THE UNIVERSITY OF CALGARY

Childhood Indicators of Developing Anti-Social Personality Disorder: A Meta-Analysis of Published Research

by

April N. Nottell

A DISSERTATION

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE

DEGREE OF DOCTOR OF PHILOSOPHY

DIVISION OF APPLIED PSYCHOLOGY

CALGARY, ALBERTA

MARCH, 2001

ACKNOWLEDGMENTS

I would like to acknowledge Dr. Claudio Violato for his willingness to take on the supervision of this research and for his guidance, particularly with respect to conducting a meta-analysis, a research methodology I initially was unfamiliar with. I found his enthusiasm for conducting research, and for mastering the statistical tools of research, encouraging and inspiring.

I would also like to express my gratitude to the Department of Applied Psychology Graduate Scholarship Committee for granting me research scholarships which have helped pay a portion of my production costs.

I wish to extend my gratitude to my family and friends who have taken an interest in my endeavors and who have supported me throughout all of my academic pursuits. In particular, I would like to thank my husband, Gordon, whose hard work has provided the financial support that has made it possible for me to continue my education. I would also like to thank my children, Amber and Jessica, for their patience and understanding as well as their cooperation. I wish to express my love and appreciation to my parents who have always believed in me, my parents-in-law, whose support of our family has meant so much to all of us, and to my extended family and friends whose continued understanding and interest have meant so much to me.

DEDICATION

I dedicate this dissertation to the one who encouraged, supported, and never stopped believing.

Table of Contents

Page

List of Tables

Abstract

A meta-analysis of published research reporting on the effects of psychobiological influences, childhood experiences or external influences, interpersonal influences, and child antisocial behavior on the development of Antisocial Personality Disorder (APD) was undertaken. Fifty three studies met the inclusion criteria. The studies included in the meta-analysis were published between 1962 and 1999, with the majority of studies being published during the 1990's (62.3%). Studies involved a total of 135,533 subjects (118,451 = male). The majority of subjects were 18 years of age or over (81%). The countries in which studies in this meta-analysis were conducted included the United States of America (66%), England (11.3%), Sweden (5.7), Australia (3.8%) Canada (1.9%), Finland (1.9%), Netherlands (1.9%), and New Zealand (1.9%). The country was not reported in three studies. The majority of studies were prospective (71.7%) and quasi-experimental (84.9%) in design. Most researchers used the Diagnostic and Statistical Manual of Mental Disorders (DSM) assessment criteria (49.1%) or their own criteria (35.8%) to assess APD. Interviews (39.6%) and questionnaires (35.8%) were the most common assessment methods.

Dependent variables in four domains (psychological, experiential/external, interpersonal, and behavioral) were coded and effect sizes (d) were computed for both weighted and unweighted effect sizes. Similarly, all of the variables within each domain were coded and both weighted and unweighted effect sizes were computed for each variable. All effect sizes decreased for weighted effect size analyses. In both the unweighted and weighted analyses, all four domains and the variables within them were

found to be potential risk factors in the development of APD. The domains showing the highest magnitude of effect on APD development were the behavioral domain ($\underline{d}$ = .67) and the psychobiological domain ($\underline{d}$ = .65). Of the twenty two variables with non-zero effects, emotional abuse by a caregiverwas the largest $\underline{d}$ for APD development (1.53). Temperament ($\underline{d}$ = .88) and family disfunction ($\underline{d}$ = .83) were the next largest effects followed by separation and loss ($\underline{d}$ = .78), and poor school achievement ($\underline{d}$ = .75). Several independent variables were found to have a moderating effect on these findings. In particular, year of study, moderated findings in the psychobiological, experiential/external, and interpersonal domains with studies conducted prior to 1990 having a stronger effect than studies conducted during the 1990's. The psychobiological domain was also moderated by methods of assessment with smaller effects being reported in studies using DSM-based assessment methods.

Findings reported in this meta-analysis indicate that there is an important emotional component to the development of APD suggesting that this disorder may be more complex than is indicated by the DSM-IV. Findings are discussed from causal developmental perspective based on attachment theory.

