*	.2577	.2093	.2859*	-.0683	.1622	-.1176
SYNQ	-.0764	-.2710*	-.0570	.1852	.2153	.1529	.2131	.2207	.1093	.1521	.0704
ARTIC	.0328	.0781	.1745	.0760	-.0773	-.0397	-.0601	-.0959	.0373	.0929	.1524
REV	.0002	.1017	-.0376	-.0343	.0771	-.0314	-.0172	-.0131	-.0199	.0680	-.1568
VER3	-.2704*	-.3261**	-.0279	-.2052	.3098*	.1579	.1451	.2070	.1164	.1210	.0095
PERC	.0537	-.2378	-.0772	.2449*	.2757*	.1757	.2145	.2738*	-.0633	-.0605	.1604
DIST	-.0372	-.3687**	-.1208	.2578*	.3436**	.1941	.2598*	.3214*	.0772	.0338	.0245
BEHAV	-.0265	-.2164	.0293	.0492	.0703	.1465	.1025	.1057	-.1346	.1769	-.0033
COUD	.3463**	.6875**	.0105	-.0327	-.1336	-.1316	.1468	.1772	-.0031	-.0737	.1687
LEARN	.2476*	.7629**	.1118	-.0070	-.0691	.0151	-.1374	.1375	.0341	.1000	-.0232
PSYCH	.2754*	.3027*	-.0023	.0840	.0601	.0637	.1424	.1263	-.0698	-.1090	-.0082
IUP	.1023	.8349**	.0776	.1294	-.0292	.1513	-.0984	.1228	-.0713	-.2044	.1193
ANX	1.0000**	.2867*	-.0927	.1084	.0845	.0879	.0233	.0428	.0523	.1010	-.0728
HYPER	.2867*	1.0000**	.0178	-.0804	-.0340	-.1343	-.1703	.784	.0035	-.0741	.1023
NAME	-.0927	.0178	1.0000**	-.0701	.1127	.0143	-.0183	-.0529	.1602	.0907	.0402
YRED	.1034	-.0804	-.0701	1.0000**	.5533**	.6115**	.4408**	.5438**	.2665*	.1990	-.0792
FOCC	.0845	-.0340	-.1127	.5533**	1.0000**	.6657**	.6054**	.8617**	.3649**	.0554	-.3172*
MOCC	.0879	-.1543	.0143	.6115**	.6657**	1.0000**	.5534**	.7422**	.2053	.0003	-.3391*
INC	.0283	-.1703	.0183	.4408**	.6056**	.5344**	1.0000**	.9130**	.2475*	.0700	-.1863
ZSES	.0428	-.1784	-.0329	.5438**	.8617**	.7422**	.9130**	1.0000**	.2015*	.0868	-.2856*
MATAGE	.0525	.0033	.1602	.2665*	.3649**	.2053	.2475*	.3015*	1.0000**	.0669	-.0694
SL	.1010	-.0741	.0907	.1890	.0564	.0083	.0700	.0853	.0669	1.0000**	-.1848
EX	-.0728	.1023	-.0402	-.0792	-.3172*	-.3391*	-.1863	-.2856*	-.0694	-.1848	1.0000**

* - SIGNIF. LE .01

** - SIGNIF. LE .001

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Appendix G: Correlation matrix for Family Environment Scales
and FES1 factor score with dependent and independent variables

Key to variable names:

Family Environment Scale

COH	Cohesion
EXP	Expressiveness
CONF	Conflict
INDEP	Independence
ACH	Achievement Orientation
ICO	Intellectual-Cultural Orientation
ARO	Active-Recreational Orientation
MRE	Moral-Religious Emphasis
ORG	Organization
CONT	Control
FES1	factor score

(Dependent and Independent variables - see Appendix F)

Appendix G: Correlation matrix for Family Environment Scales and FES1 factor score
with dependent variables

