

THEEIS







١

This is to certify that the

dissertation entitled

INFLUENCES OF MATERNAL DRUG INVOLVEMENT, PSYCHOPATHOLOGY AND PARENTING ON PRESCHOOLERS' DEVELOPMENTAL STATUS, IQ AND BEHAVIOR PROBLEMS presented by

MARIA ZEGLEN TOWNSEND

has been accepted towards fulfillment of the requirements for

PH.D. degree in PSYCHOLOGY

Major profe

Hiram E. Fitzgerald

Date October 9, 1998

MSU is an Affirmative Action/Equal Opportunity Institution

0-12771

PLACE IN RETURN BOX to removê this checkout from your record. TO AVOID FINES return on or before date due. MAY BE RECALLED with earlier due date if requested.

DATE DUE	DATE DUE	DATE DUE
MAR 2 0 2000		•
FEB 0 2 2002		
0 9:2:131 9 2:02 0V 014 8 2005		

1/98 c/CIRC/DateDue.p85-p.14

INFLUENCES OF MATERNAL DRUG INVOLVEMENT, PSYCHOPATHOLOGY AND PARENTING ON PRESCHOOLERS' DEVELOPMENTAL STATUS, IQ AND BEHAVIOR PROBLEMS

By

Maria Zeglen Townsend

A DISSERTATION

Submitted to Michigan State University in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

Department of Psychology

ABSTRACT

INFLUENCES OF MATERNAL DRUG INVOLVEMENT, PSYCHOPATHOLOGY AND PARENTING ON PRESCHOOLERS' DEVELOPMENTAL STATUS, IQ AND BEHAVIOR PROBLEMS

By

Maria Zeglen Townsend

Though many studies have examined the effects of maternal depression, parenting practices, and prenatal drinking and smoking on child development, few have focused on maternal antisociality, and postnatal smoking and drinking. This study examined the effects of maternal drinking and smoking both prenatal and postnatal, depression, antisociality, adaptive functioning, and parenting behaviors and attitudes on preschoolers' developmental status, IQ, and behavior problems. A sample of 307 mothers and their sons participating in wave one of the Michigan State University-University of Michigan Longitudinal Study of children at risk for alcoholism and antisociality due to fathers' diagnosis of these pathologies was utilized.

Both hierarchical regressions and structural equations modeling (SEM) were used. Results of hierarchical regressions showed that mothers who smoked during pregnancy or who are antisocial, depressed or adhered to negative parenting attitudes had children who exhibited more externalizing behavior problems. Mothers who provided stimulating home environments and adhered to positive parenting attitudes had children with higher IQs. A stimulating home environment was also related to more advanced language development and overall developmental quotient.

Two SEMs were run. The results of the child behavior problem model showed that mother's current level of smoking, depression, and adaptive functioning were directly related to externalizing behavior problems with an environment of alcohol being the foremost predictor. An environment of alcohol was defined as the mother's past exposure to family members' alcohol problems and alcoholism within her family of origin, her own past experiences with alcohol problems, and her current drinking levels. For child IQ, it was the amount of stimulation in the home and the mother's positive parenting attitudes that were directly related to child IQ and language development. Mother's current and lifetime drug involvement and psychopathology were directly related to her parenting behaviors and attitudes.

The findings of this study are of interest given the population-based sample and the utilization of SEMs to explore the influences of multiple maternal characteristics on a range of child outcomes. These findings provide a foundation for future research in the areas of maternal alcohol environment and the mediating effects of parenting on the relationships between maternal drug involvement and psychopathologies and child development. This study also adds to the small literature of non-incarcerated, antisocial mothers. There are implications for the investigation of the transmission of maladaptive behaviors from mothers to sons.

FOR MY MOTHER

Who encouraged my desire to excel and fostered in me the skills necessary to succeed not only in school but in relationships with others and as a mother.

FOR MY HUSBAND

Whose unending support and encouragement made it easier for me to complete this research and degree. I am forever thankful that our relationship is one built on love, support and mutual admiration and less on need.

FOR MY CHILDREN

You can do anything you want with perseverance, dedication, hard work, and the support of family and friends. I hope I have and will foster these abilities in both of you and provide each of you with the support to aid you in the pursuit of your dreams.

ACKNOWLEDGEMENTS

There are always many people who lend their expertise, encouragement and support to aid in the completion of a large project. This is even more true when the work was completed long distance.

First, I would like to thank my advisor, Hiram Fitzgerald, who believed in my ability to complete this work long distance. Thank you for being willing to work with me via any means including email, postal mail, and phone calls. Your guidance and mentoring style allowed me to develop independence as a researcher and sharpen my professional writing skills. I am also grateful for the opportunity to expand my doctoral plan of study with an emphasis on Infant Studies through the IGSIS program.

Second, I would like to thank my second reader, Robert Zucker, whose insight provided the impetus for hypothesis five and, I would say, the more interesting results of this study. With your insistence I embarked on learning Structural Equation Modeling and in so doing have expanded my skills as a researcher.

I would also like to thank both Hi and Bob for the opportunity to work on a longitudinal study. By working on the Michigan State University-University of Michigan Longitudinal Study, I was able to work with a variety of people in many different ways. I learned skills in data management, training of undergraduate students, supervisory skills, and most importantly the ability to work with a number of people of various status on a project. All of these skills will serve me well in my professional career. Also, thank you both for allowing me to bring my daughter, Sara, to work and meetings. It was an incredible opportunity for which I am very grateful.

v

I would also like to thank the other members of my committee. First, Tom Luster, who served on both my masters and doctoral committees. Thank you for your insights, comments, and editorial changes. You have always encouraged me to strive to do my best and produce a higher quality study. To Ellen Strommen, whose comments have spurred me to be clearer in both my thinking and writing. Finally, to Alex von Eye, whose statistical expertise served me well in my pursuit of both understanding and completing the structural equations modeling. I am grateful for your willingness to work with a student you had never met and to do so long distance. Your vast knowledge of statistics and ability to make one feel at ease made a very frustrating time into a rewarding learning experience. I would also like to say that it was a pleasure to work with my committee as a whole and I appreciate everyone's efforts in helping me to reach my goal of finishing, despite the long distance.

Because I did this dissertation long distance, there are many people who helped me at Michigan State who need to be thanked and recognized. Ray Bingham, the data manager who fielded my questions on the data and supported me with his friendship. To Alex Loukas, who answered my questions when Ray left and also supported me with her friendship. To other members of the MSU-UM study including Leon Puttler, Sally Gardener, Susan Refior, and other staff members who answered my questions as well as gathered and sent me codebooks, information and data. Lastly, I would like to thank Suzy Pavick, the Psychology Graduate Secretary who helped me to stay on top of the requirements and paperwork needed to finish and graduate.

I can honestly say that my time in graduate school will always hold a special place in my heart for two reasons. It was during this time that Ed and I became "us" and

vi

built a wonderful foundation for our future together. I will fondly remember living on \$25.00 a week for groceries which included my "favorites" pick of the chic chicken and Kraft macaroni and cheese, our one bedroom furnished apartment, Pot Luck Easters for 15 or more friends in the one bedroom apartment, our first house we rented including the annual Fall migration of mice, Friday nights, Sara, and all those other times we shared that are too numerous to mention.

Secondly, I will remember this time because of the long lasting friendships I created and have still maintained (thanks to email and Bell Atlantic). To Kurt and Tracy who Ed and I shared many happy times including the day and night at the hospital when Sara was born. Thank you for being our "family" during one of the most special times in our lives. To Laura Pence for her friendship and support. To many of the others including Kris Kurtz, Jason and Sue Rouse, Tim McCarthy, Jeff and Berly Gilbert, Steve and Kelly Medlin, as well as Karen and Jon Wall. I will fondly remember tailgating, football and basketball games, our annual Ski trips, the Halloween party, the many bars including but not limited to Rocky's (thanks Berly for dancing on the bar with me), line dancing on the seats at Mac's, Amaretto Sours and darts at Moriarty's, and dinners at Macri's.

To my friends from Psychology not already mentioned. To AJ Alejano for your friendship, encouragement, for being my exercise partner after Sara was born, and for going through comps with me. I am glad we were able to be there for each other. To Betty Lafan for your friendship, for being my exercise partner when I was pregnant, and for supporting my decision to bring Sara to work and for helping make it possible. To

vii

THBBIS





1293 01766 8033

This is to certify that the

dissertation entitled

INFLUENCES OF MATERNAL DRUG INVOLVEMENT, PSYCHOPATHOLOGY AND PARENTING ON PRESCHOOLERS' DEVELOPMENTAL STATUS, IQ AND BEHAVIOR PROBLEMS presented by

MARIA ZEGLEN TOWNSEND

has been accepted towards fulfillment of the requirements for

PH.D. degree in PSYCHOLOGY

Major prof

Hiram E. Fitzgerald

Date _____0ctober 9, 1998

MSU is an Affirmative Action/Equal Opportunity Institution

0-12771

PLACE IN RETURN BOX to remove this checkout from your record. TO AVOID FINES return on or before date due. MAY BE RECALLED with earlier due date if requested.

DATE DUE	DATE DUE	DATE DUE
MAR 2 0 2000		
FEB 0 2 2002		
0 912:131 9 2002 0 V 01 8 2005		
		1/98 c/CiRC/DateDus.p85-

INFLUENCES OF MATERNAL DRUG INVOLVEMENT, PSYCHOPATHOLOGY AND PARENTING ON PRESCHOOLERS' DEVELOPMENTAL STATUS, IQ AND BEHAVIOR PROBLEMS

By

Maria Zeglen Townsend

A DISSERTATION

Submitted to Michigan State University in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

Department of Psychology

ABSTRACT

INFLUENCES OF MATERNAL DRUG INVOLVEMENT, PSYCHOPATHOLOGY AND PARENTING ON PRESCHOOLERS' DEVELOPMENTAL STATUS, IQ AND BEHAVIOR PROBLEMS

By

Maria Zeglen Townsend

Though many studies have examined the effects of maternal depression, parenting practices, and prenatal drinking and smoking on child development, few have focused on maternal antisociality, and postnatal smoking and drinking. This study examined the effects of maternal drinking and smoking both prenatal and postnatal, depression, antisociality, adaptive functioning, and parenting behaviors and attitudes on preschoolers' developmental status, IQ, and behavior problems. A sample of 307 mothers and their sons participating in wave one of the Michigan State University-University of Michigan Longitudinal Study of children at risk for alcoholism and antisociality due to fathers' diagnosis of these pathologies was utilized.

Both hierarchical regressions and structural equations modeling (SEM) were used. Results of hierarchical regressions showed that mothers who smoked during pregnancy or who are antisocial, depressed or adhered to negative parenting attitudes had children who exhibited more externalizing behavior problems. Mothers who provided stimulating home environments and adhered to positive parenting attitudes had children with higher IQs. A stimulating home environment was also related to more advanced language development and overall developmental quotient.

Two SEMs were run. The results of the child behavior problem model showed that mother's current level of smoking, depression, and adaptive functioning were directly related to externalizing behavior problems with an environment of alcohol being the foremost predictor. An environment of alcohol was defined as the mother's past exposure to family members' alcohol problems and alcoholism within her family of origin, her own past experiences with alcohol problems, and her current drinking levels. For child IQ, it was the amount of stimulation in the home and the mother's positive parenting attitudes that were directly related to child IQ and language development. Mother's current and lifetime drug involvement and psychopathology were directly related to her parenting behaviors and attitudes.

The findings of this study are of interest given the population-based sample and the utilization of SEMs to explore the influences of multiple maternal characteristics on a range of child outcomes. These findings provide a foundation for future research in the areas of maternal alcohol environment and the mediating effects of parenting on the relationships between maternal drug involvement and psychopathologies and child development. This study also adds to the small literature of non-incarcerated, antisocial mothers. There are implications for the investigation of the transmission of maladaptive behaviors from mothers to sons.

FOR MY MOTHER

Who encouraged my desire to excel and fostered in me the skills necessary to succeed not only in school but in relationships with others and as a mother.

FOR MY HUSBAND

Whose unending support and encouragement made it easier for me to complete this research and degree. I am forever thankful that our relationship is one built on love, support and mutual admiration and less on need.

FOR MY CHILDREN

You can do anything you want with perseverance, dedication, hard work, and the support of family and friends. I hope I have and will foster these abilities in both of you and provide each of you with the support to aid you in the pursuit of your dreams.

ACKNOWLEDGEMENTS

There are always many people who lend their expertise, encouragement and support to aid in the completion of a large project. This is even more true when the work was completed long distance.

First, I would like to thank my advisor, Hiram Fitzgerald, who believed in my ability to complete this work long distance. Thank you for being willing to work with me via any means including email, postal mail, and phone calls. Your guidance and mentoring style allowed me to develop independence as a researcher and sharpen my professional writing skills. I am also grateful for the opportunity to expand my doctoral plan of study with an emphasis on Infant Studies through the IGSIS program.

Second, I would like to thank my second reader, Robert Zucker, whose insight provided the impetus for hypothesis five and, I would say, the more interesting results of this study. With your insistence I embarked on learning Structural Equation Modeling and in so doing have expanded my skills as a researcher.

I would also like to thank both Hi and Bob for the opportunity to work on a longitudinal study. By working on the Michigan State University-University of Michigan Longitudinal Study, I was able to work with a variety of people in many different ways. I learned skills in data management, training of undergraduate students, supervisory skills, and most importantly the ability to work with a number of people of various status on a project. All of these skills will serve me well in my professional career. Also, thank you both for allowing me to bring my daughter, Sara, to work and meetings. It was an incredible opportunity for which I am very grateful.

v

I would also like to thank the other members of my committee. First, Tom Luster, who served on both my masters and doctoral committees. Thank you for your insights, comments, and editorial changes. You have always encouraged me to strive to do my best and produce a higher quality study. To Ellen Strommen, whose comments have spurred me to be clearer in both my thinking and writing. Finally, to Alex von Eye, whose statistical expertise served me well in my pursuit of both understanding and completing the structural equations modeling. I am grateful for your willingness to work with a student you had never met and to do so long distance. Your vast knowledge of statistics and ability to make one feel at ease made a very frustrating time into a rewarding learning experience. I would also like to say that it was a pleasure to work with my committee as a whole and I appreciate everyone's efforts in helping me to reach my goal of finishing, despite the long distance.

Because I did this dissertation long distance, there are many people who helped me at Michigan State who need to be thanked and recognized. Ray Bingham, the data manager who fielded my questions on the data and supported me with his friendship. To Alex Loukas, who answered my questions when Ray left and also supported me with her friendship. To other members of the MSU-UM study including Leon Puttler, Sally Gardener, Susan Refior, and other staff members who answered my questions as well as gathered and sent me codebooks, information and data. Lastly, I would like to thank Suzy Pavick, the Psychology Graduate Secretary who helped me to stay on top of the requirements and paperwork needed to finish and graduate.

I can honestly say that my time in graduate school will always hold a special place in my heart for two reasons. It was during this time that Ed and I became "us" and

vi

built a wonderful foundation for our future together. I will fondly remember living on \$25.00 a week for groceries which included my "favorites" pick of the chic chicken and Kraft macaroni and cheese, our one bedroom furnished apartment, Pot Luck Easters for 15 or more friends in the one bedroom apartment, our first house we rented including the annual Fall migration of mice, Friday nights, Sara, and all those other times we shared that are too numerous to mention.

Secondly, I will remember this time because of the long lasting friendships I created and have still maintained (thanks to email and Bell Atlantic). To Kurt and Tracy who Ed and I shared many happy times including the day and night at the hospital when Sara was born. Thank you for being our "family" during one of the most special times in our lives. To Laura Pence for her friendship and support. To many of the others including Kris Kurtz, Jason and Sue Rouse, Tim McCarthy, Jeff and Berly Gilbert, Steve and Kelly Medlin, as well as Karen and Jon Wall. I will fondly remember tailgating, football and basketball games, our annual Ski trips, the Halloween party, the many bars including but not limited to Rocky's (thanks Berly for dancing on the bar with me), line dancing on the seats at Mac's, Amaretto Sours and darts at Moriarty's, and dinners at Macri's.

To my friends from Psychology not already mentioned. To AJ Alejano for your friendship, encouragement, for being my exercise partner after Sara was born, and for going through comps with me. I am glad we were able to be there for each other. To Betty Lafan for your friendship, for being my exercise partner when I was pregnant, and for supporting my decision to bring Sara to work and for helping make it possible. To

vii

Susan Wyssman for your friendship as well as for being understanding and helping with Sara when she was at work with us.

To the friends I made when Ed switched to the Ledford group. To Greg Noonan for being a special friend to all of us including my daughter Sara and for being at MSU to help me celebrate my defense. To Per Askeland for your friendship, for watching our cat Moses when we went out of town, for buying the MSU watermark paper for this dissertation, and for doing the leg work to get the final copy to University. To Kathy Severin for the use of your house during the times we visited MSU since moving away. You have made some very stressful times, (Ed's defense and both of my defenses) less stressful by providing us with your home, hospitality, and most importantly, your friendship.

To my friends and others here in the Pittsburgh area who have helped me to finish. To Keith Borden, who lent me his Power Mac for over a year so I could run EQS for structural equations modeling. To Dr. Cathy Ryan, who gave me access to EQS even though she did not know me. To the anonymous library workers at Western Psychiatric Clinic, Falk Medical Library, and University of Pittsburgh who gave me access to Psychlit and Medline and allowed me to check out books even though I was not a student. For my two wonderful babysitters, Shannon Hilliard and Kathleen Haughney, who entertained and cared for my children so I could have a few more hours a day during the summers (other than naps and the middle of the night) to work on this dissertation. To Sharon Elliot, Mark Fair, and Kim McLoughin who let me practice my presentation for the defense. Your comments and suggestions were very helpful in making the actual defense relatively easy (so to speak). To my friends not already mentioned who gave me

viii

encouragement and support to finish; Mike Elliot, Kurt Wolske, Pat McLoughin, Jim and Doris Birdsong, Pam Lees, and friends from our church.

I would also like to thank my mother for the times when she helped with Sara and Teddy during my comps and while writing this dissertation. Thanks also go to my sister Michele and her husband John, my brother Joe and his wife Amy, my husband's parents, and his brother Kevin and his wife Mary for their encouragement and support.

Most importantly I would like to thank the members of my family of procreation. To my husband, Ed; your support, encouragement (at times prodding), document formatting, and assistance has made doing this dissertation much easier and allowed me to finish while still being a good mom to our children. I can not thank you enough for helping make this happen. I will always be grateful that our relationship is such that we encourage each other to grow as individuals and yet we maintain "us". To my daughter Sara, who allowed mom to work on her research at times instead of playing. To Teddy (who is now 17 months old) who amazingly slept through the night since coming home from the hospital which allowed me to stay up at night to work and still get a few hours of sleep. I am glad I had the three of you to keep this time of my life balanced and to remind me of the important things in life.

In closing, I would like to say a few things for the benefit of a group of my graduate school friends.

"Imagine the psychological ramifications" of all of this as you are reading this dissertation. To Tracy and all, "Hugs and Sunshine". To Ed, now you can finally "Take me home and ...!!"

ix

TABLE OF CONTENTS

LIST OF TABLESxiv	/
LIST OF FIGURES	vi
CHAPTER 1	
IDENTIFICATION OF THE PROBLEM	
Introduction1	
Purpose of Study1	
Rationale2	
Behavior Problems2	
Developmental Status and IQ	
CHAPTER 2	
REVIEW OF LITERATURE	
Introduction	
Developmental Systems Theory	
Prenatal Influences	
Prenatal Exposure to Alcohol11	
Physiological Effects of Alcohol on Mother and Fetus	
Prenatal Alcohol Exposure, Developmental Status and IQ	
Prenatal Alcohol Exposure and Behavior Problems	
Prenatal Exposure to Smoking25	
Physiological Effects of Smoking on Mother and Fetus	
Prenatal Smoking, Developmental Status and IQ	
Prenatal Smoking and Behavior Problems	
Summary of Research on Prenatal Exposure and Child Development 36	
Postnatal I: Maternal and Familial Influences	
Maternal Psychopathology and Drug Involvement	
Maternal Depression, Developmental Status and IQ42	
Maternal Depression and Behavior Problems	
Maternal Antisociality and Behavior Problems	
Maternal Smoking and Child Development	
Maternal Alcohol Abuse and Behavior Problems53	
Additional Maternal and Familial Postnatal Influences	
on Child Development	
Summary of Maternal and Familial Influences during the Postnatal	
Period on Child Development 57	

Postnatal II: Parenting Influences	57
Parenting Influences on Developmental Status and IQ	58
Parenting Influences on Behavioral Problems	60
The Present Research	62
Research Objectives	62
Hypotheses	63
Discussion of Review of Literature	

CHAPTER 3 METHOD

ME	TI	H	D	D
••••				

Research Objectives and Hypotheses	72
Subjects	
Demographic Characteristics	73
Data Collection	
Measures	74
Maternal Independent Measures	76
Past Smoking and Alcohol Use	76
Smoking and Alcohol Use Prenatally	77
Current Smoking and Alcohol Use	78
Antisociality	78
Depression	79
Adaptive Functioning	80
Intelligence	80
Level of Education	
Familial Independent Measures	
Family Expression of Alcoholism	81
Family Socioeconomic Status	81
Parenting Measures	
Parenting Attitudes	82
Parenting Behaviors/Environmental Stimulation	83
Child Outcome Measures	83
Behavior Problems	83
Developmental Status	85
IQ	85
Attention Span/Distractibility	
Impulsivity	
Limitations of Sampling Design and Data Collection Techniques	
Data Preparation and Statistical Analyses	
Cleaning of the Data	
Probability Level, Power Analysis and Statistical Analyses	

CHAPTER 4 RESULTS

ESULIS	
Hypothesis One: Prenatal Influences on Child Development	
Prenatal Influences on child Behavior Problems	90
Externalizing Behavior Problems	90
Summary of Analyses of Hypothesis One: Prenatal Influences	
on Child Development	95
Hypothesis Two: Postnatal Influences on Child Development	
Postnatal Influences on Child Behavior Problems	96
Externalizing Behavior Problems	96
Internalizing Behavior Problems	101
Total Behavior Problems	101
Summary of Analyses of Hypothesis Two: Postnatal Influences	
on Child Development	103
Hypothesis Three: Parenting Influences on Child Development	
Parenting Influences on Developmental Status and IQ	
Parenting Influences on Developmental Status	104
Parenting Influences on IQ	
Parenting Influences on Child Behavior Problems	110
Externalizing Behavior Problems	
Total Behavior Problems	
Mother's Rating of Child's Attention Level	112
Summary of Analyses of Hypothesis Three: Parenting Influences	
 on Child Development 	112
Hypothesis Four: Prenatal and Postnatal Maternal Consumption	
Influences on Child Development	114
Prenatal and Postnatal Consumption Influences on	
Child Behavior Problems	118
Impulsivity	
Summary of Analyses of Hypothesis Four: Maternal Consumption	
Influences on Child Development	118
Hypothesis Five: Life History and Current Levels of Maternal Drug	
involvement and Psychopathology Influences on Child Developme	
Measurement Models	
Child Behavior Problems	124
Child IQ	
Summary of Results of Hypothesis Five: Life History and Current	
Levels of Maternal Drug involvement and Psychopathology	
Influences on Child Development	142

CHAPTER 5

DISCUSSION	
Introduction	143
Environment of Alcohol	143
Direct and Mediating Effects of Parenting Behaviors and Attitudes	148

	rections
Appendix A:	Missing Data Estimation157
Appendix B: S	Sample Results of Mothers and Fathers SEMs
Appendix C:	Non-trimmed Solutions for Child Behavior Problems and IQ Models
Appendix D:	Post-hoc Analysis for Child Behavior Problems Model165
Bibliography	

List of Tables

Table 1	Sociodemographic Characteristics of the Sample $(N = 307)$	74
Table 2	Variables of Interest for Present Research.	75
Table 3	Bivariate Correlations, Means, and Standard Deviations for Prenatal and Outcome Variables Used in Hypothesis One Analyses	91
Table 4	Externalizing Behavior Problems Regressed on Prenatal Variables, Standard Coefficients (Betas)	94
Table 5	Bivariate Correlations, Means, and Standard Deviations for Postnatal and Outcome Variables Used in Hypothesis Two	
Table 6	Analyses Externalizing Behavior Problems Regressed on Postnatal Variables,	97
Table 7	Standard Coefficients (Betas) Internalizing Behavior Problems Regressed on Postnatal Variables,	100
	Standard Coefficients (Betas)	102
Table 8	Total Behavior Problems Regressed on Postnatal Variables,	
Table 9	Standard Coefficients (Betas) Bivariate Correlations, Means, and Standard Deviations for Parenting and Outcome Variables Used in Hypothesis Three	102
	Analyses	105
Table 10	Developmental Quotient Regressed on Parenting Variables, Standard Coefficients (Betas)	108
Table 11	Fine Motor Development Regressed on Parenting Variables,	
Table 12	Standard Coefficients (Betas) Language Development Regressed on Parenting Variables, Standard Coefficients (Betas)	108 109
Table 13	Stanford IQ Regressed on Parenting Variables, Standard	
Table 14	Coefficients (Betas) Externalizing Behavior Problems Regressed on Parenting Variables,	111
Table 15	Standard Coefficients (Betas) Total Behavior Problems Regressed on Parenting Variables,	111
	Standard Coefficients (Betas)	113
Table 16	Mother's Rating of Child's Attention Level Regressed on Parenting Variables, Standard Coefficients (Betas)	112
Table 17	Bivariate Correlations, Means, and Standard Deviations for Prenatal and Postnatal Maternal Consumption and Outcome Variables Used	113
Table 18	in Hypothesis Four Analyses Impulsivity Regressed on Prenatal and Postnatal Maternal	115
m 11 46	Consumption Variables, Standard Coefficients (Betas)	119
Table 19	Bivariate Correlations, Means, and Standard Deviations for	101
Table 20	Hypothesis Five Analyses Solution for Child Behavior Problems Model	121 128
1 4010 20		120

Table 21	Comparison of High and Low Lifetime Drug Involvement and	
	Psychopathology to High and Low Current Drug Involvement and	
	Psychopathology Among Those Mothers' with High Levels of	
	Alcohol Pervasiveness	132
Table 22	Comparison of Medium and High Alcohol Pervasiveness to Low	
	and High Current Drug Involvement and Psychopathology	132
Table 23	Solution for Child IQ Model	137
Table 24	Comparison of Low, Medium, and High Current Drug Involvement	
	and Psychopathology to Low, Medium, and High Levels of	
	Parenting Behaviors and Attitudes Among Those Mothers' with	
	High Levels of Lifetime Drug Involvement and Psychopathology	140
Table 25	Frequencies and Percent of Valid Variables in Sample	158
Table 26	Bivariate Correlations for the Latent Constructs of Paternal (P) and	
	Maternal (M) Lifetime Psychopathologies (F2) and Paternal and	
	Maternal Drug Involvement (F3)	160
Table 27	Lagrange Multiplier Test (for adding parameters)	160
Table 28	Non-trimmed Solution for Child Behavior Problems Model	162
Table 29	Non-trimmed Solution for Child IQ Model	163
Table 30	Child Behavior Problems Model: Solution for Testing the Veracity	
	of Relationship between Alcohol Pervasiveness and Current Drug	
	Involvement and Psychopathology	165

LIST OF FIGURES

Figure 1	Hypothetical measurement model for child behavior problems and impulsivity	125
Figure 2	Child behavior problems and impulsivity standardized solution	
Figure 3	Hypothetical measurement model for child IQ and language	
	development	134
Figure 4	Child IQ and language development standardized solution	136

CHAPTER 1

Identification of the Problem

Introduction

Development is the outcome of interactive relationships between a person and his/her environment (Bronfenbrenner, 1989). These relationships take place within many contexts and at different levels of interaction including within the person, between persons in the environment, and between the person and the social/cultural context. Though there are many contexts, the central context is the family, especially for a preschooler who has limited exposure to other contexts beyond possibly a day care/preschool, community/friends, and extended family (Bronfenbrenner, 1986). Therefore, in order to understand the influences on preschoolers' development, the context of the family must be examined, and in particular, maternal influences. Though research has shown that fathers uniquely influence development in their children (Lamb, 1975; Townsend, Bingham, Loukas, & Piejak, 1995), mothers still perform the majority of child care activities with the result that preschoolers spend more time with their mothers than their fathers (McBride & Mills, 1993). By measuring various maternal and some paternal and familial characteristics of the preschoolers' families as well as child characteristics, one is able to measure the intraindividual, interindividual, and social/cultural (contextual) influences on children's development.

Purpose of Study

The purpose of the present study was to examine the influences of maternal characteristics and behaviors on child development. The following maternal variables:

past, prenatal, and present alcohol use and smoking, depression, antisociality, intelligence, and level of education, two familial variables: family expression of alcoholism and family socioeconomic status, and two parenting variables: parenting behaviors (environmental stimulation) and attitudes, were used to predict child behavior problems, developmental status, and IQ.

Rationale

Behavior Problems

Behavior problems in preschoolers have been shown to be related to continued behavior problems in school-aged children and the possibility of continued maladaptive behavior through adulthood (Egeland, Kalkoske, Gottesman, & Erickson, 1990). In a review of retrospective and prospective studies of hyperactive children, a relationship between early childhood diagnosis of Attention Deficit Hyperactivity Disorder (ADHD) and later diagnosis of conduct disorder and exhibition of aggressive behaviors in adolescence was identified (Thorley, 1984). This combination of early ADHD with conduct disorder and aggressive behaviors in adolescence was found to be highly related to alcohol and drug use in adolescence and adulthood (Pihl & Peterson, 1991). Based on these past studies, there is a link between child behavior problems, ADHD, and later aggressive behaviors and alcohol and drug use. This link is even stronger when the child lives with a mother who has the following characteristics or psychopathologies: depression, antisociality, and drug and alcohol use (Egeland et al., 1990; Ham, Fitzgerald, & Zucker, 1993a; Pihl & Peterson, 1991).

Some of the children participating in the Michigan State University - University of Michigan Longitudinal Study (Zucker & Fitzgerald, 1994; Zucker & Fitzgerald, 1996;

Zucker, Noll, & Fitzgerald, 1986) have mothers with these characteristics and are at risk for developing antisocial personalities and abusing alcohol. By examining the earlier intraindividual, interindividual, and social/cultural (contextual) influences on preschoolers' behavior problems, one could later identify future targets of intervention with the mothers, families, and children in order to change the possible trajectory of development identified in research.

Developmental Status and IQ

Developmental delays in specific areas during one age are related to delays or difficulties during later ages. The educational system operates on the idea that moderate delays in fine and gross motor, language, and social/adaptive behaviors are evidence that children are not ready to begin school with same age peers (Crnic & Lamberty, 1994). The premise is that these children are not ready for school because they do not have the basic skills necessary to learn kindergarten-level materials and they will need additional stimulation and assistance to meet the requirements.

Not only are delays in specific areas related to continued delays within the same area but also in other areas. Lower levels of cognitive development as measured by preschool tests are related to lower levels of IQ and school performance during the school years (Hale, McKay, & Neale, 1986). Delays in language development during infancy and the preschool years are related to later learning problems, lower levels of intelligence, and more behavior problems (Lockwood, 1994; Benasich, Curtiss, & Tallal, 1993; Silva, 1983). Even when early language deficits are later reduced, there is an increased chance of developing reading problems during the elementary school years (Scarborough & Dobrich, 1990).

Children who are experiencing difficulties in school are at risk for being "held back" in school. With each grade retention, there is an increase risk for school drop out, delinquency, and behavior problems (USDE, 1986). Dropping out is related to increased poverty, drug abuse, teen pregnancy, and need for social services (USDE, 1986). As previously mentioned, children in the MSU -UM Longitudinal Study are at risk for alcoholism and antisociality based on paternal alcoholism and antisociality. Developmental delays could be the source of an increase in the level of risk for these children. Identification of delays and possible areas of difficulties experienced by this group of children could be used to develop future interventions to address these areas and minimize the risks related to the delays.

CHAPTER 2

Review of Literature

Introduction

The literature review is presented in five main sections. In the first section, Developmental Systems Theory is discussed and will provide a conceptual framework for organizing the multiple influences on child outcomes of interest to this study. The second section examines the effects of maternal smoking and drinking during pregnancy on preschoolers' developmental status, IQ, and behavior problems. The third section considers the effects of a number of contemporaneous, maternal influences (present smoking and alcohol use, depression, antisociality, level of education, and intelligence), as well as familial influences (family expression of alcoholism and socioeconomic status) on children's developmental status, IQ, and behavior problems. The fourth section examines the influence of parenting behaviors and attitudes on child developmental status, IQ, and behavior problems during the postnatal period. The fifth section discusses the present research in terms of the reviewed literature. For sections two through four, the literature will be presented first for the influences on developmental status and IQ followed by child behavior problems.

Developmental Systems Theory

With a fifty year history rooted in biology, general systems theory, and researchers such as C. H. Waddington and Ludwig von Bertalanffy, a new theory, developmental systems theory (Ford & Lerner, 1992; Miller, 1978; Sameroff, 1983) was created to better meet the challenge of explaining and operationalizing the complex

processes of human development. In so doing, developmentalists have produced a theory that is useful in both conceptualizing and researching human development.

Developmental systems theory views development as "emergent, epigenetic, systemic, organized, constructive, hierarchically integrated, and potentially chaotic" (pp. 8-9, Fitzgerald, Zucker, & Yang, 1995). From conception throughout the lifespan, the person is constantly changing and developing. New structures (behavior episodes schemata), adaptive behaviors (behavior episodes), and adaptive functioning come into existence and are used to create, maintain, and reestablish organization and equilibrium within the system (Ford & Lerner, 1992). This system consists of the person including all of his/her components, such as genes, physical states, and psychological states and the person's environment including the family, neighborhood, school, work, and community/society (Lerner, 1991; Miller, 1978).

These components or subsystems are hierarchically integrated with the interactions or coactions between the subsystems being characterized as reciprocal, occurring both horizontally (within the same level) and vertically (between different levels) and bi-directional, influencing each subsystem in both directions from higher to lower and lower to higher levels (Gottlieb, 1991; Lerner, 1991; Miller, 1978). These interactions between the organism and the environment as well as within the organism itself are seen as having three roles in development: maintenance, the process of sustaining existing states and behaviors, facilitation, which includes assisting in the regulation of existing states and behaviors during the development of new states and behaviors, and induction, the creation of new states and behaviors (Gottlieb, 1991). During interactions among the components in the system, there is disequilibrium and

chaos within and between the various sources of variance within a system; intraindividual, interindividual, and contextual (Fitzgerald, Davis, Zucker, & Klinger, 1994a). These sources of variance will change depending upon which level of the system one is examining.

From this disequilibrium emerges the opportunity for development within the organism. Though chaos and disequilibrium exist, there is global structure based on the sociocultural restraints found within the interindividual and contextual levels of the system (Thelen, 1992) and there is also organism structure or boundary conditions that constrain and facilitate development in order to preserve the integrity of the organism (Ford & Lerner, 1992). Both global and organism structures guide the development of new or revised structures and behaviors in the person and later assists in the creation and re-establishment of equilibrium within the system (Fitzgerald et al., 1994a; Ford & Lerner, 1992; Thelen, 1992). The new adaptive behaviors and structures are more complex and differentiated than the previous ones and will assist the person in present and future interactions with his/her environment (Lerner, 1991).

With its integrative and wholistic assumptions, developmental systems theory provides the researcher with the means to hypothesize and test a comprehensive model of development. In terms of the present study, this theory will provide a fuller and more accurate understanding of events occurring in the prenatal and postnatal periods and their effects on children's adaptive function during the preschool period.

Within the study of prenatal development, developmental systems theory is similar to the assumptions of behavioral teratology which assumes that the affect of structural damage to the organism depends on the interactions between the organism and

the environment (Vorhees & Mollnow, 1987). Developmental systems theory views people and the environment as active and changing and development as a result of the dynamic interactions between the person and the environment which may explain the greater variation in outcomes of development. Though variations exist in development, there is also continuity in structures of the organism that are maintained by the organism's maintenance functions. It is these functions that explain the similarities found in development across organisms despite the different interactions and environments.

Within the study of postnatal development, developmental systems theory offers researchers a means to examine intraindividual, interindividual, and contextual influences on the development of the child. All three sources of influence can be examined simultaneously and in conjunction with past influences and outcomes of interactions among them. This strategy provides the best method to study the developing child by giving researchers the strategy to tease apart the influences of past environments, such as the prenatal environment and maternal past experiences and environments from those of the postnatal environment (Zuckerman & Bresnahan, 1991).

Prenatal Influences

During prenatal development, the structure of the organism goes through a series of stages beginning with disorganization and simplicity and moving towards increasing complexity and progressive differentiation (Ford & Lerner, 1992). Usually, this process proceeds unimpeded and normal development of the organism can occur. However, any restrictions on this process can act as a limiting force on later child development. These restrictions can affect the organism in two major ways, by damaging either differentiation of organ systems or the central nervous system.

Fetal injury can occur at anytime during the prenatal period, however there are periods of rapid reorganization or growth known as sensitive periods during which exposure to risk factors maximize the likelihood of damage (Bornstein, 1989). Because all major organs begin to develop during the embryonic period lasting from the end of the second week to the end of the eight week of gestation, exposure to a risk factor during this time can result in damage to a specific organ or extremity depending upon the timing of the exposure (Martin, 1976). As a result, the child can experience a set of physical handicaps and/or chronic health conditions caused by teratogenic exposure. The outcome of any one of these abnormalities could be a debilitating health condition, long-term developmental disability, and/or reduced life expectancy (Kopp, 1987).

The central nervous system (CNS) is vulnerable to structurally damaging insults not only during the embryonic period, but throughout the fetal period and during the entire lifespan. However, the sensitive period for the CNS occurs during the prenatal period and through the early childhood years when the brain is becoming more differentiated and myelination is occurring (Bornstein, 1989). At nine weeks gestation, the cerebellum is a mass of neuroblasts. Development continues during the fetal period with the appearance of fissures in the midline at fourteen weeks followed by fissures in the cerebral cortex around twenty weeks gestation. The process of myelination begins with the spinal cord around five months gestation and continues with the myelination of the fiber tracts within the brain beginning at six months prenatally (Martin, 1976). At birth, all peripheral and cranial nerves except those in the olfactory tract are myelinated (Gibson, 1991). By two years of age, all cortical areas have begun myelination and the brainstem tracts and subcortical processing areas are heavily myelinated (Gibson, 1991; Martin, 1976). Myelination of the corpous callosum, limbic system, reticular systems, and neocortex continues into middle childhood (Gibson, 1991). Due to the continuing maturation of the CNS throughout infancy and childhood, any damage to the CNS during the prenatal or postnatal periods can influence the postnatal behaviors of the child in terms of impaired or delayed cognitive, affective, social, and motorical behaviors (Vorhees & Mollnow, 1987).

There are many teratogenic influences that can cause structural damage to the developing organism. They include maternal illness or infection, maternal drug consumption (ethanol, illicit and legal drugs, and prescription drugs), environmental toxins, radiation, and injury to the embryo directly or indirectly through complications experienced as a direct result of an injury to the mother (Martin, 1976; Vorhees & Mollnow, 1987). In this section, the review of literature on prenatal influences on later developmental status, IQ, and child behavior problems will focus on the effects of two behavioral teratogens, ethanol and nicotine¹. Each review begins with a discussion of structural teratology including physiological effects of the teratogen on the mother, the process through which the teratogen comes in contact with the embryo and fetus, and the physiological effects of the teratogen on the developing fetus.

To aid understanding of the literature to be reviewed, clarification is needed between structural teratology and behavioral teratology. Structural teratology is the study of congenital malformations or birth defects, whereas behavioral teratology is the study

Teragenic influences during pregnancy have been reviewed extensively and the reader is referred to the general literature for influences not reviewed here (Brooks-Gunn, McCarton, & Hawley, 1994; Kopp, 1987; Vorhees & Mollnow, 1987; Zuckerman & Bresnahan, 1991).

of damage to the developing brain and its effect on behavior (Vorhees & Mollnow, 1987).

Prenatal Exposure to Alcohol

Physiological Effects of Alcohol on Mother and Fetus

Alcohol is a physiological depressant that operates on the central nervous system of the user. Since alcohol is water soluble it is quickly absorbed into the blood stream and rapidly dispersed throughout the body (Zuckerman & Bresnahan, 1991). As a result, chronic abusers of alcohol suffer from a variety of diseases and conditions affecting almost all parts of the body including, the liver, gastrointestinal tract with complications associated with malnutrition, heart, circulatory system, immune functions, and neurological abilities (USDHHS, 1988).