CHAPTER I

INTRODUCTION

The official system generally used for classification of mental disorders or conditions, including personality disorders, is the Diagnostic and Statistical Manual of Mental Disorders (DSM) published by the American Psychiatric Association (1994). The DSM has undergone a number of revisions with the current edition being the DSM IV. The DSM IV describes the antisocial personality as one who demonstrates a pervasive pattern of disregard for, and violation of the rights of others that begins in childhood or early adolescence, and continues into adulthood. By adulthood, the behaviors associated with the antisocial personality include those that violate society's accepted standards and laws. The consequences of these acts can be harmful for the antisocial individual (e.g. loss of employment, incarceration) and harmful to members of society who are victimized by the antisocial individual's behaviors. To be diagnosed with Antisocial Personality Disorder (APD), an individual must be at least age 18 years old and must show evidence of Conduct Disorder (CD) before age 15 years. CD involves a repetitive pattern of behavior in which the basic rights of others, or major age-appropriate societal norms or rules are violated. The DSM IV also indicates the likelihood of APD developing in adults is increased if Attention-Deficit/Hyperactivity Disorder (ADHD) accompanies the early onset of CD (before age 10 years) suggesting that APD develops from specific childhood disorders or pathology, particularly CD, and that development is enhanced by other disorders like ADHD which display significant behavioral components.

The DSM IV includes child abuse and neglect, unstable parenting, and inconsistent parental discipline as risk factors for APD development suggesting that development of APD may be influenced by abnormal parenting or inadequate caregiving. (See Appendix A for complete DSM-IV criteria for APD.)

In addition to the risk factors outlined in the DSM IV, other risk factors have been proposed in the development of APD. These risk factors include, 1) psychobiological variables such as genetics, temperament, or intelligence, 2) experiential/external influences including separation and/or loss, poor school achievement or early dropout, medical incident history (brain damage, head injuries, poor nutrition, toxins), family socio-economic status, and family dysfunction or breakdown, 3) interpersonal factors such as caregiver rejection, harsh parenting, parental delinquency or criminality, delinquent sibling(s), parent disorders/pathology, or social desirability, and 4) behavioral characteristics, specifically, child antisocial behavior not meeting the diagnostic criteria for CD.

A number of theories of APD development have been proposed. Psychobiological theorists, for example, suggest that biological factors play an important role in the development of APD (Nigg & Goldsmith, 1994; Siever & Davis, 1991). Experiential theorists point to the interaction of constitutional factors with external factors or childhood experiences as contributing to the development of APD (Crowell, Waters, Kring, & Riso, 1993; Draganic, Lecic-Tosevski, & Calovska-Hertzog, 1997; Engler, 1995; Norden, Klein, Donaldson, Pepper, & Klein, 1995; Paris, 1998a; Paris, 1998b; Rutter & Maughan, 1997; Wishnie, 1977). Interpersonal theorists hold that interactions

with significant others are most important in APD development (Adler, 1990; Benjamin, 1996a; Kernberg, 1996; Pincus & Wiggins, 1990) and behavioral theorists propose that unpunished or reinforced childhood antisocial behaviors contribute to the development of APD (Conger, 1976; Glueck & Glueck, 1950; Hirshi, 1969; Sutherland, 1939; Thornberry, 1987; Thornberry & Krohn, 1997).

Theoretically based research has also been conducted but no clear understanding of APD development has been found (Stoff, Breiling, & Maser, 1997). Unfortunately, the study of APD has been complicated by researchers and writers using a variety of definitions for antisocial personality and Antisocial Personality Disorder as well as various labels for those diagnosed with antisocial personality. In addition, many researchers equate antisocial personality with criminality, delinquency, or with antisocial behavior. Most criminals, however, do not meet criteria for APD and the majority of children and adolescents who display delinquency or antisocial behavior never develop APD.