	CON	EXP	CONF	INDEP	ACH	ICO	ARO	MRE	ORG	CONT	FES1
PEGD	-.0227	-.0128	-.0632	-.1223	-.0096	-.0547	.0792	.0727	-.0114	.1442	.0014
PECH	.0372	-.0609	-.0613	-.0366	.1313	-.1732	.0839	-.0567	.0222	.0808	-.0399
READ	-.0490	-.0253	-.1066	.0476	-.0455	.0635	-.1238	-.1857	.0418	-.0884	.0159
SPELL	-.0456	-.0079	-.0566	.0799	-.0271	.0297	-.0952	-.2153	.0211	-.1130	.0003
ARITH	.0851	.0326	-.0266	.1591	.2641*	-.0421	.0182	-.0530	.1553	.0147	.0315
SOUND	.0988	-.0658	-.1168	-.1191	-.0023	-.0967	.0627	.0286	-.0303	-.0578	.0779
PPVT	.1867	.0468	-.0843	-.1552	-.0376	.1428	.0349	.0484	-.0056	.0435	.1429
DRAW	.0637	.1975	-.1219	.1069	.0632	.0060	.1067	-.0037	.0985	.1133	.1375
MAN	-.0035	.0132	-.1333	.0314	.0487	.1739	.1054	.0017	.1249	.1333	.1246
SYNQ	.0098	-.0304	-.1042	.0096	.0249	.0666	.0128	-.0602	.1460	-.1841	.0769
ARTIC	.0244	-.0397	.0331	.0877	-.0004	.1425	.0168	.1023	.0604	-.0464	.0514
REV	-.0542	.0955	.0767	-.0913	.2075	.0337	.2236	.0437	.1365	.1346	-.0672
VERB	.0429	-.0049	.0220	.0158	-.0860	.1380	.0169	-.1007	.1573	-.0859	.0968
PERC	.1872	.0423	-.1742	.1086	-.0359	.1380	.1775	.1005	.2416	.1723	.2082
DIST	.1119	-.0621	-.0359	-.0354	.0291	-.0702	.1775	.1200	.1240	-.1106	.1330
BEHAV	-.0262	-.0386	.0833	.0940	-.0028	.0990	.2290	-.1460	-.0071	-.0815	-.0448
COND	-.1482	-.0386	.1856	-.1880	.2137	-.0429	-.1046	.0454	-.1872	.0248	.2377
LEARN	-.0830	.0102	.0350	.0013	.1072	.1158	-.0822	.0441	-.2222	.0434	-.1038
PSYCH	.0794	-.0655	.1941	-.0667	.2788*	-.0187	.0998	.0670	-.0533	.2498*	-.0949
IMP	.0437	-.0501	-.0363	.0048	.1852	-.0254	-.0245	.1333	-.0488	.0489	-.0420
ANX	-.0819	.0231	-.0140	.1253	.0464	.0109	.1433	-.0598	-.0411	.1018	-.0852
HYPER	.0358	-.0826	.0832	-.0612	.2620*	-.0188	-.0418	.1498	-.1376	.1302	-.1513
NAME	-.0483	-.1138	-.0067	-.0021	-.0890	-.0188	-.0302	.0116	.1402	.1011	.0521
YERD	.1634	.0975	-.1827	.0603	-.1135	.1770	-.1416	-.2653*	.0822	-.0693	.2891*
FOCC	.1069	-.0126	-.3119*	-.0867	.0876	.3152*	.2330	-.0532	.2625*	.0794	.2898*
NOCC	.1072	-.1011	-.3836**	.0094	.0847	.3447*	.1782	-.0332	.2148	.0813	.2893*
INC	-.1078	-.1442	-.0253	.0935	.2066	.0628	.0808	-.0582	.1484	.0416	-.0321
ZSES	.0067	-.1102	-.1841	.0246	.1587	.1982	.1623	-.0313	.2152	.0494	.1328
MATAGE	-.1537	-.0900	-.0151	-.0163	.1013	.1392	.1523	-.0911	-.0096	.0015	-.0784
SL	-.1390	-.1179	-.0191	.1077	-.1291	-.0577	-.1211	-.1993	-.0958	-.1648	-.1161
EX	-.1028	.0325	-.1729	-.1421	.0324	-.2946*	.0288	-.0077	-.2069	-.1875	-.2097