Based on various populations of pregnant women in the United States and abroad, it has been estimated that between one-half and three-fourths of women drink some alcohol during their pregnancy (Behnke & Eyler, 1993; Condon & Hilton, 1988). Studies have been conducted to determine the specific rates of drinking during pregnancy. A study of over 1,700 pregnant women found that 25% of the women had consumed alcohol in the past month, about 3% of the subjects were binge drinkers, and 0.6% of the mothers abused the substance (Day & Richardson, 1994). Women who drink heavily in the first trimester are most likely to be single, Euro-American, and to have little social support (Day, 1992). In contrast, those women who continue to drink throughout the pregnancy are most likely to be older, African-American, and experiencing many stressful events in their lives (Day, 1992). Several studies have found that women tend to reduce their drinking as the pregnancy progresses. Day and Richardson found high rates of alcohol use before and reduced rates during pregnancy. Prior to the pregnancy, 44% of the women drank one or more drinks per day (Day, 1992; Day & Richardson, 1991). This rate fell to 37% during the first month of pregnancy and continued to fall to 14% by the second trimester, and 5% in the third trimester (Day, 1992; Day & Richardson, 1991). The most striking change in drinking patterns for pregnant women was found in an Australian study (Condon & Hilton, 1988; Hilton & Condon, 1989). This study found that of the 86 pregnant women who drank prior to pregnancy, all of them reduced their intake of alcohol while pregnant and 53% of them stopped drinking (Condon & Hilton, 1988). In a replication of these results with only primiparous women, the percentage of women who stopped drinking during their pregnancy rose to 66% (Hilton & Condon, 1989).

During pregnancy, fetal exposure to alcohol is controlled by the placenta. Though referred to as the "placenta barrier", most molecules do pass through the placenta and affect the developing fetus. The rate of permeation increases with fetal age. As a result, continued exposure to large amounts of alcohol prenatally (2 to 4 drinks per day) is related to more severe effects including; growth retardation (Smith, Coles, Lancaster, Fernhoff, & Falek, 1986), lower scores on habituation measures resulting from difficulty in attending to auditory and visual stimuli (Streissguth, Barr, & Martin, 1983), lower scores on Bayley Scales of Infant Development and lower verbal comprehension scores on the Reynell Developmental Language Scales (Autti-Ramo et al., 1992).

After alcohol passes through the placenta, its distribution in the fetus will depend on the water content of the fetus' tissue (Beattie, 1986). Those tissues with higher water

contents, such as the cerebral grey matter will be exposed to higher levels of alcohol. As fetal age increases, so does the ability of the fetus to metabolize alcohol, though at birth this rate is still twice as long as the average adult rate (Beattie, 1992). After metabolizing the alcohol, the fetus is still exposed via swallowing the amniotic fluid (Beattie, 1986). Alcohol enters the amniotic fluid in two ways. Early in the pregnancy, alcohol is present in the amniotic fluid as a result of its being passed through the fetus' permeable skin. Later in the pregnancy, the alcohol enters the fluid through the fetus' urine.

Once exposed to alcohol, the fetus may experience a variety of physiological changes and increased risks of stillbirth, abruptio placentae, and premature delivery depending upon the level and timing of exposure (Behnke & Eyler, 1993). In humans, the fetus may experience respiratory changes ranging from some suppression to near complete inhibition of fetal breathing movements within thirty minutes of exposure and lasting for up to three hours (Suguihara & Bancalari, 1991), and suppression of brain activity as measured by EEG (Beattie, 1986). In sheep, exposure to high doses of alcohol administered in close intervals is related to high levels of acid found in body fluids (acidosis) which may be followed by the collapse of the umbilical blood vessels (Behnke & Eyler, 1993; Beattie, 1992). Other animal studies have found an association between prenatal exposure to ethanol and abnormal nasal and impaired optical cell development in mice; skeletal deficits, sensorineural hearing loss, and reduced fetal activity in rodents; and an increase in the number of fetal deaths for rodents, sheep, dogs, and rabbits (Behnke & Eyler, 1993; USDHHS, 1988).

Depending upon the level and timing of exposure the fetus may experience the long term effects of Fetal Alcohol Syndrome (FAS) which includes prenatal and postnatal

growth retardation; damage to the central nervous system resulting in jitteriness, developmental delays, hyperactivity, and facial dysmorphology (Jones, Smith, Ulleland, & Streissguth, 1973). The prevalence of FAS in the general population is estimated at 0.33 cases per 1,000² (Able & Sokol, 1991). Other physiological difficulties associated with FAS include abnormalities in the cardiac, immune, renal, orthopedic, respiratory, and metabolic systems (Burd & Martsolf, 1989).

When all the conditions for a FAS diagnosis can not be met, the classification of Fetal Alcohol Effects (FAE) is made (Beattie, 1992; Day & Richardson, 1991). The incidence of FAE is suggested to be about three times greater than the rate for FAS (USDHHS, 1988). However, an explicit rate for FAE is difficult to determine since it has not been operationally defined (Sokol & Clarren, 1989). Further controversy with the term FAE results from the suggestions by some researchers that using FAE has created an artificial dichotomy of the effects of alcohol (Burd & Martsolf, 1989), which violates the principles of behavioral teratology³. Still others believe that the use of FAE is incorrect and instead the term Alcohol-Related Birth Defects (ARBD) should be used (NIAAA, 1991).

The effects of prenatal exposure to various levels of alcohol on later child development have been found and studies that are pertinent to the present research will be reviewed in the following sections. In order to reduce confusion, the broad terms FAE or ARBD will not be used to explain the literature to be presented or the purpose of this

² This more conservative estimate by Able and Sokol is based upon both prospective and retrospective studies, whereas a previous estimate of 1.9 cases per 1,000 was based upon only retrospective studies.

³ The principles of behavioral teratology state that there is a range of effects for any given teratogen based on doseresponse, genetic milieu of the organism, timing of the exposure, and influence of the environment (Vorhees & Mollnow, 1987).

study. It is not the task of the present research to find support for the use of the terms FAE or ARBD. Instead the present research will examine along with other effects, the effects of prenatal alcohol exposure on the developmental status, IQ, and child behavior problems of preschoolers.

Pre- and post-natal growth retardation resulting from prenatal exposure to alcohol has been documented in a number of studies conducted on a variety of populations drawn from Canada, Finland, Italy, Mexico, and the United States (Autti-Ramo et al., 1992; Borges, Lopez-Cervantes, Medina-Mora, Tapia-Conyer, & Garrido, 1993; Cavallo et al., 1992; Day et al., 1991; Flores-Huerta, Hernandez-Montes, Argote, & Villalpando, 1992; Godel et al., 1992; Greene et al., 1991b; Jacobson et al., 1993). Deficits in birthweight, length, and head circumference have been reported for as little as two drinks per week (Greene et al., 1991b), whereas others have only found a reduction associated with binge drinking for birthweight (Borges et al., 1993) and head circumference (Godel et al., 1992). Continued growth retardation has been shown at six months of age (Jacobson et al., 1993) and later during the preschool years (Day et al., 1991; Greene et al., 1991b). When compared to preschoolers whose mothers did not drink during their pregnancy, preschoolers whose mothers drank more than one drink per day were on average 1.6 pounds lighter, .9 inches shorter, and had a head circumference that was .2 inches smaller (Day et al., 1991). These researchers have suggested that these children may never "catch-up" to their peers in terms of growth (Day et al., 1991; Greene et al., 1991b).

Other long term effects associated with prenatal exposure to alcohol include cognitive deficits, hyperactivity, behavior problems, language difficulties, motorical delays, and learning disabilities (Zuckerman & Bresnahan, 1991).

The following sections will address the effects of prenatal exposure to varied levels of alcohol on preschoolers' developmental status, IQ, and behavior problems.

Prenatal Alcohol Exposure, Developmental Status and IQ

Studies examining the effects of prenatal exposure on later developmental outcomes have found conflicting results depending upon the method for measuring exposure, specific outcomes measured, choice of confounding variables examined, and ages of children in the study. However, one study conducted in Seattle has consistently found a relationship between exposure to alcohol and children's cognitive development and fine and gross motor development (Barr, Streissguth, Darby, & Sampson, 1990; Streissguth, Barr, & Sampson, 1990; Streissguth, Barr, Sampson, Darby, & Martin, 1989).

Streissguth's longitudinal study of middle-class Euro-Americans found a relationship between mothers prenatal drinking and children's IQ at age four and seven years old (Streissguth et al., 1989; Streissguth et al., 1990). A five point decrement on the Wechsler Preschool and Primary Scales of Intelligence (WPPSI) at age four years was associated with an average of over three drinks per day consumption by the mother during her pregnancy (Streissguth et al., 1989). An even larger decrement of seven points was found at age seven with the Wechsler Intelligence Scale for Children-Revised (WISC-R) associated with an average consumption of more than one ounce of absolute alcohol per day⁴ during pregnancy (Streissguth et al., 1990). The subtests most highly

⁴ 0.50 ounces of absolute alcohol = one standard drink ≅ 12 oz beer ≅ 5 oz wine ≅ 1.25 oz liquor (Jacobson & Jacobson, 1994).

correlated (negatively) with mothers prenatal drinking were the Block Design at age four, the Digit Span at age seven and mathematics at both ages. It is unclear from the results reported by Streissguth and colleagues why a larger decrement at age seven was found for a smaller amount of alcohol exposure compared to the findings at age four.

Streissguth's study is one of the few studies to assess fine and gross motor skills in children exposed prenatally to an average of one to three drinks of alcohol per day (Barr et al., 1990; Forrest, Florey, Taylor, McPherson, & Young, 1991). Using a variety of standardized measures, Streissguth and colleagues found that four year old children who were exposed to alcohol early in the pregnancy exhibited poorer balance in gross motor tasks compared to children whose mothers did not drink (Barr et al., 1990). In the fine motor tests, these same children were slower to self-correct, took longer to complete the tasks, and made more errors (Barr et al., 1990).

Though the Seattle study has produced significant relationships between prenatal exposure to alcohol and child outcomes, Streissguth and her colleagues have been criticized by others for not controlling for potentially confounding variables (Zuckerman & Bresnahan, 1991). In their study on child IQ, they did not control for race or parental IQ, though they crudely controlled for mother-child interaction by using nine global assessments each on a five point scale scored when the child was 8 and 18 months of age (Steissguth et al., 1989; Streissguth et al., 1990). They did control for the effects of parental education. In their study of fine and gross motor development, they did not control for the effects of birthweight and gestational age, though they controlled for mother-infant interaction with the same crude measure noted above. These potentially

confounding variables could alter the results of these studies especially since the effects on some of the outcomes were small (Zuckerman & Bresnahan, 1991).

The lack of measurement of mother-child interaction in the Seattle study is a problem because studies have shown moderate correlations (ranging from 0.30 to 0.60) between the Home Observation for the Measurement of the Environment (HOME) and the Stanford-Binet, Bayley Mental Development Index, McCarthy Scales of Children's Abilities scores, and the Reynell Language Comprehension scores for children who were low birthweight babies or children with mental disabilities (Bradley, 1993). Though few studies have measured or even controlled for the effects of home environment on child outcomes for children exposed to alcohol prenatally, one study that did control for it found the quality of the home environment to be a key determinant for IQ (Greene et al., 1991a) and language development (Greene, Ernhart, Martier, Sokol, & Ager, 1990).

However, another study found that significant relationships between exposure to alcohol prenatally and children's language development and IQ continued to remain significant even after controlling for HOME. The Ottawa Prenatal Prospective Study of middle class mothers and their children found relationships between verbal development and moderate levels of prenatal exposure to alcohol when the children were one and three years of age but not after they reached five and six years of age (Fried, O'Connell, & Watkinson, 1992a; Fried & Watkinson, 1990; Gusella & Fried, 1984). At 13 months, social drinking -- defined as between 0.14 and 0.85 ounces of absolute alcohol per day during pregnancy -- was significantly associated with lower scores on the Mental Developmental Index of the Bayley Scales of Infant Development, specifically in the areas of spoken language and verbal comprehension (Gusella & Fried, 1984). A

continuation of language delays were found at 36 months with lower scores on the Reynell Developmental Language Scales in the areas of comprehension and expression (Fried & Watkinson, 1990).

Though the previously reviewed studies were measuring the effects of prenatal exposure to varied levels of alcohol, none of these studies attempted to control for mothers' current drinking. As mentioned previously, mothers do reduce their drinking during their pregnancy (Condon & Hilton, 1988; Fried, Innes, & Barnes, 1984; Hilton & Condon, 1989; Little et al., 1983). It is unclear from these studies whether the women resumed their higher prepregnancy levels of drinking after delivery. However, a longitudinal study in Pittsburgh found that by the time children reached three years of age, mothers had increased their level of drinking to their prepregnancy levels (Day et al., 1991). In any case, current drinking is a factor that may influence the outcomes of studies on the long term effects of prenatal exposure to alcohol.

One small study (N = 68) of early school-aged children in Atlanta controlled for mothers' current level of drinking while examining the effects of prenatal exposure to moderate and heavy levels of alcohol (Coles et al., 1991). This study of predominately black, low SES mother-child pairs compared two groups of children whose mothers drank on average two to five drinks per day throughout the pregnancy or only during the first trimester, with other mothers who never drank. Children whose mothers drank during pregnancy had lower IQ scores on the Kaufman Assessment for Children as well as lower scores in the specific areas of math skills, prereading identification of words and letters, and sequential processing skills measuring short-term memory (Coles et al.,

1991). This relationship was duration dependent with children whose mothers drank throughout the pregnancy scoring the lowest.

Another study of low income mothers from the Pittsburgh area also controlled for mothers' current substance use as well as maternal socioeconomic and psychological characteristics and the home environment (Goldschmidt, Richardson, Stoffer, Geva, & Day, 1996). This study examined the effects of alcohol exposure during each trimester of pregnancy on the academic achievement of six year olds. It was found that those mothers who drank heavily in the first and second trimesters had children who scored lower on the Wide Range Achievement Test-Revised (WRAT-R), compared to children whose mothers only drank heavily in the first or third trimester (Goldschmidt et al., 1996). Decrements were found in the spelling, reading, and arithmetic scales of the WRAT-R with the largest decrement associated with the arithmetic scale.

In summary, the studies reviewed in this section have found negative effects for exposure to various levels of alcohol prenatally in the areas of language acquisition (Autti-Ramo et al., 1992; Fried & Watkinson, 1990; Fried et al., 1992a; Gusella & Fried, 1984; Streissguth et al., 1989; Streissguth et al., 1990) fine and gross motor skills (Barr et al., 1990), and cognitive development (Coles et al., 1991; Goldschmidt et al., 1996; Gusella & Fried, 1984; Streissguth et al., 1989; Streissguth et al., 1990). The Seattle study has shown a continuation of negative effects as the child ages, for IQ in terms of verbal, mathematical, and performance skills (Streissguth et al., 1989; Streissguth et al., 1990). In contrast, another study has shown a continuation then finally a loss of effects as the child ages in terms of language development (Fried et al., 1992a; Fried & Watkinson, 1990; Gusella & Fried, 1984).

Prenatal Alcohol Exposure and Behavioral Problems

The area of research examining the effects of prenatal exposure to alcohol on later child behavior problems is also plagued with conflicting results. Differences in the levels of exposure to alcohol, sources of the data (teacher, parent, or child), measures used, specific outcomes examined, and sample size have been mentioned as reasons for the contrasting results (Boyd, Ernhart, Greene, Sokol, & Martier, 1991). One prospective study measured attention and impulsivity at two different ages and found significant results for each age (Streissguth et al., 1984; Streissguth et al., 1986). In order to better understand the literature reviewed in this area, an explanation of the technique of Continuous Performance Task (CPT) often used in this research will be given

CPT was created by Mackworth in 1950 to measure sustained attention by using a critical stimulus presented infrequently over a long period of time (Streissguth et al., 1984). Task length for children ranges from nine to thirteen minutes with a critical or noncritical stimulus presented approximately every one to every three seconds (Boyd et al., 1991; Kristjansson, Fried, & Watkinson, 1989; Streissguth et al., 1984). Traditionally the stimulus is often a number, letter, or word or it could even be a pattern in presentation of numbers, letters, or words that is projected onto a screen or appears on a computer screen. A non-traditional version or children's version of the CPT has been created that uses a house as background with animals or objects appearing in the windows as the stimuli (Herman, Kirchner, Streissguth, & Little, 1980; Streissguth et al., 1984). For each of the preceding reviewed studies, there is only one critical stimulus and a range of three to nine noncritical stimuli (Herman et al., 1980; Fried, Watkinson, & Gray, 1992b; Streissguth et al., 1984).

When the critical stimulus appears subjects are instructed to react in a certain way, such as push a button, strike a key on a keyboard, or raise their hands. Researchers count the number of correct and false responses as well as reaction time which is the time span between the appearance of a stimulus and the subjects' responses. An efficiency ratio can be computed by dividing the total number of correct responses by the total number of all responses.

There are two types of false responses, omission and commission. An omission error occurs when subjects fail to respond to the appearance of the critical stimulus. The number of omission errors provides researchers with a measure of inactivity. A measure of impulsivity is created by counting the number of commission errors or times when subjects respond to a noncritical stimulus.

In addition to measuring responses, reaction times and efficiency ratio, researchers can also measure the amount of time subjects spend looking at the screen for a measure of orientation. A measure of activity can be created by using movement sensors placed under the subjects' chairs to record the amount of time the subjects spend fidgeting or moving in their chairs (Kristjansson et al., 1989). The use of the CPT to measure vigilance in children has added to the literature on children's attention span and behavior problems.

The Seattle study conducted by Streissguth and associates used a CPT to measure vigilance in children ages four and seven years (Streissguth et al., 1984; Streissguth et al., 1986). A modified version of the traditional CPT using a house and pictures was used with the four year old sample (Herman et al., 1980; Streissguth et al., 1984) and the traditional version using numbers as stimuli was used with the seven year olds. Children

exposed to alcohol prenatally had a higher number of omission errors (inactivity) at age four years, higher number of commission errors (impulsivity) at ages four and seven, and overall slower reaction times at both ages (Streissguth et al., 1984; Streissguth et al., 1986).

These results have been supported by another study using in-home observations of four year old children from middle class homes. The researchers found that children exposed to an average of one drink per day prenatally were more likely to have instances of inattention and shorter attention spans, compared to those children whose mothers' were classified as infrequent drinkers or abstainers (Landesman-Dwyer, Ragozin, & Little, 1981).

Two other studies failed to find any significant relationship between prenatal alcohol exposure and children's performances on the CPT vigilance task (Boyd et al., 1991; Fried et al., 1992b), an impulse control task, or an observational measure of behavior, once all confounding variables were controlled (Fried et al., 1992b). These authors suggest that Streissguth's results may be suspect due to their failure to control for maternal intelligence and the quality of the home environment. These critics also cite as a limitation the large amount of missing data in Streissguth's studies due to equipment failures including power outages and printer failure. Due to these equipment failures, the sample sizes in the reaction and errors trials have been affected. Specifically, Streissguth's reaction time trials have a sample size ranging from 66 to 172 compared to the sample for the errors trial with a range of 284 to 360 (Boyd et al., 1991; Streissguth et al., 1984). This represents a loss of 23 to 48% of the total sample.

Prenatal exposure to alcohol has also been related to both externalizing and internalizing behavior problems. In a small sample (n = 68) of low-income five year olds, teachers rated children exposed to alcohol prenatally as more destructive, inattentive, nervous/overactive, and aggressive on the Child Behavior Checklist (CBCL) than children who were not exposed (Brown et al., 1991).

Another small pilot study (n = 41) of six year olds from upper-middle class homes found that children whose mothers drank an average of two to four drinks of alcohol per week prior to and during their pregnancy were more likely to endorse depressive items on the Pictorial Depression Scale, than those children whose mothers did not drink (O'Connor, 1995). This relationship was dose-dependent with the average depression score below the clinical range.

One study used retrospective, prenatal data and found a relationship between mother's substance use during pregnancy including alcohol and her preschooler's total behavior problems based on the CBCL (Bingham, Fitzgerald, Fitzgerald, & Zucker, 1996a). Parental alcoholism and both maternal substance use and environmental insults during prenatal period were predictive of total behavior problems. However, it was found that mother's current depression and lifetime antisociality accounted for the variance associated with prenatal exposure to substance use and behavior problems. The data were drawn from the first wave of data from the MSU-UM Longitudinal Study of children at risk for alcoholism and antisociality based on fathers' diagnoses of these pathologies. Children were preschoolers at the time of data collection.

Though the effects for prenatal exposure to alcohol on children's behavior problems have not consistently been found, there are results in a number of areas

encompassed by behavior problems. Children exposed to various levels of alcohol prenatally do less well on the continuous performance task measuring vigilance (sustained attention) (Streissguth et al., 1984; Streissguth et al., 1986), are more likely to be inattentive, impulsive, and display shorter attention spans during natural observations (Landesman-Dwyer et al., 1981). When compared to children who were not prenatally exposed to alcohol, the exposed children were more likely to be rated by their teachers as destructive, aggressive, inattentive, nervous/overactive, and depressed (Brown et al., 1991). Finally, children exposed prenatally to alcohol are more likely to rate themselves as depressed (O'Connor, 1995).

Prenatal Exposure to Smoking

Physiological Effects of Smoking on Mother and Fetus

Unlike alcohol which acts as a depressant, smoking exposes the user to the toxin nicotine which is a central nervous system activator. Smokers experience rapid increases in their heart rate, blood pressure, and respiration due to the constriction of blood vessels and release of adrenaline (Martin, 1976). Along with nicotine, smokers are also exposed to polycyclic aromatic hydrocarbon products or "tar" which are all known carcinogens as well as carbon monoxide, carbon dioxide, cyanides, aldehydes, and many other toxic compounds (Abel, 1980). With long term exposure, smokers increase their risk of developing cancers including lung, cervical, and endometrial, cardiovascular diseases, gastrointestinal diseases, renal diseases, high cholesterol levels, abnormal EEGs, weight loss, malnutrition, reduced fertility, and reduced life expectancy (USDHHS, 1990).

Like alcohol, nicotine is highly water-soluble as well as lipid-soluble (Abel, 1980). As a result, it is rapidly distributed throughout all body tissues and fluids.

Highest levels are found in the brain, pituitary and adrenal glands with high levels in the liver, kidneys, salivary glands, stomach, and bone marrow (Abel, 1980). With an initial overall distribution of nine minutes after first inhalation, nicotine remains in the body for over two hours with its major metabolite, continine, remaining for at least twenty hours (Russell, 1989).

Nicotine not only affects the person who is smoking, but it also affects the fetus of pregnant smokers. It has been estimated that between 25 to 33% of all pregnant women smoke (Behnke & Eyler, 1993; Condon & Hilton, 1988). Unlike drinkers, smokers are less likely to reduce their smoking while pregnant (Condon & Hilton, 1988; Hilton & Condon, 1989). An aforementioned Australian study found that of the 35 pregnant women who smoked prior to pregnancy, only 34% of them reduced their intake of nicotine while pregnant and 23% of them stopped smoking (Condon & Hilton, 1988). In a replication of these results with only primiparous women, the percentage of women who stopped smoking during their pregnancy rose to only 29% (Hilton & Condon, 1989). The percentage of pregnant women who tried to stop smoking but could not was 40% in the first study (Condon & Hilton, 1988) and 53% in the follow-up study (Hilton & Condon, 1989). Though these percentages only represent 14 and 15 pregnant women respectively, when compared to the number of women in the same study who tried to stop drinking during their pregnancy but failed the numbers are more telling of the difficulty in ceasing to smoke. In the first study no woman failed to stop drinking that wanted to and only five women failed to reach their goal of not drinking in the follow-up study. Women who continue to smoke throughout the pregnancy are more likely to be classified

as lower SES, as having completed fewer years of schooling, and as heavy drinkers (0.85 ounces of absolute alcohol per day) (Fried et al., 1984).

Nicotine is delivered to the fetus via the placenta and is distributed throughout the tissues and fluids. In rats, nicotine crosses the placenta within five minutes of drug administration, reaching peak levels at 30 minutes and remaining in the organism for 20 hours (Abel, 1980). High concentrations were found in the adrenals, kidneys, gonads, brain, and liver of the rat fetuses, though the actual levels are lower than those found in the placenta or maternal blood (Abel, 1980).

A recent study involving 139 newborns and their mothers in Belgium found infants of smokers were exposed to high levels of continine⁵ while in utero. Three day old neonates whose mothers smoked while pregnant were found to have continine levels averaging 551 nanograms/milligrams in their urine (Hanet, 1997). This level compares to 583 nanograms/milligrams which is the average continine level found in urine samples of adult smokers (Hanet, 1997). The fetus of a smoker is at greater risk for spontaneous abortion, premature labor, stillbirth, growth retardation, congenital heart defects, anencephaly, cleft palate and harelip, malformations of the central nervous system, eyes, and ears, and having a weaker sucking reflex (Abel, 1980).

Growth retardation in infants and children exposed to smoking prenatally has been documented in a number of studies drawn from several countries including Canada, Finland, Italy, and the United States (Autti-Ramo et al., 1992; Butler & Goldstein, 1973; Cavallo et al., 1992; Fogelman, 1980; Godel et al., 1992; Hardy & Mellits, 1972; Jacobson et al., 1993). Low birthweigths were found in infants whose mothers smoked

⁵ Continine is a major metabolite of nicotine.

as little as five or fewer cigarettes per day while pregnant (Godel et al., 1992). Exposure to more than ten cigarettes per day in utero was related to lower weight, length, and head circumference at birth (Jacobson et al., 1993), smaller head circumference at age four years (Hardy & Mellits, 1972), and an average of one centimeter deficit in height at ages seven and eleven years (Butler & Goldstein, 1973).

Some exposed children who had deficits in growth at birth have later shown a growth rate increase that has them within normal range for their ages. Studies have found this "catch-up" growth trend as early as six months (Jacobson et al., 1993), at seven years (Hardy & Mellits, 1972), and later at sixteen years of age for the girls (Fogelman, 1980). This latter finding suggests that the boys may also "catch-up" after they finish puberty (Fogelman, 1980). These results suggest that it may be the rate of growth that is decreased due to prenatal exposure to cigarettes and not the eventual height.

Other long term effects of prenatal exposure to mothers' smoking include; increased risk of Sudden Infant Death Syndrome (SIDS), higher rates of bronchitis and pneumonia, hyperactivity, lower scores on cognitive measures, and learning disabilities (Abel, 1980).

The following sections will address the effects of prenatal exposure to mothers' smoking on preschoolers' developmental status, IQ, and behavior problems.

Prenatal Exposure to Smoking, Developmental Status and IQ

Like research on the effects of prenatal exposure to alcohol, research on the effects of prenatal exposure to mothers' smoking and child outcomes have produced contradictory results. There are several reasons for the contradictory results related to the age of the child, the outcome measured, and the practice of controlling or not

controlling for confounding variables. In particular, whether or not one covarys social class or birthweight can affect the outcomes. Social class is highly correlated with smoking and smokers tend to have infants with lower birthweights. Failure to control for social class leads to an overestimation of the effects of prenatal exposure, whereas controlling for birthweight leads to an underestimation (Rush & Callahan, 1989).

An example of overestimation of the effects of prenatal exposure to smoking was found in a study of toddlers from lower-middle class homes in Dundee, Scotland. The researchers found a negative relationship between smoking and the Bayley Mental Developmental Index, only to have the relationship become non-significant after controlling for social class, maternal age, sex of child, birthweight, and gestational age (Forrest et al., 1991). The researchers suggest that the loss of significance was related to the strong association between maternal smoking and social class, however they did not publish the results of their regression analyses on smoking only their analyses on alcohol consumption.

The aforementioned, prenatal study conducted in Ottawa with middle class mothers and their children found relationships between nicotine exposure and language development even after controlling for a wide range of confounding variables including parental education, income, socioeconomic status, age, weight gain and nutrition during pregnancy, other drug use, quality of the home environment, and sex of the child (Fried et al., 1992a; Fried & Watkinson, 1990; Gusella & Fried, 1984). Using three categories of exposure based on milligrams of nicotine: nonsmoker, light smoker (< 15 mg/day⁶) and heavy smoker (> 15 mg/day), Fried and colleagues documented language delays in the

⁶ The average cigarette contains between 1.5 and 2.5 mg of nicotine (Schuckit, 1995).

area of verbal comprehension at 13 months of age with the Bayley Scales of Infant Development (Gusella & Fried, 1984) and in the area of expression for children ages three to six years with the McCarthy Scales of Children's Abilities and the Reynell Developmental Language Scales (Fried et al., 1992a; Fried & Watkinson, 1990). When these children were six to nine years of age, relationships were found between both maternal smoking and exposure to passive smoke during the pregnancy and the scores for comprehension, articulation, sound blending, and syntax using the Test of Language Development and Sound Blending (Makin, Fried, & Watkinson, 1991). Children whose mothers were active smokers scored the lowest on these verbal measures even after controlling for maternal age, education, and socioeconomic status, all of which were not significantly related to the outcome variables.

The negative association between prenatal exposure to smoking and verbal comprehension was also found in another study which defined exposure as the number of cigarettes smoked per day (Bauman, Flewelling, & LaPrelle, 1991). In a subsample of 5,000 five year olds from the Child Health and Development Studies of thousands of children from across the United States, a relationship was found between mothers' smoking two packs of cigarettes per day during pregnancy and their children's lower levels of receptive vocabulary as measured by the Quick Test (Bauman et al., 1991). This study controlled for a wide range of confounding variables including; social class, sex of child, parental education, prenatal exposure to alcohol, and birthweight⁷.

⁷ Because Bauman and colleagues controlled for birthweight in their analyses, they may have underestimated their effects (Rush & Callahan, 1989).

Along with delays in language development, delays have also been found in fine and gross motor skills from infancy to school age. On timed motorical tasks, such as Peg Board and Hand Dominance, four year old children whose mothers smoked more than 16 mg of nicotine per day took longer to complete the tasks than those children whose mothers did not smoke (Fried & Watkinson, 1990). However, when these children were six years old, the relationship changed. Having a mother who smoked or was exposed to smoking during pregnancy was related to faster rates of completion on timed motor tasks (Fried et al., 1992a).

Children exposed to smoking prenatally did not fare as well on the more complex motor tasks, such as the Draw-a-Man and Developmental Drawings Test which required more attention to detail (Bauman et al., 1991; Makin et al., 1991). On these tasks, children exposed prenatally to more than two packs of cigarettes per day (Bauman et al., 1991) or 16 mg of nicotine per day (Makin et al., 1991) scored below those children who were not exposed. To explain the conflicting results, the authors suggest that children of mothers who smoked while pregnant were found to have higher activity levels which enabled them to perform better on timed motor tasks but these same children had less developed skill of attending to details which hindered completion of the more complex drawing tests.

There also is evidence pointing to a negative relationship between prenatal exposure to smoking and cognitive development. In the Ottawa study, Fried and researchers found significant associations between prenatal exposure and deficits in cognitive development when the children were between the ages of three and nine years even after controlling for a wide range of confounding variables including parental

education, income, age, weight gain and nutrition during pregnancy, other drug use, quality of the home environment, and sex of the child (Fried et al., 1992a; Fried & Watkinson, 1990; Makin et al., 1991). Prenatal exposure to smoking was negatively related to three specific subscales of the McCarthy Scales of Children's Abilities, perceptual at age three years, qualitative at ages three and five years, and memory for three through six years of age (Fried et al., 1992a; Fried & Watkinson, 1990). The relationships were dose dependent with the largest decrements found in children who were exposed to more than 16 mg of nicotine per day. The negative relationship between perceptual skills and exposure to smoking continued during the early elementary years with children of mothers who smoked or passively smoked during pregnancy scoring the lowest on the perceptual subscale of the WISC-R with nonsmokers scoring the highest (Makin et al., 1991).

The continuation into the school years of delays in cognitive development was also found for academic skills in the National Child Development Study involving 17,000 children and their mothers born in Britain (Butler & Goldstein, 1973; Fogelman, 1980). The researchers found a four month decrement in reading scores in seven year old children whose mothers were heavy smokers (>10 cigarettes/day) during their pregnancy (Butler & Goldstein, 1973). For these same children at age eleven, the decrement in reading scores remained the same and additional delays in general ability (three months) and math (five months) were also documented (Butler & Goldstein, 1973).

Other studies of prenatal exposure to nicotine have found no relationship between exposure and developmental status at ages 6 months to four years (Greene et al., 1990; Greene et al., 1991a; Streissguth et al., 1989). Mothers' smoking during pregnancy was

found to be unrelated to children's cognitive and language development for a sample of low SES mother and child pairs ages 6 months to four years (Greene et al., 1990; Greene et al., 1991a) and for four year old children from middle class homes (Streissguth et al., 1989). Streissguth and colleagues' study of motor development also found no relationship between mothers' smoking during pregnancy and their children's fine and gross motor development after controlling for the confounding variables of parental education, age, race, nutrition, and other drug use and omitting two outliers from the regressions (Barr et al., 1990). This trend of insignificance was found even though these studies (Barr et al., 1990; Greene et al., 1990; Greene et al., 1991a; Streissguth et al., 1989) had similar rates of smoking compared to the previously examined studies in this section that did find significant relationships.

In summary, prenatal exposure to smoking has been shown to be related to language delays and negatively related to cognitive development in children. These associations are documented from infancy through the school years using a variety of specific and general measures (Bauman et al., 1991; Butler & Goldstein, 1973; Fogelman, 1980; Fried et al., 1992a; Fried & Watkinson, 1990; Gusella & Fried, 1984; Makin et al., 1991). Several of these studies may have even underestimated their effects by controlling for birthweight (Bauman et al., 1991; Fried & Watkinson, 1990; Gusella & Fried, 1984). The conflicting results concerning timed measures of motor development are ambiguous, with children exposed to prenatal smoking doing better (Makin et al., 1991) or worse (Fried & Watkinson, 1990) than those not exposed depending upon the children's ages. These results are hard to explain especially since these two samples

(Fried & Watkinson, 1990; Makin et al., 1991) were drawn from the same prenatal study conducted in Ottawa.

It is difficult to explain the lack of findings in the longitudinal studies conducted by Streissguth and Greene when other studies reviewed did find results. However, most of the studies that found results used ANOVAs, an orthogonal design based on exposure levels for their analyses. Whereas, Streissguth and Greene used regression analyses, a nonorthogonal design. The use of ANOVAs in this literature attenuates the effects associated with maternal smoking during pregnancy, because it does not take into account the possibility of other variables or maternal behaviors that may also be associated with maternal smoking and developmental status, such as parenting behaviors or maternal depression. Therefore, it is possible that the results in the reviewed studies using ANOVAs that were attributed to maternal smoking during pregnancy may in fact be due to postnatal parenting behaviors or maternal depression.

Prenatal Exposure to Smoking and Behavioral Problems

Unlike research examining the effects of prenatal exposure to alcohol in children without FAS, studies examining the effects of prenatal exposure to maternal smoking on child behavior problems have produced consistent results. Effects were found for children ages four to seven years with all but one study comprised of mostly Euro-America, middle class mother-child pairs (Fried et al., 1992b; Kristjansson et al., 1989; Streissguth et al., 1984; Weitzman, Gortmaker, & Sobol, 1992). The exception is a large national study which oversampled mother-infant pairs from low SES and minority households (Weitzman et al., 1992).

Several studies found significant relationships between exposure to smoking and sustained attention and impulsivity control on the Continuous Performance Task (CPT) measuring vigilance (Fried et al., 1992b; Kristjansson et al., 1989; Streissguth et al., 1984). Two of the studies used a modified version of the CPT which utilized pictures instead of letters or numbers as the stimuli (Kristjansson et al., 1989; Streissguth et al., 1984). Both of these studies found a relationship between prenatal exposure to smoking and difficulty attending to the task either by increased activity levels such as fidgeting (Kristjansson et al., 1989) or spending less time oriented towards the screen (Streissguth et al., 1984). Only the Seattle study of middle class families found an increase in omission errors and a decrease in the number of correct responses on the vigilance test for those children who were exposed (Streissguth et al., 1984). In another study, the relationship between omission errors and mothers' prenatal smoking became insignificant after the researchers controlled for family income, maternal education, and postnatal exposure to smoking (Kristjansson et al., 1989). Using letters as the stimuli, a subsample of the Ottawa study of middle class families found higher levels of impulsive behavior and activity in children exposed to smoking prenatally (Fried et al., 1992b).

Additional studies of attention dependent memory were conducted on the subsamples of the Ottawa study (Fried et al., 1992b; Kristjansson et al., 1989). Children exposed to smoking prenatally had more difficulty with verbal memory measured by the word, sentence, and story recalls of the McCarthy Scales of Children's Abilities and least difficulty with nonverbal tasks of pictorial memory and finger tapping on the McCarthy Scales (Fried et al., 1992b). Similar results were found with an auditory vigilance task. Specifically, a positive relationship was found between exposure to smoking and the

number of commission errors (impulsivity) (Kristjansson et al., 1989). These results remained significant even after researchers controlled for a wide variety of variables including, family income and maternal education (Fried et al., 1992b; Kristjansson et al., 1989), maternal age, other drug use, quality of home environment, and sex of child (Fried et al., 1992b), and postnatal exposure to smoking (Kristjansson et al., 1989).

Besides showing differences in attention and impulsivity, children who were exposed to smoking prenatally were rated by their parents as having more behavior problems as measured by the Connors' Parenting Rating Scale (Fried et al., 1992b) and the Behavior Problem Index, a short questionnaire modeled after the Child Behavior Checklist (Weitzman et al., 1992). Children whose mothers smoked less than one pack of cigarettes per day during pregnancy were 1.6 times more likely to exhibit extreme behavior problems compared to those children whose mothers did not smoke during their pregnancy (Weitzman et al., 1992). Exposure to smoking prenatally was specifically related to higher scores on the immaturity subscale of the Behavior Problem Index (Weitzman et al., 1992).

Children exposed to smoking prenatally have been shown to exhibit difficulty in several areas encompassed by the term behavior problems. These children are more likely to be inattentive (high number of omission errors) and impulsive (high number of commission errors) thus, they had fewer correct responses on the vigilance tasks (Fried et al., 1992b; Kristjansson et al., 1989; Streissguth et al., 1984). Exposed children are also more likely to experience difficulty in auditory memory tasks and less likely to experience difficulty in visual or motorical memory tasks (Fried et al., 1992b; Kristjansson et al., 1989). Finally, children exposed to smoking prenatally are more

likely to exhibit extreme behavior problems as rated by their parents (Weitzman et al., 1992).

Summary of Research on Prenatal Exposure and Child Development

Though some of the results in the literature presented is limited due to the practice of controlling for or not controlling for potentially confounding variables, there are significant relationships and trends when found in the literature that were consistent for prenatal exposure to approximately 1 to 3 drinks per day of alcohol and smoking one pack of cigarettes per day and child development. Mothers who drank or who smoked during their pregnancy are more likely to have children who are experiencing delays in cognitive development. Mothers who drank during their pregnancy are more likely to have children who are experiencing problems with language acquisition as opposed to women who smoked during pregnancy are more likely to have children who are experiencing difficulties with comprehension and speech. Mothers who drank or smoked during their pregnancy are more likely to have children who had less developed fine and gross motor skills. This relationship continued except for simple timed motor tasks, in which children exposed to nicotine prenatally performed better than those children who were not exposed to nicotine. Mothers who drank or smoked during pregnancy are more likely to have children who are exhibiting more externalizing behavior problems and inattentiveness. Lastly, children who were rated by teachers as nervous and depressed are more likely to have mothers that drank during their pregnancy. It is important to note that some of the reviewed studies have failed to find these relationships or trends or they lost significance after controlling for the quality of the home environment and parenting practices.

Postnatal I: Maternal and Familial Influences

In contrast to the prenatal period, the postnatal period is characterized by numerous influences on development occurring both directly and indirectly along with a greater number of opportunities for interaction with different contexts at different levels. Influences on child development originate from a wide variety of sources, such as, self (intraindividual), parents, siblings, peers, and other adults (all interindividual). The different contexts of home, school, neighborhood, community, and society that a child interacts with are also influenced by parental and family characteristics such as education and socioeconomic status. The complexity and inter-relationships of the postnatal environment make it difficult to understand the effects it has on child development.

By focusing on a few key influences and one context, one can begin to disentangle the effects. The following sections will examine the influences of mothers' psychopathology, specifically depression and antisociality, and their current use of alcohol and smoking on the developmental status, IQ, and behavior problems of children. The influences of family socioeconomic status, maternal IQ and maternal level of education will also be examined. This will be followed by the fourth section of the review, which examines the influences of maternal parenting behaviors (environmental stimulation) and attitudes on the child outcomes of developmental status, IQ, and behavior problems.

Maternal Psychopathology and Drug Involvement

Since the early 20th century, a link between parental mental health and child development has been noted in studies involving people with schizophrenia, depression, and an assortment of other mental illnesses (Janet, 1925; Kraepelin, 1921; Rutter, 1966).

Within the past thirty years, studies with similar populations have examined the effects of maternal psychopathology on the following child outcomes; mental health, attachment to the mother, cognitive development, and social behaviors (Downey & Coyne, 1990; Elder, Caspi, & Downey, 1983; Rutter, 1990). Of particular interest to the current study is the effects of maternal depression, antisociality, and drug involvement on child development.

Rates for these three maternal behaviors vary depending upon the pathology and circumstances of the women. In general the percentage of young women who are experiencing major depression is 9% (Puckering, 1989). However, two London studies found rates of depression among non-working mothers of preschoolers as high as 40% (Brown & Harris, 1978; Richman, Stevenson, & Graham, 1982). The percentage of women who are only experiencing depressive symptoms but are not diagnosablely depressed is between 25% and 30% (Robins & Regier, 1991). The rates for antisociality in the general population are lower compared to depression at 1.9% lifetime prevalence rate, though the rate is increasing rapidly (Mulder, Wells, Joyce, & Bushnell, 1994).