Rationale for the Present Dissertation

Most personality development theorists such as Otto Kernberg, Theodore Millon, Roger Davis, Lorna Smith Benjamin, Richard Depue, James Pretzer, and Aaron Beck, suggest that early childhood factors contribute to the development of APD (Clarkin & Lenzenweger, 1996). Research examining these factors has provided mixed results so that it is unclear whether or not certain factors influence the development of APD. Also unclear is the strength of contribution for particular early indicators. To gain a richer understanding of the development of APD, the relative importance of specific early childhood indicators of a developing APD need to be assessed.

A useful approach to determining which early childhood factors are the strongest indicators of later APD development, is to perform a meta-analysis of existing research. Specifically, a meta-analysis of research regarding the impact of behavioral risk factors including CD and ADHD, and other risk factors as described in the DSM-IV including child abuse, neglect, unstable parenting, or inconsistent parental discipline, should be conducted to determine potential indicators of a developing APD. In addition, research of other possible risk factors in APD development should be examined including psychobiological, experiential, interpersonal, and behavioral risk factors. Psychobiological risk factors include such variables as genetics, temperament, intelligence (Nigg & Goldsmith, 1994; Siever & Davis, 1991), and disorders/pathology that may manifest in childhood other than CD or ADHD, such as Oppositional Defiant Disorder or Adjustment Disorder (American Psychiatric Association, 1994). Experiential or external factors such as physical and/or sexual abuse, emotional abuse, separation from or loss of a significant other, poor school achievement or early dropout, medical incident history, socio-economic status, and family dysfunction or breakdown in terms of the stability of the family unit are also possible risk factors for developing APD (Farrington, 1996; Robins, 1966). In addition, interpersonal influences may contribute to APD development. These risk factors include neglect, rejection by a caregiver, unstable or erratic parenting, inconsistent discipline, harsh parenting, parent delinquency or criminality, delinquent sibling(s), parent disorders/pathology, and/or social desirability (Farrington, 1996; Robins, 1966). Finally, child antisocial behavior not meeting diagnostic criteria for CD may be a risk factor for APD development.

Examples of studies examining these risk factors include the work of Robins (1966) who examined information derived from clinical, Juvenile Court, police, and school records and from self-report in interview and examined childhood factors characteristic of the pre-sociopathic personality in terms of both internal and external factors at different ages (less than 8, 8-10, 11-13, and greater than 13 years). Significant risk factors identified by Robins included having few friends (social desirability) ($p < .0009$), being separated from a caregiver ($p < .0001$), low non-verbal IQ ($p < .0009$), low school attainment ($p < .0001$) and dropping out before age 15 ($p < .0007$), poor supervision (neglect) ($p < .0001$), having a parent convicted of a crime ($p < .001$) and antisocial behavior ($p < .0001$). In addition, Robins found 35% of pre-sociopaths were impulsive and 29% were reckless and irresponsible. Although Robins found an association between a Stanford-Binet IQ score below 100 and sociopathy, he found the disorder appeared even among intellectually gifted children. Robins' study included an examination of medical incident history. Pre-sociopaths were found to have a somewhat higher rate of head injuries and physical defects but these results were below significance. Robins suggested head injuries may be a consequence of recklessness and impulsivity characteristic of antisocial children rather than a cause of APD. Generally speaking, Robins found antisocial behavior to be the greatest risk factor for APD development. Specifically, he found over half of adults diagnosed with APD were those who exhibited a variety of antisocial behaviors as children.

Another example of research investigating childhood risk factors for APD is a study by Farrington (1996). Farrington examined similar internal and external risk factors

for APD development as those studied by Robins (1966) but instead of studying clinical and delinquent or criminal populations, Farrington chose to measure APD in a general population sample. Like Robins, Farrington also examined risk factors at different ages (10, 14, 18 and 32 years). Those risk factors found to be significant included low family income ($p < .05$), low social class ($p < .05$), convicted parent ($p < .001$), a behavior problem sibling ($p < .05$), poor supervision ($p < .05$), separation from a caregiver ($p < .001$), low non-verbal IQ ($p < .05$), low scholastic attainment ($p < .001$), being unpopular ($p < .05$), dropping out of school ($p < .001$), high neuroticism ($p < .05$) and antisocial behavior ($p < .001$). Farrington also found continuity in antisocial personality from childhood to adulthood with about half of antisocial males at one age continuing to be antisocial at a later age.