- SIGNIF. LE .01 ** - SIGNIF. LE .001 (1-TAILED) " " PRIN

Appendix H: Means, Standard Deviations and ANOVA F for total sample and three groups on
Dependent Variables not selected for further analysis

Variable Name	Total Sample		Valid N	Nonsmoking Group		Passive Smoking		Active Smoking		Univariate F/Signif.
	Mean	S.D.		Mean (n=23)	S.D.	Mean (n=35)	S.D.	Mean (n=33)	S.D.	
Grammatic understanding	11.86	2.26	91	13.00	2.30	11.83	2.36	11.09	2.13	4.84 **
Sentence Imitation	12.27	2.53	90	13.04	2.60	12.43	2.64	11.53	2.34	2.51 +
Grammatic Closure	11.51	2.19	90	12.17	2.21	11.40	2.09	11.16	2.27	1.52
French (EFI) Spelling	29.36	26.55	28	37.00	34.31	17.90	19.73	35.08	27.36	1.46
WISC-R subtests:										
Information	12.46	2.40	90	13.26	2.93	12.49	2.21	11.84	2.17	2.34 +
Similarities	13.72	2.85	90	14.57	3.09	13.34	2.82	13.53	2.70	1.39
Arithmetic	11.65	2.89	89	12.82	2.64	11.23	3.09	11.31	2.81	2.39 +
Vocabulary	13.98	2.59	90	14.70	2.93	13.97	2.47	13.47	2.45	1.51
Comprehension	13.22	2.77	87	13.86	2.92	13.11	2.60	12.87	2.85	0.86
Digit Span	11.47	2.51	90	12.48	2.73	11.20	2.48	11.03	2.38	2.55 +
Picture Completion	12.90	2.77	91	13.30	3.20	12.46	2.45	13.09	2.78	0.77
Picture Arrangement	12.98	2.65	91	13.78	2.73	12.57	2.32	12.82	2.92	1.53
Block Design	13.32	2.70	91	14.39	2.61	12.86	2.85	13.06	2.59	2.48 +
Object Assembly	12.78	2.57	91	13.91	2.84	12.20	2.03	12.61	2.87	3.20 *
Coding	11.24	2.94	91	11.83	2.92	11.14	2.50	10.94	3.35	0.65
Verbal IQ	118.24	13.41	90	123.83	15.45	117.06	12.88	115.53	12.39	2.78 +
Performance IQ	118.33	12.81	91	123.91	12.99	115.51	10.58	117.42	14.70	3.11 *
Full Scale IQ	120.57	12.32	90	126.74	13.37	118.23	11.11	118.69	12.76	3.89 *

** $p < .01$ * $p < .05$ + $p < .10$

Appendix I: Error checklist for scoring Slingerland
Copying Test

<u>Error</u>	<u>Example</u>
letter substitution	"as" for us
letter reversal	"bolls" for dolls
letter or word insertion/omission	"Bobb " for Bobby
letter inversion	"we" for me
mixed capitals and lowercase	"BookS" for books
sequencing of letters	"wnet" for went
spatial orientation on line	"bir th day" for birthday
letter formation/size	"m ^{ade} " for made
omission of punctuation	
spacing between letters/words	"theair" for the air
failure to erase	"books" for books

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FAMILY ENVIRONMENT SCALE

FORM R

RUDOLF H. MOOS

INSTRUCTIONS

Appendix J

There are 90 statements in this booklet. They are statements about families. You are to decide which of these statements are true of your family and which are false. Make all your marks on the separate answer sheets. If you think the statement is True or mostly True of your family, make an X in the box labeled T (true). If you think the statement is False or mostly False of your family, make an X in the box labeled F (false).

You may feel that some of the statements are true for some family members but false for others. Mark T if the statement is true for most members. Mark F if the statement is false for most members. If the members are evenly divided, decide what is the strongest overall impression and answer accordingly.