Rates of substance abuse also vary. Approximately 60% of all women drink alcohol (NIAAA, 1990) with 6.4% of the women meeting the classification for alcoholabuse and it was also found that 8.2% of the women were classified as alcohol dependence (Kessler et al., 1997). Chronicity of alcohol problems for women tend to peak during their late twenties and early thirties, but the incidence of alcohol dependence is greatest for women ages 35-49 years (NIAAA, 1990). Alcoholic women are twice as likely as alcoholic men to be classified as binge drinkers, 28% compared to 14% in an intreatment population (Dunne, Galatopoulos, & Schipperheijn, 1993). A subsample of alcoholic mothers (n = 128) from the MSU-UM Longitudinal Study of children at risk for

alcoholism and problem behaviors found that 38% of the mothers met the criteria for alcohol abuse, 23% for mild dependence, 28% for moderate dependence, and 11% for severe dependence (Caplan, 1996).

There is also a positive relationship between drinking and smoking. Based on the 1990 National Health Interview Survey, 55.5% of those women classified as heavy drinkers regularly smoke and it was also found that 27% of female drinkers are former smokers (NIAAA, 1992). These numbers are striking when compared to the smoking habits of women who abstain from alcohol, only 9% smoke and 7% are former smokers (NIAAA, 1992). In terms of smoking, over 22% of all adult women regularly smoke (USDHHS, 1990). This statistic is stable throughout the adult years, due to the fact that between 80-90% of all adult smokers began smoking by the age of twenty-one years (USDHHS, 1990).

Depression, antisociality, and substance abuse can be detrimental to child development. Their influence is both direct and indirect due to their relationships with other additional risk factors. These risk factors are lower levels of social support, marital conflict and violence, unemployment, and comorbid psychiatric disorders (Downey & Coyne, 1990; Ellis, Bingham, Zucker, & Fitzgerald, under review; Hesselbrock, Hesselbrock, & Workman-Daniels, 1986; Kendler et al., 1993; Mulder et al., 1994; Reider, Zucker, Maguin, & Fitzgerald, 1996; Rutter, 1990; Zucker, Ellis, Fitzgerald, Bingham, & Sanford, 1996b).

The interrelatedness of these three psychopathologies can be illustrated by a few of their comorbidity rates. The rates for depression are reported between 8 and 31% among alcoholics (Dunne et al., 1993; Schuckit, 1995), 48.5% among those women

classified as alcohol dependent (Kessler et al., 1997), and as high as 63% for alcoholics intreatment (Hesselbrock et al., 1986). Fifteen percent of depressed, alcoholic women report having their first depressive episode prior to their first major alcoholic life problem (Schuckit, 1995). Using data from the MSU-UM Longitudinal Study to determine alcoholic typologies in women, it was found that mothers classified as negative affect alcoholic were less likely to met the criteria for alcohol abuse than those women classified as primary alcoholic (Caplan, 1996).

Over half of the people classified as depressed are smokers (Kendler et al., 1993; Schuckit, 1995) and about 20% of smokers met the classification for clinical depression (Breslau, Kilbey, & Andreski, 1993). The relationship between smoking and depression is described by some as reciprocal (Kendler et al., 1993) and by others as unidirectional with smoking leading to depression (Newhouse & Hughes, 1991), or smoking as the consequence of depression (Covey, Glassman, & Stetner, 1990). One prospective study of young adults found that a nicotine dependence classification based on the DSM-III-R criteria was related to a higher likelihood of a first-incidence of major depression occurring within a fourteen month time period (odds ratio = 2.45) (Breslau et al., 1993).

Other comorbidity rates are reported to further illustrate the interrelatedness of antisociality and alcoholism and alcoholism and smoking. Women who are diagnosed with antisocial personality disorder are ten to thirteen times more likely to abuse alcohol (Mulder et al., 1994) and 5% of alcoholic women meet the classification of antisocial personality disorder (Schuckit, 1995). A comparison of gender comorbidity rates found a 2.5 times greater rate of antisociality and alcohol abuse/dependence in women than men (Zucker & Gomberg, under review). Lastly, women who are alcoholics are more likely

to have started smoking at a younger age and are more likely to be currently classified as a heavy smoker (Gomberg, 1993).

The following sections will examine the relationships among depression, antisociality, and substance abuse and the child outcomes of developmental status, IQ, and behavior problems.

Maternal Depression, Developmental Status and IQ

In the literature, depression is used to refer to either depressive symptomology or a diagnosis of depression. Depressive symptomology includes feelings of sadness, emptiness, and a flattening of affect. The diagnosis of clinical depression is made if these depressive symptoms exist for at least two weeks and are accompanied by changes in body functioning (e.g. insomnia and lethargy) and changes in mental functioning (e.g. inability to concentrate and loss of interest in usual activities) (Schuckit, 1995; Zuckerman & Beardslee, 1987). Research studies use both self-reported measures and clinical interviews to determine level of depression among the participants. The practice of using self-reported measures to diagnosis depression instead of clinical interviews has been criticized by some who feel that these measures are identifying depressive symptomology and not clinical depression (Downey & Coyne, 1990). Both depressive symptomology and clinical depression are valid areas of study, but for generalization purposes it is important to note which level of depression was assessed in each study.

Maternal depression is negatively related to cognitive, motorical, and language development throughout childhood. Preschoolers and early elementary children of depressed mothers were found to have lower scores on all subscales and the full scale of the McCarthy Scales of Children's Abilities compared to children of non-depressed

mothers (Coghill, Caplan, Alexandra, Robson, & Kumar, 1986; Goodman & Brumley, 1990; Hay & Kumar, 1995; Puckering, 1989). A longitudinal study of 119 London families found that maternal depression during the child's first year of life predicted the children's performances on the McCarthy Scales at age four years (Coghill et al., 1986; Hay & Kumar, 1995). All five subscales of the McCarthy Scales were negatively affected with the largest deficits measured by the perceptual-performance and motor subscales. Mothers' level of depression was assessed by a clinical interview.

A smaller study of 48 mother-child pairs offers support for the association between cognitive development and maternal depression diagnosed with the Diagnostic and Statistical Manual of Mental Disorders, third edition (DSM-III) but not for motor development and maternal depression. Infants and preschoolers of depressed mothers had lower scores on the Bayley Scales of Infant Development and mental development subscales of the McCarthy Scales of Children's Abilities when compared to children of non-depressed mothers (Goodman & Brumley, 1990). However, the children of depressed mothers scored higher on the psychomotor subscales of the Bayley Scales and McCarthy Scales when compared to the control group. After further analysis of these conflicting results, the authors found that maternal parenting practices mediated the relationship between maternal depression and the child's motorical development. The previous studies did not include an assessment of parenting practices in their analyses (Coghill et al., 1986; Hay & Kumar, 1995).

Other studies have found deficits in language development for children of clinically depressed mothers. This trend has been found using the standardized WISC and home observations on children ranging from two years of age through school-age

(Cox, Puckering, Pound, & Mills, 1987; Kaplan, Beardslee, & Keller, 1987). The London longitudinal study found that 2 to 2 1/2 year old children of depressed mothers demonstrated delays in expressive language based on observations in the home (Cox et al., 1987).

Delays in language development are also found in older children. A smaller study of school-aged children (n = 41) found scores on the verbal subscale of the WISC-R to be negatively related to mothers' diagnosis of depression (Kaplan et al., 1987). For approximately 20% of these children, there was a 12 point or larger difference between their scores on the verbal subscales when compared to their scores on the performance subscales. Another study found that seven year old children of depressed mothers were more likely to be experiencing reading problems than children of non-depressed mothers (Puckering, 1989).

A negative relationship between maternal depression and both cognitive and language development has been found in the literature for preschool and school-aged children. However, the research is scarce and utilizes only a few standardized assessments of the outcomes. The relationship between maternal depression and motor development in preschoolers is unclear. It is possible that parenting practices may account for the differences in motorical development for children of depressed mothers.

Maternal Depression and Behavioral Problems

Unlike the relationship between maternal depression and developmental status, the association between maternal depression and child behavior problems has been the focus of many studies. These studies encompass samples of children from infancy through the school years with a majority of the literature focusing on children between

the ages of three and nine years. In order to better review this literature, an overview of the problem of maternal bias will precede the review.

Several studies including reviews of past research findings have been conducted in order to determine if depressed mothers are biased in their assessment of their children's behavior. One study compared clinically depressed and non-depressed mothers' reports of their children's symptoms and found higher rates of reported symptoms by the depressed mothers (Breslau, Davis, & Prabucki, 1988). Children of depressed mothers reported more symptoms in the areas of depression and hyperactivity, but their mothers reported higher symptoms in all areas of assessment including anxiety, behavior problems, and antisociality. Another study of children ages three to eight years found that depressed mothers reported high rates of behavior problems in their children, though both teacher reports and in-home observations by the researchers found no significant differences between children of depressed and non-depressed mothers (Webster-Stratton & Hammond, 1988).

Meta-analysis of past findings conducted by Richters (1992) examined the issue of maternal accuracy in reporting child behavior. Based on his review of 22 studies⁸, he found no evidence to support the finding that depressed mothers are inaccurate informants of their children's behavior. Based on his own criteria for determining a distortion influence of maternal depression, Richters found that the 17 studies that found a bias did not meet the criteria for reaching this conclusion. Poor methodology, lack of observer assessment of the children's behavior, and not calculating agreement among the assessments all discredit the reviewed studies' findings of a bias. Richters (1992) stresses

⁸ Breslau et al., 1988 and Webster-Stratton & Hammond, 1988 studies are included in this analysis.

the need for additional studies utilizing stricter methodologies that compare reports of child behavior problems from a variety of sources including independent observers, mothers, fathers, and teachers.

It is important to note that differences in parental ratings of child behavior problems are found in other studies beyond the maternal depression literature. Several studies from the MSU-UM Longitudinal Study of children at risk for alcoholism and problem behaviors compared mothers' and fathers' reports of child behavior problems (Bingham, Loukas, Fitzgerald, & Zucker, under review-a; Fitzgerald et al., 1990; Fitzgerald, Zucker, Maguin, & Reider, 1994b). Both alcoholic and nonalcoholic fathers tended to rate their preschool sons as less attentive and more active than the mothers (Fitzgerald et al., 1990). Mothers who were classified as antisocial and aggressive were more likely to rate their children as distractible and less attentive than those mothers who were not classified as antisocial or aggressive. In general, it was found that low parental agreement was associated with greater parental psychopathology including antisociality, alcohol consumption, current depression, and low adaptive functioning (Bingham et al., under review-a). Another study utilizing a subsample of the same longitudinal study found that as paternal time with sons increased, the fathers and mothers ratings of aggression and conduct problems became more similar, but mothers still continued to rate their sons as more aggressive and as having more conduct problems than fathers (Fitzgerald et al., 1994b).

Bias in reporting child behaviors is a concern in any literature utilizing information obtained from a secondary source. When reviewing results based on data from secondary sources (parental reports on offsprings), it is important to remember two

things. The first point is that it may be the fathers and not the mothers that are biased in rating their children. Secondly, biases whether maternal or paternal may not just be a product of depression but may be related to other parental characteristics, such as antisociality and aggression as well as other more mundane factors such as time spent with the child.

Maternal depression has been found to be associated with a variety of internalizing and externalizing behavior problems in infants, toddlers, preschoolers, and school-aged children. (Breznitz & Friedman, 1988; Cox et al., 1987; Downey & Coyne, 1990; Field, 1992; Fitzgerald et al., 1993; Forehand, McCombs, & Brody, 1987; Kaplan et al., 1987; Lovejoy, 1991; O'Connor, 1995; Puckering, 1989; Richman et al., 1982; Stein & Newcomb, 1994; Webster-Stratton & Hammond, 1988; Zahn-Waxler, Jannotti, Cummings, & Denham, 1990). Children of depressed mothers were found to have higher rates of depression, anxiety, fearfulness, neurosis, aggression, and antisociality compared to children of non-depressed mothers (Downey & Coyne, 1990; Kaplan et al., 1987; Stein & Newcomb, 1994). When preschoolers were split into two groups, high and low behavior problems based on the 75th percentile of total behavior problems on the Child Behavior Checklist, it was found that 49% of the high group had a depressed mother based on the Beck Depression Inventory compared to only 21% of the children in the low group (Mun, Bingham, Fitzgerald, & Zucker, 1997). Lastly, a survey of existing literature found a positive, linear relationship between maternal depression and child behavior problems with children of clinically depressed mothers exhibiting more behavior problems than children of mothers with depressed mood (Forehand et al., 1987).

In two longitudinal studies, one conducted in the United States (Zahn-Waxler et al., 1990) and one in London (Richman et al., 1982), it was found that this linear relationship continues to exist as the child develops. In the United States, a small sample (n = 44) of clinically depressed and non-depressed mothers and their preschoolers were followed for four years. Children of depressed mothers were more likely to exhibit aggression towards an adult at age two years and to exhibit more externalizing and internalizing behavior problems at ages five and six years compared to children of non-depressed mothers (Zahn-Waxler et al., 1990).

In London, seven hundred families with preschoolers were followed through the children's first years of school (Richman et al., 1982). Maternal depression was diagnosed during an interview with researchers. It was found that maternal depression predicted both internalizing and externalizing behavior problems at ages three, four, and eight years. Mother's level of depression when the child was a preschooler predicted both the children's level of antisociality and neurosis at age eight years. The authors found that the more severe the mothers' depression, the more antisocial and neurotic the children were at age eight.

Maternal depression is positively related to both internalizing and externalizing behavior problems in preschoolers and continues to affect the children's behavior beyond school entry. This relationship is linear with children of clinically depressed mothers exhibiting more behavior problems than children of mothers with depressed moods. There is support in the literature that this relationship continues to exist even after the depressive episode ends and the symptoms have disappeared (Forehand et al., 1987; Hay & Kumar, 1995; Rutter, 1990).

Maternal Antisociality and Behavior Problems

Unlike depressed women, women with Antisocial Personality Disorder are rarely studied and when they are, the sample is often restricted to those women who are incarcerated. In a review article examining the effects of maternal antisociality on children, five of the eight studies examined the outcome of juvenile delinquency and one studied school behavior problems (Martin & Burchinal, 1992). Only six studies were found that are useful to the present research (Bingham et al., 1996a; Bingham, Smith, Fitzgerald, & Zucker, 1997; Ham et al., 1993a; Jansen, Fitzgerald, Ham, & Zucker, 1995; Loukas, Piejak, Bingham, Milburn, & Fitzgerald, 1995; Martin & Burchinal, 1992). These were the only studies that used a population-based sample instead of a restricted sample of incarcerated women. The first study examined the effects of mothers' antisociality on the behavior problems of their preschoolers and early elementary schoolaged children (Martin & Burchinal, 1992). The other five studies also examined the relationship between mothers' antisociality and behavior problems of preschoolers using subsamples of children whose fathers are alcoholics compared to a subsample of children whose fathers are not alcoholics (Bingham et al., 1996a; Bingham et al., 1997; Ham et al., 1993; Jansen et al., 1995; Loukas et al., 1995).

Utilizing information from over fourteen hundred mother and child pairs from the National Longitudinal Survey of Youth, Martin and Burchinal (1992) found a relationship between mothers' self-reported levels of delinquency and their children's level of behavior problems assessed six years later. Mothers' non-drug offenses were positively related to their children's total score on the Behavior Problem Index and all six subscale scores. There was a linear but non-significant trend for a relationship between

mothers' drug offenses and child behavior problems. The authors suggest that this lack of finding was related to the smaller subscale of drug offenses compared to the non-drug offenses subscale (3 items to 13 items) and the small subsample of women (n = 43) who committed only drug offenses and did not commit any other crimes (Martin & Burchinal, 1992).

The following five studies were all drawn from the same longitudinal study of alcoholism and child development and all used the Child Behavior Checklist (CBCL) to measure child behavior problems. The first study had the largest sample of 328 mothers and sons including those families with an alcoholic father (Bingham et al., 1996a). In this study both maternal antisociality and depression were related to children's behavior problems. Those mothers that scored high on measures of lifetime antisociality and current depression were more likely to have children with higher total behavior problems scores than those mothers with lower levels of antisociality and depression. An association between maternal lifetime antisociality and current depression was found in another sample of 313 mothers and sons, both maternal influences predicted higher number of behavior problems in the sons (Loukas et al., 1995).

A smaller study utilizing 69 alcoholic families and 32 comparison families found that maternal lifetime antisociality was related to ADHD problem behaviors and aggression in preschool boys (Ham et al., 1993). ADHD problem behavior was a composite score of the following factors created from the CBCL and the Dimensions of Temperament Scale (DOTS); hyperactivity, attention span/distractibility, and impulsivity.

Utilizing Structural Equations Modeling to examine the influences of family history of alcoholism and parental antisociality on child behavior problems, the next study found that maternal antisociality mediated the relationship between maternal family history of alcoholism and child behavior problems (Bingham et al., 1997). This study included a sub-sample of 177 mothers, fathers and sons.

The last study included 191 mothers, fathers (alcoholic and nonalcoholic), and their preschool children. When examining maternal influences alone, it was found that child reactivity, maternal antisociality, and family socioeconomic level combined to predict 22% of the variance associated with children's total behavior problems scores (Jansen et al., 1995).

Though limited in scope and generalizability, this area of research suggests a positive relationship between maternal antisociality and child behavior problems. Additional research is needed to examine this relationship as well as the relationship between maternal antisociality and developmental status of the preschoolers.

Maternal Smoking and Child Development

The majority of the studies examining the effects of postnatal, maternal smoking focus on the effects of exposure on children's health, specifically respiratory ailments. However, examination of children's saliva has shown that children are absorbing the byproducts of tobacco in body systems other than the respiratory system. A study of over 500 school-aged children found that children of smokers had a mean continine concentration of 3.38 ng/ml compared to children of non-smokers (0.44 ng/ml) with mothers' smoking affecting the children's continine levels more strongly than fathers' (Jarvis et al., 1985). Children of mothers who smoked had a continine level equivalent to

smoking 50 cigarettes per year compared to an equivalent of 30 cigarettes per year for children of smoking fathers. Exposure to second-hand smoke is more detrimental to young children for two reasons; the immaturity of their bodily systems and the dose of second-hand smoke is large in relation to their body size (Tong & McMichael, 1992).

Based on the statistics that most people start smoking before the age of twentyone years (USDDH, 1990) and that only approximately one quarter of the women quit smoking during pregnancy (Condon & Hilton, 1988; Hilton & Condon, 1989), it is difficult to study the effects of postnatal maternal smoking on child development without the confounding variable of prenatal exposure. A study previously reviewed in this chapter examined the effects of postnatal exposure to maternal smoking while controlling for the effects of prenatal exposure (Bauman et al., 1991). This study found that children whose mothers smoked postnatally or continued to smoke after the pregnancy scored lower on measures of reasoning and vocabulary than those children whose mothers smoked only during their pregnancy or never smoked (Bauman et al., 1991).

Another study reviewed previously examined the effects of postnatal exposure to maternal smoking and child behavior problems (Weitzman et al., 1992). When mothers smoked less than one pack of cigarettes per day postnatally, they were more likely to describe their children as headstrong and hyperactive compared to children whose mothers did not smoke (Weitzman et al., 1992). This study found that the following child characteristics, antisociality, immaturity, and depression as well as a greater risk of exhibiting extreme behavior problems (odds ratio of 2.01) were positively related to mothers smoking more than one pack per day postnatally.

Though the amount of literature in the area of postnatal effects of smoking on developmental status and child behavior problems is small, it does suggest that a possible trend of maternal smoking being negatively related to cognitive and language development and positively related to behavior problems may exist. These are areas that need to be explored further.

Maternal Alcohol Abuse and Behavior Problems

The effects of maternal postnatal drinking on preschool children is also a small area of study that has only found effects on child behavior problems. Generalizability of the results of the four studies reviewed for this section are limited due to sampling selection. Only two studies (Brown et al., 1991; Dawson, 1992) are drawn from the general population with one being a longitudinal prenatal study (Brown et al., 1991) that also examined current, postnatal drinking by the mothers. Of the remaining studies, one used a treatment population of children with Aggressive Conduct Disorder (Hamdan-Allen, Stewart, & Beeghly, 1989) and the other involved a sub-sample of children from families with an alcoholic father (Fitzgerald et al., 1993). All of these studies show a relationship between maternal, postnatal drinking and increased behavior problems.

A small study (n = 68) of five year olds discussed previously (Brown et al., 1991) found that current, maternal drinking accounted for the variance measured in internalizing behavior problems as well as a decrease in attention levels during a Continuous Performance Task.

Another study of mothers' current drinking found a relationship between mothers' diagnosis of alcohol dependence in the past year and child behavior problems. This study of children, ages five to eleven years, found that having a mother who was diagnosed as

alcohol dependent increased the children's risk of being rated by their mothers as having serious behavior problems (odds ratio of 2.19) (Dawson, 1992). A child was classified as having serious behavior problems if his/her mother endorsed 15 or more of the 28 items in a checklist of behavior problems as having occurred in the past three months.

A study conducted on three year old sons of male alcoholics and controls found that mothers' current as well as past drinking habits were related to behavior problems exhibited by their child. In this study, the mothers' Lifetime Alcohol Problems score (LAPS) was positively related to the number of internalizing and externalizing behavior problems endorsed by the mother on the Child Behavior Checklist List (Fitzgerald et al., 1993).

Lastly, a retrospective study of school-aged children in treatment for severe cases of aggressive conduct disorder also found a relationship between child behavior problems and mothers' postnatal drinking that was diagnosis dependent. In this study, children were in treatment for either Pervasive Aggressive Conduct Disorder (PACD), a diagnosis based on aggressive behavior exhibited at both school and home or Situational Aggressive Conduct Disorder (SACD), a diagnosis based on aggressive behavior being exhibited in only one of the settings. Children diagnosed as PACD were more likely to have a mother who abused alcohol than either children diagnosed as SACD or the controls (Hamdan-Allen et al., 1989).

A positive relationship between maternal drinking and child behavior problems has been shown in several studies drawn from different populations. In the past, more attention has been given to the effects of prenatal drinking by the mother and postnatal drinking by the father. Additional research is warranted with emphasis on mothers'

current as well as past drinking behaviors beginning after the delivery of her child and continuing through the entire postnatal period.

Additional Maternal and Familial Postnatal Influences on Child Development

Family Socioeconomic Status, maternal education, and maternal IQ have been shown in numerous studies to affect child development. These familial and maternal variables have been shown to be positively related to the developmental status of children and negatively related to child behavior problems. Child IQ has been shown to be positively related to maternal IQ (Bouchard & McGue, 1981), maternal education (Bee et al., 1982; McGowan & Johnson, 1984), and family SES (Nichols & Anderson, 1973; Silva & Fergusson, 1976). Child behavior problems has been shown to be negatively associated with maternal IQ (McGee, Silva, & Williams, 1984), family SES (Fitzgerald et al., 1993; Hamdan-Allen et al., 1989).

Research on risk and protective factors of childhood have used these variables to determine their effect on child development (Sameroff, Seifer, Baldwin, & Baldwin, 1993). Risk is defined as the low end of the ranges of these variables and increased risk is associated with the accumulation of more than one risk factor. Children are perceived to be at risk for lower IQ scores and higher number of behavior problems if they live in poverty, have a mother who did not finish high school and who has lower mental abilities (Liaw & Brooks-Gunn, 1994; Sameroff et al., 1993). On the other hand, children at risk for developing a wide range of problems may be protected by the higher end of these variables. For example, a child who lives in poverty but has a mother with high mental abilities that has finished high school may be less likely to drop out of school than

another child who is also living in poverty but whose mother has lower mental abilities and did not finish high school.

Maternal education has been shown to be positively related to IQ and language development using a variety of measures with samples drawn from the United States and other countries. In the United States, maternal education is associated with IQ measured by the Stanford Binet during preschool and performance IQ measured by Wechsler Preschool and Primary Scales of Intelligence- Revised (WPPSI-R) and WISC in the early elementary years (Bee et al., 1982; McGowan & Johnson, 1984). A positive relationship was also found for Israeli five year olds using the Peabody Picture Vocabulary Test and a measure of academic competence (Auerbach, Lerner, Barasch, & Palti, 1992). Maternal education has been shown to be related to the following specific areas of preschooler and early elementary school children's language development: expressive, receptive, and spoken language as measured by the Peabody Test, Test of Language Development (TOLD), and the Sequenced Inventory of Communication Development (Bee et al., 1982; Walker, Greenwood, Hart, & Carta, 1994).

Though maternal education has been shown to be positively related to child IQ and language development, it may indirectly influence child development through its association with parenting practices, specifically the quality of the home environment. Quality of the home environment has been shown to be positively related to maternal education as well as the other variables of family SES, and maternal IQ in numerous studies throughout the past thirty years (Bradley, 1993). In addition to the relationship with the home environment, specific types of parenting practices are related to economic characteristics of the family. For example, authoritarian and inconsistent parenting styles

are more commonly found in lower SES families (Darling & Steinberg, 1993). The influences of parenting on child development will be explored further in the next section. Summary of Maternal and Familial Influences during the Postnatal Period on Child Development

Though the literature is often sparse and limited in generalizability, there are relationships and trends for these maternal and familial influences on child development. Mothers who are depressed and who smoke as well as have a low IQ, and low levels of education, and SES are more likely to have children who are experiencing delays in cognitive and language development. Mothers who are depressed, antisocial, and who smoke and abuse alcohol are more likely to have children who exhibit internalizing and externalizing behavior problems. This relationship also exists for mothers who have lower IQs and for children who live in a family that has little money and social prestige. Several of the studies reviewed have suggested that parenting practices may mediate the relationship between maternal depression, maternal IQ, family SES, and family income and both child outcomes of behavior problems and IQ.

Postnatal II: Parenting Influences

During the past sixty years, research studies have been examining the influences of parenting practices on child development. This area of research was altered by Baumrind's theoretical model (1968) and subsequent typologies of parenting that stressed one broad aspect of parenting, control, and defined parenting as a parent characteristic and not a characteristic of the parent-child relationship (Darling & Steinberg, 1993). Present research in this area continues with these assumptions and adds to them the ideas

of responsiveness including such concepts as involvement and affect as well as demandingness which includes the ideas of conformity and rigidity.

While Baumrind was changing the study of parenting, researchers such as Bloom and Caldwell were exploring the effects of early experiences and the home environment on child development (Bradley, 1993). Building upon past research findings, Caldwell and colleagues created an objective measure of the home environment called the Home Observation for the Measurement of the Environment (HOME) that has spurred a large amount of research in the area of child development and health. The HOME focuses on the child within the environment and measures the inputs, events, and transactions occurring with the child in this specific context.

The following sections will examine the effects of parenting practices, parenting attitudes, and environmental stimulation on the developmental status and behavior problems of preschoolers.

Parenting Influences on Developmental Status and IQ

Parenting practices that are characterized by warmth, acceptance, and encouragement of independent and competent behaviors were found to be associated with higher scores on cognitive and language tests during the preschool years and better test performances and reading and writing skills in the early elementary school years (Bee et al., 1982; Belsky & MacKinnon, 1994). Child IQ, as measured by the Stanford Binet, was positively related to maternal attitudes of independence and reciprocity at age three years among Mexican-American children (McGowan & Johnson, 1984). In a longitudinal study of children, from four months to four years of age, mother's actions during a teaching task with her three year old child were related to the child's current

language development and later IQ. Mothers who used positive messages, encouraged task facilitation, and used non-intrusive techniques when assisting their children, had children who scored higher on the subscales of receptive and expressive language on the Sequenced Inventory of Communication Development at age three years and had higher IQs based on the McCarthy Scales of Children's Abilities at age four years (Bee et al., 1982).

On the other hand, parental attitudes of rigidity and conformity and an authoritarian parenting style were negatively related to IQ. Parents of early elementary children who valued conformity in their children and whose parenting techniques were rigid and non-flexible, had children with lower IQs as measured by the WPPSI-R (Sameroff et al., 1993). Similar results were found with a sample of four year old children from New Zealand, in which a negative relationship was found between the adherence to the authoritarian style of parenting and child IQ (Silva & Fergusson, 1976).

Quality of the home environment as measured by the HOME has also been found to be positively related to IQ and language development (Bradley, 1993). Across a variety of studies, HOME scores were found to be moderately related to IQ beginning at age two years and continuing through the age of seven years (Auerbach et al., 1992; Bee et al., 1982; Bradley, 1993). The subscales of learning materials, language stimulation, responsivity, academic stimulation, and variety are more strongly correlated across this age span compared to the other subscales of physical environment and modeling. Early elementary school performance in the areas of math and language arts are associated with higher scores on the subscales of learning materials and responsivity (Belsky & MacKinnon, 1994; Bradley, 1993).

An association between HOME scores and language development has been found using a variety of measures. In a longitudinal study of children from infancy to four years of age, HOME scores from each age period were predictive of both receptive and expressive language measured at age three years with the Sequenced Inventory of Communication Development (Bee et al., 1982). Vocabulary scores on the WPSSI were found to be positively related to scores on the HOME for children ages three to five years (Jordan, 1978). Lastly, verbal IQ measured by the WISC-R was predicted by higher HOME scores for a group of five to eight year old Mexican-American children (McGowan & Johnson, 1984).

In summary, mothers who are warm, accepting, flexible, non-intrusive, and encouraging of independent and competent behaviors in their children have children who score higher on IQ tests, who have advanced language skills, who do well in math and language arts in school, and who perform well on academic tests. Likewise, children who live in homes with an abundance of age appropriate toys and materials, where they are allowed to partake in a variety of experiences, and with a mother who is responsive to their needs and encourages them to talk, score higher on IQ tests and measures of language development.

Parenting Influences on Behavioral Problems

Parenting practices that are characterized by disapproval, negativity, and nonacceptance were found to be positively related to behavior problems in children. In a comparison study of two groups of elementary children, one diagnosed with ADHD and one without a diagnosis, it was found that boys with ADHD had mothers who expressed more negative parenting practices (Anderson, Hinshaw, & Simmel, 1994). During a taskorientated session, mothers were told to supervise their sons doing specific tasks. Mothers who were rated by observers to be discouraging, show disapproval, and be nonaccepting of her son's behavior had sons who were later rated by observers to be noncompliant in a playground setting and more likely to steal in a laboratory situation (Anderson et al., 1994). Non-compliance included disobedience, rule violations, and defying authority. Another study utilizing data from the Berkley Guidance Study of four generations, found that inconsistent and extreme punishment by parents was related to increased levels of aggression and delinquency among children ages three to eight years (Elder et al., 1983).

HOME scores were found to be negatively related to child behavior problems. More specifically, homes of children with behavior problems were characterized as having few appropriate learning materials and low levels of maternal involvement (Bradley, 1993). In Ireland, a small study (n = 52) of three and four year olds also found deficiencies in the home environment that were related to behavior problems in the children (Mulhall, Fitzgerald, & Kinsella, 1988). Specifically, low levels of warmth, acceptance, and affection as measured by the HOME were positively related to the number of behavior problems reported by the mothers.

A longitudinal study found that low levels of maternal warmth predicted child externalizing behavior problems several years later. Data from wave one and two of a longitudinal study of children of alcoholic and non-alcoholic fathers found low levels of maternal warmth at T1 (when the sons were preschoolers) predicted higher number of child externalizing behavior problems at T2 (when the sons were early elementary) (Wilen, 1997). Additional analyses showed that maternal warmth mediated the

relationship between maternal current depression and externalizing child behavior problems.

Based on the previously reviewed studies, children with behavior problems are more likely to have a mother who is disapproving, non-accepting, and offers little warmth and affection and to come from homes characterized by lack of age appropriate toys and material.

The Present Research

Based on the review of the literature and the purpose of the study stated in Chapter one pages one and two, specific research objectives and hypotheses were developed to guide the present research of multiple risk effects. This section will state the research objectives and hypotheses. This will be followed by a discussion on how the results and recommendations as well as the shortcomings of the previously reviewed studies were drawn together to guide the present research.

Research Objectives

In order to achieve the purpose of the study, more specific objectives were developed and were used as guides for this research:

- To determine whether maternal smoking and alcohol use during pregnancy predict later behavior problems, developmental status, and IQ of preschoolers.
- To determine whether maternal smoking and alcohol use, psychopathology, and adaptation during the postnatal period predict behavior problems, developmental status, and IQ of preschoolers.

- To determine whether maternal parenting attitudes and environmental stimulation (parenting behaviors) predict behavior problems, developmental status, and IQ of preschoolers.
- 4) To determine whether prenatal exposure to alcohol and nicotine persist and predict behavior problems, developmental status, and IQ of preschoolers or whether current maternal consumption of alcohol and cigarettes are more influential than prenatal exposure in predicting behavior problems, developmental status, and IQ of preschoolers.
- 5) To determine whether maternal life history of alcoholism, antisociality, smoking, and depression influence mothers' current levels of alcohol use, antisociality, smoking, and depression as well as parenting attitudes and environmental stimulation (parenting behaviors) which in turn influence behavior problems, IQ, and language development of their children.

Hypotheses

In order to accomplish the objectives of this study, several hypotheses were tested.

- Mothers who smoked and used alcohol during their pregnancy are more likely to have preschoolers with behavior problems, less advanced developmental status and lower levels of IQ.
- 2) Mothers who currently smoke and use alcohol, who are depressed and antisocial, and who have lower levels of education, intelligence, and socioeconomic status are more likely to have preschoolers with behavior problems, less advanced developmental status and lower levels of IQ.

- Preschoolers from homes with lower levels of environmental stimulation and whose mothers adhere to negative parenting attitudes will exhibit more behavior problems and have less advanced developmental status and lower levels of IQ.
- Mothers' current smoking and alcohol use will be better predictors of preschoolers' behavior problems, developmental status, and IQ than mothers' smoking and alcohol use during pregnancy.
- 5) Mothers with a life history of alcoholism, antisociality, smoking and depression will be more likely to currently be alcoholic, antisocial, depressed and smoke and they will also have more negative attitudes about parenting and will provide a less stimulating environment for their preschoolers than mothers who do not have a life history of alcoholism, antisociality, smoking and depression and are not currently alcoholic, antisocial, depressed and smoke and thus the former mothers' preschoolers will exhibit more behavior problems and have lower levels of IQ and language development.

Discussion of Review of Literature

Several important issues can be gleaned from the review of the current research on prenatal exposure to alcohol and smoking and the postnatal influences of maternal and familial characteristics on child development. The first one is the importance of measuring all areas of development status: motorical, language, and cognitive performance, and an overall measure of IQ as well as behavior problems including measures of internalizing and externalizing behaviors, impulsivity, and attention. By measuring these specific areas of developmental status along with an overall measure of IQ and a number of behavior problems, a more complete picture of the possible deficits experienced by children prenatally exposed to maternal smoking and alcohol use as well as the possible delays found in children of mothers who are depressed, antisocial, and drug involved was achieved.

Second, parenting behaviors (environmental stimulation) and attitudes are important components in children's development. In the prenatal literature, researchers have found that the quality of the home environment may be even more predictive of development at the preschool age than prenatal exposure to alcohol (Greene et al., 1990; Greene et al., 1991). Several of the reviewed studies in the postnatal literature have found that the relationship between maternal depression and child behavior problems is mediated by the effect of maternal depression on parenting practices (Downey & Coyne, 1990; Goodman & Brumley, 1989; Puckering, 1989; Richman et al., 1982; Rutter, 1990). It has also been suggested that parenting practices may influence the relationship between maternal depression and psychomotor development (Goodman & Brumley, 1990). In addition, a theoretical paper on the origins of antisociality in older children suggests that ineffective and negative parenting may mediate the relationship between maternal antisociality and child behavior problems and antisociality (Patterson, DeBaryshe, & Ramsey, 1989; Reid & Patterson, 1989). Parenting practices and quality of the home environment may also mediate the relationships between maternal IQ and level of education and the child outcomes of developmental status (Bradley, 1993), as well as the relationship between family SES and child behavior problems (Darling & Steinberg, 1993).

Third, when examining the effects of prenatal exposure to mothers' drinking and smoking, it is important to measure and possibly control for several important variables. When studying the effects of prenatal exposure to alcohol, researchers need to measure or control for current levels of maternal drinking. It is also important to control for social class when examining the effects of prenatal exposure to smoking on preschoolers' developmental status.

Fourth, the present research postulates several relationships between smoking and present parental psychopathology. Some researchers believe that smoking leads to depression. Physiological evidence cited by researchers to support this belief include abnormalities in thyroid functioning among smokers that are similar to those found in people with major depression and lower levels of natural antidepressants in smokers' blood (Newhouse & Hughes, 1991). This belief can also be supported temporally by the fact that between 80-90% of all adult smokers began smoking by the age of twenty-one years prior to or at the beginning of adulthood (USDHHS, 1990) and as adults 20% of smokers are clinically depressed (Breslau et al., 1993). This compares to the rate of depression for the general population which is only 9% (Puckering, 1989). Between one half and two-thirds of smokers express the desire to stop smoking but due to the psychologically and physiologically addictive nature of cigarettes and the uncomfortable withdrawal symptoms, only 44% of ever-smokers have quit for more than a year (USDHHS, 1990). There is also a high relapse rate among ex-smokers (Schuckit, 1995). It could be hypothesized that being unsuccessful in attempting to stop smoking could lead to an increase in depressive symptomology.

Finally, it should be noted again that the sample selection of the present study is from the MSU-UM Longitudinal Study of children at risk for alcoholism and antisociality due to fathers' diagnosis of these pathologies. Some background of the Longitudinal Study and its findings to date that pertain to the present study is helpful.

The family study began in 1982 and involved a pilot study of twenty-five families. The work has grown into a longitudinal study of over 300 families (See chapter three for information on sample selection) and includes data on the mother, father, and male target child and has more recently added data on a female target child and siblings for both male and female target children. In order to study these children at risk for the later development of alcoholism and alcohol abuse, there was a need to measure nonalcohol-specific factors that pertain to later alcohol use (Zucker, 1987; Zucker, Ellis, Bingham, & Fitzgerald, 1996a; Zucker, 1994; Zucker & Fitzgerald, 1994, Zucker & Fitzgerald, 1996).

A topological framework that allows for the examination of the heterogeneity of the developmental course of alcoholism was adapted early on in the study (Zucker, 1987). The classification of alcoholism with high levels of antisociality has been used prior to the MSU- UM Longitudinal Study by other researchers utilizing a variety of samples including Cloninger's (1987) and Hesselbrocks' work (Hesselbrock et al., 1984). Although earlier work (Zucker et al., 1994) from the MSU-UM Longitudinal Study involved utilization of the Cloninger typology, problems with that system lead to the decision to use other strategies in subtyping. This new approach established group membership based on a measure of both child and adulthood antisocial behavior (Zucker et al., 1996a); with classification of antisoical alcoholic based on the parents' scores.

This approach has identified two groups: antisocial alcoholics (AALs) and nonantisocial alcoholics (NAALs) with the classification of antisocial alcoholic being associated with earlier onset of alcoholism and more severe alcohol related difficulties (Zucker, Ellis, & Fitzgerald, 1994; Zucker et al., 1996a; Zucker et al., 1996b). Other differences include: AALs have a greater number of relatives who can be classified as alcoholics (family history of alcoholism), AALs experience more lifetime depressive symptoms as well as current depressive symptoms (Zucker et al., 1994; Zucker et al., 1996a), and AALs have lower levels of SES as well as lower paternal and maternal IQs (Zucker et al., 1996a). It is important to note that the NAALs represent a heterogeneous group (Zucker, 1994) whose purpose in the Longitudinal Study is to distinguish one type, the AALs from all other types that can not be distinguished further than NAALs because the Longitudinal Study's sample though large is not large enough to support the further classification of alcoholisms into "a finer-grained set of distinctions" and still maintain power in analyses.

Previous research from the Longitudinal Study has examined the paternal influences of alcoholism and antisociality on the maternal and familial characteristics as well as the child outcomes in this study. This research is summarized below for the benefit of the reader.

Antisocial alcoholic families are often characterized by assortative mating which aggregates the amount of risk experienced by the children (Zucker et al., 1996a). This increased risk is a result of a number of maternal characteristics including being linked to parenting patterns of lower nurturance (Ellis et al., under review). When comparing wives of AALs with wives of NAALs and comparison wives, it was found that the wives of AALs were found to drink more alcohol, be more depressed, be more antisocial, have

lower IQs, have fewer years of education, and have lower levels of adaptive functioning and income (Ellis et al., under review; Zucker et al., 1996a). Both wives of AALs and NAALs were more likely to have higher levels of lifetime alcohol problems and worst– ever depressive episode compared to comparison wives (Zucker et al., 1996a).

The same pattern of characteristics were found when comparisons were made between wives of court alcoholics⁹ with wives of community alcoholics and wives of comparisons. It was found that wives of court alcoholics were more antisocial, depressed, and experienced more economic and legal problems (Ichiyama et al., 1995). Wives of court alcoholics were also found to provide a less stimulating home environment for their preschoolers when compared to wives of non-alcoholic men (Noll, Zucker, Fitzgerald, & Curtis, 1992).

Additional data on only court alcoholic families shows trends of aggression and low nurturance. A study of violence towards children found that the severity of maternal aggression towards her son was positively related to the mother's Lifetime Alcohol Problems Score, antisociality, worst-ever depressive episode and current depression (Reider et al, 1996). The mean level of aggression for these mothers was threatening to hit or throw something at the child. Another study found that high levels of maternal antisociality predicted low levels of nurturance in court alcoholic families (Davis, et al., 1991).

Differences between groups were also found in child outcomes. When comparing children in antisocial alcoholic, non-antisocial alcoholic, and comparison families, it was

⁹ 58.5% of the court recruited alcoholics were classified as antisocial alcoholics (AALs) and 24.5% were classified as nonantisocial alcoholics (NAALs) (Bingham, Fitzgerald, Townsend, & Zucker, 1995; Zucker et al., 1994).

found that children in antisocial alcoholic families exhibited more behavior problems both internalizing and externalizing (Bingham, Fitzgerald, & Zucker, 1996b; Ellis et al., under review), were rated as more hyperactive (Ellis et al., under review), and scored higher on a risky temperament index measuring high activity level, emotional reactivity, and approach to life situations (Zucker et al., 1996a). Children in non-antisocial alcoholics were similar in the aforementioned areas to children in comparison families, except that children in non-antisocial alcoholic families exhibited more externalizing behavior problems (Zucker et al., 1996a). Another study found that preschool boys of court alcoholics scored significantly lower than matched controls on the Revised Yale Developmental Schedules overall developmental quotient, as well as the subscales of fine motor and personal/social behavior, after controlling for chronological age, family SES, and HOME score (Noll et al., 1992). Children in court alcoholic families were also more likely to be impulsive and to be rated in the extreme clinical range for behavior problems (Fitzgerald et al., 1993).

It is clear from these studies that there exists a positive relationship between paternal antisociality and alcoholism and both maternal psychopathology and child behavior problems. There also exists a negative relationship between paternal antisociality and alcoholism and both maternal parenting practices and child outcomes of IQ, developmental status, and behavior problems. Therefore, this study will add to the knowledge of this sample by focusing on maternal influences and their effects on child development.

Specifically of interest to this researcher are the effects of maternal influences during the prenatal period of development, the maternal characteristics of depression,

antisociality, substance abuse, IQ, and education, the familial characteristics of family expression of alcoholism and SES, and the influences of parenting behaviors (environmental stimulation) and attitudes on preschoolers' IQ, developmental status, and behavior problems.

CHAPTER 3

Method

Research Objectives and Hypotheses

The research objectives and questions were stated in Chapter two pages 62 to 64. These objectives and hypotheses defined the focus of the study.

Subjects

The sample was drawn from the first wave of a longitudinal study of alcoholism and its influence on child development (n = 308 families) (Zucker et al., 1986; Zucker & Fitzgerald, 1994; Zucker & Fitzgerald, 1996). This ongoing study utilizes a populationbased sample of alcoholic men and their families, along with a comparison group of nonalcoholic families.

Alcoholic families were located using two methods. First, families of convicted, male, drunk drivers in the Mid-Michigan area were referred to the study by probation officers (court referred group; n = 157). In order for the families to be asked to participate in the study, the alcoholic father had to have a blood alcohol level (BAL) of at least .15% (150 mg / 100 ml) for first time offenses or .12% (120 mg / 100 ml) if the current arrest was not the first alcohol-related arrest. Second, families of alcoholic fathers were recruited by neighborhood canvassing of households in the Mid-Michigan area (community alcoholic group; n = 60). In order for either the court or community recruited alcoholic families to be included in the study, they had to meet the following criteria: the father had to be diagnosed as probable or definite alcoholism based on Feighner criteria (Feighner et al., 1972); the father had to be living with a biological male

child between the ages of 3.0 and 6.0; and the family had to be intact (non-divorced and non-separated) at the time of the first contact. The fathers' alcohol diagnosis was later confirmed during two separate visits, first with the Short Michigan Alcohol Screening Test (SMAST: Selzer, 1975) and then, with the Diagnostic Interview Schedule (DIS: Robins, Helzer, Croughan, & Ratcliff, 1981).

A comparison group of non-alcoholic families were also recruited by neighborhood canvassing in the same area (n = 91). In order to be considered for participation in the study, both the father and mother had to be confirmed as nonalcoholic (according to Feighner criteria for alcoholism) and non-illicit drug abusing/dependent and the family was intact. These families were then matched to alcoholic families based on the following criteria: the families had to have male sons who were within six months of age from each other; and the families had to have similar SES based on living in the same census tract. The latter criteria was expanded to include similar census tracts due to the prevalence of drug and/or alcohol abuse in certain neighborhoods. The families in this study are characterized as lower-middle class, Euro-American.

Demographic Characteristics

The present study sample (n = 307 families) consisted of mothers and their sons from all three recruitment groups: court referred alcoholics, community alcoholics, and comparisons. Sociodemographic characteristics of the sample are presented in Table 1. The measure of socioeconomic status (SES) used in this study was the Revised Duncan Socioeconomic Index (Stevens & Featherman, 1981) described in the Measures Section. In this sample the mean occupational attainment score was 341.9. This score is reflective

of lower level white-collar occupations such as a skilled office worker. Scores range from 130.0 (unemployed) to 885.0 (physician).

Variable	Minimum	Maximum	Mean	Standard Deviation
Family SES	130	874	341.9	150.8
Education (yrs)				10010
mothers	9	20	13.2	2.0
fathers	7	20	13.5	2.3
Mother's age at				
birth of child	16.79	41.83	26.51	4.32
Child's age	2.3	7.1	4.41	1.06

Table 1.Sociodemographic Characteristics of the Sample (N = 307)

Data Collection

Data were collected by trained project staff who were blind to family risk status. Because a large volume of data were collected from each family, multiple contacts were necessary. Wave One data were collected during eight separate contacts with each family, six of which took place in the family home and two of which took place on the campus of a large local university. The visits involved approximately 11 hours with each parent, and seven hours with each target child. Contacts included questionnaire sessions, semi-structured interviews, and interactive tasks. The families were assured that the study was not connected to the court system and that all data they provided would be confidential. They received financial compensation for their participation.

Measures

For the present study, the independent variables were measures of maternal, familial, and parenting influences on child development (see Table 2).

Table 2. Var	Variables of Interest for Present Research.	ch.
Variable Group	Specific Variables	Measurement of Variables
Independent Variables Maternal Influences	past smoking	Diagnostic Interview Schedule (DIS), Drinking and Drug History (DDH)
	past alcohol use	DIS, DDH, Lifetime Alcohol Problems Score, SMAST
	smoking during pregnancy	Health History Prenatal Form
	alcohol use during pregnancy	Health History Prenatal Form
	present smoking	Drinking and Drug History
	present alcohol use	Drinking and Drug History
	worst ever depression	Hamilton Rating Scale for Depression
	current depression	Beck Depression Inventory, short form
	adaptive functioning	DSM-III-R Axis V, Global Assessment of Adaptive Functioning
		Scale
	intelligence	Wechsler Adult Intelligence Information and Digit Symbol Scales
Familial Influences	family expression of alcoholism socioeconomic status	Family Expression of Alcoholism Revised Duncan Socioeconomic Index
, , ,		
Parenting Influences	parenting attitudes environmental stimulation	Child Rearing Practices Report Home Observation for Measurement of the Environment Scale
Dependent Variables		
Child Functioning	child behavior problems	Child Behavior Checklist, Dimensions of Temperament,
		Delay of Gratification Task
	developmental status child IQ	Revised Yale Developmental Schedules Stanford-Binet Intelligence Scale

The maternal variables included past smoking and alcohol use, smoking and alcohol use during pregnancy, current and worst ever depression, lifetime antisociality, present alcohol use and smoking, level of education, and intelligence. Family expression of alcoholism and socioeconomic status were the familial variables. Parenting attitudes and environmental stimulation were the parenting variables. The dependent variables were child behavior problems, developmental status, and IQ. This section will summarize the specific measures used in this study and define the concepts the measures assess.

Maternal Independent Measures

<u>Past Smoking and Alcohol Use</u> The Diagnostic Interview Schedule (DIS; Robins et al., 1981) is a structured interview that obtains physical, alcohol and drug related, and mental health (symptomatic) information. For this study only those sections pertaining to smoking behaviors and alcohol use are discussed. The DIS examines the onset, frequency, and cessation of smoking and drinking behaviors as well as physical problems associated with smoking and drinking and other problems related to alcohol use. A measure of smoking dependency was computed by summing the following variables: the squared inverse of the age at which the respondent first started to smoke regularly, the number of years the respondent has smoked, and the numeral ten to insure a positive number. This variable of smoking dependency was found to be predictive of the measure of current smoking, smoking in the past month (r = .41, p < .000).

The Lifetime Alcohol Problems Score (LAPS; Zucker, 1991; Zucker, Davis, Kincaid, Fitzgerald, & Reider, 1997) measures the extent of alcohol-related difficulties over the lifetime by examining the onset (primacy), variety, and life invasiveness of problems associated with drinking. The information is compiled from three different

sources; the Drinking and Drug History (DDH), the DIS (Robins et al., 1981), and the short Michigan Alcoholism Screening Test (SMAST; Selzer, 1975) and computed into three subscores: primacy, variety, and life percent. These subscores are derived using the following information: primacy is the squared inverse of the age at which the respondent reported first drinking enough to get drunk; variety involves the number of areas in which drinking problems are reported; and the life percent is a measure of the intervals between the most recent and earliest drinking problems, corrected for current age. The scores are standardized with high scores indicating greater severity of lifetime alcohol problems. This measure is unrelated to current alcohol consumption patterns and has been shown to differentiate between DSM-III-R levels of alcohol dependence, abuse, and no abuse (Zucker, 1991; Zucker et al., 1997). LAPS was also found to be positively related to antisociality, current and worst-ever depression, and family violence including both spousal and child (Zucker et al., 1997).

Smoking and Alcohol Use Prenatally The Health History--Prenatal Form (HHPF; Carpenter & Lester, 1980) was used to obtain information on mothers' smoking and alcohol use during pregnancy. The Prenatal form of the questionnaire assesses maternal health, illness and drug use (including illicit and prescription drugs), length of gestation, difficulties during labor and method of delivery as well as problems and illnesses during the neonatal period, mediations or treatment for the target child during the neonatal period, and the method and length of feeding (bottle or breast). The HHPF asks the mothers how many cigarettes they smoked per day during the pregnancy and how much they drank per day and week. Two variables were used, number of cigarettes smoked per day and number of alcoholic drinks drank per week.

<u>Current Smoking and Alcohol Use</u> The Drinking and Drug History (DDH) questionnaire provides detailed information on current consumption of alcohol and smoking patterns and problems related to excessive alcohol use. The DDH is comprised of items from the 1978 National Institute on Drug Abuse Survey (Johnston, Bachman, & O'Malley, 1978), the American Drinking Practice Survey (Cahalan, Cisin, & Crossley, 1969), and the Veterans Administration Medical Center Research Questionnaire for Alcohol (Schuckit, 1978). Questions pertaining to patterns of alcohol use include data on quantity (Q), frequency (F), and variability (V) of consumption. This information is then computed using z-scores into QFV-R indexes of heavy, moderate, light, and infrequent drinker and abstainer (Cahalan et al., 1969).

The DDH asks the respondent if they ever smoked and how much they smoked in the past year and month. These questions have predetermined categories on the questionnaire for a response to each question. Only the question pertaining to smoking in the past month has categories based on number of cigarettes instead of regularity of smoking. Therefore, only the variable smoked in the past month was used in this study. It was coded as 0 = never smoked, 1 = less than one cigarette per day, 2 = 1-5 cigarettes per day, 3 = a half a pack per day, 4 = one pack per day, 5 = one and one half packs per day, 6 = two or more packs per day.

<u>Antisociality</u> The Antisocial Behavior Checklist (ASB; Zucker & Noll, 1980) was used to measure maternal antisociality. This 46-item questionnaire measures the frequency of the mother's participation in a variety of delinquent, criminal, and antisocial activities in child- and adulthood. Responses range from 0 = never to 3 = often. The items are summed for an overall measure of antisociality. The ASB has an adequate test-retest reliability of .91 over four weeks and an internal consistency coefficient alpha of .93 (Zucker & Noll, 1980). It has been shown to differentiate between inmates, minor offenders in the district court system, and university students (Zucker & Noll, 1980) as well as differentiating between the alcoholic and non-alcoholic males (Fitzgerald, Jones, Maguin, Zucker, & Noll, 1991; Ham, Zucker, & Fitzgerald, 1993b).

Depression Mothers' current depression was measured by the short form of the Beck Depression Inventory (BDI; Beck, 1961). This 13-item self-report instrument assesses concurrent cognitive, emotional, motivational, and physical manifestations of depression. By emphasizing the psychological aspects of depression, this measure tends to be more sensitive to moderate levels of depression than more profound states. Respondents endorse items on this questionnaire on a scale of 0 to 3, with a higher score indicating more depression. Depending upon the population, internal consistency reliability ranges from .73 to .92, with a mean alpha of .81 for nonpsychiatric subjects. The BDI has been shown to have adequate validity. Scores on the short and long forms of the BDI have been found to correlate between .89 and .97 (Beck, Steer, & Garbien, 1988).

Mother's most severe depressive episode was assessed using the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960; Hamilton, 1967). Ratings on levels of current and worst ever for a variety of behavioral, affective, somatic, and psychological dimensions of depression were made by a clinician during a semi-structured interview with the mother. Depending on the item, the ratings range from 0 to 2 or 0 to 4. Using Hamilton's scoring instructions, a score for worst ever depressive episode was computed

from the sum of the first seventeen items of this 24 item scale. Inter-rater reliability for the HRSD ranges from .80 to .90 (Hamilton, 1960).

<u>Adaptive functioning</u> Maternal adaptive functioning was measured using the DSM-III-R Axis V, Global Assessment of Adaptive Functioning Scale (GAF; American Psychiatric Association, 1987). This measure evaluates the mother's highest level of functioning during the previous year. The GAF was scored immediately following the administration of the DIS and measures the aspects of social/personal competence that may be semi-independent of symptomatic status. It has been shown to be inversely related to alcohol abuse/dependence diagnosis and drug use (Gonzalez, Zucker, Fitzgerald, 1993a; Gonzalez, Zucker, & Fitzgerald, 1993b). Inter-rater reliability for the GAF scoring in the MSU-UM Longitudinal Study has been established at r = .85.

Intelligence Maternal intelligence was measured using the Wechsler Adult Intelligence Information and Digit Symbol Scales (Wechsler, 1955, 1981). The scores on the scales were multiplied by a constant to obtain prorated estimates of adult IQ. The Information scale measures general knowledge, verbal skill, and mental alertness. It is reliable (coefficient $\alpha = .88$) and strongly correlated with the full-scale IQ (r = .83; Wechsler, 1981). The Digit Symbol scale measures attention, visual-motor coordination, motor persistence, and response speed. It is also reliable (coefficient $\alpha = .86$) and correlates moderately with the full-scale IQ (r = .61; Wechsler, 1981).

<u>Level of Education</u> The Demographic Questionnaire assesses basic background information on self and family of origin, including, age, education, income, number of marriages, number of children, etc. The mothers were asked on the Demographic questionnaire to identify the highest grade completed or the number of years she attended after high school graduation. The answers were coded as 01= first grade to 12= twelfth grade and 13=1 year after high school etc.

Familial Independent Measures

<u>Family Expression of Alcoholism</u> The Family Expression of Alcoholism (FEA) is a computed score based on the data collected from the genogram interview (Ellis, 1994). During the interview the mother reported her family history of a variety of medical and psychological diagnoses and problems found in her immediate and extended family which is defined as her mother, father, siblings, grandparents, aunts, uncles, and first cousins. Based on the genogram data, a weight equivalent to the coefficient of relationship value for each alcoholic family member was assigned with a .5 value assigned to first degree relatives, .25 value for second degree relatives, etc. FAE scores were then computed as follows: first, within each generation the weightings for all alcoholic relatives were summed; second, the sum computed in the first step was multiplied by the ratio of alcoholics to the total number of relatives in that generation; and third the subscores across the generations were summed. Thus, the FAE score reflects the density of alcoholism in the subject's immediate and extended family as well as the degree of relatedness of these family members to herself (Zucker et al., 1994).

<u>Family Socioeconomic Status</u> The Revised Duncan Socioeconomic Index (Stevens & Featherman, 1981) was used to measure family socioeconomic status. The Duncan measures social prestige and economic rewards of occupations and is based on self-reported type of work, most important work-related duties, and the kind of business or industry where the subject is employed. A higher score indicates higher prestige and salary. Due to the Index's occupation-based approach, it is consider a better measure of

SES than previous measures that did not account for occupational prestige (Mueller & Parcel, 1981).

Parenting Measures

Parenting Attitudes The Child Rearing Practices Report (CRPR; Block, 1965) was used to measure maternal parental attitudes with respect to child rearing. The CRPR uses a forced-choice procedure to have parents rate 91 statements as most descriptive to least descriptive of their attitudes towards childrearing. The parents sort the cards into seven categories of 13 items each. The CRPR is comprised of 21 scales which provide information on four primary domains of socialization: 1) child's autonomy, independence, and emergence self, 2) parental authority, discipline, and control strategies, 3) parental goals and aspirations for their child, and 4) expression of emotion in parent-child interactions (Susman, Trickett, Iannotti, Hollenbeck, & Zahn-Waxler, 1985). The CRPR has been found to provide an evaluation of current parental socialization practices and to predict the future adaptation of children (Block, Block, & Keyes, 1988).

For the present study three scales were used, positive affect, negative affect, and openness to experience. The scale for positive affect included the following statements: I feel a child should be given comfort and understanding when he is scared or upset; I express affection by hugging, kissing, and holding my child; I joke and play with my child; and my child and I have warm, intimate times together. The negative affect scale included the following items: I often feel angry with my child; I feel my child is a bit of a disappointment to me; and there is a good deal of conflict between my child and me. Finally, the openness to experience scale included the following statements: I encourage

my child to wonder and think about life; I feel a child should have time to think, daydream, and even loaf sometimes; I encourage my child to be curious, to explore, and question things; and I encourage my child to talk about his troubles. A measure of relatedness was calculated for each of these three scales for the present study. The alphas were .74 for the positive affect scale, .70 for the negative affect scale, and .64 for the openness to experience scale.

Parenting Behaviors/Environmental Stimulation The Home Observation for Measurement of the Environment Scale (HOME; Caldwell & Bradley, 1984) was used to measure environmental stimulation. The HOME is a measure of cognitive, emotional, and social stimulation provided to the child in their home. It is based on observations by trained clinical students and reports by the child's primary caregiver. The score is based on the sum of 55 items composing eight subscales, with the higher score indicating more stimulation in the home. The validity for varied groups, as well as reliability has been well established in the literature (Bradley et al., 1989; Bradley, Caldwell, & Rock, 1988; Hollenbech, 1978).

Child Outcome Measures

Behavior Problems The Child Behavioral Dysfunction Index (CBDI; Bingham et al., under review-a), an extension of the Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1983) was used to measure behavior problems and the severity of child behavioral dysfunction by utilizing both the mother's and father's scoring on the CBCL. The CBDI measures child behavior across different contexts thus offering a more complete characterization of the full range of child behavior problems. In order to understand the CBDI, the CBCL is discussed first.

The CBCL is a 113-item checklist in which the parent rates his/her child's behavior over the past six months. It provides an objective measure of the child's social and emotional functioning. It has been normed and standardized on children aged four to sixteen. The items range from 0 to 2, with a higher score indicating more behavior problems. The CBCL provides a total behavior problems score (TBPS), internalizing and externalizing behaviors scores, and eight narrow band behaviors indexes: withdrawn, anxiety depression, somatic complaints, social problems, thought problems, attention problems, aggressive behavior, and delinquent behaviors. In non-clinical population studies, the CBCL has been found to have a test-retest reliability of .84 for a three month interval and an interrater reliability of .79 (Achenbach & Edelbrock, 1983).

The CBDI used the CBCL data for both mothers and fathers and was calculated in a two step process. First, the number of contexts (mother-child and father-child) were counted in which the 113 behavior problems listed in the CBCL occurred. The occurrence of each behavior on the CBCL was indicated by either parent giving the behavior a rating of at least 1. Scores on any single behavior ranged from 0 to 2: 0 if neither parent gave the behavior a rating of at least 1; 1 if one parent gave the rating of at least 1; and, 2 if both parents gave the behaviors were summed. The CBDI was found to have good internal validity ($X^2(16) = 18.75$, p <.29; GFI = .99; CFI = 1.00), moderate construct validity (maternal model $X^2(236) = 354.97$, p <.000; GFI = .90; CFI = .96), and is internally consistent (Bingham et al., under review-a). For the present study, the TBPS and both the internalizing and externalizing behavior scores were used as dependent variables. Developmental Status The Revised Yale Developmental Schedules (RYDS; Provence & Naylor, 1983) was used to measure developmental status. The RYDS assesses child development in five areas; gross motor, fine motor, adaptive, language, and personal/social behavior. The items are a compilation of test items from the Stanford-Binet, Bayley, Gesell, Viennese, and Merrill Palmer scales. The assessment is completed by a trained clinician during a two hour intervention with the child in the child's home. It provides a developmental age for each of the five areas, as well as an overall evaluation of developmental status. In each of the five areas a baseline is obtained when the child has mastery of the items, thereafter, items are presented to the child until the child is no longer able to succeed and a ceiling is obtained.

IQ The Stanford Binet Intelligence Scale (Terman & Merrill, 1973) was used to measure overall intellectual functioning. The Stanford Binet has been revised three times, with the Third Revision competed in 1960 and renormed in 1972 using a representative sample. An overall IQ is obtained by comparing the subject's mental age obtained from the Stanford Binet with his or her chronological age. The Stanford Binet has been shown to be highly reliable and stable. It predicts academic achievement for children of majority population groups as well as minority children (Munday & Rosenberg, 1979).

Attention Span/Distractibility The Dimensions of Temperament (DOTS; Lerner, Palermo, Spiro, & Nesselroade, 1982) was used to measure attention span/distractibility. The DOTS is a 34-item true/false questionnaire in which the mother rates her child's temperament. It provides measures of activity level, attention span/distractibility, adaptability/approach/withdrawal, rhythmicity, and reactivity. Based on samples

utilizing infants, preschoolers, school-aged children, and young adults, reliability coefficients on the five scales ranged from .31 to .96, with reactivity the only factor that was consistently below .60 (Lerner et al., 1982a; Lerner, Belsky, & Windle, 1982b). The score on those subscale of interest was used in this research.

Impulsivity The Delay of Gratification Task (DGT; Funder, Block, & Block, 1983) was used to measure impulsivity. Following the child's intellectual assessment session, the examiner gives the child a present but insists that the child must complete one more complex task prior to opening the present. The examiner then times the child's delay interval and evaluates the child's behavior and performance on the task using Funder et al (1983) scoring procedures. Children who are able to delay on this task are described as being more attentive, able to concentrate, reasonable, and cooperative (Funder et al., 1983). For the present study, the number of seconds the child is able to delay will be used as the dependent variable.

Limitations of Sampling Design and Data Collection Techniques

The greatest limitation of the analyses concerns the information about mothers' smoking and alcohol use during their pregnancy. There are several reasons why the data to be used in this study are limited. One, the measure, Health History--Prenatal Form (HHPF) used to assess maternal health during the pregnancy and the neonate health is retrospective, completed by the mothers when the target children were preschoolers. The difficulty associated with this is that the mothers may not remember all of the information asked about a pregnancy that happened 4 to 7 years earlier, especially if she has had other subsequent pregnancies. The second reason the data are weaker is that there are no medical records to validate and add to the information collected on the HHPF.

There is another limitation of the present analyses, beyond the limitation of the prenatal data. The Home Observation for the Measurement of the Environment (HOME) was added later to the data collection protocol for the Longitudinal Study, consequently, 71 families did not compete the HOME during Time One data collection. A result of this and the recruitment strategy of identifying families through DUI offenses, the majority of the families that did not complete a HOME were alcoholic and possibly antisocial as well. This has been confirmed by the analysis which showed that the majority of these families have a father that has been classified by the principal investigators as an antisocial alcoholic (C. R. Bingham, personal communication, December 12, 1995).

Though the unavailability of the HOME data is known to occur more heavily in the AALs families, the missing data protocol discussed in the next section and in more detail in Appendix A addresses this concern thus attempts to reduce the possible adverse effects on the present study's analyses.

Lastly, the present study has defined its focus to be the examination of only maternal influences on child outcomes. The researcher is aware that there are other influences on child outcomes both distal and proximal including: paternal influences of alcoholism, antisociality, depression, smoking, psychological functioning, and parenting behaviors and attitudes, parental influences of aggression and genetic risk for alcoholism, and familial influences of cohesion and levels of stress as well as sibling influences and the more distal influences of neighborhood, community and society (Rende & Plomin, 1993; Rende & Plomin, 1990). These influences are valid areas to study and are possible areas to explore in the future. However, the researcher feels that examining maternal influences on child outcomes is also a valid study and the results of the present study

adds to the knowledge of the MSU-UM Longitudinal Study in which the focus has most often been to examine the paternal influences of alcoholism and antisociality on maternal, familial, and child outcomes.

Data Preparation and Statistical Analyses

Cleaning of the Data

The data were prepared, cleaned, and entered into the Advanced Version of SPSS for Windows 95 (v 7.0, SPSS, Inc.). Missing data were handled with a two-step process. The first step dealt with missing items and the second step dealt with missing subscales or scores. For missing items within an individual's instrument, mean data estimation was used. Missing subscales and scores were estimated using Stepwise Regression. A more detailed explanation of this two-step process for missing data estimation can be found in Appendix A.

Probability Level, Power Analysis and Statistical Analyses

For all analyses, statistical significance was set at the probability level of less than 0.05. Based on the sample size n = 307 (Zucker & Fitzgerald, 1996), power analysis was conducted with estimated effects size and revealed a level of power of greater than 0.90 for finding both small effects ($\Delta = 0.20$) and moderate effects ($\Delta = 0.50$) (Shavelson, 1988).

Two different statistical analyses were used to test the hypotheses. Hierarchical regression was used to test the individual and combined, direct contributions of the independent variables to the prediction of children's developmental status, IQ, and behavior problems for hypotheses one through four. Structural equation modeling, specifically the statistical program EQS Structural Equations Program for PowerMac (v

5.6, Multivariate Software, Inc.) was used to determine both direct and indirect relationships among the latent constructs of interest to this study for hypothesis five (The reader is referred to Figures 1 and 3 in chapter 4 for a visual representation of these relationships).

•

CHAPTER 4

Results

Hypothesis One: Prenatal Influences on Child Development

Table 3 displays zero-order correlations of the child outcome variables with demographic and prenatal variables referred to in this section.

Hierarchical regressions were run for all outcomes using various combinations of three groups of variables: parental IQ/demographics, mother's age at birth of child/parity, and prenatal exposure for a total of 7 models, one of which included all three groups of variables. The results showed that prenatal smoking was positively related to externalizing behavior problems in children. There was no support for the prediction that children whose mothers' smoked or drank during pregnancy would have lower IQs or less advanced developmental status.

Prenatal Influences on Child Behavior Problems

Externalizing Behavior Problems

The results of multiple regression models designed to show the separate and combined effects between the prenatal variables and externalizing behavior problems as measured by the CBCL are seen in Table 4. In the final model (Model 7), mother's level of education, her age at birth of target child, and the amount of cigarettes she smoked per day during her pregnancy all had a significant effect on externalizing behavior problems accounting for 10.7% of the variance. Trimming this model to include only the three variables that were significant revealed a loss of significance for maternal level of education. The final trimmed model included prenatal smoking and mother's age at

Variables	Father IQ	Father Educ	Mother IQ	Mother Educ	Family SES	Age at birth	Parity	Prenatal Smok
Father IQ	1.00							
Father Educ	.509***	1.00						
Mother IQ	.358***	.414***	1.00					
Mother Educ	.349***	.486***	.516***	1.00				
Family SES	.432***	.647***	.439***	.565***	1.00			
Age at birth	.279***	.353***	.156***	.381***	.340***	1.00		
Parity	.029	.020	.024	128*	029	.318***	1.00	
Prenatal Smok	228***	262***	277***	229***	223***	134*	013	1.00
Prenatal Drink	.001	.022	040	.070	022	.027	020	.163**
External BPs	167***	203***	142*	056	177**	188***	068	.200***
Internal BPs	104	100	081	061	112	173**	047	.071
Total BPs	147**	169**	118*	067	164**	**/77	075	.166**
Child IQ	.319***	.271***	.295***	.318***	.319***	.195***	088	134*
Dev Quotient	111.	.048	.046	.114*	.102	.019	038	019
Fine Motor	.120*	.061	.053	660.	.087	012	.023	.026
Gross Motor	.049	.023	.014	060.	.054	008	.015	.012
Language	.141*	.128*	.085	.150**	.170**	.063	037	100.
Impulsivity	.103	.123*	.124*	.039	.038	.020	025	157**
F Rate Attent	.154**	.164**	.034	.035	.085	.031	.077	039
M Rate Attent	.113*	*611.	.107	.140*	.182**	.061	024	093
Mean	102.74	13.53	96.20	13.21	341.85	26.51	1.81	4.60
Standard Dev	13.85	3 28	14 23	1 06	150.75	1 27	20	0 71

.01, 2 °°,

	(ninu).							
Variables	Prenatal Drink	External BPs	Internal BPs	Total BPs	Child IQ	Dev Quotient	Fine Motor	Gross Motor
Father IQ								
Father Educ								
Mother IQ								
Mother Educ								
Family SES								
Age at birth								
Parity								
Prenatal Smok								
Prenatal Drink	1.00							
External BPs	.127*	1.00						
Internal BPs	.025	.549***	1.00					
Total BPs	.094	.849***	.793***	1.00				
Child IQ	050	153**	059	180**	1.00			
Dev Quotient	007	.016	.167**	.014	.404***	1.00		
Fine Motor	040	.019	.209***	.038	.358***	.904***	1.00	
Gross Motor	038	.054	.199***	.060	.264***	.866***	.884***	1.00
Language	030	031	.130*	035	.529***	***106'	.876***	.840***
Impulsivity	059	051	.086	023	.175**	.308***	.320***	.326***
F Rate Attent	041	170**	086	193***	.073	.068	.072	.014
M Rate Attent	053	116*	014	122*	.276***	.185***	.184***	.071
Mean	.22	21.71	9.79	54.28	103.84	56.07	55.01	53.56
Standard Dev	.42	8.63	5.98	20.94	14.16	12.64	13.09	14.57
*p ≤ .05: **p ≤ .01: ***p ≤ .001 n = 307	01·***n < 001	n = 307						

ut'd) Table 3

Table 3.	(cont'd)			
Variables	Language	Impulsivity	F Rate Attent	M Rate Attent
Father IQ		•		
Father Educ				
Mother IQ				
Mother Educ				
Family SES				
Age at birth				
Parity				
Prenatal Smok				
Prenatal Drink				
External BPs				
Internal BPs				
Total BPs				
Child IQ				
Dev Quotient				
Fine Motor				
Gross Motor				
Language	1.00			
Impulsivity	.313***	1.00		
F Rate Attent	.057	.005	1.00	
M Rate Attent	.201**	.041	.236***	1.00
Mean	56.07	64.86	4.72	5.13
Standard Dev	13.69	37.60	2.99	2.84
*p≤.05; **p≤	*p ≤ .05; **p ≤ .01; ***p ≤ .001	n = 307		

p ≤ .001 n = 307 ; IO. 2 d ;cu. < q

Table 4. E	m	iehavior Problems Regressed on Prenatal Variables, Standard Coefficients (Betas).	Regressed on	Prenatal Varial	oles, Standard (Coefficients (Be	tas).
	Model	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Variables							
Father IQ	073			060	062		049
Father Educ	132			112	121		-101
Mother IQ	087			085	056		054
Mother Educ	.133			.167	.125		.160*
Family SES	-096			086	086		076
Age at birth		185***		145*		166**	147*
Parity		-000		.003		011	.005
Prenatal Smoke			.185**		.133*	.161**	.132*
Prenatal Drink			.097		.095	.105	.097
R ²	.061	.035	.049	.078	060.	.078	.107
· · · · · · · · · · · · · · · · · · ·							

*p ≤ .05; **p ≤ .01; ***p ≤ .001 n = 307

birth of target child which together accounted for 6.7% of the variance associated with externalizing behavior problems.

Though mother's smoking during pregnancy and her age at birth of target child were significant in the final trimmed model, the effects of prenatal smoking and maternal age on externalizing behavior problems were reduced. A twenty-nine percent reduction in magnitude for mother's smoking during pregnancy was found when mother's smoking was considered with maternal and paternal IQ and level of education and family SES as in Models 5 and 7. This reduction in the effect of mother's smoking on externalizing behavior problems appears to be due to the moderate associations between mother's smoking and the following variables; maternal and paternal IQ and level of education and family SES (rs range from -.223 to -.277; Table 3). The twenty-one percent reduction (Models 4 and 7) for the effects of mother's age externalizing behavior problems was a result of the moderate association between mother's age at birth of target child and mother's level of education (r = .340).

Summary of Analyses of Hypothesis One: Prenatal Influences on Child Development

The results of analyses for hypothesis one support the prediction that children of mothers who smoked during pregnancy exhibit more behavior problems. In this study, it was found that prenatal exposure to mothers' smoking was positively related to externalizing behavior problems in preschoolers. These children were more likely to exhibit 'acting out' behaviors such as arguing, fighting, restlessness, and temper tantrums.

In addition, it was found that younger mothers were more likely to have children who exhibited externalizing behavior problems compared to older mothers as measured by the CBCL. The median for the mothers' age at birth of target child was 26.5 years.

Hypothesis Two: Postnatal Influences on Child Development

The zero-order correlations of the child outcome variables with demographic and postnatal variables referred to in this section are displayed in Table 5. Hierarchical regressions were run for all outcomes using various combinations of four groups of independent variables: antisociality/alcohol problems, parental IQ/demographics, current smoking/drinking, and current depression for a total of 15 models, one of which included all four groups of variables.

The analyses showed that maternal depression, antisociality and LAPS (Lifetime Alcohol Problems score) were positively related to children's behavior problems. There was no support for the prediction that children whose mothers currently smoked, drank, or who were antisocial or depressed would have lower IQs or less advanced developmental status.

Postnatal Influences on Child Behavior Problems

Externalizing Behavior Problems

When externalizing behavior problems was regressed against all variables in Model 15, adult antisociality, mother's level of education, and current depression all had significant effects on externalizing behavior problems, accounting for 17% of the variance (Table 6). However, the effects of adult antisociality and depression were weakened as shown by the 30% drop in their effects in the final model compared to their effects in Models 1 and 4, respectively.

Variables	Father IQ	Father Educ	Mother IQ	Mother Educ	Family SES	External BPs	Internal BPs	Total BPs
Father IQ	1.00							
Father Educ	.509***	1.00						
Mother IQ	.358***	.414***	1.00					
Mother Educ	.349***	.486***	.516***	1.00				
Family SES	.432***	.647***	.439***	.565***	1.00			
External BPs	167**	203***	142*	056	177**	1.00		
Internal BPs	104	100	081	061	122	.549***	1.00	
Total BPs	147**	169**	118*	067	164**	.894***	.793***	1.00
Child IQ	.319***	.271***	.295***	.318***	.319***	153**	059	180**
Dev Quotient	111.	.048	.046	.114*	.102	.016	.167**	.014
Fine Motor	.120*	.061	.053	660.	.087	.019	.209***	.038
Gross Motor	.049	.023	.014	060.	.054	.054	.199***	.060
Language	.141*	.128*	.085	.150**	.170**	031	.130*	035
Impulsivity	.103	.123*	.124*	.039	.038	051	.086	023
F Rate Attent	.154**	.164**	.034	.035	.085	170**	086	193***
M Rate Attent	.113*	.119*	.107	.140*	.182***	-,116*	014	122*
Adlt Antisocal	127*	251***	140*	219***	303***	.298***	.138*	.269***
LAPS	106	118*	067	066	083	.246***	.136*	.227***
Currnt Smoke	270***	364***	349***	347***	305***	***661.	.134*	.179**
Currnt Drink	079	059	077	103	125*	.161**	.083	.112
Currnt Depress	004	182***	031	126*	137*	.214***	.179**	.233***
Mean	102.74	13.53	96.20	13.21	341.85	21.71	9.79	54.28
Standard Dev	12 05	000	14 33	101	150.75	0.00		

Bivariate Correlations, Means, and Standard Deviations for Postnatal and Outcome Variables Used in Hypothesis Two Table 5.