Remember, we would like to know what your family seems like to you. So do not try to figure out how other members see your family, but do give us your general impression of your family for each statement.

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80. Rules are pretty inflexible in our household.
81. There is plenty of time and attention for everyone in our family.
82. There are a lot of spontaneous discussions in our family.
83. In our family, we believe you don't ever get anywhere by raising your voice.
84. We are not really encouraged to speak up for ourselves in our family.
85. Family members are often compared with others as to how well they are doing at work or school.
86. Family members really like music, art and literature.
87. Our main form of entertainment is watching T.V. or listening to the radio.
88. Family members believe that if you sin you will be punished.
89. Dishes are usually done immediately after eating.
90. You can't get away with much in our family.

- In our family each person has different ideas about what is right and wrong.
- Each person's duties are clearly defined in our family.
- We can do whatever we want to in our family.
- We really get along well with each other.
- We are usually careful about what we say to each other.
- Family members often try to one-up or out-do each other.
- It's hard to be by yourself without hurting someone's feelings in our household.
- "Work before play" is the rule in our family.
- Watching T.V. is more important than reading in our family.
- Family members go out a lot.
- The Bible is a very important book in our home.
- Money is not handled very carefully in our family.

- Family members really help and support one another.
- Family members often keep their feelings to themselves.
- We fight a lot in our family.
- We don't do things on our own very often in our family.
- We feel it is important to be the best at whatever you do.
- We often talk about political and social problems.
- We spend most weekends and evenings at home.
- Family members attend church, synagogue, or Sunday School fairly often.
- Activities in our family are pretty carefully planned.
- Family members are rarely ordered around.
- We often seem to be killing time at home.
- We say anything we want to around home.
- Family members rarely become openly angry.
- In our family, we are strongly encouraged to be independent.
- Getting ahead in life is very important in our family.
- We rarely go to lectures, plays or concerts.
- Friends often come over for dinner or to visit.
- We don't say prayers in our family.
- We are generally very neat and orderly.
20. There are very few rules to follow in our family.
21. We put a lot of energy into what we do at home.
22. It's hard to "blow off steam" at home without upsetting somebody.
23. Family members sometimes get so angry they throw things.
24. We think things out for ourselves in our family.
25. How much money a person makes is not very important to us.
26. Learning about new and different things is very important in our family.
27. Nobody in our family is active in sports, Little League, bowling, etc.
28. We often talk about the religious meaning of Christmas, Passover, or other holidays.
29. It's often hard to find things when you need them in our household.
30. There is one family member who makes most of the decisions.
31. There is a feeling of togetherness in our family.
32. We tell each other about our personal problems.
33. Family members hardly ever lose their tempers.
34. We come and go as we want to in our family.
35. We believe in competition and "may the best man win."
36. We are not that interested in cultural activities.
37. We often go to movies, sports events, camping, etc.
38. We don't believe in heaven or hell.
39. Being on time is very important in our family.
40. There are set ways of doing things at home.
41. We rarely volunteer when something has to be done at home.
42. If we feel like doing something on the spur of the moment we often just pick up and go.
43. Family members often criticize each other.
44. There is very little privacy in our family.
45. We always strive to do things just a little better the next time.
46. We rarely have intellectual discussions.
47. Everyone in our family has a hobby or two.
48. Family members have strict ideas about what is right and wrong.
49. People change their minds often in our family.
50. There is a strong emphasis on following rules in our family.
51. Family members really back each other up.
52. Someone usually gets upset if you complain in our family.
53. Family members sometimes hit each other.
54. Family members almost always rely on themselves when a problem comes up.
55. Family members rarely worry about job promotions, school grades, etc.
56. Someone in our family plays a musical instrument.
57. Family members are not very involved in recreational activities outside work or school.
58. We believe there are some things you just have to take on faith.
59. Family members make sure their rooms are neat.
60. Everyone has an equal say in family decisions.
61. There is very little group spirit in our family.
62. Money and paying bills is openly talked about in our family.
63. If there's a disagreement in our family, we try hard to smooth things over and keep the peace.
64. Family members strongly encourage each other to stand up for their rights.
65. In our family, we don't try that hard to succeed.
66. Family members often go to the library.
67. Family members sometimes attend courses or take lessons for some hobby or interest (outside of school).