*p ≤ .05; **p ≤ .01; ***p ≤ .001 n = 307

Child IQ Dev Quotient Fine Motor Gross Motor Language Impulsivity 100	Child IQ Dev Quotient Fine Motor Gross Motor Language Impulsivity n h h h h h h n h h h h h h n h h h h h h n 1.00 h h h h h h n 1.00 h h h h h h n 264************************************	I able 5.	(cont'd)						
100 100 100 1100 100 100 1100 100 100 1100 100 100 1100 100 100 1100 100 100 1110 100 100 1111 100 100 1111 100 100 1111 100 100 1111 100 100 1111 1100 100 1111 1110 100 1111 1110 100 1111 1110 100 1111 1110 100 1111 1110 100 1111 1110 101 1111 1110 101 1111 1110 101 1111 1110 101 1111 1110 101 1111 1110 101 1111 1110 1011 1111	• 1.00 1.00 • 1.00 1.00 • 884*** 1.00 • 3276*** 340*** • 320*** 1.00 • 320*** 1.00 • 320*** 0.14 • 0.12 0.14 • 0.11 0.01 • 0.01 0.01 • 0.01 0.01 • 0.01 0.01 • 0.01 0.01 • 0.01 0.01 • 0.01 0.01 • 0.01 0.01 • 0.01 0.01 • 0.03 0.03 • 0.04 0.04 • 0.03 0.04 • 0.03 0.04 • 0.03 0.04 • 0.03 0.05 • 0.04 0.05 • 0.05	Variables	Child IQ	Dev Quotient	Fine Motor	Gross Motor	Language	Impulsivity	F Rate Attent
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	• 1.00 1.00 • 1.00 1.00 • 8.600 1.00 • 8.600 1.00 • 3.1300 1.00 • 3.1300 1.00 • 3.1300 1.00 • 3.1300 1.00 • 1.1840 0.01 • 0.01 2014 • 0.01 2.014 • 0.014 0.014 • 0.014 0.014 • 0.014 0.014 • 0.014 0.014 • 0.014 0.014 • 0.014 0.014 • 0.014 0.014 • 0.018 0.014 • 0.018 0.014 • 0.018 0.014 • 0.018 0.014 • 0.018 0.014 • 0.018 0.014 • <	her IQ					2		+
100 404*** 1.00 100 1.00 1.00 258*** 366**** 884*** 258*** 366**** 884*** 252*** 366*** 884*** 252*** 301*** 370*** 252*** 301*** 370*** 270*** 310*** 100 173** 308*** 372*** 173** 308*** 370*** 173** 308*** 370*** 173** 308*** 371*** 170** 100 100 173** 308*** 371*** 310*** 371 374** 310*** 371 374 310*** 309 034 310** 309 334* 310** 364** 364** 3116** 360** 364** 310** 354** 360** 310** 3550** 3556** 310** 3550** 3556**	• 100 100 • 100 100 • 384*** 100 • 370*** 313** • 320*** 100 • 320*** 100 • 320*** 100 • 320*** 014 007 • 011 207* 005 • 011 207* 004 • 011 207* 004 • 011 207* 004 • 011 207* 004 • 012 014 057 • 013 014 057 • 014 207* 041 • 014 014 056 • 014 018 • • 013 • 036 • 014 016 • • 013 • 056 • 058	her Educ							
100 100 100 100 358*** 100 358*** 904*** 100 358*** 904*** 100 358*** 904*** 100 358*** 308*** 370*** 359*** 901*** 100 359*** 308*** 372*** 175** 308*** 372*** 175*** 301*** 100 372*** 313*** 100 175** 308*** 372*** 175** 308*** 372*** 170 100 071 051* 007 010 006 014 007 010 006 014 007 013 009 014 016 014 013 013 017 014 013 014 017 013 014 013 018 014 014 013 016 014 014	• 100 100 • 1300 100 • 88400 100 • 88400 100 • 37500 31300 • 37000 31300 • 37000 31700 • 37000 31700 • 37000 31700 • 100 005 • 18400 005 • 18400 005 • 18400 005 • 014 057 • 014 057 • 014 057 • 014 057 • 014 057 • 014 005 • 014 005 • 014 005 • 014 005 • 014 005 • 014 014 • 014 014	ther IQ							
100 100 100 100 100 100 530*** 904*** 1.00 554*** 866*** 884*** 529*** 901*** 876*** 529*** 901*** 876*** 529*** 301*** 876*** 529*** 301*** 320*** 173** 001*** 320*** 073 068** 072 073 068** 071 071 201*** 100 1700* 001 014 180** 071 321*** 071 201*** 041 1700* 005 001 014 160 -014 -084 -073 077 006 -014 -084 079 -013 006 -014 -073 049* -014 -026 -073 -034 164 -014 -018 -074 -075 116*	• 100 100 • 100 100 • 384*** 100 • 376*** 334*** • 376*** 313*** • 376*** 313*** • 376*** 313*** • 100 005 • 101* 201** • 014 201** • 014 201** • 014 201** • 014 201** • 014 201** • 014 201** • 014 201** • 014 201** • 014 205* • 014 056 • 014 056 • 014 056 • 018 -006 • 018 -005 • 018 -005 • 018 -056	ther Educ							
100 100 100 100 100 100 358*** 964**** 100 358*** 964**** 100 358*** 964**** 100 358*** 964**** 100 359*** 964**** 100 359*** 384*** 100 375*** 384*** 100 175*** 308*** 372*** 175*** 308** 372*** 175*** 308** 372 175** 308** 372 175** 308** 372 175** 308* 372 175** 309 071 371** 185** 013 006 014 107* 013 006 014 167** 013 013 014 107* 013 014 013 107* 014 014 013 167** 014 014 014	100 100 873**** 100 874**** 100 874**** 100 874**** 100 874**** 100 922**** 335*** 922**** 100 922**** 013 922**** 014 923**** 013 923**** 014 923**** 013 934**** 014 935*** 013 935*** 014 935*** 013 935*** 014 935*** 013 935*** 014 935*** 035 935*** 935** 935*** 13.09 13.09 14.57 13.60 37.60	nily SES							
100 100 100 1.00 1.00 1.00 3.69**** 9.94**** 1.00 2.56**** 86.6*** 88.4*** 2.59**** 9.94**** 1.00 2.50**** 9.94**** 1.00 1.75**** 3.84*** 1.00 1.75**** 3.84*** 1.00 1.75**** 3.84*** 1.00 1.75**** 3.84*** 1.00 1.75*** 3.84*** 1.00 1.75*** 3.84*** 1.00 1.75*** 3.84*** 1.00 1.75*** 3.84*** 1.00 1.75*** 3.84*** 1.00 1.75*** .014 .094 1.70*** .012 .014 .094 1.10*** .013 .009 .014 .094 1.70*** .013 .004 .014 .094 1.10*** .014 .029 .013 .014 1.10**** .014 .026	• 1.00 1.00 • 854 1.00 • 875 1.00 • 875 1.00 • 37 0.05 • 37 0.05 • 1.00 1.00 • 3.13 1.00 • 3.13 1.00 • 1.014 0.05 • 0.014 0.014 • 0.014 0.014 • 0.014 0.014 • 0.014 0.05 • 0.014 0.05 • 0.014 0.05 • 0.014 0.05 • 0.014 0.05 • 0.014 0.05 • 0.014 0.05 • 0.014 0.05 • 0.014 0.05 • 0.014 0.05 • 0.014 0.05 • 0.014	ernal BPs							
100 100 100 358*** 366**** 100 100 252*** 366**** 384*** 100 358*** 366**** 384*** 100 359*** 366*** 384*** 100 359*** 360*** 372*** 313*** 373*** 320*** 320*** 313*** 173** 368*** 372*** 313*** 173** 368*** 320*** 313*** 173** 368*** 320*** 313*** 173** 368*** 320*** 313*** 185*** 367** 313*** 100 173** 368*** 372 041 1007 010 006 014 064 1007 013 009 034 035 035 1016* -048 -033 -034 -035 035 1017* 018 -034 -034 -034 1010 014	• 100 100 • 100 100 • 844 100 • 876* 840* • 876* 840* • 876* 840* • 876* 840* • 876* 840* • 100 100 • 071 057 • 074 057 • 012 006 • 014 057 • 014 057 • 013 • • 014 057 • 013 • • 014 075 • 013 • • 014 • • 014 • • 014 • • 014 • • • • • • • •<	rnal BPs							
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	100 100 100 88444 1.00 1.00 93764 3.004 1.00 93764 3.104 1.00 93764 3.134 1.00 9375 0.14 0.37 9112 0.01 0.05 9122 0.14 0.37 909 .014 0.01 909 .014 0.03 918 .005 .014 919 .005 .014 910 .014 .026 911 .014 .034 912 .014 .044 913 .026 .013 913 .026 .014 913 .026 .035 913 .035 .035 913 .035 .035 913 .035 .035 913 .035 .035 913 .035 .035 913 .037.60 .037.60	al BPs							
40**** 1.00 2.54*** 8.66*** 1.00 2.54*** 8.66*** 1.00 2.54*** 8.66*** 1.00 1.75*** 3.01*** 8.76*** 1.75*** 3.01*** 8.76*** 1.75*** 3.01*** 3.76*** 1.75*** 3.01*** 3.74*** 1.75*** 3.01*** 3.74*** 1.75*** 3.01*** 3.74*** 1.71*** 0.01 0.05 1.71*** 0.01 0.04 1.71*** 0.05 -0.01 1.71*** 0.06 -0.01 -0.94 1.70*** 0.06 -0.01 -0.94 1.70*** 0.06 -0.01 -0.94 1.70*** 0.06 -0.01 -0.02 1.70*** 0.04** -0.02 -0.74 1.70*** 0.04** -0.75 -0.75 1.70*** 0.04** -0.02** -0.75 1.70*** 0.04** -0.76	100 100 884*** 1.00 9 320*** 320*** 310*** 9 320*** 100* 100 9 320*** 101* 007 101* 007 101* 014 101* 014 011* 014 011* 014 010* 014 011* 014 011* 014 010 014 011* 014 010 014 010 014 010 014 010 014 010 014 010 014 010 014 010 014 018 -010 018 -010 018 -010 018 -010 018 -010 019 -035 010 -036	DI PI	1.00						
358*** 964*** 1.00 522*** 961*** 1.00 522*** 961*** 376*** 732*** 961*** 376*** 773** 981*** 272*** 175** 981*** 272*** 175** 388*** 272*** 175** 313*** 1.00 175** 313*** 1.00 170** 0.01 0.01 0.01 0.07 0.00 0.02 0.01 0.04 0.07 0.03 0.06 0.01 0.04 0.07 0.03 0.06 0.04 0.04 0.07 0.03 0.06 0.04 0.04 0.07 0.03 0.06 0.04 0.03 0.07 0.03 0.04 0.03 0.03 106******* 0.03 0.04 0.03 0.03 107*********************** 0.03 0.04 0.03 0.03 108************************************	 100 876*** 387*** 370*** 370**** 	Quotient	.404***	1.00					
2.64*** 8.66*** 8.84*** 1.00 .75*** .30**** .53*** 1.00 .75*** .308*** .320*** .100 .75*** .308*** .320*** .313*** 1.00 .75*** .308*** .320*** .313*** 1.00 .75*** .308*** .320*** .313*** 1.00 .70** .068 .072* .014 .057 .005 .07** .013 .006 .011 .004 .094 .094 .07** .013 .006 .001 .018 .094 .094 .07* .013 .006 .001 .018 .094 .094 .07* .013 .006 .014 .058 .094 .0756 .0756 .0756 .049 .016* .018 .029 .026 .056 .0756 .0756 .0756 .0756 .0756 .0756 .0756 .0756 .0756 .0756 .	 884 1100 876 840 1100 375 100 372 100 373 100 373 100 101 326 313 100 101 327 005 101 313 100 101 313	e Motor	.358***	.904***	1.00				
520*** 901*** 876*** 840*** 100 175** 368*** 220*** 2326*** 100 073 368*** 220*** 2326*** 100 175** 368*** 72 041 100 177** 368*** 77 041 061 1 107 036 014 056 1 107 030 006 014 064 107 010 006 014 064 104 107 010 006 014 064 104 107 013 009 024 018 073 108 -048 -014 018 075 075 108 -014 018 026 075 075 108 -014 018 075 075 075 108 -014 018 075 075 075 109 016 -018 076	8 3/6*** 8,0*** 1,00 •	ss Motor	.264***	.866***	.884***	1.00			
175** 308*** 320*** 326*** 100 073 072 014 057 005 1 -107 072 014 057 005 1 -107 006 014 007 006 1 -107 006 014 087 004 1 -107 006 014 086 094 1 -107 006 014 072 094 -167* -013 006 -014 -086 -094 -167* -013 006 -014 -076 -075 -014 -018 -029 -014 -076 -075 -019 -014 -018 -026 -075 -075 -016 -014 -026 -036 -075 -075 -018 -028 -048 -076 -075 -075 -018 -036 -036 -036 -075 -076	330*** 3326*** 313*** 100 0722 014 057 005 0122 014 201** 041 012 014 201** 041 012 014 201** 041 012 014 201** 043 009 -014 -080 -034 009 -014 -016 -034 018 -018 -016 -034 018 -018 -026 -073 018 -018 -026 -073 018 -048 -100 -036 018 -048 -100 -036 018 -048 -100 -036 13.09 14.57 13.69 37.60 -	guage	.529***	***106"	.876***	.840***	1.00		
073 068 072 014 057 005 1 007 .071 .001 .001 .001 1 007 .002 .005 .001 .001 .007 .003 .006 .001 .004 .004 .007 .003 .006 .001 .018 .094 .0 .007 .003 .006 .001 .018 .094 .0 .007 .013 .006 .001 .018 .094 .0 .007 .013 .006 .001 .018 .094 .0 .014 .006 .001 .018 .003 .014 .094 .0 .014 .018 .029 .016 .036 .035 .035 .035 .035 .016 .018 .029 .016 .036 .035 .035 .035 .035 .035 .035 .035 .035 .035 .035 .035<	1072 014 057 005 1012 014 057 005 1012 011 2011 011 1012 006 -014 0.05 1012 006 -014 0.06 006 -0101 -018 -094 009 -034 -026 -074 008 -034 -026 -076 -018 -028 -026 -056 -038 -048 -100 -036 -053 -048 -100 -036 -13.09 14.57 13.69 37.60 -	ulsivity	.175**	.308***	.320***	.326***	.313***	1.00	
1 276*** 185*** 184*** 071 201*** 041 0.07 0.07 0.02 0.06 0.14 0.86 0.07 0.00 0.02 0.06 0.14 0.80 1.67** 0.03 0.06 0.04 0.03 0.93 1.16** 0.03 0.09 0.04 0.03 0.03 1.16** 0.08 0.09 0.04 0.03 0.03 1.16* 0.48 0.08 0.048 0.03 0.03 1.03 1.16* 0.48 0.08 0.048 0.036 1.05 </td <td>• 184*** 071 201*** 041 012 006 -014 -080 - 009 -001 -018 -093 - 009 -024 -026 -073 - 018 -024 -026 -073 - 018 -029 -036 -056 - 018 -048 -100 -036 - 55.01 33.56 56.07 54.86 - 55.01 13.56 56.07 54.86 -</td> <td>ate Attent</td> <td>.073</td> <td>.068</td> <td>.072</td> <td>.014</td> <td>.057</td> <td>.005</td> <td>1.00</td>	• 184*** 071 201*** 041 012 006 -014 -080 - 009 -001 -018 -093 - 009 -024 -026 -073 - 018 -024 -026 -073 - 018 -029 -036 -056 - 018 -048 -100 -036 - 55.01 33.56 56.07 54.86 - 55.01 13.56 56.07 54.86 -	ate Attent	.073	.068	.072	.014	.057	.005	1.00
1 .107 .020 .012 .006 .014 .080 .107 .006 .001 .006 .011 .003 .004 .013 .167** .013 .006 .014 .018 .004 .013 .167** .013 .006 .014 .018 .004 .014 .167** .013 .006 .014 .029 .013 .004 .013 .049 .014 .018 .029 .016 .056 .075 .075 .075 .075 .075 .075 .075 .075 .076 .0756 <td< td=""><td>012 006 .014 .080 006 .001 .018 .094 009 .031 .024 .034 009 .034 .026 .074 009 .034 .026 .074 018 .038 .026 .076 018 .048 .100 .056 . 053 .3356 .100 .036 . 55.01 33.56 56.07 54.86 . 13.09 14.57 13.69 37.60 .</td><td>tate Attent</td><td>.276***</td><td>.185***</td><td>.184***</td><td>.071</td><td>.201***</td><td>.041</td><td>.236***</td></td<>	012 006 .014 .080 006 .001 .018 .094 009 .031 .024 .034 009 .034 .026 .074 009 .034 .026 .074 018 .038 .026 .076 018 .048 .100 .056 . 053 .3356 .100 .036 . 55.01 33.56 56.07 54.86 . 13.09 14.57 13.69 37.60 .	tate Attent	.276***	.185***	.184***	.071	.201***	.041	.236***
.007* .010 .006 .001 .018 .094 .167** .013 .009 .014 .026 .073 .049** .013 .009 .014 .026 .073 .049 .014 .026 .073 .075 .073 .041 .018 .018 .026 .075 .073 .043 .018 .028 .048 .076 .036 .116* .048 .038 .046 .036 .036 .101 .100 1.100 .036 .036 .036 .1013 .550 .553 .106 .136 .136	006 .001 .018 .094 009 .024 .073 .073 -018 .024 .075 .075 -018 .029 .026 .075 -018 .048 .100 .036 -038 .048 .100 .036 -350 -31.56 56.07 64.86 -13.09 14.57 13.69 37.60	t Antisocal	107	020	012	.006	014	080	094
.167** .003 .009 .024 .026 .073 .167** .014 .018 .029 .075 .073 .167** .014 .018 .029 .075 .075 .116** .014 .018 .029 .026 .075 .116** .048 .010 .036 .036 103 .350 .3536 .5607 .64.86 14.5 .5607 .13.60 .13.60 .14.86	009 .034 .026 .073 .018 .029 .036 .035 .018 .048 .100 .036 .031 .348 .100 .036 .031 .35.01 .35.60 .6607 64.86 .13.09 14.57 13.69 37.66	Sc	.007	010	.006	001	018	094	107
-049 -014 -018 -029 -026 -056 -056 -056 -1056 </td <td>.018 .029 .026 .056 .038 .048 .100 .036 .051 .3.56 56.07 64.86 .13.09 14.57 13.69 37.60</td> <td>rnt Smoke</td> <td>167**</td> <td>033</td> <td>600.</td> <td>034</td> <td>026</td> <td>073</td> <td>- 069</td>	.018 .029 .026 .056 .038 .048 .100 .036 .051 .3.56 56.07 64.86 .13.09 14.57 13.69 37.60	rnt Smoke	167**	033	600.	034	026	073	- 069
s 116* 048 058 048 036 10.3.84 5607 5.501 5.536 5607 64.86 10.1.6 12.64 13.00 14.57 13.60 7.648	058 048 100 036 55.01 53.56 56.07 64.86 13.09 14.57 13.69 37.60	rnt Drink	049	014	018	029	026	056	083
103.84 56.07 55.01 53.56 56.07 64.86 14.16 12.64 13.09 14.57 13.60 37.60	55.01 53.56 56.07 64.86 13.09 14.57 13.69 37.60	rnt Depress	116*	048	058	048	100	036	035
14.16 12.64 13.09 14.57 13.69 37.60	13.09 14.57 13.69 37.60	u	103.84	56.07	55.01	53.56	56.07	64.86	4.72
10.7C 20.CI 10.LI 20.CI		idard Dev	14.16	12.64	13.09	14.57	13.69	37.60	2.99

11.4 Tobla 5 p ≤ .001 n = 307 p ≤ .01; ;cu. 2 d

	(cont'd)					
Variables	M Rate Attent	Adlt Antisocal	LAPS	Currnt Smoke	Currnt Drink	Currmt Depress
Father IQ						-
Father Educ						
Mother IQ						
Mother Educ						
Family SES						
External BPs						
Internal BPs						
Total BPs						
Child IQ						
Dev Quotient						
Fine Motor						
Gross Motor						
Language						
Impulsivity						
F Rate Attent						
M Rate Attent	1.00					
Adlt Antisocal	115*	1.00				
LAPS	055	.431***	1.00			
Currnt Smoke	108	.213***	.186***	1.00		
Currnt Drink	055	.236***	.339***	.206***	1.00	
Currnt Depress	019	.282***	.132*	.102	004	1.00
Mean	5.13	4.56	9.93	1.43	423.96	3.22
Standard Dev	2.84	3.47	2.01	1.96	1366.95	3.29
$*n \le .05: **n <$	$*b \le 05$; $**b \le 01$; $***b < 001$ $n = 307$	n = 307				

= 307 c p ≤ .001 p≤.01; ;c0.≥q

Table 6.	Externalizir	nalizing	g Beha	vior Pro	blems l	Regres	sed on	Postnai	al Varia	ables, S	tandard	Coeffic	ng Behavior Problems Regressed on Postnatal Variables, Standard Coefficients (Betas)	setas).	
	Model 1	Model	Model	Model 4	Model	Model	Model	Model	Model	Model	Model	Model	Model	Model	Model
		7	m		s	9	7	**	6	10	Ξ	12	13	14	15
Variables															
Adult Antisocl	.236***				.210***	.211***	•••861.				.200	.176**	.172**		.164
LAPS	.144*				.136*	.117	.143•					.133*	.112		104
Father IQ		073			073			061	. 098		066	089		086	083
Father Educ		132			-100			117	090		089	074		078	064
Mother IQ		087			-060			064	-101		073	6 60'-		080	081
Mother Educ		.133			.147			.156	.149•		.162*	.156*		.171•	.171•
Family SES		. 096			- 049			082	088		046	051		073	046
Current Smoke			.173**			.123•		.121		.152**	160		.115*	601	.087
Current Drink	-		.125*			.046		.125•		.130	.052		.059	.130*	.065
Depression				.214***			.139•		.200***	•••661		.142*	.139*	197***	.146*
R²	.106	.061	.055	.046	.142	.123	.124	.093	.099	.094	.152	.160	.141	.130	.170

*p < .05; **p < .01; ***p < .001 n = 307

A trimmed model revealed a loss of significance for maternal level of education. In the final trimmed model, adult antisociality and maternal depression account for 10.7% of the variance associated with externalizing behavior problems. Magnitude for mother's current depression also was reduced by 9% when mother's current drinking was trimmed from the model.

Both current smoking and drinking were found to have significant effects on externalizing behavior problems (Model 3). But the effect for current smoking was lost when it was considered jointly with the following variables: maternal and paternal IQs and levels of education and family SES (Models 8, 11, 14, and 15). Mother's current level of drinking also lost significance when it was considered jointly with LAPS (Models 6, 11, 13, and 15).

Internalizing Behavior Problems

Mother's current depression also had a significant effect on internalizing behavior problems in Model 4 accounting for 3.2% of the variance (Table 7). Current depression continued to maintain significance with only a small reduction in magnitude across all Models and was the only significant predictor of internalizing behavior problems in the final Model (Model 15) accounting for 6.4% of the variance.

Though current smoking was found to have a significant effect on internalizing behavior problems in Model 3, this effect was lost in all other Models.

Total Behavior Problems

In Model 15, only adult antisociality and current depression had significant effects on total behavior problems though their magnitudes were reduced by 36% and 19%, respectively (Table 8). These variables accounted for 14.3% of the variance associated

Table 7.	Intern	Internalizing Beh	Behavic	or Probl	ems Re	gressed	on Pos	tnatal V	avior Problems Regressed on Postnatal Variables, Standard Coefficients (Betas)	, Stand	ard Coe	officient	s (Beta:	s).	
	Model	Model	Model	Model	Model	Model	Model	Model	Model	Model	Model	Model	Model	Model	Model
	-	7	m	4	Ś	6	٢	00	6	01	Ξ	12	13	4	15
Variables															
Adult Antisocl	.097				.075	.079	.055				.068	.037	.038		.029
LAPS	.094				.092	.078	.092				.078	.089	073		.072
Father IQ		059			057			052	- 081		052	075		073	070
Father Educ		023			007			008	014		.006	.021		.026	.032
Mother IQ		034			034			016	046		020	044		030	031
Mother Educ		.030			.034			.046	044		.047	.045		.059	.057
Family SES		073			057			067	066		056	059		060	057
Current Smoke			.122*			660.		960.		.104	180.		060	.086	.076
Current Drink			.058			.018		.054		.063	.016		.032	.058	.030
Depression				••641.			.152**		.177**	••691.		.157**	.150*	.174**	.158**
R ^z	.026	.018	.021	.032	.037	.036	.047	.029	.047	.049	.042	.059	.057	.058	.064
4 4 4 C 1 4	;	· · · · · · · · · · · · · · · · · · ·													

*p ≤ .05; **p ≤ .01; ***p ≤ .001 n = 307

Table 8.	Total I	Total Behavior I	or Proble	ems Re	gressed	on Post	tnatal V	/ariable:	Problems Regressed on Postnatal Variables, Standard Coefficients (Betas).	ard Coe	fficient	s (Beta	S). Madal	Model	Model
		2	3	4	5	6	7	8	9	10	11	12	13	14	15
Variables		I	I			I		,	·			•	!		:
Adult Antisocl	.210***				.184**	••161	.163**				.178**	.141*	.145•		135
LAPS	.137*				.132*	.123	.135•				.120	.128•	.118		.113*
Father IQ		068			067			058	096		062	087		086	082
Father Educ		087			058			069	041		042	026		026	013
Mother IQ		057			060			035	073		044	071		053	056
Mother Educ		.086			860.			.107	.104		III.	.110		.123	.122
Family SES		101			060			093	092		061	062		083	062
Current Smoke			.163**			.115•		611.			060		.106	.106	.085
Current Drink			078			100		.075			.003		.017	080	.018
Depression				.233***			••691.		.223***	.219***		.174**	.166**	.220***	.174**
R ¹	.088	.042	.038	.054	.109	.100	.114	.062	060	.085	.116	.136	.125	.108	.143
*p ≤ .05; **p ≤ .01; ***p ≤ .001	. * ;10.≥c	00.≥d**	1 n = 307	5											
•		•													

with the outcome. Both variables maintained significance in the trimmed model with adult antisociality showing an increase in magnitude to the original level found in Model

1. Additional analyses revealed that adult antisociality was suppressed by the

combination of LAPS and depression along with father's level of education. These three variables were moderately correlated with adult antisociality (*rs* range from -.251 to .431; Table 5).

Once again, mother's smoking was found to have a significant effect on total behavior problems (Model 3), but was lost when considered jointly with the following: maternal and paternal IQs and levels of education and family SES (Models 8, 11, 14, and 15).

Summary of Analyses of Hypothesis Two: Postnatal Influences on Child Development

The results of analyses for hypothesis two support the prediction that children of mothers who are depressed, antisocial or scored high on the LAPS exhibit more behavior problems. Specifically, children of depressed or antisocial mothers exhibit more externalizing behavior problems, whereas only maternal depression was related to children's internalizing behavior problems. Compared to non-depressed mothers, depressed mothers were more likely to have children who exhibited both 'acting out' behaviors such as fighting, restlessness, and temper tantrums as well as internalizing behaviors such as crying, clinginess, and nervousness. Whereas, mothers who were classified as antisocial or have a history of alcohol problems were more likely to have children who engaged in the 'acting out' behaviors.

Hypothesis Three: Parenting Influences on Child Development

Table 9 displays the zero-order correlations of the child outcome variables with demographic and postnatal variables referred to in this section. Hierarchical regressions were run for all outcomes using various combinations of three groups of variables: parental IQ/demographics, parenting attitudes, and parenting behaviors for a total of 7 models, one of which included all three groups of variables.

The analyses showed that the HOME was positively related to fine motor, language, and overall Developmental Quotient, as well as child IQ and mother's rating of child's attention level. It was also found that the parenting attitude subscale of Positive Affect was positively related to child IQ and the subscale of Negative Affect was positively related to behavior problems. No support was found for the prediction that children from less stimulating homes would exhibit more behavior problems.

Parenting Influences on Developmental Status and IQ

Parenting Influences on Developmental Status

Unlike the lack of significant results for both prenatal and postnatal influences on developmental status, significant influences were found for parenting. Due to similar trends in significant effects for HOME score on the outcomes of Developmental Quotient, Fine Motor Skills, and Language, the results for these outcomes will be presented together (See Tables 10 through 12).

In Model 7 for the outcomes of Developmental Status, only HOME score had a significant effect on: Developmental Quotient accounting for 3.6% of the variance (Table 10), fine motor accounting for 2.2% of the variance (Table 11), and language development, accounting for 5.3% of the variance (Table 12).

Variables	Father IQ	Father Educ	Mother IQ	Mother Educ	Family SES	External BPs	Internal BPs	Total BPs
Father IQ	1.00							
Father Educ	***605.	1.00						
Mother IQ	.358***	.414***	1.00					
Mother Educ	.349***	.486***	.516***	1.00				
Family SES	.432***	.647***	.439***	.565***	1.00			
External BPs	167**	203***	142*	056	177**	1.00		
Internal BPs	104	100	081	061	122	.549***	1.00	
Total BPs	147**	169**	118*	067	164**	.894***	.793***	1.00
Child IQ	.319***	.271***	.295***	.318***	.319***	153**	059	180**
Dev Quotient	111.	.048	.046	.114*	.102	.016	.167**	.014
Fine Motor	.120*	.061	.053	660.	.087	610.	.209***	.038
Gross Motor	.049	.023	.014	060.	.054	.054	***661.	.060
Language	.141*	.128*	.085	.150**	.170**	031	.130*	035
Impulsivity	.103	.123*	.124*	.039	.038	051	.086	023
F Rate Attent	.154**	.164**	.034	.035	.085	170**	086	193***
M Rate Attent	.113*	*611.	.107	.140*	.182***	116*	014	122*
Open Expernc	.161***	.178**	.277***	.250***	.295***	122*	093	133*
Positive Affct	.130*	.113*	.176**	.144*	.110	093	127*	108
Negative Affct	.007	.026	.040	034	023	.194***	.137*	***661.
HOME	.282***	.341***	.296***	.403***	.404***	183***	097	166**
Mean	102.74	13.53	96.20	13.21	341.85	21.71	9.79	54.28
Standard Dev	13.85	2 28	14 23	1 06	150.75	0 62	6 00	10 00

Bivariate Correlations, Means, and Standard Deviations for Parenting and Outcome Variables Used in Hypothesis Three Table 9.

*p ≤ .05; **p ≤ .01; ***p ≤ .001 n = 307

Father (Q Hather Educ Monter (Q Monter Educ Monter Educ External BPs External BPs					
Faither Educ Monter Educ Family SES Femily SES External BPs Internal BPs					
Mother Educ Mother Educ Family SES External BPs Internal BPs					
Mother Educ Family SES External BPs Internal BPs Total BPs					
Family SES External BPs Internal BPs Total BPs					
External BPs Internal BPs Total BPs					
Internal BPs Total BPs					
Total BPs					
Child IQ 1.00					
Dev Quotient .404*** 1.00					
	1.00				
	.884***	1.00			
.529***		.840***	1.00		
Impulsivity .175** .308***	.320***	.326***	.313***	1.00	
F Rate Attent .073 .068	.072	.014	.057	.005	1.00
.276***	.184***	.071	.201***	.041	.236***
.196***	063	066	.033	.028	.092
Positive Affct 256***035	-096	098	014	022	.018
: Affct	.020	.005	035	.023	109
HOME .390*** .189***	.148**	.103	.230***	.088	.092
Mean 103.84 56.07	55.01	53.56	56.07	64.86	4.72
Standard Dev 14.16 12.64	13.09	14.57	13.69	37.60	2.99

 $p \le .05$; ** $p \le .01$; *** $p \le .001 n = 307$

Table 9.	(cont'd)				
Variables	M Rate Attent	Open Expernc	Positive Affct	Negative Affct	HOME
Father IQ					
Father Educ					
Mother IQ					
Mother Educ					
Family SES					
External BPs					
Internal BPs					
Total BPs					
Child IQ					
Dev Quotient					
Fine Motor					
Gross Motor					
Language					
Impulsivity					
F Rate Attent					
M Rate Attent	1.00				
Open Expernc	.120*	1.00			
Positive Affct	.059	.357***	1.00		
Negative Affct	087	131*	277***	1.00	
HOME	.318***	.204***	.173**	165**	1.00
Mean	5.13	23.20	26.14	5.83	44.76
Standard Dev	2.84	3.04	2.01	2.56	4.60
*p≤.05; **p≤	* $p \le .05$; ** $p \le .01$; *** $p \le .001$ n = 307	n = 307			

Table 10. Developmental		uotient Regress	ed on Parenting	Variables,	Juotient Regressed on Parenting Variables, Standard Coefficients (Betas)	ents (Betas).	
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Variables							
Father IQ	.101			.106	.088		.092
Father Educ	082			082	060'-		092
Mother IQ	038			022	047		031
Mother Educ	.097			660.	.061		.064
Family SES	.074			.078	.041		.046
Positive Affect		050		059		067	068
Negative Affect		043		043		018	019
Open Experience		.008		033		024	038
HOME			.189***		.168**	.203***	.176**
R²	.024	.003	.036	.030	.046	.041	.053
#~ / VC: ##~ / VI: ###~ / VVI	01. 444. / 001 -	- 201					

 $p \le .05$; $p \le .01$; $p \le .00 \le 00$ n = 307

(Betas).	` `
Coefficients	
Standard (
Variables,	
Parenting	•
uo	
Regressed	•
Development)
Fine Motor]	
Table 11.	

I ADIC 11. L	Fine Motor Devel	opment kegres	elopment Regressed on Parenting Variables, Standard Coefficients (Betas)	Variables, St	andard Coeffic	ients (Betas).		
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	-
Variables								
Father IQ	.109			.118	.100		.107	-
Father Educ	046			050	052		059	
Mother IQ	021			.005	027		002	
Mother Educ	.072			.082	.046		.054	_
Family SES	.038			.053	.014		.028	-
Positive Affect		086		100		101	107	
Negative Affect		008		014		.014	900.	
Open Experience		034		077		062	081	
HOME			.148**		.121	.180**	.142*	
R ²	.020	.010	.022	.038	.031	.041	.053	
** / 02: ***	*n < 05. **n < 01. ***n < 001 n = 307	- 307						

 $p \le .05$; $p \le .01$; $p \le .001$ n = 307

Table 12. L	Table 12. Language Develo	pment Regresse	pment Regressed on Parenting Variables, Standard Coefficients (Betas).	Variables, Sta	ndard Coefficie	nts (Betas).	
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Variables							
Father IQ	.081			.085	.067		.070
Father Educ	011			- .008	020		020
Mother IQ	029			017	038		026
Mother Educ	080			.081	.041		.044
Family SES	.109			601.	.074		.076
Positive Affect		040		052		060	062
Negative Affect		040		046		012	019
Open Experience		.043		014		.005	019
HOME			.230***		.182**	.237***	.187**
R²	.038	.003	.053	.042	.064	.056	.068
** / US: *** /	#~ / US: ##~ / UI: ###~ / UU ~	- 307					

*p ≤ .05; **p ≤ .01; ***p ≤ .001 n = 307

In trimmed models for the three developmental status outcomes, significance was maintained for the HOME and magnitude for the Betas increased for all three outcomes. The largest increase in magnitude (23%) occurred when language development was regressed against HOME.

Additional analyses revealed that family SES was moderately correlated with the HOME with r = .40 and was suppressing HOME's effect on the developmental status outcomes in the non-trimmed Models. This suppression effect was shown in Model 5 for all outcomes with the smallest reduction (11.1%) occurring for the outcome of Developmental Quotient (Table 10) and the largest reduction (23.6%) occurring for the outcome of language development (Table 12).

Parenting Influences on IQ

Table 13 presents the results of multiple regression models for the parenting variables and Stanford Binet IQ. In Model 7, father's IQ, positive affect, and the HOME score all had a significant effect on child's IQ accounting for 25.2% of the variance. A trimmed model revealed continued significance for all three predictors and an increase in magnitude for both father's IQ (27%) and the HOME score (16%). Further analyses showed that the effects of both father's IQ and the HOME score were suppressed by the effects of mother's IQ (r = .296) and family SES (r = .404).

Parenting Influences on Child Behavior Problems

Externalizing Behavior Problems

In Table 14, Model 7, both negative affect and maternal level of education were found to have significant effects on externalizing behavior problems accounting for 11.2% of the variance. In the trimmed model, significance and magnitude were

Model 1 Model 2 Model 3 Model 4 Model 6 Model 7 Variables Variables .187** .174** .166** .155** Father IQ .187** .174** .166** .031 Father IQ .187** .174** .166** .155** Father IQ .187** .014 .033 .097 .031 Mother IQ .111 .083 .097 .070 .070 Mother Educ .113 .121 .075 .073 .071 Family SES .124 .223*** .123 .073 .076 .078 Positive Affect .124 .223*** .189*** .073 .078 .078 Negative Affect .124 .223*** .194*** .176** .078 Negative Affect .122* .017 .073 .073 .078 .055 Negative Affect .122* .123* .124 .176** .056 .010 Negative Affect<	tble 13. St	Table 13. Stanford IQ Regressed on Parenting Variables, Standard Coefficients (Betas).	ssed on Parenti	ng Variables, S	standard Coeffi	cients (Betas).		
		Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
.187** .174** .166** -014 .016 .027 .013 .016 .027 .111 .083 .097 .112 .083 .097 .124 .121 .076 .124 .123 .073 .124 .123 .073 .124 .123 .073 .124 .123 .073 .124 .123 .073 .124 .223*** .194*** .125* .017 .083 .122* .017 .066 .170 .080 .152 .204 .23***	/ariables							
014027 .111	er IQ	.187**			.174**	.166**		.155**
.111	er Educ	014			016	027		031
.133 .121 .076 .124 .123 .073 .124 .223*** .189*** .073 .040 .019 .083 .122* .017 .083 .122* .017 .066 .170 .080 .152 .204 .194***	her IQ	.111			.083	<i>1</i> 60.		.070
.124 .123 .073 .123 .073 .144** .223*** .189*** .073 .194*** .040 .019 .083 .083 .017 .083 .083 .017 .066 .083 .066 .083 .066 .066 .066 .066 .066 .066 .066 .06	her Educ	.133			.121	.076		.071
.223*** .189*** .194*** .040 .019 .083 .122* .017 .083 .122* .017 .066 .170 .080 .152 .204 .224	ily SES	.124			.123	.073		.078
e	tive Affect		.223***		.189***		.194	.176**
.122*	ative Affect		.040		010		.083	.055
.390***	n Experience		.122*		.017		.066	.010
.152	МЕ			.390***		.263***	.357***	.252***
	R²	.170	080.	.152	.204	.224	.198	.252

*p≤.05; **p≤.01; ***p≤.001 n = 307

Table 14. E	Externalizing Behavior Problems Regressed on Parenting Variables, Standard Coefficients (Betas).	navior Problems	Regressed on H	Parenting Varia	bles, Standard	Coefficients (B	etas).
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Variables							
Father IQ	073			072	062		064
Father Educ	132			151	126		145
Mother IQ	087			095	080		060
Mother Educ	.133			.150*	.162*		•169*
Family SES	-096			073	070		055
Positive Affect		010		.010		.002	.015
Negative Affect		••641.		.201		.161	.188***
Open Experience		095		050		072	047
HOME			183***		136*	142*	097
R ²	.061	.047	.034	.105	.075	.066	.112
*p ≤ .05; **p ≤	r 100.≥q***;10.≥q**;50.≥q*	= 307					

maintained for negative affect, but were lost for maternal level of education which was enhanced by both openness to experience (r = .250) and the HOME score (r = .403). Alone, negative affect accounted for 3.8% of the variance associated with externalizing behavior problems.

Total Behavior Problems

Table 15 presents the results of multiple regression models designed to show the separate and combined effects between the parenting variables and total behavior problems. In Model 7, only negative affect had a significant influence on total behavior problems accounting for 9.3% of the variance. A trimmed model revealed continued significance and maintenance of magnitude for negative affect which accounted for 4% of the variance associated with total behavior problems.

Mother's Rating of Child's Attention Level

The HOME score was found to have a significant influence on mother's rating of child's attention level (Table 16). Significance was maintained with only a slight (the largest being 9%) decrease in magnitude across the models. In the final model, HOME accounted for 10.9% of the variance associated with mother's rating of child's attention levels (Model 7).

Summary of Analyses of Hypothesis Three: Parenting Influences on Child Development

The results of analyses for hypothesis three support the prediction that children who live in stimulating environments are more advanced in terms of fine motor and language development as well as overall development as measured by the Yale Developmental Quotient. These same children also have higher IQs and are rated by

ole 15. To	Table 15. Total Behavior P	Problems Regressed on Parenting Variables, Standard Coefficients (Betas).	sed on Parentin	g Variables, St	andard Coeffic	ients (Betas).	
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Variables							
Father IQ	068			066	059		060
Father Educ	087			106	082		102
Mother IQ	057			061	051		057
Mother Educ	.086			.105	.112		.120
Family SES	-101			076	078		062
Positive Affect		022		007		012	003
Negative Affect		.180**		197***		.165**	.186**
Open Experience		-101		062		082	060
HOME			166**		120	120*	078
R²	.042	.052	.028	.088	.054	.065	.093
	#~ / VS. ##~ / VI. ###~ / VV1 -						

 $p \le .05$; $p \le .01$; $p \le .01$; $p \le .001$ n = 307

÷		
s (Betas).		
lard Coefficients		
ard Cc		
les, Standard		1 1 1 1 1 1
on Parenting Variables,		1-1-1 5
ating		2
Parent		
l on		VI-L-I
tention Level Regressed on P		
el Re		ſ
Levi		NICACI 3
tion		4
tten	ļ	
's A		
of Child's Att		N.S.
ofC		
ting		_
s Ra		a po
her':		
Mot		
e 16		
Tabl		
•	L	

.

	0				forming given		(cmar) curain
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Variables							
Father IQ	.040			.039	.016		.017
Father Educ	026			016	041		033
Mother IQ	.013			010	002		005
Mother Educ	.048			.039	016		-019
Family SES	.149			.131	160 [.]		.079
Positive Affect		001		008		026	023
Negative Affect		072		077		036	037
Open Experience		111.		.058		.063	.051
HOME			.318***		.298***	.304***	.289***
R²	.037	.020	101.	.046	.106	.106	.109
t 100. ≥ q*** ;10. ≥ q** ;50. ≥ q*		= 307					

their mothers as having increased levels of attention. Support was found for the prediction that mothers who scored high on the positive affect parenting attitude subscale have children with higher IQs. Specifically, mothers who provided an abundance of age appropriate toys, a variety of experiences and responded to their children's needs with warmth and affection had children who did well on fine motor tasks, such as tracing objects and draw shapes, who scored high on language tests that measured use of plurals in speech and comprehension, as well as have the ability to stay on task and follow directions.

Finally, support was also found for the prediction that children whose mothers' scored high on the negative affect parenting attitudes subscale are more likely to exhibit behavior problems, specifically, externalizing behavior problems. In this study, mothers who felt anger towards their children and characterized their relationship as being conflicted had children that exhibited 'acting-out' behaviors, such as fighting, destruction of property, restlessness, and temper tantrums.

Hypothesis Four: Prenatal and Postnatal Maternal Consumption Influences on Child Development

Table 17 displays zero-order correlations of the child outcome variables with demographic and postnatal variables referred to in this section. Hierarchical regressions were run for all outcomes using various combinations of three groups of variables: parental IQ/demographics, prenatal exposure, and current smoking/drinking for a total of 7 models, one of which included all three groups of variables.

Results of the regressions showed that prenatal smoking was the better predictor of child impulsivity. No support was found for this hypothesis in terms of the outcomes

Consumption and Outcome	
17. Bivariate Correlations, Means, and Standard Deviations for Prenatal and Postnatal Matern	Variables Used in Hypothesis Four.
Table 1	

Variables	Father IQ	Father Educ	Mother IQ	Mother Educ	Family SES	External BPs	Internal BPs	Total BPs
Father IQ	1.00							
Father Educ	.509***	1.00						
Mother IQ	.358***	.414***	1.00					
Mother Educ	.349***	.486***	.516***	1.00				
Family SES	.432***	.647***	.439***	.565***	1.00			
External BPs	167**	203***	142*	056	177**	1.00		
Internal BPs	104	100	081	061	122	.549***	1.00	
Total BPs	147**	169**	118*	067	164**	****68.	.793***	1.00
Child IQ	.319***	.271***	.295***	.318***	.319***	153**	059	180**
Dev Quotient	111.	.048	.046	.114*	.102	.016	.167**	.014
Fine Motor	.120*	.061	.053	660.	.087	.019	.209***	.038
Gross Motor	.049	.023	.014	060.	.054	.054	***661	.060
Language	.141*	.128*	.085	.150**	.170**	031	.130*	035
Impulsivity	.103	.123*	.124*	.039	.038	051	.086	023
F Rate Attent	.154**	.164**	.034	.035	.085	170**	086	193***
M Rate Attent	.113*	.119*	.107	.140*	.182***	116*	014	122*
Prenatal Smok	228***	262***	277***	229***	223***	.200***	.071	.166**
Prenatal Drink	100.	.022	040	.070	022	.127*	.025	.094
Currnt Smoke	270***	364***	349***	347***	305***	***661.	.134*	.179**
Currnt Drink	.079	059	077	103	125*	.161**	.083	.112
Mean	102.74	13.53	96.20	13.21	341.85	21.71	9.79	54.28
Standard Dev	13.85	2.28	14.23	1.96	150.75	8.63	5.98	20.94

50/ = 100. 2 d :10. 2 d ... 'p ≤ .05;

		(n ilinn)						
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	bles	Child IQ	Dev Quotient	Fine Motor	Gross Motor	L'anguage	Immileivity	E Data Attant
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	r IQ					00	furnandan	I IVAILY VIICIII
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	r Educ							
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	er IQ							
100 100 100 130 100 100 359 100 100 359 904*** 100 359 904*** 100 359 904*** 100 356 884*** 100 357 904*** 100 175*** 308*** 100 175*** 308*** 100 175*** 308*** 100 175*** 308*** 100 175*** 308*** 100 175*** 308*** 100 175*** 308*** 100 175*** 308*** 100 175*** 308** 313** 100 012 011 011 101 012 012 013 101 013 013 013 101 013 013 013 101 013 013 013 101 013 01	er Educ							
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	y SES							
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	nal BPs							
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	al BPs							
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	BPs							
40+*** 1.00 256*** 866*** 884*** 1.00 256*** 866*** 884*** 1.00 256*** 866*** 884*** 1.00 759*** 901*** 576*** 384*** 1.00 759*** 901*** 576*** 384*** 1.00 757** 308** 377 0.12 0.01 1.00 757** 108** 377 0.12 0.01 1.57** 0.05 7 1034 0.12 0.01 0.12 0.01 1.57** 6 .033 .090 .034 .030 .059 .059 6.05 .013 .014 .018 .026 .079 .059 6.09 .014 .018 .029 .026 .056 .056 .056 6.09 .014 .026 .026 .056 .056 .056 .056 .056 .056 .056 .056 .056 .056 <td< td=""><td>IQ</td><td>1.00</td><td></td><td></td><td></td><td></td><td></td><td></td></td<>	IQ	1.00						
358*** 904*** 1.00 359*** 904*** 1.00 259*** 368*** 1.00 259*** 301*** 376*** 375** 308*** 313*** 175** 308*** 326*** 175** 308*** 326*** 175** 308*** 313*** 174 071 201** 276*** 1100 005 174 071 201** 184*** 071 201** 184*** 071 201** 1007 205 012 001 1017 205 012 001 1017 009 014 056 073 105*** 013 009 034 076 073 105*** 013 009 024 076 073 1015 014 012 010 076 073 1018 013 014 014 076	Duotient	.404***	1.00					
264*** 866*** 884*** 100 175*** 306*** 884*** 100 175*** 308*** 370 104 175*** 308*** 370 313** 2013 308*** 370 313** 2014 313** 100 1710 314** 313** 2013 301 313** 2014 313** 100 2134** 301 301* 305 2013 301 313** 301 305 2014 301 301 301 305 2014 301 301 301 303 2014 301 303 303 303 2015 303 303 303 303 2013 401 -018 -025 056 2013 301 3556 5607 5607 2014 3550 3556 5607 566	Aotor	.358***	.904***	1.00				
5.20*** 901*** 876*** 840*** 1.00 175*** 308*** 322*** 336*** 1.00 075 308*** 322*** 336*** 1.00 073 308** .05 .01 .00 175** .068 .071 .001 .001 185** .185*** .071 .001 .013 1 .074 .012 .001 .015 1 .007 .033 .009 .034 .035 1 .071 .012 .001 .035 .073 1 .073 .003 .034 .036 .073 1 .071 .012 .011 .015 .075 1 .073 .009 .034 .026 .076 .076 .049 .018 .029 .026 .056 .056 .056 .049 .013 .013.0 .13.09 .074 .076 .075	Motor	.264***	.866***	.884***	1.00			
175** 308*** 326*** 326*** 108 100 7 313** 101 313** 100 7 276*** 38*** 101 307 005 7 184** 071 201* 014 005 6 -134* 071 201* 041 041 6 -019 .026 .012 .001 .157** 045 6 -019 .026 .012 .001 .059 .059 6 -014 .018 .026 .033 .099 .056 6 -014 .018 .029 .026 .056 .056 1049 .014 .018 .029 .026 .056 .056 103 .012 .013 .029 .026 .056 .056 1049 .014 .018 .023 .026 .056 .056 1049 .014 .018 .023 .026	age	.529***	***106"	.876***	.840***	1.00		
073 0.68 072 0.14 0.57 005 276*** 188*** 184*** 071 201** 065 5 -13** 188*** 184*** 071 201** 065 5 -13** 007 -001 0.17 -001 1.17** 5 -030 -007 -040 -0.38 -0.39 -0.99 5 -16** -014 -0.38 -0.34 -0.76 -0.73 6 -014 -018 -029 -0.76 -076 -056 -049 -014 -018 -029 -076 -056 -056 10.5 5.01 55.07 56.07 64.86 - 10.5 56.07 13.09 73.56 56.07 64.86 -	sivity	.175**	.308***	.320***	.326***	.313***	1.00	
226*** 18**** 18**** 071 201*** 041 134* 019 026 012 001 1.57** 6 -039 007 -032 001 0.57** 167** -031 009 -034 -035 073 -167** -033 009 -034 -026 073 -049 -014 -018 -026 -076 -075 -049 -014 -018 -026 -056 -056 103.84 5.01 53.56 5.07 5.67 5.486 103.84 5.01 13.59 14.50 17.60 15.66 164.86	: Attent	.073	.068	.072	.014	.057	005	1 00
(*	e Attent	.276***	.185***	.184***	.071	.201***	041	236***
x -007 -040 -038 -039 -059 -167** -033 -009 -034 -035 -073 -167** -013 -009 -034 -076 -073 -049 -018 -014 -076 -075 -075 -049 -018 -026 -026 -056 103.84 56.07 55.01 53.56 56.07 64.86 14.15.64 15.09 13.59 13.69 73.69 74.86	tal Smok	134*	019	.026	.012	.001	157**	- 039
167** 033 .009 014 026 073 049 014 018 029 073 049 014 018 029 075 013 018 029 076 075 013 018 029 076 075 013 018 029 076 076 013 018 029 076 076 013 013 025 076 076 013 013 023 076 076 013 013 013 014 076	al Drink	050	007	040	038	030	059	-041
014 018 029 026 036 103.84 5607 5501 53356 5607 64.86 14.16 12.64 13.09 14.57 13.60 77.66	Smoke	167**	033	600.	034	026	073	- 069
103.84 56.07 55.01 53.56 56.07 64.86 14.16 12.64 13.09 14.57 13.69 37.60	Drink	049	014	018	029	026	056	083
14.16 12.64 13.09 14.57 13.69 37.60		103.84	56.07	55.01	53 56	56.07	98 19	CE F
	ird Dev	14.16	12.64	13.09	14.57	13.69	37.60	200 0

Table 17. (cont'd)

* $p \le .05$; ** $p \le .01$; *** $p \le .001$ n = 307

	ke Curmt Drink	┢																-			1.00	423.96	1366.95
	Currnt Smoke																			1.00	.206***	1.43	1.96
	Prenatal Drink																		1.00	.148**	.126*	.22	.42
	Prenatal Smok																	1.00	.163**	.678***	.220***	4.60	8.71
(cont'd)	M Rate Attent																1.00	093	053	108	055	5.13	2.84
~.	Variables	Father IQ	Father Educ	Mother IQ	Mother Educ	Family SES	External BPs	Internal BPs	Total BPs	Child IQ	Dev Quotient	Fine Motor	Gross Motor	Language	Impulsivity	F Rate Attent	M Rate Attent	Prenatal Smok	Prenatal Drink	Currnt Smoke	Currnt Drink	Mean	Standard Dev

 $p \le .05; w \ge .01; w \ge .001 n = 307$

of developmental status, child IQ, and behavior problems as measured by the CBCL or for the effects of mothers' prenatal or current drinking levels.

Prenatal and Postnatal Maternal Consumption Influences on Child Behavior Problems Impulsivity

In Table 18, Model 2, impulsivity was regressed against smoking and drinking during pregnancy. Only prenatal smoking was found to have a significant effect accounting for 2.6% of the variance. Significance effects for prenatal smoking were maintained across Models except for a loss of significance of p = .055 in Model 4 and magnitude was increased in Models 6 and 7.

A trimmed model of only smoking during pregnancy was run and results revealed continued significant effect for prenatal smoking that accounted for 2.5% of the variance in impulsivity.

Summary of Analyses of Hypothesis Four: Maternal Consumption Influence on Child Development

The results of analyses for hypothesis four do not support the prediction that mother's current smoking or alcohol use is the better predictor child outcomes than mother's smoking or alcohol use during the prenatal period. Instead, it was found that mother's prenatal smoking was the better predictor than mother's current smoking in terms of child's impulsivity. Mothers who smoked during their pregnancy were more likely to have children that were unable to delay gratification. In a simple task, these children were more likely to reach for the wrapped present prior to the end of a timed one-minute delay.

le 18. In	Table 18. Impulsivity Regressed on Prenatal and Postnatal Maternal Consumption Variables, Standard Coefficients (Betas)	ssed on Prenata	il and Postnatal	Maternal Con	sumption Varia	bles, Standard	Coefficients (E
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Variables							
Father IQ	.051			.041	.049		.041
Father Educ	.134			.122	.136		.139
ğ	.117			.094	.115		.103
Educ	052			052	056		040
SES	093			860	-099		103
Smoke		152**		117		192*	177*
Drink		035		038		035	042
Current Smoke			064		-009	.068	.113
Current Drink			043		052	023	032
R ²	.031	.026	.007	.046	.034	.029	.053

 $p \le .05$; $p \le .01$; $p \le .01$; $p \le .001$ n = 307

Hypothesis Five: Life History and Current Levels of Maternal Drug Involvement and Psychopatholgy Influences on Child Development

Table 19 displays zero-order correlations of the child outcome variables with maternal life history and current variables referred to in this section.

Raw data were entered into EQS Structural Equations Program for PowerMac (v 5.6, Multivariate Software, Inc.) and a covariance matrix was created and analyzed in order to obtain the maximum likelihood estimates of the model coefficients. Two structural equations models (SEMs) were run with the sample of mothers (n = 307). The first model examined the effects of maternal life history as well as current psychopathology and substance use on the child outcomes of behavior problems and impulsivity and the second model examined the same maternal influences on the child outcomes of IQ and language development. The adequacy of fit for both models was determine by considering the following indices in combination, the Chi-Square statistic, the Bentler-Bonett's normed fit index (NFI), nonnormed fit index (NNFI), and comparative fit index (CFI). In general, a good fitting model is one that has a nonsignificant Chi-Square and a NFI, NNFI, and CFI of .90 or larger. In addition to the results of each subsequent run of the models, Lagrange Multiplier and Wald Test were also used as starting points in editing the original models in order to improve the fit.

Though this study's focus was the examination of maternal influences on child outcomes, an exploratory analysis was conducted which included the appropriately paired fathers from the MSU-UM Longitudinal Study. Both behavior problems and IQ models were run with results indicating that the factor structure was not stable due to colinearity among the father and mother variables between the latent constructs. A simplified model

		Mother IQ	M Total ASB	M Worst Depr	Mother LAPS	M Smoke Dep	Mother AxisV
	,			•			
143 1.00	0						
	.358***	1.00					
.232***20	201***	271***	1.00				
	.030	137*	.299***	1.00			
.303***10	106	067	.513***	.243***	1.00		
224***21	217***	380***	.488***	.212***	.390***	1.00	
116*	.197***	.319***	430***	554***	252***	381***	1.00
.093004	04	031	.302***	.330***	.132*	.116*	349***
	62	077	.236***	.059	.339***	.220***	186***
.180**20	207***	349***	.345***	.195***	.186***	.636***	332***
.017 .00	.007	.040	.136*	014	.134*	037	089
026 .13	.130*	.176**	075	186***	350.	160'-	**751.
	.282***	.296***	362***	173**	175**	231***	.393***
.140*16	167**	142*	.333***	.116*	.246***	.264***	221***
053 .14	.141*	.085	037	.049	018	.014	.112
062 .1(.103	.124*	094	076	094	960'-	.164**
.022 .31	319***	.295***	121+	-119*	.007	118*	.240***
.55 102.74	4	96.20	11.18	15.24	9.93	5.94	61.17
.48 13.85	5	14.23	7.37	11.71	2.01	4.92	10.81

Bivariate Correlations. Means, and Standard Deviations for Hypothesis Five Analyses. Table 19.

The second sec	Variables	Mother Depres	Mother OFVR	Mother Smoke	M Neg Affect	M Pos Affect	HOME	Extanual DDa	I among the
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	Express Alcoh				0		TIONE	EXIGINAL DES	Language Dev
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	ather IQ								
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	Aother IQ								
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	A Total ASB								
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	A Worst Depr								
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	Aother LAPS								
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	A Smoke Dep								
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	Aother AxisV								
8 165* 100 6 165* 100 116* 209 -0.3 -116* 309 -0.3 -116* 309 -0.3 -116* 309 -0.3 -116* 309 -0.3 -116* 309 -1.67* -116* 100 100 -116* 199*** 100 -116* 199*** 100 -116* 199*** 100 -116* 199*** 100 -116* 199*** 100 -116* 161** 199*** -116* 199*** 100 -116* 161** 103* -116* 013 210** -116* 161** 163* -116* 164 167* -116* 164 167* -116* 164 167* -116* 164 167*	Aother Depres	1.00							
e 102 206*** 100 116* .089 .0.03 1.00 .010 .089 .0.03 1.00 .011 .013 .013 .00 .011 .013 .013 .00 .011 .013 .013 .010 .014*** .199*** .173** 1.00 .104** .035 .013 .014 .105** .196*** .033 .013 .106* .025 .033 .013 .010 .106 .026 .033 .012 .011 1 .106 .026 .033 .023 .031 .011 .106 .056 .033 .022 .038 .051 .116* .049 .16** .038 .051 .133* .116* .049 .16** .038 .051 .051 .116* .049 .16** .038 .051 .051 .116* <td>Aother QFVR</td> <td>.165**</td> <td>1.00</td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td>	Aother QFVR	.165**	1.00						
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	Aother Smoke	.102	.206***	1.00					
.107 .021 .102 .2.27*** 1.00 .298*** .193*** .288*** .165** .173** 1.00 .298*** .193*** .588*** .165** .173** 1.00 .298*** .193*** .59*** .165** .173** 1.00 .2104 .016 .026 .035 .014 .230*** .031 .100 .026 .026 .033 .014 .230*** .031 1 .036 .073 .023 .023 .023 .031 1 1 .116* .049 .167** .038 .256** .390*** .153** .56 .116* .049 .167** .038 .256*** .300*** .513* .56 .125 .423.56 1.43 .583 .26 .417* .217* .56 .329 .326** .065 .36* .31* .46* .37* .55	A Neg Affect	.116*	.089	033	1.00				
298*** 19**** 258*** 165** .165** .165** .100 * .101 .05 .099*** .166** .100 .001 * .106 .056 .037 .049 .011 .01 * .006 .026 .033 .012 .012 .011 1 * .016 .056 .073 .023 .012 .031 .011 1 * .049 .167** .038 .256*** .30*** .151** .113** * .049 .167** .038 .256*** .30*** .113** .51 * .049 .167** .038 .264* .416 .173** .56 3.22 423.56 .166 .56 .740 .460 .57 .56	A Pos Affect	107	.021	102	277***	1 00			
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	HOME	298***	193***	258***	165**	173**	1 00		
v -100 026 035 014 2.30*** 031 1 .036 .035 .013 .023 .023 .031 1 .036 .073 .023 .023 .031 .031 1 .116* .049 .167** .038 .051 .153** .153** .116* .049 .167** .038 .256*** .390*** .153** .513 .127 .323 .256*** .390*** .153** .513 .55 .517 .55 .128 .325 .6.4 .4176 .217 .56 .56 .55 .56 .56 .55 .55 .56 .56 .55 .56 .56 .55 .55 .56 .56 .56 .55 .55 .56 .56 .56 .56 .56 .56 .56 .56 .57 .56 .57 .56 .57 .56 .57 .57 .56 .56	xternal BPs	.214***	.161**	***661.	.196***	093	183***	1 00	
-036 073 023 022 088 051 -116* 049 167** .038 .256*** .390*** .051 -116* 049 167** .038 .256*** .390*** .173** -116* 049 167** .038 .256*** .390*** .173** -116 049 167** .038 .264 .476 .173 -123 433 264 .4176 .217 .56 -123 .166 .363 .264 .4176 .217 .56	anguage Dev	100	026	026	035	014	.230***	- 031	1 00
	mpulsivity	036	056	073	.023	022	088	- 051	312###
3.22 423.96 1.43 5.83 26.14 44.76 21.71 3.29 1366.95 1.96 2.56 2.01 4.60 e.23	Child IQ	116*	049	167**	038	.256***	.390***	153**	.529***
3.29 1366.95 1.96 2.56 7.01 4.60 2.171	fean	3.22	423.96	1.43	5.83	26.14	44.76	12.10	LV 73
	tandard Dev	3.29	1366.95	1.96	2.56	2 01	4 60	0 63	10.00

(cont'd) Table 19 *p ≤ .001 n = 307 10. < d 'cn < 2

	Child IO																		1.00	103.84	14.16	n = 307
(cont'd)	Impulsivity																	1.00	.175**	64.86	37.60	⁺ p ≤ .05; ⁺⁺ p ≤ .01; ⁺⁺⁺ p ≤ .001
Table 19.	Variables	Express Alcoh	Father IQ	Mother IQ	M Total ASB	M Worst Depr	Mother LAPS	M Smoke Dep	Mother AxisV	Mother Depres	Mother QFVR	Mother Smoke	M Neg Affect	M Pos Affect	HOME	External BPs	Language Dev	Impulsivity	Child IQ	Mean	Standard Dev	*p ≤ .05; **p ≤ .

ſ

was not possible due to the number of variables for each factor (See Appendix B for an example of these difficulties). The following results are based on this study's sample which included 307 mothers and their male preschoolers.

Measurement Models

Child Behavior Problems

The child behavior problems model consisted of five latent variables and 14 indicator variables (See Figure 1 for hypothetical behavior problems measurement model). The latent constructs were alcohol pervasiveness, lifetime drug involvement and psychopathology, current drug involvement and psychopathology, parenting behaviors and attitudes, and child behavior problems. Alcohol pervasiveness was composed of family expression of alcoholism, lifetime alcohol problems, and current alcohol consumption. Lifetime drug involvement and psychopathology was composed of smoking dependency, lifetime antisociality, and worst ever depression. Current level of smoking, depression, and adaptive functioning comprised the latent construct of current drug involvement and psychopathology. Parenting behaviors and attitudes was composed of the HOME and two factors of parenting attitudes, positive affect and negative affect. Child externalizing behavior problems and impulsivity comprised the latent construct of child behavior problems.

Examination of the final trimmed model¹⁰ for behavior problems found the overall model fit was acceptable with fit indices larger than .90 and a NFI of .91, even though the Chi-Square was significant $[X^2(64, n = 307) = 101.88, p < .01]^{11}$ (See

¹⁰ The path from parenting behaviors and attitudes to child behavior problems was not significant (z = -.757) and was dropped from the final trimmed model. See Appendix C for non-trimmed model.

¹¹ Many researchers accept a chi-square value if it is less than twice the degrees of freedom.

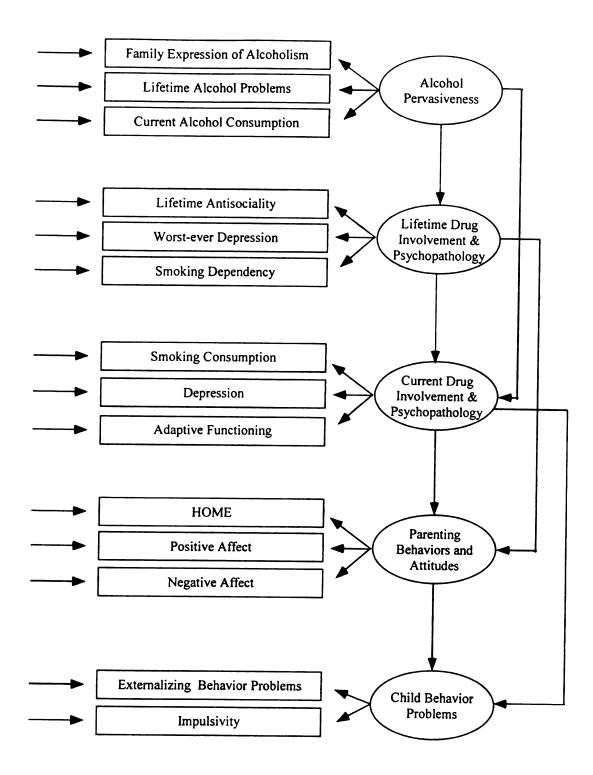


Figure 1. Hypothetical measurement model for child behavior problems and impulsivity.

Figure 2 for the standardized solution and Table 20 for the z values). The model revealed that as alcohol pervaded more of the mother's life, she was more likely to have experienced a severe episode of depression and a history of antisociality and smoking addiction. The model showed that high levels of lifetime drug involvement and psychopathology predicted high levels of current drug involvement and psychopathology¹². Alcohol pervasiveness directly predicted lower levels of current depression and smoking and higher levels of adaptive functioning. An unexpected result which will be discussed more fully below. High maternal levels of depression and smoking as well as low levels of adaptive functioning were related to the mother providing a less stimulating environment and adhering to more negative parenting attitudes. Finally, mother's depression, level of smoking and lower levels of adaptive functioning predicted higher levels of behavior problems exhibited by her son.

An examination of the measurement model showed that all indicators loaded significantly on their respective constructs. Within alcohol pervasiveness, both family expression of alcoholism and lifetime alcohol problems loaded strongly $[-.75 (-6.10)^{13}$ and -.90-6.11), respectively] with current alcohol consumption loading much smaller $[-.37(0.0)^{14}]$. A similar trend of combinations of strong and weak loadings occurred for the constructs of parenting behaviors and attitudes with HOME loading the strongest [.74 $(0.0)^{14}$ compared to .23 (2.75) for positive affect and -.23 (-2.78) for negative affect] and

¹² Due to the negative loadings of the indicators onto lifetime drug involvement and psychopathology, a negative path coefficient between this construct and current drug involvement and psychopathology is supportive of the hypothesis.

¹³ Z values for loadings are in parentheses.

¹⁴ Variance of the parameter was set to zero indicating that the statistical variability of the parameter estimate can not be accurately computed. Because the solution converged, the parameter estimates are considered correct.

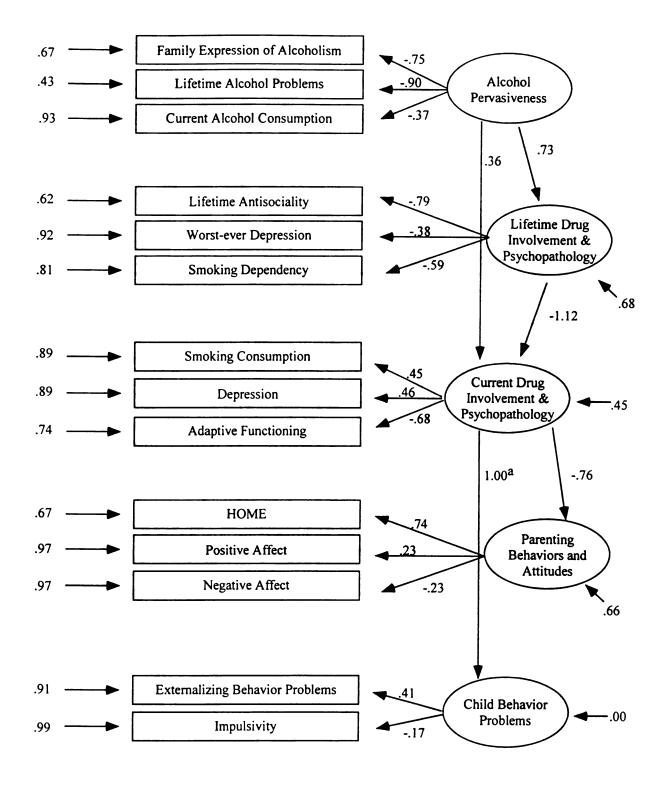


Figure 2. Child behavior problems and impulsivity standardized solution.

^a This parameter was estimated at 1.00 by EQS, it was not fixed.

	Estimates	Z values	Error Estimates	Z values
Variables				
Family Express. Alcoholism	75	-6.10	.67	8.43
Lifetime Alcohol Problems	90	-6.11	.43	3.24
Current Alcohol Consumption	37	0.0ª	.93	12.03
Antisociality	79	-9.51	.62	6.90
Worst Ever Depression	38	-5.71	.92	11.96
Smoking Dependency	59	0.0*	.81	10.82
Adaptive functioning	68	-2.53	.74	8.76
Current Depression	.46	2.44	.89	11.24
Current Smoking	.45	2.44	.89	11.33
HOME	.74	0.0ª	.67	2.73
Positive Affect	.23	2.75	.97	12.01
Negative Affect	23	-2.78	.97	12.00
Child Behavior Problems	.41		.91	11.52
Impulsivity	17	-2.42	.99	12.25
Factors				
Lifetime Drug Involvement	.73 F1	5.28	.68	4.21
and Psychopathology F2				
Current Drug Involvement and	.36 F1	0.0 ^a	.45	.93
Psychopathology F3	-1.12 F2	-3.29		
Parent Behavior Attitude F4	76 F3	-2.48	.66	1.45
Child Behavior Problems F5	1.00	2.4		
Correlations				
Worst Ever Depression	47	-6.49		
Adaptive functioning				
Worst Ever Depression	.20	3.58		
Current Depression				
Smoking Dependency	.55	7.61		
Current Smoking				
Positive Affect	24	-3.90		
Negative Affect	at to some indian	1		California de la companya de la comp

 Table 20.
 Solution for Child Behavior Problems Model.

^{*}Variance of the parameter was set to zero indicating that the statistical variability of the parameter estimate can not be accurately computed. Because the solution converged, the parameter estimates are considered correct.

child behavior problems with externalizing behavior problems loading stronger than impulsivity [.41 compared to -.17 (-2.42)]. Lifetime drug involvement and psychopathology indicators loaded over a wide range with antisociality loading strongest at -.79 (- 9.51) and worst ever depression loading weakest at -.38 (-5.71). Finally, current drug involvement and psychopathology indicators all loaded similarly onto this construct.

Overall, the results from the child behavior problems model demonstrated that the majority of the hypothesized relationships were verified. However, one finding was contrary to what was hypothesized, the positive relationship between alcohol pervasiveness and current drug involvement and psychopathology. Based on the negative loadings of the indicators on alcohol pervasiveness and the positive loadings of smoking consumption and depression on current drug involvement and psychopathology, a negative path coefficient would provide support for the hypothesis that more alcohol pervasiveness predicts higher levels of current drug involvement and lower levels of adaptive functioning.

Because this relationship was contrary to what was expected, recoding and further analysis was conducted to test the veracity of the relationship. The path from alcohol pervasiveness to current drug involvement and psychopathology was fixed to a value of -1.0 in order to test a negative relationship. The results showed a reversal of the loading signs of the alcohol pervasiveness indicators onto the alcohol pervasiveness construct, thus making a negative path coefficient from alcohol pervasiveness to current drug involvement and psychopathology non-supportive of the hypothesis (See appendix D for this result).

A plausible explanation is that the main path between alcohol pervasiveness and current drug involvement and psychopathology is an indirect path through lifetime drug involvement and psychopathology. Thus, the path from alcohol pervasiveness to current drug involvement and psychopathology is what is left over from the indirect path and represents the variance that is not explained by the indirect path. This is supported by the difference in magnitude in the path coefficients between alcohol pervasiveness and lifetime drug involvement and psychopathology ($\mu = .73$, z = 5.28) and lifetime drug involvement and psychopathology to current drug involvement and psychopathology ($\mu = .1.12$, z = -3.29) compared to the path coefficient from alcohol pervasiveness to current drug involvement and psychopathology ($\mu = .36$, $z = 0.0^{15}$).

Mothers who were characterized as having high levels of alcohol pervasiveness but not high levels of lifetime addiction and psychopathology were currently not smoking or depressed and are functioning at a higher adaptive level. This was supported by crosstabulations using median breakpoints for each of the indicators within the three constructs: alcohol pervasiveness, lifetime drug involvement and psychopathology and current drug involvement and psychopathology. Each mother was a assigned a 1.0 for each variable value that was greater than the median value for that variable. The recoded variables were summed within each construct with a range of 0 to 3 for the summed computed values for each of the constructs.

The median was chosen as the breakpoint instead of the upper quartile versus the lower three quartile approach (extreme groups approach) for two reasons. First this

¹⁵ Variance of the parameter was set to zero indicating that the statistical variability of the parameter estimate can not be accurately computed. Because the solution converged, the parameter estimates are considered correct.

approach generates a more representative measure of the total range of change experienced by the subjects. Second, this approach is less likely to truncate the variance. This approach has been used by other studies in the MSU-UM Longitudinal Study with good results (Bingham, Zucker, & Fitzgerald, under review-b; Moses, Gonzalez, Zucker, & Fitzgerald, 1993).

The results of the first crosstabulation show that mothers who have a family history of alcoholism and who have many lifetime alcohol problems, currently drink, have a high smoking dependency, are antisocial, and have had a severe worst-ever depressive episode are more likely to currently be depressed, smoke, and have lower adaptive functioning compared to those mothers who have a family history of alcoholism and who have many lifetime alcohol problems, currently drink, but never smoked, are not antisocial, and were never depressed. See Table 21 for the crosstabulation results. For increased clarity and ease of comparison, low and high categories in Table 21 for both current and lifetime included the scores of 0 to 1 for low and 2 to 3 for high.

In the second crosstabulation, high alcohol pervasiveness was split into two groups medium alcohol pervasiveness for those who scored above the median for two of the three variables and high alcohol pervasiveness for those who scored above the median for all three variables and was entered into a crosstabulation with low and high current drug involvement and psychopathology. It was found that for those 83 mothers who were classified as medium alcohol pervasiveness, they were almost equally divided between low and high current drug involvement and psychopathology (See Table 22). In other words, a mother who scored above the median for two of the three alcohol pervasiveness variables can just as likely be currently smoking, depressed, and

Table 21.Comparison of High and Low Lifetime Drug Involvement and
Psychopathology to High and Low Current Drug Involvement and
Psychopathology Among Those Mothers' with High Levels of Alcohol
Pervasiveness.

	Low Lifetime	High Lifetime	Totals
Low Current	45	28	73
	29.4	18.3	47.7
High Current	15	65	80
	9.8	42.5	52.8
Totals	60	93	153
$I^{2}(1 - 152) = 2$	39.2	60.8	100.0

 $X^{2}(1, n = 153) = 29.46, p < .000.$

Table 22.Comparison of Medium and High Alcohol Pervasiveness to Low and High
Current Drug Involvement and Psychopathology.

	Medium. Alcohol	High Alcohol	Totals
Low Current	41	32	73
	26.8	20.9	47.7
High Current	42	38	80
	27.5	24.8	52.3
Totals	83	70	153
	54.3	45.7	100.0

 $X^{2}(df = 3, n = 153) = 8.12, p < .04.$

functioning at a low adaptive level or not be currently smoking and depressed as well as functioning at a high adaptive level. Therefore, a negative relationship (in this case represented as a positive path coefficient in Figure 2) between alcohol pervasiveness and current drug involvement and psychopathology is an accurate reflection of the data.

Child IQ

The child IQ model consisted of five latent variables and 15 indicator variables (See Figure 3 for hypothetical IQ measurement model). The latent constructs were parental IQs, lifetime drug involvement and psychopathology, current drug involvement and psychopathology, parenting behaviors and attitudes, and child IQ. Parental IQs was composed of paternal IQ and maternal IQ. Lifetime drug involvement and psychopathology was composed of the maternal variables of lifetime alcohol problems, smoking dependency, lifetime antisociality, and worst ever depression. Maternal current levels of smoking, drinking, depression, and adaptive functioning comprised the latent construct of current drug involvement and psychopathology. Parenting behaviors and attitudes was composed of the HOME and two factors of parenting attitudes, positive affect and negative affect. Child IQ and language development comprised the latent construct of child IQ.

Examination of the final trimmed model¹⁶ for IQ found a significant Chi-Square $[X^{2}(56, n = 307) = 104.15, p < .001]^{17}$, however, the overall model fit was acceptable with

¹⁶The path from current drug involvement and psychopathology to child IQ was not significant at the two-tail level (z = 1.91) and the path failed the one-way test of significance loading positively instead of negatively (F5 = .689F3), therefore it was dropped from the final trimmed model along with the variable negative affect which did not load significantly onto the parenting behaviors and attitudes construct (z = 1.68). See Appendix C for non-trimmed model.

¹⁷Many researchers accept a chi-square value if it is less than twice the degrees of freedom.

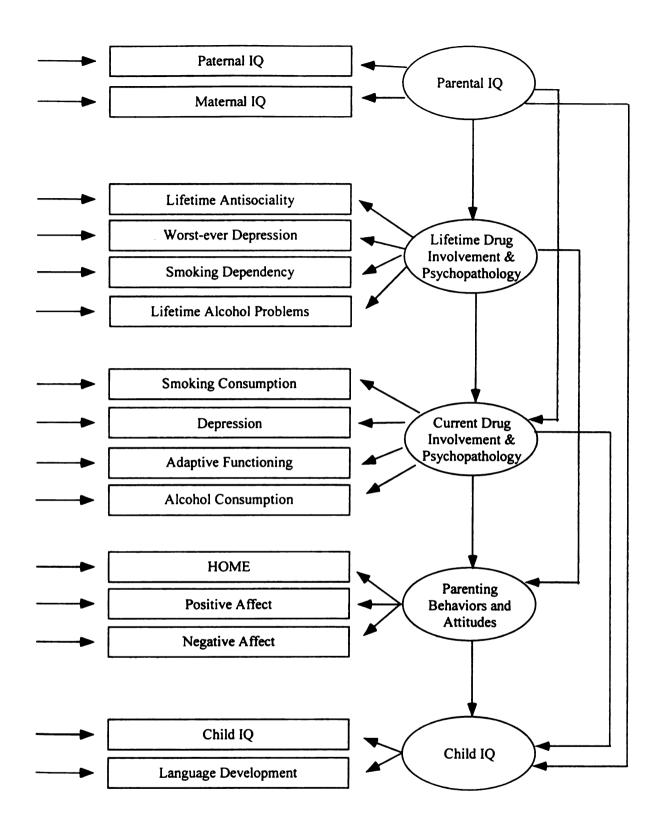


Figure 3. Hypothetical measurement model for child IQ and language development.

fit indices of .90 or larger and a NFI = .91, (See figure 4 for the standardized solution and Table 23 for the z values). The model revealed that lower parental IQs predicted higher maternal levels of lifetime antisociality, alcohol problems, and smoking dependency and an increased likelihood of a past, severe episode of depression¹⁸. Current maternal levels of depression, drinking, and smoking were higher and adaptive functioning were lower when parental IQ was lower. Higher levels of lifetime antisociality, alcohol problems, smoking dependency, and a severe depressive episode predicted both higher levels of current depression, drinking, smoking, and lower levels of adaptive functioning¹⁹ as well as a more stimulating home environment and more positive parenting attitudes adhered to by the mother. However, current drinking, depression, and smoking as well as lower levels of current adaptive functioning predicted a less stimulating home environment and less positive parenting attitudes adhered to by the mother. Finally, a stimulating home environment and less positive parenting attitudes adhered to by the mother IQ and more advanced language development for the child.

An examination of the final trimmed measurement model revealed similar trends as those found in the behavior problems model, that is a combination of strong and weak loadings on a construct. Lifetime and current drug involvement and psychopathology, ,attitudes, and child IQ all have a wide range of loading values. Once again, antisociality has the largest loading and worst ever depression has the smallest loading on

¹⁸ Due to the negative loadings of the indicators onto lifetime drug involvement and psychopathology, a positive path coefficient between parental IQ and this construct is supportive of the hypothesis.

¹⁹ Due to the negative loadings of the indicators onto lifetime drug involvement and psychopathology, a negative path coefficient between this construct and current drug involvement and psychopathology is supportive of the hypothesis.

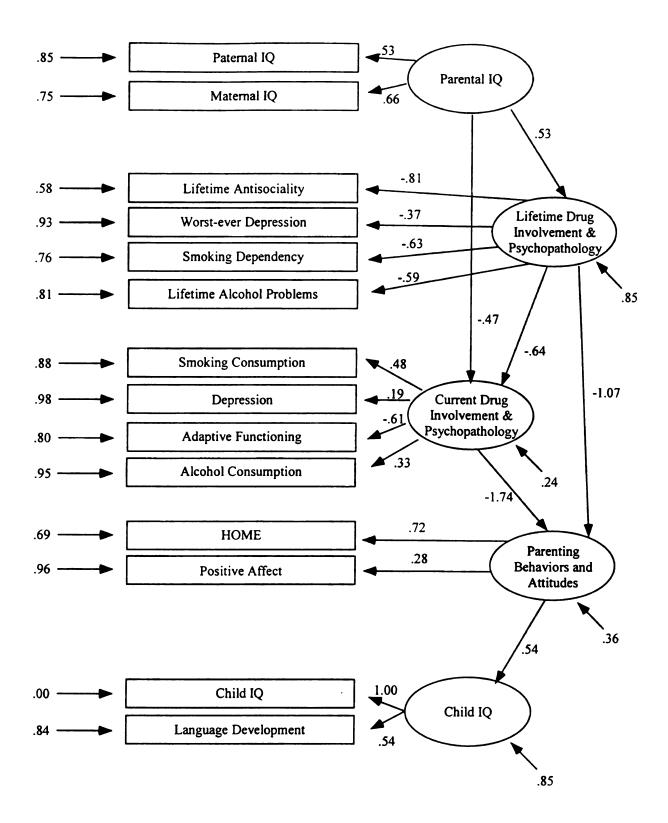


Figure 4. Child IQ and language development standardized solution.

	Estimates	Z values	Error Estimates	Z values
Variables				
Paternal IQ	.53	4.80	.85	10.00
Maternal IQ	.66	4.73	.75	6.77
Antisociality	81	0.0*	.58	5.55
Worst Ever Depression	37	-5.71	.93	11.91
Smoking Dependency	63	-9.19	.76	9.95
Lifetime Alcohol Problems	59	-8.71	.81	10.47
Adaptive functioning	61	-7.14	.80	10.70
Current Depression	.19	2.67	.98	12.28
Current Smoking	.48	0.0*	.88	11.53
Current Alcohol Consumption	.33	4.69	.95	12.04
HOME	.72	3.98	.69	5.99
Positive Affect	.72	0.0*	.96	12.00
Child IQ	1.00	0.0	.70	12.00
Language Development	.54	11.56	.84	12.37
Factors		11.50	7 0.	12.37
	(2.5)	0.04	0.5	
Lifetime Drug Involvement	.53 F1	0.04	.85	5.66
and Psychopathology F2	47.51	2.00	24	
Current Drug Involvement and	47 F1	-3.88	.24	1.15
Psychopathology F3	64 F2	-5.03	26	
Parent Behavior Attitude F4	-1.07 F2	-2.32 -3.02	.36	.72
Child IO Es	-1.74 F3 .54 F4	3.92	.85	10.27
Child IQ F5	.54 F4	3.92	۲۵.	10.27
Correlations				
Maternal IQ	18	-1.65		
HOME				
Current Depression	.25	3.16		
Antisociality		() (
Worst Ever Depression	48	-6.96		
Adaptive functioning		4.22		
Worst Ever Depression	.28	4.77		
Current Depression	12	2.5(
Worst Ever Depression Positive Affect	13	-2.56		
	14	2.72		
Worst Ever Depression	.14	2.72		
Language Development	62	7.02		
Smoking Dependency	.52	1.02		
Current Smoking Lifetime Alcohol Problems	.23	3.64		
	.23	3.04		
Current Drinking Adaptive functioning	29	-4.52	<u>├</u>	
	27	-4.52		
Current Depression Current Depression	22	-3.13		
HOME	22	-3.15		
Positive Affect	18	-3.06	<u>├</u>	
Language Development	10	-5.00		
Variance of the parameter was s	at the many indian	ting that the sta		Cab a management

Table 23.Solution for Child IQ Model.

^{*} Variance of the parameter was set to zero indicating that the statistical variability of the parameter estimate can not be accurately computed. Because the solution converged, the parameter estimates are considered correct.

lifetime drug involvement and psychopathology [-.81 (0.0)^{20.21} and -.37 (-5.71),

respectively]. HOME [.72 (3.98)] loads stronger on parenting behaviors and attitudes than positive affect [.28 $(0.0)^{21}$] and Child IQ loading is almost double that of language development [1.00 and .54 (11.56), respectively]. Unlike the loadings of the indicators for current drug involvement and psychopathology construct in the child behavior problems model, the loadings on this construct in the child IQ model are wide ranging with -.61 (-7.14) for adaptive functioning and .19 (2.67) for current depression. Finally, both paternal and maternal IQ loadings on parental IQ are similar in magnitude [.53 (4.80) and .66 (4.73), respectively].