3

of/de

3

MicroD

183

T											T
F	1-	2-	3-	4-	5-	6-	7-	8-	9-	10-	F
T											T
F	11-	12-	13-	14-	15-	16-	17-	18-	19-	20-	F
T											T
F	21-	22-	23-	24-	25-	26-	27-	28-	29-	30-	F
T											T
F	31-	32-	33-	34-	35-	36-	37-	38-	39-	40-	F
T											T
F	41-	42-	43-	44-	45-	46-	47-	48-	49-	50-	F
T											T
F	51-	52-	53-	54-	55-	56-	57-	58-	59-	60-	F
T											T
F	61-	62-	63-	64-	65-	66-	67-	68-	69-	70-	F
T											T
F	71-	72-	73-	74-	75-	76-	77-	78-	79-	80-	F
T											T
F	81-	82-	83-	84-	85-	86-	87-	88-	89-	90-	F

[illegible]

FAMILY ENVIRONMENT SCALE

DIRECTIONS

Look at your test booklet and check the Form printed on it here:

Form R ____ E ____ I ____

Please provide the information requested below.

Your Name _____ Age _____

Address _____ Sex: M F
(circle)

Please indicate your position in the family (check one):

Mother (wife) ____ Father (husband) ____ Son or Daughter ____

Other ____ (Please specify): _____

Today's Date _____ Other _____

Now, please read each statement in your booklet and then, in the boxes on the other side of this sheet, mark T (true) if you think the statement is true of your family, and F (false) if the statement is not true of your family.

Use a heavy X, as in the example: Please use a pencil with an eraser, not a pen. Be sure to match each number in the booklet with each one on this sheet.

EXAMPLE ONLY

T	X	
F	1	2 X

Designed by Rudolf H. Moos

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Appendix K: Classification results of Discriminant analysis for total sample (3 groups) 184

Actual group	N	Predicted group					
		Nonsmoking		Passive		Active	
		N	%	N	%	N	%
Nonsmoking	23	14	60.9	6	26.1	3	13.0
Passive Smoking	35	5	14.3	24	68.6	6	17.1
Active Smoking	32	4	12.5	4	12.5	24	75.0

Total percent of cases correctly classified = 68.9%

Crossvalidation Results

Percent of cases correctly classified using 2/3 sample = 69.2%

Percent of cases correctly classified using 1/3 sample = 52.0%

Appendix L: Classification results of Discriminant analysis for Active and Nonsmoking groups

Actual group	N	Predicted group			
		Nonsmoking		Active	
		N	%	N	%
Nonsmoking	23	19	82.6	4	17.4
Active Smoking	32	3	9.4	29	90.6

Total percent of cases correctly classified = 87.3%

Crossvalidation Results

Percent of cases correctly classified using 2/3 sample = 82.5%

Percent of cases correctly classified using 1/3 sample = 86.7%

Appendix M: Classification results of Discriminant analysis for
Passive and Nonsmoking groups

Actual group	N	Predicted group			
		Nonsmoking		Passive	
		N	%	N	%
Nonsmoking	23	18	78.3	5	21.7
Passive Smoking	35	5	14.3	30	85.7

Total percent of cases correctly classified = 82.8%

Crossvalidation Results

Percent of cases correctly classified using 2/3 sample = 81.0%

Percent of cases correctly classified using 1/3 sample = 62.5%

Appendix N: Classification results of Discriminant analysis for
Passive and Active Smoking groups

Actual group	N	Predicted group			
		Passive		Active	
		N	%	N	%
Passive smoking	35	31	88.6	4	11.4
Active Smoking	32	4	12.5	28	87.5

Total percent of cases correctly classified = 88.1%

Crossvalidation Results

Percent of cases correctly classified using 2/3 sample = 92.0%

Percent of cases correctly classified using 1/3 sample = 64.7%

END

0|3·0|3·90

FIN