Overall, the results from the child IQ model demonstrated that the majority of the hypothesized relationships were verified. However, one finding was contrary to what was hypothesized, the negative relationship between lifetime drug-involvement and psychopathology and parenting behaviors and attitudes. Because the indicators for lifetime drug involvement and psychopathology loaded negatively onto this construct the path coefficient from lifetime drug involvement and psychopathology to parenting behaviors and attitudes should be positive in order to support the hypothesis. Instead the model revealed that higher levels of lifetime antisociality, alcohol problems, smoking dependency, and a severe depressive episode predicted a more stimulating home environment and more positive parenting attitudes adhered to by the mother.

²⁰Z values for loadings are in parentheses.

²¹ Variance of the parameter was set to zero indicating that the statistical variability of the parameter estimate can not be accurately computed. Because the solution converged, the parameter estimates are considered correct.

Because this relationship was contrary to what was expected, recoding and further analysis were conducted to test the veracity of the relationship. The path from lifetime drug involvement and psychopathology to parenting behaviors and attitudes was fixed to a value of +1.0 in order to test a positive relationship. Convergence for the model was lost and never regained even when the fixed value for the path from lifetime drug involvement and psychopathology to parenting behaviors and attitudes was lowered from +1.0 to +0.7, to +0.5, to +0.3, and finally, to +0.1. Additional start values were added and allowed to vary for other paths and indicators, but convergence was never obtained even though all indices of fit were above .85.

As with the behavior problems model, a plausible explanation is that the main path between lifetime drug involvement and psychopathology and parenting behaviors and attitudes is an indirect path through current drug involvement and psychopathology. Thus, the path from lifetime drug involvement and psychopathology to parenting behaviors and attitudes is what is left over from the indirect path and represents the variance that is not explained by the indirect path. This is supported by the difference in magnitude in the path coefficients between lifetime drug involvement and psychopathology and current drug involvement and psychopathology ($\mu = -.64$, z = -5.03) and current drug involvement and psychopathology to parenting behaviors and attitudes ($\mu = -1.74$, z = -3.02) compared to the path coefficient from lifetime drug involvement and psychopathology to parenting behaviors and attitudes ($\mu = -1.07$, z = -2.32).

A mother who had both high levels of lifetime drug involvement and psychopathology and current drug involvement and psychopathology was less likely to provide a stimulating environment for her son and to adhere to positive parenting

attitudes compared to a mother who had high levels of life time drug involvement and psychopathology but had low levels of current drug involvement and psychopathology. This is supported by the results of a crosstabulation created using the same procedure as discussed for the behavior problems model. The ranges for the constructs were from 0 to 4 for both lifetime and current drug involvement and psychopathology and 0 to 2 for parenting behaviors and attitudes. For increased clarity and ease of comparison, low, medium, and high categories in Table 24 for current drug involvement and psychopathology included the scores of 0 to 1 for low, 2 for medium, and 3 to 4 for high.

Table 24.Comparison of Low, Medium, and High Current Drug Involvement and
Psychopathology to Low, Medium, and High Levels of Parenting Behaviors
and Attitudes Among Those Mothers' with High Levels of Lifetime Drug
Involvement and Psychopathology.

	Low Current	Med Current	High Current	Totals
T D		21	26	(0)
Low Parenting	3	21	36	60
	2.8	19.8	34.0	56.6
Med Parenting	5	7	17	29
	4.7	6.6	16.0	27.4
High Parenting	2	6	9	17
	1.9	5.7	8.5	16.0
Totals	10	34	62	106
	9.4	32.1	58.5	100.0

 $X^{2}(df = 4, n = 106) = 4.12, p < .39.$

For parenting, low category was assigned for a score of zero, medium category was assigned for a score of one, and high category was assigned for a score of two. A high level of lifetime drug involvement and psychopathology was defined as a score of 3 or 4. The results of the crosstabulation show that high levels of both lifetime and current drug involvement and psychopathology predicted lower levels of parenting behaviors and attitudes. An odds ratio of 1.39 is computed for the likelihood that a mother with high levels of both lifetime and current drug involvement and psychopathology would provide a less stimulating environment and be less likely to adhere to positive parenting attitudes. However, there is an odds ratio of .64 that she would be able to at least provide a stimulating environment or adhere to positive parenting.

Due to the small number of mothers who have high levels of lifetime drug involvement and psychopathology but low levels of current drug involvement (10 mothers or 9.4%), an odds ratio comparison may not be accurate. However, one can see that of the ten mothers in this category, seven are categorized at the medium or high levels of parenting. In other words, these mothers were able to provide a stimulating environment, or adhere to positive parenting attitudes, or both.

Based on the crosstabulation results, two trends were identified. A high level of lifetime drug involvement and psychopathology can predict high levels of parenting behaviors and attitudes when low levels of current drug involvement and psychopathology exist. Second, for a small group of mothers a high level of lifetime drug involvement and psychopathology as well as high levels of current drug involvement and psychopathology can predict medium to high levels of parenting. Therefore, it is important to note that high levels of lifetime and current drug involvement and psychopathology does not always predict low levels of parenting. Summary of the Results of Hypothesis Five: Life History and Current Levels of Maternal Drug Involvement and Psychopathology Influences on Child Development

The overall results of the child behavior problems and child IQ structural equation models supported the hypothesized relationships. In general, child behavior problems are directly related to mother's current drug involvement and psychopathologies and indirectly related to her lifetime drug involvement and psychopathologies with the foremost predictor of child behavior problems being an environment of alcohol. The parenting behaviors and attitudes construct was not significantly related to child behavior problems.

On the other hand, child IQ is only indirectly affected by both mother's current and lifetime drug involvement and psychopathology. Instead, the parenting behaviors and attitudes that the mother adhered to are directly related to her son's IQ and language development with both paternal and maternal IQs foremost in the model.

CHAPTER 5

Discussion

Introduction

The results of the present study can be classified into two areas of influences each affecting a specific child outcome. First, a complex environment of alcohol was found to predict child behavior problems and impulsivity. In the child behavior problems SEM, this environment of alcohol was the foremost predictor of child behavior problems. Second, parenting behaviors and attitudes predicted child IQ and developmental status and mediated the effects of mothers' drug involvement and psychopathology on these same child outcomes.

Environment of Alcohol

In the present study, an environment of alcohol was based on three variables: family expression of alcoholism, lifetime alcohol problems, and current alcohol consumption. These variables measured the mother's past exposure to alcohol problems and alcoholism within her family of origin, her own past experiences with alcohol problems, and her current drinking levels. Also, by the nature of the sampling for the larger longitudinal study of which this study is a sub-sample, there is the influence of the mother's partner's alcohol problems and alcoholism.

An environment of alcohol would be characterized as follows, a mother who reported a high number of her relatives currently suffering or having suffered from alcoholism or problems with alcohol, who had personal history of lifetime alcohol problems, and who was currently consuming a high level of alcohol. Based on previous findings from the same longitudinal study, the mother was also likely to be married to a man very similar to her in terms of his family of origin being marred by alcoholism and alcohol problems as well as his own history of alcohol problems and high current levels of drinking (Caplan, 1996; Ichiyama et al., 1995).

Based on the results of the current study and previous studies utilizing subsamples of the MSU-UM Longitudinal Study (Caplan, 1996; Ellis et al., 1996; Ichiyama et al., 1995), a more complete picture of this environment of alcohol can be hypothesized. As a child, the mother was exposed to family members who drank in excess and whose lives were troubled by alcohol related problems as well as depression (Caplan, 1996) and lower SES (Ichiyama et al., 1995). As the mother entered school and her teen years, she was more likely to have taken part in antisocial behaviors and was less likely to have received additional education or training beyond high school (if she finished). As an adult, she was more likely to continue to drink and to have experienced more alcohol related problems including, regular physical and social consequences of alcohol use. The mother also continued to take part in antisocial behaviors. She was also more likely to be depressed. When she married, her partner was more likely to be an alcoholic compared to other mothers in the sample who did not experience this environment of alcohol (Ellis et al., 1996; Ichiyama et al., 1995). Thus, her family of procreation was more likely to be characterized by drinking, antisociality, lower SES, more economic and legal problems, and domestic violence (Caplan, 1996; Ellis et al., 1996; Ichiyama et al., 1995).

As the mother's life became entangled with alcohol, this set into motion a series of relationships for the mother. An environment of alcohol found within both families of origin and procreation was the setting for the mother's learning of the drug-related

behaviors of smoking and drinking as well as the beginning and continued reinforcement of certain psychopathologies, such as antisociality and depression. These behaviors and structures became more maladaptive and the mother increased her current levels of smoking and drinking and concurrently her level of depression increased as her level of adaptive functioning decreased. Finally, her increased levels of current maladaptive behaviors and structures resulted in higher levels of externalizing behavior problems and impulsivity (maladaptive behaviors) exhibited by her son.

On the other hand, there are many pathways the mother could have taken during her lifetime. The following is an example of one alternative pathway. A mother who was raised in an alcoholic family could have "adopted" a friend's family and spent most of her time in this friend's household. This exposure to a non-alcoholic family was an impetus for the mother to chose friends who did not drink or take part in antisocial behaviors. With the social support of this "adopted" family, the mother obtained additional education or training beyond high school. When the mother married, her spouse was not alcoholic and her family of procreation was characterized by normative drinking behaviors and lower levels of psychopathology as well as higher SES and fewer legal and economic problems compared to the previous mother whose family of procreation was marred by alcoholism.

An environment of alcohol based on the mother's postnatal drinking has been examined in only a few studies (Brown et al., 1991; Dawson, 1992; Hamdan-Allen et al., 1989). Usually the focus is on mother's prenatal drinking and the father's postnatal drinking. The latter has been the focus for a number of papers within the MSU-UM Longitudinal Study. It has been found that children of fathers who were classified as

antisocial alcoholics exhibited more externalizing behavior problems (Ellis et al., 1996). Children of court alcoholics were more likely to be impulsive compared to control families (Ham, Fitzgerald, & Piejak, 1994; Fitzgerald et al, 1993) and to be rated in the extreme clinical range for behavior problems (Fitzgerald et al, 1993; Jansen et al, 1995). Another study found that just having one parent with the diagnosis of alcoholism was related to the child being rated in the extreme clinical range for behavior problems (Bingham, Fitzgerald, Townsend, & Zucker, 1995).

One cross-generational study from the MSU-UM data set examined the effects of both mothers and fathers combined, reported substance abuse and psychopathology on child behavior problems (Piejak & Loukas, 1995). It was found that both parental and grandparental effects of alcohol abuse predicted child/grandchild behavior problems. In other words, an environment of alcohol within the mother's family of origin and procreation is related to more behavior problems in her child.

In addition, the literature outside the longitudinal study that does exist on mother's postnatal drinking (Brown et al., 1991; Dawson, 1992; Hamdan-Allen et al., 1989) supports the findings of the current study in terms of mother's current drinking being related to a higher number of externalizing behavior problems exhibited by her child.

One possible counter argument to the above scenario is a biological basis for the child behavior problems. This argument builds on the environment of alcohol idea but instead focuses on the behavioral teratology aspects of the relationship between maternal alcohol use and child behavior problems. Behavioral teratology is the study of damage to the developing brain in utero and its affect on later behavior (Vorhees & Mollnow, 1987).

It could be hypothesized that the preschoolers in the present study are exhibiting behavior problems not as a social reaction to the mother's environment of alcohol but as a result of a biological predisposition to externalizing behavior problems and impulsivity related to the children's exposure to alcohol during the prenatal period.

The present study examined this affect in terms of the influences of prenatal exposure to mother's drinking and smoking. However, the present study was limited by the use of retrospective prenatal data. Results did show that mother's prenatal smoking was predictive of externalizing behavior problems in her son and that her levels of prenatal smoking were more predictive of her preschool son's impulsivity than her current levels of smoking.

However, there was no significant relationship between mother's prenatal drinking levels and her son's behavior problems and impulsivity. This may be more reflective of the nature of prenatal drinking consumption compared to prenatal smoking. As previously mentioned in chapter two, results from several studies found that women are more likely to maintain their pre-pregnancy levels of smoking throughout their entire pregnancies making it easier to recall the quantity smoked, whereas nearly all the women reduced their levels of drinking and over half stopped drinking (Condon & Hilton, 1988; Day, 1992; Day & Richardson, 1991; Hilton & Condon, 1989). Due to this trend, retrospective data on prenatal drinking may not be as accurate as retrospective data on prenatal smoking. Therefore, the lack of significant findings in the present study for prenatal exposure to drinking may be a result of using retrospective data. A longitudinal study beginning prior to conception and continuing through the preschool years would examine this counter argument.

Direct and Mediating Effects of Parenting Behaviors and Attitudes

The other area of influence identified in the present study is the direct and mediating effects of parenting attitudes and behaviors on child IQ and developmental status. The results of this study showed that the quality of stimulation the mother provided in the home (parenting behaviors) and the positive parenting attitudes she adhered to were directly related to her son's IO and both his language and fine motor development, as well as the overall developmental quotient. More specifically, the mother who provided age appropriate toys including musical toys and records/cassettes as well as books, who provided her preschooler with a variety of experiences such as trips to the museum and grocery store, and responded to her child's needs with warmth and affection had a child who scored high on the IQ test. The child also did well on fine motor tasks, such as drawing objects and tracing figures and scored high on language tests that measured such areas as use of plurals in speech and comprehension. In addition, the mother who adhered to positive parenting attitudes had a son who scored high on the IQ test. The positive parenting attitudes are; the mother felt her child should be given comfort and understanding when scared or upset, she should openly express affection for her child in terms of hugging, kissing, and holding him, and her time spent with her child should include warm and intimate times together.

The findings in this study support those findings in the literature that a high quality of environmental stimulation and positive parenting attitudes are positively related to child IQ and language development (Bee et al., 1982; Belsky & MacKinnon, 1994; Bradley, 1993; McGowan & Johnson, 1984). When the quality of environmental stimulation is poor and inadequate and the mother does not adhere to positive parenting

attitudes, the preschooler scored lower on IQ tests and experienced lower levels of language and fine motor development.

Other studies have suggested that maternal, parenting behaviors may mediate the relationship between the maternal influences and child development (Bradley, 1993). One small study found that mother's parenting practices mediated the relationship between maternal depression and motorical delays in children (Goodman & Brumley, 1990).

The present study also found evidence of the mediating effects of parenting attitudes and behaviors on child development. The results showed that parenting behaviors and attitudes mediated the effects of both lifetime and current drug involvement and psychopathology on child IQ and language development. More specifically, mother's current levels of drinking, smoking, depression, and adaptive functioning as well as her lifetime drinking, smoking, and antisociality levels and worst depressive episode directly affected her parenting behaviors and attitudes and indirectly affected her son's IQ and language development.

The following is an hypothetical example of this mediated relationship. The mother's life is characterized by a history of drug involvement and psychopathologies. This history would include alcohol problems, both physical and social, smoking dependency characterized by starting to smoke prior to adulthood and many years of continued smoking, and antisocial behaviors exhibited in both childhood and adult years along with a worst-ever depressive episode. It was this history of drug involvement and psychopathology that affected the mother's current levels of drug involvement and psychopathology. Thus, a history of drug involvement and psychopathology led to high

levels of current drug involvement and psychopathology. Specifically, the mother smokes, drinks, and is characterized as depressed and functioning at a low adaptive level which affected her parenting behaviors and attitudes. Consequently, she was more likely to exhibit poorer parenting behaviors such as not providing age appropriate toys, a variety of experiences, and used physical punishment when dealing with her son's misbehavior and/or to be less likely to adhere to positive parenting attitudes. These poor parenting behaviors and negative parenting attitudes resulted in her son's lower IQ scores and delays in language development.

There is also a more direct, mediated path from lifetime drug involvement and psychopathology through parenting behaviors and attitudes to child IQ and language development. However, this mediated relationship is less clear. It appears from additional analyses that 43%(n = 46 of 106, see Table 26) of the mothers with a high level of lifetime drug involvement and psychopathology are able to provide a stimulating home environment for their sons and/or adhere to positive parenting attitudes. Of these 46 mothers, 57% (n = 26 of 106) have high levels of current drug involvement or psychopathology. That is to say that these mothers are above the median for three or four of the following measures, alcohol consumption, smoking, depression, and low levels of adaptive functioning. It is important to remember that these 26 mothers are only 8.5% of the present study's total sample (n = 307). However, they do provide an interesting point of future study to determine how these mothers are able to still provide a stimulating home environment and/or adhere to positive parenting attitudes even though they have a history of and continue to be drug involved and psychopathic.

A plausible explanation is that these mothers are not as hindered by their drug involvement and psychopathology as the other mothers who are not providing a stimulating home environment and/or adhering to positive parenting attitudes. The difference could be the number of protective factors the former mothers have. They could have more social supports including supports who are not drug involved or experiencing psychopathology themselves. The families of these 26 mothers may be experiencing less crises and be characterized as more cohesive and have more adaptive conflict resolution techniques that are less violent and aggressive. These protective factors could provide a starting point for a future study that would add to the results of the present study whose focus was maternal influences that would be characterized as risk factors not protective factors.

Support for this study's findings of mediated effects of mother's current and lifetime drinking and smoking, as well as depression and antisociality on child development is difficult to find since previous research overlooks the influence of these maternal drug-related behaviors and psychopathologies on parenting practices and thus on child development outcomes. In terms of the relevant literature reviewed in chapter two of this dissertation, only two studies, Goodman and Brumley's study of depression (1990) and Wilen's study of risk factors (1997) included a parenting measure. The other studies did not, even though a developmental model of antisocial behavior which included a parenting component was published in 1989 (Patterson et al.; Reid & Patterson).

Studies involving sub-samples from the MSU-UM Longitudinal Study of children at risk for alcoholism and antisociality have focused on the effects of being married to

alcoholic men on the mothers' parenting behaviors and attitudes. These studies have found several trends for wives of court alcoholics in terms of their parenting (Davis et al., 1991; Noll et al., 1992; Reider et al., 1996). It was found that the mothers provided a less stimulating home environment for their preschoolers compared to mothers married to non-alcoholics (Noll et al., 1992). It was also found that women married to court alcoholics were aggressive towards their sons with the severity of aggression being positively related to the mothers' lifetime alcohol problems, antisociality, worst-ever depressive episode and current depression (Reider et al, 1996). Another study found that high levels of maternal antisociality predicted low levels of nurturance in these families (Davis et al., 1991).

Other studies from the MSU-UM Longitudinal Study have found that mother's current and lifetime levels of drug involvement and psychopathologies were related to her husband's own current and lifetime levels. More specifically, wives of antisocial alcoholics drank more alcohol, experienced more alcohol related problems, and were more depressed and antisocial than women married to non-antisocial alcoholics or comparisons (Ellis et al., 1996; Zucker et al., 1996a). This same trend existed for women married to court alcoholics compared to community alcoholics and comparison wives (Ichiyama et al., 1995).

In order to understand how pervasive the drug involvement and psychopathologies are for the wives of alcoholic men, additional prevalence rates are cited. Forty-four percent of the women married to alcoholics can be classified as alcoholics (Ellis et al., 1996). This compares to a prevalence rate of 4.6% for alcohol abuse/dependence diagnosis for women in the U.S. (Zucker & Gomberg, under review). There is also a high prevalence rate of depression for wives of alcoholics as shown by the following numbers, 48% of the women married to antisocial alcoholics were currently depressed compared to only 23% of those women married to non-antisocial alcoholics or non-alcoholics (Mun et al., 1997).

Past research within the longitudinal study has shown that father's alcoholism and antisociality affect mother's drug involvement and psychopathology as well as parenting behaviors and attitudes. Now, the present study has shown that both mother's lifetime and current drug-involvement and psychopathology influence her parenting behaviors and attitudes, and thus, indirectly affect her son's IQ and language development. It was also shown that there is a direct effect for mother's parenting behaviors and attitudes on her son's IQ and language development.

Future Directions

The results of the present study suggest two different directions to explore further. First, the influences of the environment of alcohol needs to be studied in-depth. More specifically, what are the degrees of influence for the environment of alcohol on the mother? Is one part of this environment more influential than another part? Is the influence different depending upon the outcomes (maternal or child) studied and in terms of the time relationship between the environmental variables and the outcomes? It would be helpful to examine the effects of the environment of alcohol longitudinally. The answers to these and similar questions using both cross-sectional and longitudinal methods can increase the understanding of the maternal influences on maladaptive behaviors in children and possibly identify areas in which interventions can be tried.

Secondly, the area of parenting behaviors and attitudes needs to be examined further. Of interest would be to type certain maternal parenting behaviors most often associated with different combinations of maternal characteristics. In particular, are the parenting behaviors of a depressed alcoholic mother different or similar to the parenting behaviors of a depressed smoking mother, or an antisocial alcoholic mother? Are there specific ways in which each mother provides less adequate stimulation in the home environment? Can addressing the issues of the maternal drug involvement and psychopathologies reduce negative parenting behaviors and attitudes? By examining these questions with both cross sectional and longitudinal research a more complete understanding of the relationship between maternal characteristics, parenting behaviors and attitudes, and child outcomes can be gained and the dynamics of the mother-child relationship can be charted with possible areas of intervention identified.

Also, the small group of mothers who manage to provide a stimulating environment or/and adhere to positive parenting attitudes despite having a past history and currently being drug involved and experiencing psychopathologies need to be studied further. Though the small sample would mean low power in terms of analyses and will affect the results of a study, possible trends could be identified that may provide direction for future, intervention strategies to assist the other mothers who are not providing a stimulating environment for their children or adhering to positive parenting attitudes.

Conclusions

The present study of mothers and their preschool sons examined the influences of maternal characteristics including the following behaviors, lifetime, prenatal, and current levels of drinking, prenatal and current levels of smoking as well as a measure of

smoking dependency, antisociality, and parenting behaviors along with the following structures, worst ever and current depression, adaptive functioning, and parenting attitudes on the child outcomes of behavior problems, IQ, and developmental status.

In testing the first four hypotheses, it was found that mothers who smoked during pregnancy or who are characterized as antisocial or adhered to negative parenting attitudes had children who exhibited more externalizing behavior problems. Additional analyses showed that prenatal smoking was a better predictor of children's impulsivity than current smoking. Only depressed mothers had children who exhibited a higher number of both externalizing and internalizing behavior problems. In terms of the child outcomes of IQ and developmental status, mothers who provided stimulating home environments had children with higher IQs as well as more advanced language and fine motor development and a higher overall developmental quotient. Lastly, an adherence to positive parenting attitudes by the mother was also related to higher IQ in her son.

When structural equation modeling (SEM) was utilized to examine the influences on child behavior problems, it was found that an environment of alcohol was the foremost predictor. An environment of alcohol based on mother's family history of alcoholism, as well as her high levels of both past and current drinking was related to higher levels of both lifetime and current maternal drug involvement and psychopathology. Finally, it was the mother's high, current levels of smoking and depression as well as a low level of adaptive functioning that directly predicted more behavior problems in her son.

In terms of child IQ and language development, results of the SEM found that the mother's parenting behaviors and attitudes mediated the relationships between both

lifetime and current, maternal drug involvement and psychopathologies. High maternal levels of current smoking, drinking, and depression and a low level of adaptive functioning was related to the mother providing a less stimulating home environment and adhering to fewer positive parenting attitudes, this negatively affected her son's IQ and language development.

The findings of this study are of considerable interest given the population-based, multivariate nature of the sample, and the fact that the study utilized SEM to explore the influences of multiple maternal characteristics on child development. These findings provide a foundation for future research in the areas of maternal alcohol environment and the mediating effects of parenting behaviors and attitudes on the relationships between maternal drug involvement and psychopathologies and child outcomes. This study adds to the small literature of non-incarcerated antisocial mothers and their children's development. There are also strong implications for the investigation of the transmission of maladaptive behaviors from mothers to sons and for future interventions. Appendix A

APPENDIX A

Missing Data Estimation

The first step in missing data estimation involved using mean data estimation to complete missing items within an individual's instrument. When an instrument included subscales, means were computed for each subscale or score that had missing data. Once all the individual items were estimated for each instrument, a master data file was created.

The second step in handling missing data involved data estimation using regression. Frequencies were run for all variables of interest in this study and a count was made of available data for each subject. Results of the frequencies revealed the number of people missing a variable ranged from 0 to 102, with the T2 WAIS subscores having the highest amount of missing data. A count of how many variables each mother and father had completed revealed a missing number of 0 to 31 variables from a total of 47 possible variables (see Table 25). Based on the results of the frequencies and count, one family that did not complete wave one data collection were deleted from the final sample. The final sample included 307 families.

The sample was then split into three groups based on fathers' diagnosis, antisocial alcoholic (AAL), nonantisocial alcoholic (NAAL), and nonalcoholic. The split was made utilizing a procedure first outlined by Zucker (1987). This has since been revised utilizing a developmental approach which established group membership among adult alcoholics based on a measure of both child and adulthood antisocial behavior (Zucker et al., 1996a). Stepwise Regressions were then run for each of the three groups. This

assured a more accurate reflection of the missing data. Additional data from the MSU-UM Longitudinal Family Study not used in this study were utilized as independent variables in the Stepwise Regressions in order to increase the amount of variance used to predict the missing data and therefore increase the accuracy of data estimation.

Number of Valid Variables	Frequency	Percent	Cumulative Percent
16	1	.3	.3
17	1	.3	.6
26	1	.3	.9
30	2	.6	1.5
33	1	.3	1.8
34	1	.3	2.1
35	1	.3	2.4
36	1	.3	2.7
39	1	.3	3.0
40	4	1.3	4.3
41	3	1.0	5.3
42	8	2.6	7.9
43	10	3.2	11.1
44	29	9.4	20.5
45	37	12.0	32.5
46	64	20.8	53.3
47	143	46.4	99.7
Total	308	100	

 Table 25.
 Frequencies and Percent of Valid Variables in Sample.

In order to check the integrity of the data after estimations, a comparison of correlations without the missing data and with estimated data was done. A difference of .05 or larger between values in correlation matrixes of unestimated data and estimated data was investigated. Only the HOME scores had differences of .05 to .069 for seven of the 48 correlations. Data estimation using regressions were rerun on the HOME and the

newly estimated HOME scores were examined. It was found that only five of the 48 correlations differed in the magnitude of .05 to .067. These correlations involved the following variables; the developmental quotient from the Revised Yale Developmental Schedules (RYDS) (change between correlations of .064), the subscales on the RYDS: fine motor (change between correlations of .066), gross motor (.066), language development (.065), and age of mother at birth of target child (.053).

One possible explanation for why the correlations between the estimated HOME scores and the overall score and subscales of the RYDS differed the most may be related to the trend in the missing HOME scores. The majority of the missing HOMES were for families in which the fathers' were classified as antisocial alcoholics (AALs). This was a result of a change in data collection protocol and the timing of the recruitment of these families. On the basis of earlier analyses in the larger study, we already know that the sons of AAL fathers are different from the other boys in the sample in terms of development measured by the Yale Developmental Schedules. An earlier paper utilizing a subsample from the MSU-UM Longitudinal Study supports this interpretation (Noll et al., 1992). In this earlier study, sons of alcoholics were found to have delays in fine motor development and to have lower overall developmental quotients as measured by the RYDS compared to sons of non-alcoholic fathers (Noll et al., 1992).

Though the differences in correlations between the estimated HOME scores and the RYDS overall score and subscales are worth noting, the second estimated HOME scores were better and were used in the analyses. Therefore the results, especially those involving the overall developmental quotient and the subscales of interest to this study, are viewed with these differences in mind. Appendix B

APPENDIX B

Sample Results of Mothers and Fathers SEMs

Table 26.Bivariate Correlations for the Latent Constructs of Paternal (P) and Maternal
(M) Lifetime Psychopathologies (F2) and Paternal and Maternal Drug
Involvement (F3).

Variables	P Antisociality	M Antisociality	P Worst-ever	M Worst-ever
			Depression	Depression
P Antisociality	1.00			
M Antisociality	.31***	1.00		
P Worst Depression	.31***	.20***	1.00	
M Worst Depression	.14*	.30***	.36***	1.00
P Alcohol Problems	.56***	.31***	.42***	.20***
M Alcohol Problems	.16**	.51***	.17**	.24***
P Smoking				
Dependency	.41***	.31***	.29***	.19***
M Smoking	.30***	.49***	.24***	.21***
Dependency				
* $p \le .05$. ** $p \le .05$	$01. ***p \le .001$	n = 307		-

 Table 27.
 Lagrange Multiplier Test (for adding parameters).

Parameter	Chi-square	Probability	Parameter Change
Behavior Problems Model			
P Alcohol Problems F2	4.27	.04	.61
P Antisociality F3	1.3	.25	-2.33
M Antisociality F3	1.1	.30	-52.07
M Alcohol Problems F2	.82	.36	.74
Child IQ Model			
M Antisociality F3	10.99	.001	.27
M Alcohol Problems F2	2.45	.12	-46.91

Tables 26 and 27 illustrate just one of the difficulties encountered in the behavior problems and child IQ models in which both maternal and paternal variables were analyzed together. The maternal and paternal variables of antisociality and alcohol problems would be "happier" if they were loaded on the same latent construct which combine both lifetime drug involvement and psychopathologies instead of being separated as they were in the exploratory analyses. However, combining these two constructs into one is not feasible since the new construct would consist of eight variables instead of four each. Also as stated in Chapter one of this dissertation, only maternal characteristics are of interest in this study. Therefore, models analyzing both maternal and paternal variables simultaneously or paternal variables separately were not explored further in this study. Appendix C

APPENDIX C

Non-trimmed Solutions for Child Behavior Problems and IQ Models

	Estimates	Z values	Error Estimates	Z values
Variables				
Family Express. Alcoholism	.75	8.0 *107	.67	8.44
Lifetime Alcohol Problems	.90	11.56	.43	3.25
Current Alcohol Consumption	.37	6.11	.93	12.03
Antisociality	.79	9.55	.61	6.75
Worst Ever Depression	.38	5.69	.93	11.99
Smoking Dependency	.59	0.0	.80	10.79
Adaptive Functioning	.67	4.97	.74	9.01
Current Depression	47	-4.40	.88	11.11
Current Smoking	47	-4.65	.88	11.08
HOME	76	0.0	.65	2.37
Positive Affect	22	-2.69	.98	12.03
Negative Affect	.22	2.67	.98	12.04
Child Behavior Problems	.43		.90	10.80
Impulsivity	17	-2.41	.99	12.21
Factors				
Lifetime Drug Involvement	.73 F1	8.01	.68	4.25
and Psychopathology F2				
Current Drug Involvement	.353 F1	2.01	.48	2.4*107
and Psychopathology F3	-1.10 F2	-3.41		
Parent Behavior Attitude F4	75 F3	-4.75	.66	1.45
Child Behavior Problems F5	-1.19 F3	-2.94	.00	.00
	28 F4	76		
Correlations				
Worst Ever Depression	48	-6.64		
Adaptive Functioning				
Worst Ever Depression	.22	3.82		
Current Depression				
Smoking Dependency	.55	7.42		
Current Smoking				
Current Depression	10	-1.79		
Current Smoking				
Positive Affect	24	-3.94		
Negative Affect				
NFI = .91, $NNFI = .95$, $CFI = .9$	$6 X^{2}(62 n =$	307) = 98.07	$\mathbf{p} < 01$	

 Table 28.
 Non-trimmed Solution for Child Behavior Problems Model.

NFI = .91, NNFI = .95, CFI = .96, $X^{2}(62, n = 307) = 98.07, p<.01$

Table 29. Non-trimmed Solution	Estimates	Z values	Error Estimates	Z values
Variables				
Paternal IQ	57	-8.47	.82	9.35
Maternal IQ	63	-9.13	.78	7.88
Antisociality	82	-5.76	.58	5.59
Worst Ever Depression	39	0.0	.93	11.96
Smoking Dependency	63	-5.60	.78	10.08
Lifetime Alcohol Problems	59	-5.45	.81	10.55
Adaptive Functioning	61	-7.12	.79	10.59
Current Depression	.20	2.86	.98	12.23
Current Smoking	.49	0.0	.87	11.45
Current Alcohol Consumption	.33	4.65	.94	12.01
HOME	64	-4.03	.77	8.70
Positive Affect	27	-3.07	.96	12.15
Negative Affect	.12	1.68	.99	12.32
Child IQ	1.00		.00	.00
Language Development	.54	11.53	.84	12.37
Factors				
Lifetime Drug Involvement and	50 F1	-4.30	.86	2.92
Psychopathology F2				
Current Drug Involvement and	.46 F1	4.28	.24	1.23
Psychopathology F3	66 F2	-4.45		
Parent Behavior Attitude F4	.75 F2	3.22	.36	1.17
	1.53 F3	0.0		
Child IQ F5	.69 F3	1.91	.70	3.63
	-1.22 F4	-3.97		
Correlations				
Current Depression	.25	3.11		
Antisociality				
Worst Ever Depression	48	-7.01		
Adaptive Functioning				
Worst Ever Depression	.28	4.79		
Current Depression				
Worst Ever Depression	16	-3.20		
Positive Affect				
Worst Ever Depression	.14	2.75		
Language Development				
Smoking Dependency	.52	7.01		
Current Smoking				
Lifetime Alcohol Problems	.22	3.38		
Current Drinking				
Lifetime Alcohol Problems	.11	1.96		
Positive Affect				

Table 29.Non-trimmed Solution for Child IQ Model.

Table 29. (cont'd)

	Estimates	Z values	Error Estimates	Z values
Correlations				
Adaptive Functioning	28	-4.36		
Current Depression				
Current Depression	21	-3.30		
HOME				
Positive Affect	28	-4.80		
Negative Affect				
Positive Affect	18	-3.15		
Language Development				

NFI = .90, NNFI = .93, CFI = .95, $X^{2}(67, n = 307) = 120.03, p<.001$

Appendix D

APPENDIX D

Post-hoc Analysis for Child Behavior Problems Model

Table 30.Child Behavior Problems Model: Solution for Testing the Veracity of
Relationship between Alcohol Pervasiveness and Current Drug Involvement
and Psycopathology.

	Estimates	Z values	Error Estimates	Z values
Variables				
Family Express.	.75	2.72	.67	8.43
Alcoholism				
Lifetime Alcohol	.90	2.66	.43	3.24
Problems				
Current Alcohol	.37	2.53	.93	12.03
Consumption				
Antisociality	.79	6.05	.62	6.90
Worst Ever Depression	.38	0.0 ^a	.92	11.96
Smoking Dependency	.59	5.71	.81	10.82
Adaptive functioning	68	0.0 ^a	.74	8.76
Current Depression	.46	6.44	.89	11.24
Current Smoking	.45	6.51	.89	11.33
HOME	74	-7.58	.67	2.75
Positive Affect	23	-2.71	.97	12.01
Negative Affect	.23	2.74	.97	12.00
Child Behavior Problems	.41		.91	11.52
Impulsivity	17	-2.42	.99	12.25
Factors				
Lifetime Drug	.73 F1	2.74	.68	2.90
Involvement and				
Psychopathology F2				
Current Drug	36 F1		.45	2.17
Involvement and	1.12 F2	5.59		
Psychopathology F3				
Parent Behavior Attitude	.76 F3	0.0^{a}	.66	1.35
F4				
Child Behavior Problems	1.00	5.97		
F5				
Correlations				
Worst Ever Depression	47	-6.49		
Adaptive functioning				

^{*} Variance of the parameter is set to zero indicating that the statistical variability of the parameter estimate can not be accurately computed. Because the solution converged, the parameter estimates are considered correct.

Table 30. (cont'd)

	Estimates	Z values	Error Estimates	Z values
Correlations				
Worst Ever Depression	.20	3.58		
Current Depression				
Smoking Dependency	.55	7.61		
Current Smoking				
Positive Affect	24	-3.90		
Negative Affect				

NFI = .91, NNFI = .95, CFI = .96, $X^{2}(65, n = 307) = 101.88, p<.001$

Bibliography

Bibliography

Abel, E. L. (1980). Smoking during pregnancy: A review of effects of growth and development of offspring. Human Biology, 52(4), 593-625.

Abel, E. L. & Sokol, R. J. (1991). A revised conservative estimate of the incidence of FAS and its economic impact. <u>Alcoholism: Clinical and Experimental</u> Research, 15(3), 514-524.

Achenbach, T. & Edelbrock, C. (1983). <u>Manual for the Child Behavior Checklist</u> and Revised Child Behavior Profile. Burlington, Vermont: University of Vermont.

American Psychiatric Association (1987). <u>Diagnostic and statistical manual of</u> mental disorders, 3rd Ed., revised. Washington, DC: American Psychiatric Association.

Anderson, C. A., Hinshaw, S. P., & Simmel, C. (1994). Mother-child interactions in ADHD and comparison boys: Relationships with overt and covert externalizing behavior. Journal of Abnormal Child Psychology, 22(2), 247-265.

Auerbach, J., Lerner, Y., Barasch, M., & Palti, H. (1992). Maternal and environmental characteristics as predictors of child behavior problems and cognitive competence. American Journal of Orthopsychiatry, <u>62(3)</u>, 409-420.

Autti-Ramo, I., Korkman, M., Hilakivi-Clarke, L., Lehtonen, M., Halmesmaki, E., & Granstrom, M. L. (1992). Mental development of 2-year-old children exposed to alcohol in utero. The Journal of Pediatrics, 120(5), 740-746.

Barr, H. M., Streissguth, A. P., Darby, B. L., & Sampson, P. D. (1990). Prenatal exposure to alcohol, caffeine, tobacco, and aspirin: Effects on fine and gross motor performance in 4-year-old children. <u>Developmental Psychology</u>, 26(3), 339-348.

Bauman, K. E., Flewelling, R. L., & LaPrelle, J. (1991). Parental cigarette smoking and cognitive performance of children. Health Psychology, 10(4), 282-288.

Baumrind, D. (1968). Authoritarian v. authoritative parental control. Adolescence, 3, 255-272.

Beattie, J. O. (1986). Transplacental alcohol intoxication. <u>Alcohol and</u> Alcoholism, 21(2), 163-166.

Beattie, J. O. (1992). Alcohol exposure and the fetus. <u>European Journal of</u> <u>Clinical Nutrition</u>, <u>46</u> (Suppl. 1), 7-17.

Beck, A. (1961). An inventory for measuring depression. <u>Archives of General</u> Psychiatry, 4, 561-571.

Beck, A., Steer, R., & Garbien, M. (1988). Psychometric properties of the Beck Depression Inventory. Clinical Psychology Review, 8, 77-100.

Bee, H. L., Barnard, K. E., Eyres, S. J., Gray, C. A., Hammond, M. A., Spietz, A. L., Snyder, C., & Clark, B. (1982). Prediction of IQ and language skills from perinatal status, child performance, family characteristics, and mother-infant interaction. <u>Child</u> Development, 53, 1134-1156.

Belsky, J. & MacKinnon, C. (1994). Transition to school: Developmental trajectories and school experiences. <u>Early Education and Development</u>, 5(2), 106-119.

Behnke, M. & Eyler, F. D. (1993). The consequences of prenatal substance use for the developing fetus, newborn, and young child. <u>The International Journal of the</u> Addictions, 28(13), 1341-1391.

Benasich, A. A., Curtiss, S., & Tallal, P. (1993). Language, learning, and behavioral disturbances in childhood: A longitudinal perspective. Journal of the American Academy of Child and Adolescent Psychiatry, 32(3), 585-594.

Bingham, C. R., Fitzgerald, H. E., Townsend, M. Z., & Zucker, R. A. (June, 1995). Childhood temperament and problem behavior: The early etiology of alcoholism. Poster presented at the annual meeting of the Research Society on Alcoholism Scientific Conference, Steamboat Springs, CO.

Bingham, C. R., Fitzgerald, W. W., Fitzgerald, H. E., & Zucker, R. A. (1996a, June). Prenatal history, parental alcoholism, and the development of preschool-aged sons of alcoholics. Poster presented at the Joint Scientific Meeting of the Research Society of Alcoholism and the International Society for Biomedical research on Alcoholism, Washington, DC.

Bingham, C. R., Fitzgerald, H. E., & Zucker, R. A. (1996b, June). Behavior problem development among children at risk for alcoholism. Poster presented at the Head Start's Third National Conference, Washington, DC.

Bingham, C. R., Loukas, A., Fitzgerald, H. E., & Zucker, R. A., (Under review-a). Parent agreement about child behavior problems: The cross-contextual assessment of child behavioral dysfunction. Psychological Assessment.

Bingham, C. R., Smith, A. M., Fitzgerald, H. E., & Zucker, R. A. (1997, July). Family history of alcoholism and parental antisociality comorbidity as predictors of parental psychopathology and child temperament and behavior problems. Poster presented at the Annual Meeting of the Research Society on Alcoholism, San Francisco, CA. Bingham, C. R., Zucker, R. A., & Fitzgerald, H. E. (under review-b). Risk load and problems behavior development across the preschool and early school years among sons of alcoholics: Variation across subtypes of alcoholic families.

Block, J. H. (1965). <u>The Child-rearing Practices Report (CRPR)</u>: A set of Q items for the description of parental socialization attitudes and values. Unpublished manual, University of California, Institute of Human Development, Berkley.

Block, J., Block, J. H., & Keyes, S. (1988). Longitudinally foretelling drug usage in adolescence: Early childhood personality and environmental precursors. <u>Child</u> Development, 59(2), 336-355.

Borges, G., Lopez-Cervantes, M., Medina-Mora, M. A., Tapia-Conyer, R., & Garrido, F. (1993). Alcohol consumption, low birth weight, and preterm delivery in the National Addiction Survey (Mexico). <u>The International Journal of Addiction</u>, <u>28(4)</u>, 355-368.

Bornstein, M. H. (1989). Sensitive periods in development: Structural characteristics and causal interpretations. Psychological Bulletin, 105(2), 179-197.

Bouchard, T. J. & McGue, M. (1981). Familial studies of intelligence: A review. Science, 212, 1055-1059.

Boyd, T. A., Ernhart, C. B., Greene, T. H., Sokol, R. J., & Martier, S. (1991). Prenatal alcohol exposure and sustained attention in the preschool years. Neurotoxicology and Teratology, 13, 49-55.

Bradley, R. (1993). Children's home environments, health, behavior, and intervention efforts: A review using the HOME Inventory as a marker measure. <u>Genetic</u>, Social, & General Psychology Monographs, <u>119</u>, 437-490.

Bradley, R., Caldwell, B. M., & Rock, S. (1988). Home environment and school performance: A 10-year follow-up and examination of three models of environmental action. Child Development, <u>59</u>, 852-967.

Bradley, R. H., Caldwell, B. M., Rock, S. L., Barnard, K. E., Gray, C., Hammond, M. A., Mitchell, S., Siegek, L., Ramey, C. T., Bottfried, A. W., & Johnson, D. L. (1989). Home environment and cognitive development in the first three years of life: A collaborate study involving six sites and three ethnic groups in North America. Developmental Psychology, 28, 217-235.

Breslau, N. Davis, G. C., & Prabucki, K. (1988). Depressed mothers as informants in family history research--Are they accurate? <u>Psychiatry Research</u>, 24, 345-359.

Breslau, N., Kilbey, M., & Andreski, P. (1993). Nicotine dependence and major depression: New evidence from a prospective investigation. <u>Archives of General</u> Psychiatry, <u>50</u>, 31-35.

Breznitz, Z. & Friedman, S. L. (1988). Toddlers' concentration: Does maternal depression make a difference? Journal of Child Psychology and Psychiatry, 29(3), 267-279.

Bronfenbrenner, U. (1986). Ecology of the family as a context for human development: Research perspectives. Developmental Psychology, 22, 723-742.

Bronfenbrenner, U. (1989). Ecological Systems Theory. <u>Annals of Child</u> Development, 6, 187-249.

Brooks-Gunn, J., McCarton, C., & Hawley T. (1994). Effects of in utero drug exposure on children's development: Review and recommendations. <u>Archives of</u> Pediatric Adolescent Medicine, 148, 33-39.

Brown, R. T., Coles, C. D., Smith, I. E., Platzman, K. A., Silverstein, J., Erickson, S., & Falek, A. (1991). Effects of prenatal alcohol exposure at school age. II. Attention and behavior. Neurotoxicology and Teratology, 13, 369-376.

Brown, G. & Harris, T. (1978). <u>The Social Origins of Depression</u>. London: Travistock.

Burd, L. & Martsolf, J. T. (1989). Fetal Alcohol Syndrome: Diagnosis and syndromal variability. Physiology and Behavior, 46, 39-43.

Butler, N. R. & Goldstein, H. (1973). Smoking in pregnancy and subsequent child development. British Medical Journal, 4, 573-575.

Cahalan, D., Cisin, I., & Crossley, H. (1969). <u>American Drinking Practices:</u> <u>National Study of Drinking Behavior and Attitudes</u>. New Brunswick, NJ: Publications Division, Rutgers Center of Alcohol Studies.

Caldwell, B. M. & Bradley, R. (1984). <u>Home Observation for Measurement of</u> the Environment. Little Rock, AR: University of Arkansas at Little Rock.

Caplan, H. M. (1996). Subtypes of Alcoholic Women. Unpublished doctoral dissertation, Michigan State University, East Lansing, MI.

Carpenter, J. & Lester, D. (1980). <u>The Etiology of Alcoholism: A Longitudinal</u> Study. Mimeo New Brunswick, NJ: Rutgers Center of Alcohol Studies.

Cavallo, F., Ruggenini, A. M., Zotti, C., Gagliardi, L., Tibaldi, C., La Vista, A., Vanini, G., Fusco, A., Gullotti, A., Oliver, R., Casuccio, A., Casuccio, N., Del Corno, G.,

Primatesta, P., & Bonazzi, M. C. (1992). Does moderate alcohol consumption reduce fetal growth? Alcologia, <u>4(2)</u>, 113-124.

Cloninger, R. (1987). Neurogenetic adaptive mechanisms in alcoholism. Science, 236, 410-416.

Coghill, S. R., Caplan, H. L., Alexandra, H., Robson, K. M., & Kumar, R. (1986). Impact of maternal postnatal depression on cognitive development of young children. British Medical Journal, 292, 1165-1167.

Coles, C. D., Brown, R. T., Smith, I. E., Platzman, K. A., Erikson, S., & Falek, A. (1991). Effects of prenatal alcohol exposure at school age. I. Physical and cognitive development. Neurotoxicology and Teratology, 13, 357-367.

Condon, J. T. & Hilton, C. A. (1988). A comparison of smoking and drinking behaviours in pregnant women: Who abstains and why. <u>The Medical Journal of</u> Australia, 148, 381-384.

Covey, L., Glassman, A., & Stetner, F. (1990). Depression and depressive symptoms in smoking cessation. Comprehensive Psychiatry, 31, 350-354.

Cox, A. D., Puckering, C., Pound, A., & Mills, M. (1987). The impact of maternal depression in young children. Journal of Child Psychology and Psychiatry, 28(6), 917-928.

Crnic, K. & Lamberty, G. (1994). Reconsidering school readiness: Conceptual and applied perspectives. Early Education and Development, 5(2), 91-105.

Darling, N. & Steinberg, L. (1993). Parenting style as context: An integrative model. Psychological Bulletin, <u>113(3)</u>, 487-496.

Davis, W. H., Zucker, R. A., Noll, R. B., & Fitzgerald, H. E. (1991). Early socialization practices in alcoholic families: The relationship of child-rearing patterns to demographics and parental psychopathology. Unpublished manuscript, Michigan State University, East Lansing, MI.

Dawson, D. A. (1992). The effect of parental alcohol dependence on perceived children's behavior. Journal of Substance Abuse, 4, 329-340.

Day, N. L. (1992). The effects of prenatal exposure to alcohol. <u>Alcohol Health</u> and Research World, 16(3), 238-244.

Day, N. L. & Richardson, G. A. (1991). Prenatal alcohol exposure: A continuum of effects. Seminars in Perinatology, 15(4), 271-279.

Day, N. L. & Richardson, G. A. (1994). Comparative teratogenicity of alcohol and other drugs. Alcohol Health and Research World, 18 (1), 42-48.

Day, N. L., Robles, N., Richardson, G., Geva, D., Taylor, P., Scher, M., Stoffer, D., Cornelius, M., & Goldschmidt, L. (1991). The effects of prenatal alcohol use on the growth of children at three years of age. <u>Alcoholism: Clinical and Experimental Research</u>, 15(1), 67-71.

Downey, G. & Coyne, J. C. (1990). Children of depressed parents: An integrative review. Psychological Bulletin, 108(1), 50-76.

Dunne, F. J., Galatopoulos, C., & Schipperheijn, J. M. (1993). Gender differences in psychiatric morbidity among alcohol misusers. <u>Comprehensive Psychiatry</u>, <u>34(2)</u>, 95-101.

Egeland, B., Kalkoske, M., Gottesman, N., & Erickson, M. F. (1990). Preschool behavior problems: Stability and factors accounting for change. Journal of Child Psychology and Psychiatry, 31 (6), 891-909.

Elder, G. H., Jr., Caspi, A., & Downey, G. (1983). Problem behavior in family relationships: A multigenerational analysis. In A. Sorensen, F. Weinert, & L. Sherrod (Eds.), <u>Human Development: Interdisciplinary Perspective</u> (pp., 93-118). Hillsdale, NJ: Erlbaum.

Ellis, D. A. (1994). Typological differences in patterns of risk among young alcoholic families. Unpublished doctoral dissertation, Michigan State University, East Lansing, MI.

Ellis, D. A., Bingham, C. R., Zucker, R. A., & Fitzgerald, H. E. (under review). Typological differences in patterns of risk load and child adaptation among young alcoholic families.

Feighner, J., Robins, E., Guze, S., Woodruff, R., Winokur, G., & Munoz, R. (1972). Diagnostic criteria for use in psychiatric research. <u>Archives of General</u> <u>Psychiatry</u>, <u>26(1)</u>, 57-63.

Field, T. (1992). Infants of depressed mothers. <u>Development and</u> Psychopathology, 4(1), 49-66.

Fitzgerald, H. E., Davis, W. H., Zucker, R. A., & Klinger, M. (1994a). Developmental systems theory and substance abuse: A conceptual and methodological framework for analyzing patterns of variation in families. In L. L'Abate (Ed.), <u>Handbook</u> of Developmental Family Psychology and Psychopathology (pp. 350-372). New York: Wiley. Fitzgerald, H. E., Jones, A., Maguin, E., Zucker, R. A., & Noll, R. B. (1991). Assessing parental antisocial behavior in alcoholic and nonalcoholic families. Unpublished manuscript, Michigan State University, East Lansing, MI.

Fitzgerald, H. E., Sullivan, L. A., Grover, M. R., Maguin, E., Zucker, R. A., and Noll, R. B. (1990, June). Parents differ in their temperament ratings of children at risk for alcoholism. Poster presented at the annual meeting of the American Psychological Society, Dallas, TX.

Fitzgerald, H. E., Sullivan, L. A., Ham, H. P., Zucker, R. A., Bruckel, S., Schneider, A. M., and Noll, R. B. (1993). Predictors of behavior problems in three-yearold sons of alcoholics: Early evidence for the onset of risk. <u>Child Development</u>, <u>64</u>, 110-123.

Fitzgerald, H. E., Zucker, R. A., Maguin, E. T., & Reider, E. E. (1994b). Time spent with child and parental agreement about preschool children's behavior. <u>Perceptual</u> and Motor Skills, 79, 336-338.

Fitzgerald, H. E., Zucker, R. A., & Yang, H. Y. (1995). Developmental systems theory and alcoholism: Analyzing patterns of variation in high-risk families. <u>Psychology</u> of Addictive Behaviors, 9(1), 8-22.

Flores-Huerta, S., Hernandez-Montes, H., Argote, R. M., & Villalpando, S. (1992). Effects of ethanol consumption during pregnancy and lactation on the outcome and postnatal growth of the offspring. <u>Annals of Nutrition and Metabolism</u>, 36, 121-128.

Fogelman, K. (1980). Smoking in pregnancy and subsequent development of the child. Child: Care, Health and Development, 6, 233-249.

Ford, D. H. & Lerner, R. M. (1992). <u>Developmental Systems Theory</u>. Newbury Park, California: Sage.

Forehand, R., McCombs, A., & Brody, G. H. (1987). The relationship between parental depressive mood states and child functioning. <u>Advance Behavioral Research</u> and Therapy, <u>9</u>, 1-20.

Forrest, F., Florey, C. du V., Taylor, D., McPherson, F., & Young, J. A. (1991). Reported social alcohol consumption during pregnancy and infants' development at 18 months. British Medical Journal, 303, 22-26.

Fried, P. A., Innes, K. S., & Barnes, M. V. (1984). Soft drug use prior to and during pregnancy: A comparison of samples over a four-year period. <u>Drug and Alcohol</u> Dependence, 13, 161-176.

Fried, P. A., O'Connell, C. M., & Watkinson, B. (1992a). 60- and 72-month follow-up of children prenatally exposed to marijuana, cigarettes, and alcohol: Cognitive and language assessment. Developmental and Behavioral Pediatrics, 13(6), 383-391.

Fried, P. A. & Watkinson, B. (1990). 36- and 48-month neurobehavioral followup of children prenatally exposed to marijuana, cigarettes, and alcohol. <u>Developmental</u> and Behavioral Pediatrics, 11(2), 49-58.

Fried, P. A., Watkinson, B., & Gray, R. (1992b). A follow-up study of attentional behavior in 6-year-old children exposed prenatally to marihuana, cigarettes, and alcohol. Neurotoxicology and Teratology, 14, 299-311.

Funder, D. C., Block, J. H., & Block, J. (1983). Delay of gratification: Some longitudinal personality correlates. Journal of Personality & Social Psychology, 44(6), 1198-1213.

Gibson, K. R. (1991). Myelination and behavioral development. In K. R. Gibson & A. C. Petersen (Eds.), <u>Brain Maturation and Cognitive Development:</u> <u>Comparative and Cross-Cultural Perspectives</u>. (pp. 25-61). New York: Aldine De Gruyter.

Godel, J. C., Pabst, H. F., Hodges, P. E., Johnson, K. E., Froese, G. J., & Joffres, M. R. (1992). Smoking and caffeine and alcohol intake during pregnancy in a northern population: Effect on fetal growth. <u>Canadian Medical Association Journal</u>, <u>147(2)</u>, 181-188.

Goldschmidt, L., Richardson, G. A., Stoffer, D. S., Geva, D., & Day, N. L. (1996). Prenatal alcohol exposure and academic achievement at age six: A nonlinear fit. Alcoholism: Clinical and Experimental Research, 20(4), 763-770.

Gomberg, E. S. L. (1993). Women and alcohol: Use and abuse. <u>The Journal of</u> Nervous and Mental Disease, 181(4), 211-219.

Goodman, S. H. & Brumley, H. E. (1990). Schizophrenic and depressed mothers: Relational deficits in parenting. Developmental Psychology, 26(1), 31-39.

Gottlieb, G. (1991). Experiential canalization of behavioral development: Theory. <u>Developmental Psychology</u>, <u>27(1)</u>, 4-13.

Gonzalez, F., Zucker, R. A., & Fitzgerald, H. E. (1993a, June). Severity of drug involvement among alcoholic women: Relationships to other psychopathology and to adaptation. Poster presented at the annual meetings of the American Psychological Association, Toronto, Canada.

Gonzalez, F., Zucker, R. A., & Fitzgerald, H. E. (1993b, August). Differences between alcoholic men and women as a function of severity of other drug involvement. Poster presented at the Research Society on Alcoholism Meetings, San Antonio, TX.

Greene, T., Ernhart, C. B., Ager, J., Sokol, R., Martier, S., & Boyd, T. (1991a). Prenatal alcohol exposure and cognitive development in the preschool years. Neurotoxicology and Teratology, 13, 57-68.

Greene, T., Ernhart, C. B., Martier, S., Sokol, R., & Ager, J. (1990). Prenatal alcohol exposure and language development. <u>Alcoholism: Clinical and Experimental</u> Research, 14(6), 937-945.

Greene, T., Ernhart, C. B., Sokol, R. J., Marler, M. R., Boyd, T. A., & Ager, J. (1991b). Prenatal alcohol exposure and preschool physical growth: A longitudinal analysis. Alcoholism: Clinical and Experimental Research, 15(6), 905-913.

Gusella J. L. & Fried, P. A. (1984). Effects of maternal social drinking and smoking on offspring at 13 months. <u>Neurobehavioral Toxicology and Teratology</u>, 6, 13-17.

Hale, M., McKay, M. F., & Neale, M. D. (1986). Extending pre-school experience as preventive intervention: Relationships with later reading achievement. Exceptional Child, 33(2), 116-124.

Ham, H. P., Fitzgerald, H. E., & Piejak, L. A. (1994, June). Attention-deficit hyperactivity disorder and conduct disorder in sons of male alcoholics: Evidence of an alcohol related biobehavioral dysregulatory mechanism. Poster presented at the annual meeting of the Research Society on Alcoholism Scientific Conference, Maui, Hawaii.

Ham, H. P., Fitzgerald, H. E., & Zucker, R. A. (1993a, June). Recent evidence of behavioral dysregulation in sons of male alcoholics. Poster presented at the annual meeting of the Research Society on Alcoholism Scientific Conference, San Antonio, TX.

Ham, H. P., Zucker, R. A., & Fitzgerald, H. E. (1993b, June). Assessing antisocial behavior with the Antisocial Behavior Checklist: Reliability and validity studies. Poster presented at the annual meeting of the American Psychological Society, Chicago, IL.

Hamdan-Allen, G., Stewart, M. A., & Beeghly, J. H. (1989). Subgrouping conduct disorder by psychiatric family history. Journal of Child Psychology and Psychiatry, 30(6), 889-897.

Hamilton, M. (1960). A rating scale for depression. Journal of Neurology, Neurosurgery, and Psychiatry, 23, 56-62.

Hamilton, M. (1967). Development of a rating scale for primary depressive illness. British Journal of Social and Clinical Psychology, 6, 278-296.

Hanet, C. (1997, March). <u>Continine levels found in newborns of smokers</u>. Paper presented at the meeting of the American College of Cardiology, Anaheim, CA.

Hardy, J. B. & Mellits, E. D. (1972). Does maternal smoking during pregnancy have long-term effect on the child? The Lancet, 2, 1332-1336.

Hay, D. F. & Kumar R. (1995). Interpreting the effects of mothers' postnatal depression on children's intelligence: A critique and re-analysis. <u>Child Psychiatry and Human Development</u>, 25(3), 165-181.

Herman, C. S., Kirchner, G. L., Streissguth, A. P., & Little, R. E. (1980). Vigilance paradigm for preschool children used to relate vigilance behavior to IQ and prenatal exposure to alcohol. Perceptual and Motor Skills, 50, 863-867.

Hesselbrock, V. M., Hesselbrock, M. N., & Workman-Daniels, K. L. (1986). Effect of major depression and antisocial personality on alcoholism: Course and motivational patterns. Journal of Studies on Alcohol, 47(3), 207-212.

Hilton, C. A. & Condon, J. T. (1989). Changes in smoking and drinking during pregnancy. <u>Australian and New Zealand Journal of Obstetrics and Gynaecology</u>, 29, 18-21.

Hollenbech, A. (1978). Early infant home environments: Validation of the Home Observation for Measurement of the Environment inventory. <u>Developmental</u> Psychology, 14, 416-418.

Ichiyama, M. A., Zucker, R. A., Nye, C. L., Bingham, C. R., Brouwer, R. D., & Fitzgerald, H. E. (1995, June). Women married to alcoholic men: Characteristics of a community sample. Poster presented at the annual meeting of the Research Society on Alcoholism Scientific Conference, Steamboat Springs, CO.

Jacobson, J. L. & Jacobson, S. W. (1994). Prenatal alcohol exposure and neurobehavioral development: What is the threshold? <u>Alcohol, Health, and Research</u> World, 18(1), 30-35.

Jacobson, J. L., Jacobson, S. W., Sokol, R. J., Martier, S., Ager, J. W., & Shankaran, S. (1993, June). Pre- and postnatal growth retardation in infants exposed prenatally to alcohol. Poster presented at the 1993 Research Society on Alcoholism, San Antonio, Texas.

Janet, P. (1925). Psychological Healing. (Vol. 1). London: Allen and Unwin.

Jansen, R. E., Fitzgerald, H. E., Ham, H. P., & Zucker, R. A. (1995). Pathways into risk: Temperament and behavior problems in three- to five-year-old sons of alcoholics. Alcoholism: Clinical and Experimental Research, 19(2), 501-509.

Jarvis, M. J., Russell, M. A., Feyerabend, C., Eiser, J. R., Morgan, M., Gammage, P., & Gray, E. M. (1985). Passive exposure to tobacco smoke: Saliva continine concentrations in a representative population sample of non-smoking school-children. British Medical Journal of Clinical Research and Education, 291(6500), 927-929.

Johnston, L. D., Bachman, J. G., & O'Malley, P. M. (1978). <u>Drugs and the Class</u> of '78: Behaviors, Attitudes, and Recent National Trends. National Institute on Drug Abuse: Division of Research, U.S. Department of Health, Education, and Welfare.

Jones, K. L., Smith, D. W., Ulleland, C. N., & Streissguth, A. P. (1973). Patterns of malformation in offspring of chronic alcohol mothers. Lancet, 1, 1267-1271.

Jordan, T. E. (1978). Influences on vocabulary attainment: A five-year prospective study. Child Development, <u>49</u>, 1096-1106.

Kaplan, B. J., Beardslee, W. R., & Keller, M. B. (1987). Intellectual competence in children of depressed parents. Journal of Clinical Child Psychology, 16(2), 158-163.

Kendler, K., Neale, M., MacLean, C., Health, A., Eaves, L., & Kessler, R. (1993). Smoking and major depression: A casual analysis. <u>Archives of General</u> Psychiatry, 50, 36-43.

Kessler, R. C., Crum, R. M., Warner, L. A., Nelson, C. B., Schulenberg, J. & Anthony, J. C. (1997). Lifetime co-occurrence of DSM-III-R alcohol abuse and dependence with other psychiatric disorders in the national comorbidity survey. <u>Archives of General Psychiatry</u>, 54, 313-321.

Kopp, C. B. (1987). Developmental risk: Historical reflections. In J. Osofsky, Handbook of Infant Development (2nd ed.). New York: John Wiley & Sons.

Kraepelin, E. (1921). <u>Manic-depressive Insanity and Paranoia</u>. Edinburgh, Scotland: Livingstone.

Kristjansson, E. A., Fried, P. A., & Watkinson, B. (1989). Maternal smoking during pregnancy affects children's vigilance performance. <u>Drug and Alcohol</u> <u>Dependence</u>, <u>24</u>, 11-19.

Lamb, M. (1975). Fathers: Forgotten contributors to child development. <u>Human</u> <u>Development</u>, <u>18</u>, 245-266. Landesman-Dwyer, S., Ragozin, A. S., & Little, R. E. (1981). Behavioral correlates of prenatal alcohol exposure: A four-year follow-up study. <u>Neurobehavioral</u> Toxicology and Teratology, 3, 187-193.

Lerner, R. M. (1991). Changing organism-context relations as the basic process of development: A developmental contextual perspective. <u>Developmental Psychology</u>, 27(1), 27-32.

Lerner, R. M., Palermo, M., Spiro, A., & Nesselroade, J. R. (1982a). Assessing the dimensions of temperament individuality across the life span: The Dimensions of Temperament Survey (DOTS). Child Development, 53, 141-159.

Lerner, R. M., Belsky, J., & Windle, M. (1982b). <u>The Dimensions of</u> <u>Temperament Survey for Infancy (DOTS-Infancy)</u>: Assessment of its psychometric properties. Unpublished manuscripts, Pennsylvania State University, University Park, Pennsylvania.

Liaw, F. & Brooks-Gunn, J. (1994). Cumulative familial risks and lowbirthweight children's cognitive and behavioral development. Journal of Clinical Child Psychology, 23(4), 360-372.

Little, R. E. & Sing, C. F. (1986). Association of father's drinking and infant's birthweight. New England Journal of Medicine, 314, 1644-1645.

Little, R. E., Streissguth, A. P., Guzinski, G. M., Grathwohl, H. L., Blumhagen, J. M., & McIntyre, C. E. (1983). Change in obstetrician advice following a two-year community educational program on alcohol use and pregnancy. <u>American Journal of Obstetrics and Gynecology</u>, <u>146</u>, 23-28.

Lockwood, S. L. (1994). Early speech and language indicators for later learning problems: Recognizing a Language Organization Disorder. Infants and Young Children, 7(2), 43-52.

Loukas, A., Piejak, L. A., Bingham, C. R., Milburn, B. J., & Fitzgerald, H. E. (1995, June). Lifetime parental health and alcohol problems: Influences on child behavior problems. Poster presented at the annual meeting of the Research Society on Alcoholism Scientific Conference, Steamboat Springs, CO.

Lovejoy, M. C. (1991). Maternal depression: Effects on social cognition and behavior in parent-child interactions. Journal of Abnormal Child Psychology, 19(6), 693-707.

Makin, J., Fried, P. A., & Watkinson, B. (1991). A comparison of active and passive smoking during pregnancy: Long-term effects. <u>Neurotoxicology and Teratology</u>, 13, 5-12.

Martin, J. C. (1976). Drugs of abuse during pregnancy: Effects upon offspring structure and function. Signs: Journal of Women in Culture and Society, 2(2): 357-368.

Martin, S. L. & Burchinal, M. R. (1992). Young women's antisocial behavior and the later emotional and behavioral health of their children. <u>American Journal of</u> <u>Public Health, 82(7), 1007-1010</u>.

McBride, B. A. & Mills, G. (1993). A comparison of mother and father involvement with their preschool age children. Early Childhood Research Quarterly, <u>8(4)</u>, 457-477.

McGee, R., Silva, P. A., & Williams, S. (1984). Perinatal, neurological, environmental and developmental characteristics of seven-year-old children with stable behaviour problems. Journal of Child Psychology and Psychiatry, 25(4), 573-586.

McGowan, R. J. & Johnson, D. L. (1984). The mother-child relationship and other antecedents of childhood intelligence: A causal analysis. <u>Child Development</u>, <u>55</u>. 810-820.

Miller, J. G. (1978). Living Systems. New York: McGraw-Hill.

Moses, H. D., Gonzalez, F., Zucker, R. A., & Fitzgerald, H. E. (1993, June). Predictors of behavior problems in children at risk for later alcohol abuse. Poster presented at the Annual Meeting of the Research Society on Alcoholism, San Antonio, TX.

Mueller, C. & Parcel, T. (1981). Measures of socioeconomic status: Alternatives and recommendations. Child Development, 52, 13-80.

Mulder, R. T., Wells, J. E., Joyce, P. R., & Bushnell, J. A. (1994). Antisocial women. Journal of Personality Disorders, <u>8(4)</u>, 279-287.

Mulhall, D., Fitzgerald, M., & Kinsella, A. (1988). A study of the relationship between the home environment and psychiatric symptoms in children and parents. <u>Irish</u> Journal of Psychiatry, 9, 13-16.

Mun, E., Bingham, C. R., Fitzgerald, H. E., & Zucker, R. A. (1997, July). Associations between paternal alcoholism subtype, maternal psychopathology, and child risk. Poster presented at the Annual Meeting of the Research Society on Alcoholism, San Francisco, CA.

Munday, L. A. & Rosenberg, G. L. (1979). Social concerns and the Stanford-Binet. Chicago: Riverside Publishing. National Institute on Alcohol Abuse and Alcoholism. (1990). Alcohol and women. <u>Alcohol Alert.</u> (No. 10 PH 290). Washington DC: U. S. Department of Health and Human Services.

National Institute on Alcohol Abuse and Alcoholism. (1991). Fetal alcohol syndrome. <u>Alcohol Alert</u> (Issue No. 13, pp. 1-4). Rockville, Maryland: National Institutes of Health.

National Institute on Alcohol Abuse and Alcoholism. (1992). Data from the 1990 NHIS show that smoking status is clearly related to drinking status. Epidemiological Report (February/March, p. 3). Washington DC: Cygnus.

Newhouse, P. & Hughes, J. (1991). The role of nicotine and nicotinic mechanisms in neuropsychiatric disease. British Journal of Addiction, 86, 521-525.

Nichols, P. L. & Anderson, V. E. (1973). Intellectual performance, race, and socioeconomic status. Social Biology, 20(4), 367-374.

Noll, R. B., Zucker, R. A., Fitzgerald, H. E., & Curtis, W. J. (1992). Cognitive and motoric functioning of sons of alcoholic fathers and controls: The early childhood years. Developmental Psychology, 28, 665-675.

O'Connor, M. J. (1995, March). Depressive features of children exposed to alcohol prenatally. Poster presented at the 1995 biennial meeting of the Society for Research in Child Development, Indianapolis, IN.

Patterson, G. R., DeBaryshe, B. D., & Ramsey, E. (1989). A developmental perspective on antisocial behavior. <u>American Psychologist</u>, <u>44(2)</u>, 329-335.

Piejak, L. A. & Loukas, A. (1995, March-April). Effects of cross-generational substance abuse and psychopathology on child behavior problems. Poster presented at the biennial meeting of the Society for Research in Child Development, Indianapolis, IN.

Pihl, R. O. and Peterson, J. B. (1991). Attention-deficit hyperactivity disorder, childhood conduct disorder and alcoholism: Is there an association? <u>Alcohol Health and</u> <u>Research World, 15(1), 25-31</u>.

Provence, S. & Naylor, A. (1983). <u>Working with disadvantaged parents and their</u> children: Scientific and practice issues. New Haven, CT: Yale University Press.

Puckering, C. (1989). Annotation: Maternal depression. Journal of Child Psychology and Psychiatry, 30(6), 807-817. Rende, R. & Plomin, R. (1990). Quantitative genetics and developmental psychopathology: Contributions to understanding normal development. <u>Development</u> and Psychopathology, 2, 393-407.

Rende, R. & Plomin, R. (1993). Families at risk for psychopathology: Who becomes affected and why? Development and Psychopathology, 5, 529-540.

Reid, J. B. & Patterson, G. R. (1989). The development of antisocial behaviour patterns in childhood and adolescence. European Journal of Personality, 3, 107-119.

Reider, E. E., Zucker, R. A., Maguin, E. T., & Fitzgerald, H. E. (1996). Alcohol involvement and violence towards children among alcoholic families in the community. Unpublished manuscript, Michigan State University, East Lansing, MI.

Richman, N., Stevenson, J., & Graham, P. J. (1982). <u>Preschool to School: A</u> Behavioral Study. New York: Academic Press.

Richters, J. E. (1992). Depressed mothers as informants about their children: A critical review of the evidence for distortion. <u>Psychological Bulletin</u>, 112(3), 485-499.

Robins, L., Helzer, J., Croughan, J., & Ratcliff, K. (1981). National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics, and validity. Archives of General Psychiatry, 38, 381-389.

Robins, L. N. & Regier, D. A. (1991). <u>Psychiatric disorders in America: The</u> epidemiologic catchment area study. New York: Free Press.

Rush, D. & Callahan, K. (1989). Exposure to passive cigarette smoking and child development: A critical review. <u>Annals of the New York Academy of Sciences</u>, 562, 74-100.

Russell, M. A. (1989). The addiction research unit of the Institute of Psychiatry University of London. II. The work of the unit's smoking section. <u>British Journal of Addiction</u>, 84, 853-863.

Rutter, M. (1966). <u>Children of Sick Parents</u>. Oxford, England: Oxford University Press.

Rutter, M. (1990). Commentary: Some focus and process considerations regarding effects of parental depression on children. <u>Developmental Psychology</u>, 26(1), 60-67.

Sameroff, A. J. (1983). Developmental systems: Contexts and evolution. In P. H. Mussen (Ed.), <u>Handbook of Child Psychology Vol 1: History, Theory, and Methods</u> (pp. 237-294). New York: Wiley.

Sameroff, A. J., Seifer, R., Baldwin, A., & Baldwin, C. (1993). Stability of intelligence from preschool to adolescence: The influence of social and family risk factors. Child Development, 64, 80-97.

Scarborough, H. S. & Dobrich, W. (1990). Development of children with early language delay. Journal of Speech and Hearing Research, 33(1), 70-83.

Schuckit, M. A. (1978). <u>Research Questionnaire</u> (mimeograph). Alcoholism Treatment Program, VA Medical Center. University of California, San Diego.

Schuckit, M. A. (1995). <u>Drug and alcohol Abuse: A Clinical Guide to Diagnosis</u> and Treatment. New York: Pleman Medical Book.

Shavelson, R. J. (1988). <u>Statistical Reasoning for the Behavioral Sciences</u> (2nd ed.). Boston: Allyn and Bacon.

Selzer, M. (1975). A self-administered short Michigan Alcoholism Screening Test (SMAST). Journal of Studies on Alcohol, 36, 117-126.

Silva, P. A. & Fergusson, D. M. (1976). Socio-economic status, maternal characteristics, child experience, and intelligence in pre-school children: A path analytic model. New Zealand Journal of Educational Studies, 11(2), 180-188.

Silva, P. A. (1983). Developmental language delay from three to seven years and its significance for low intelligence and reading difficulties at age seven. <u>Developmental</u> Medicine and Child Neurology, 25(6), 783-793.

Smith, I. E., Coles, C. D., Lancaster, J., Fernhoff, P. M., & Falek, A. (1986). The effect of volume and duration of prenatal ethanol exposure on neonatal physical and behavioral development. <u>Neurobehavioral Toxicology and Teratology</u>, 8, 375-381.

Sokol, R. & Clarren, S. K. (1989). Guidelines for use of terminology describing the impact of prenatal alcohol on the offspring. <u>Alcoholism: Clinical and Experimental</u> <u>Research</u>, 13(4), 597-598.

Stein, J. A. & Newcomb, M. D. (1994). Children's internalizing and externalizing behaviors and maternal health problems. Journal of Pediatric Psychology, 19(5), 571-594.

Stevens, G. & Featherman, D. (1981). A revised socioeconomic index of occupational status. <u>Social Science Research</u>, <u>10</u>, 364-395.

Streissguth, A. P., Barr, H. M., & Martin, D. C. (1983). Maternal alcohol use and neonatal habituation assessed with the Brazelton Scale. <u>Child Development</u>, 54, 1109-1118.

Streissguth, A. P., Barr, H. M., & Sampson, P. D. (1990). Moderate prenatal alcohol effects on child IQ and learning problems at age 7 ½ years. <u>Alcoholism: Clinical</u> and Experimental Research, 14 (3), 662-669.

Streissguth, A. P., Barr, H. M., Sampson, P. D., Darby, B. L., & Martin, D. C. (1989). IQ at age 4 in relation to maternal alcohol use and smoking during pregnancy. Developmental Psychology, <u>25(1)</u>, 3-11.

Streissguth, A. P., Barr, H. M., Sampson, P. D., Parrish-Johnson, J. C., Kirchner, G. L., & Martin, D. C. (1986). Attention, distraction, and reaction time at 7 years and prenatal alcohol exposure. Neurobehavioral Toxicology and Teratology, 8, 717-725.

Streissguth, A. P., Martin, D. C., Barr, H. M., Sandman, B. M., Kirchner, G. L., & Darby, B. L. (1984). Intrauterine alcohol and nicotine exposure: Attention and reaction time in 4-year-old children. <u>Developmental Psychology</u>, 20(4), 533-541.

Suguihara, C. & Bancalari, E. (1991). Substance abuse during pregnancy: Effects on respiratory function in the infant. Seminars in Perinatology, 15(4), 302-309.

Susman, E. J., Trickett, P. K., Iannotti, R. J., Hollenbeck, B. E., & Zahn-Waxler, C. (1985). Child-rearing patterns in depressed, abusive, and normal mothers. <u>American</u> Journal of Orthopsychiatry, <u>55</u>, 237-251.

Terman, L. M. & Merrill, M. A. (1973). <u>Stanford-Binet Intelligence Scale:</u> Manual for the third revision form L-M. Chicago: Riverside Publishing.

Thelen, E. (1992). Development as a dynamic system. <u>Current Directions in</u> Psychological Science, 1(6), 189-193.

Thorley, G. (1984). Review of follow-up and follow-back studies of childhood hyperactivity. Psychological Bulletin, <u>96(1)</u>, 116-132.

Tong, S. & McMichael, A. J. (1992). Maternal smoking and neuropsychological development in childhood: A review of the evidence. <u>Developmental Medicine and</u> Child Neurology, 34, 191-197.

Townsend, M. Z., Bingham, C. R., Loukas, A., & Piejak, L. A. (1995, March). Mothers' and fathers' differential influences on preschoolers' developmental status. Poster presented at the biennial meeting of the Society for Research in Child Development, Indianapolis, IN.

U. S. Department of Education. (1986). <u>Dropping out of school in New York</u> state: The invisible people of color. A report of the Task Force on the New York State dropout problem. Washington, DC: Office of Educational Research and Improvement. U. S. Department of Health and Human Services. (1988). <u>Seventh Special</u> <u>Report to the U.S. Congress on Alcohol and Health</u>. (DHHS Publication. No. ADM 88-0002). Washington, DC: U.S. Government Printing Office.

U. S. Department of Health and Human Services. (1990). <u>Smoking and Health</u> <u>Bulletin</u>. (DHHS Publication. No. ADM 88-0372 - 88-0638). Washington, DC: U.S. Government Printing Office.

Vorhees, C. & Mollnow, E. (1987). Behavioral teratogenesis: Long-term influences on behavior from early exposure to environmental agents. In J. Osofsky (Ed.), <u>Handbook of Infant Development</u> (2nd ed., pp. 931-971). New York: John Wiley & Sons.

Walker, D., Greenwood, C., Hart, B., & Carta, J. (1994). Prediction of school outcomes based on early language production and socioeconomic factors. <u>Child</u> Development, 65, 606-621.

Webster-Stratton, C. & Hammond, M. (1988). Maternal depression and its relationship to life stress, perceptions of child behavior problems, parenting behaviors, and child conduct problems. Journal of Abnormal Child Psychology, 16(1), 299-315.

Wechsler, D. (1955). <u>Manual of Wechsler Adult Intelligence Scale</u>. San Antonio, TX: The Psychological Corporation.

Wechsler, D. (1981). <u>Wechsler Adult Intelligence Scale--revised</u>. New York: Psychological Corporation.

Weitzman, M., Gortmaker, S., & Sobol, A. (1992). Maternal smoking and behavior problems of children. Pediatrics, 90(3), 342-349.

Wilen, S. R. (1997). The role of protective factors in the relationship between risk and child externalizing behavior problems over a continuum of risk levels. Unpublished master's thesis, Michigan State University, East Lansing, MI.

Zahn-Waxler, C., Iannotti, R., Cummings, E., & Denham, S. (1990). Antecedents of problem behaviors in children of depressed mothers. <u>Development and</u> Psychopathology, 2, 271-291.

Zucker, R. A. (1987). The four alcoholisms: A developmental account of the etiologic process. In P. C. Rivers (Ed.), <u>Alcohol and Addiction Behaviors Nebraska</u> <u>Symposium on Motivation, 1986, 34</u> (pp. 27-83). Lincoln, NE: University of Nebraska Press.

Zucker, R. A. (1991). Scaling the developmental momentum of alcoholic process via the Lifetime Alcohol Problem Score (LAPS). <u>Alcohol and Alcoholism</u> (Suppl. 1), 505-510.

Zucker, R. A. (1994). Pathways to alcohol problems and alcoholism: a developmental account of the evidence for multiple alcoholisms and for contextual contributions to risk. In R. A. Zucker, J. Howard, & G. M. Boyd (Eds.). <u>The development of alcohol problems: Exploring the biopsychosocial matrix of risk</u>. (NIAAA Research Monograph No. 26). (pp. 255-289, Ch. 13). Rockville, MD: USHHS.

Zucker, R. A., Davies, W. H., Kincaid, S. B., Fitzgerald, H. E., & Reider, E. E. (1997). Conceptualizing and scaling the developmental structure of behavior disorder: The Lifetime Alcohol Problems Score as an example. <u>Development and</u> Psychopathology, 9, 453-471.

Zucker, R. A., Ellis, D. A., Bingham, C. R., & Fitzgerald, H. E. (1996a). The development of alcoholic subtypes: Risk variation among alcoholic families during early childhood. <u>Alcohol Health & Research World</u>, 20, 46-54.

Zucker, R. A., Ellis, D. E., & Fitzgerald, H. E. (1994). Developmental evidence for at least two alcoholisms: I. Biopsychosocial variation among pathways into symptomatic difficulty. Annals of the New York Academy of Sciences, 708, 134-146.

Zucker, R. A., Ellis, D. E., Fitzgerald, H. E., Bingham, C. R., & Sanford, K. (1996b). Other evidence for at least two alcoholism, II: The case for lifetime antisociality as a basis of differentiation. <u>Development and Psychopathology</u>, 8, 831-848.

Zucker, R. A. & Fitzgerald, H. E. (1994). <u>Risk and Coping in Children of</u> <u>Alcoholics: Years 6 to 10 of the Michigan State University-University of Michigan</u> <u>Longitudinal Study</u>. NIAAA grant AA-07065 proposal, Department of Psychology, Michigan State University, East Lansing, MI.

Zucker, R. A. & Fitzgerald, H. E. (1996). <u>Risk and Coping in Children of</u> <u>Alcoholics: Years 11 to 15 of the Michigan State University-University of Michigan</u> <u>Longitudinal Study</u>. NIAAA grant AA-07065 proposal, University of Michigan Alcohol Research Center, Ann Arbor, MI.

Zucker, R. A. & Gomberg, E. S. L. (under review). Alcoholism and antisocial comorbidity in women: A note on a hot spot and some hypotheses.

Zucker, R. A. & Noll, R. B. (1980). <u>Assessment of antisocial behavior:</u> <u>Development of an instrument</u>. Unpublished Manuscript, Michigan State University, East Lansing, MI.

Zucker, R. A., Noll, R. B., & Fitzgerald, H. E. (1986). <u>Risk and Coping in</u> <u>Children of Alcoholics</u>. NIAAA grant AA-07065, Department of Psychology, Michigan State University, East Lansing, MI. Zuckerman, B. S. & Beardslee, W. R. (1987). Maternal Depression: A concern for pediatricians. <u>Pediatrics</u>, <u>79(1)</u>, 110-117.

Zuckerman, B. S. & Bresnahan, K. (1991). Developmental and behavioral consequences of prenatal drug and alcohol exposure. <u>Development and Behavior: The Very Young Child</u>, 38(6), 1387-1406.