Statement of the Problem

The major purpose of this meta-analysis is to determine which variable, or combination of variables, indicate the greatest risk in childhood for subsequent development of APD. The development of APD is not well-understood. While substantive research has been conducted on the early childhood antecedents of antisocial personality, the results are contradictory, confused, and inconclusive (Paris, 1996). It has been suggested that development of APD in adulthood may be related to psychobiological factors, childhood experiences or external influences, interpersonal relationships during childhood, and/or a child's antisocial behavior (Stoff, Breiling, & Maser, 1997). An integration of published research is required, however, to determine which early childhood risk factors are the strongest indicators of developing APD. The present study examined

research that has been conducted in each of these domains to determine which childhood factors constitute a risk for developing APD and which risk factors are the strongest indicators of APD development.

Chapter II of this dissertation contains a summary of the relevant theories and supporting evidence for the development of APD. The methodology employed in the present meta-analysis is described in Chapter III and the results are summarized in Chapter IV. The final chapter (V) includes a discussion of the results, the delimitations and limitations of the present study, and the summary and conclusions of this dissertation.

CHAPTER II

LITERATURE REVIEW

The following chapter provides a brief historical overview of Antisocial Personality Disorder and the various systems used in personality disorder classification. A number of theories of personality development and supporting evidence are discussed, and the proposed research is presented. This chapter concludes with a summary and integration of the evidence reviewed. It concludes with several research questions that are addressed by the subsequent meta-analysis.

Introduction

Individual differences research focuses on personality, a person's more or less persistent set of tendencies to behave in a given manner in relation to other persons, across situations and over time (Mischel, & Shoda, 1995). Personality may be construed as a more or less integrated system of attitudes toward the self, originating in experiences with other persons and, in turn, expressed toward others (Adams-Webber, 1992). While these attitudes are more or less modifiable throughout life, the degree of modifiability is influenced by age and other factors, such as the absence of cultural opportunities and inadequate inherent assets (Kogan, 1990; Sanderson, & Cantor, 1999). Also, attitudes experienced earliest in life are the most firmly integrated into the self and are therefore the least influenced or modified by interpersonal occurrences later in life (Szurek, 1969). Thus, disorders in personality development are more likely to be treatable if they are identified during childhood before attitudes become fixed.

Antisocial Personality Disorder

Research examining how personality disorders (PDs) develop is still in its infancy. Over time, preferences for labels, definitions, and diagnostic criteria for personality disorders, and for APD in particular, have changed as views regarding PD's and their development have changed (Sutker, Bugg, & West, 1993). Differences in views regarding "personality disorder" as a construct relate primarily to differentiation (personality disorder versus normal personality) resulting in disagreement regarding the best approach to classification (Lilienfeld, Purcell, & Jones-Alexander, 1997). In addition, a number of theoretical perspectives have been proposed regarding development of PDs generally, and with respect to APD development in particular (Engler, 1995).

Labels and Definitions

Labels for individuals who exhibit antisocial behavior and definitions of APD have evolved over the years as beliefs about etiology have changed. In 1801, Phillipe Pinel described the disorder as characterized by aberrant affect, proneness to impulsive rage, and no deficit in reasoning. In 1835, Benjamin Rush described antisocial individuals as constitutionally deficient in moral faculties and in the same year, J. C. Prichard popularized the label "moral insanity" and promoted the belief that antisocial behaviors resulted from organic or constitutional factors with poor prognosis for change (Sutker, Bugg, & West, 1993). As a result, the label "psychopathic personality" became popular during the late 1800's and early 1900's, and this term was included in the DSM I.

By the 1930's, researchers had begun to examine the role of environmental and cultural factors in the etiology of behavioral deviance and in 1930, Partridge coined the

term sociopathic personality emphasizing the failure to conform to societal demands. This perspective gradually gained popularity into the 1970's (Sutker, Bugg, & West, 1993) as did the term sociopath.

During the late 1960's and early 1970's, many theorists and clinicians retained their constitutional view of APD. Szurek (1969), for example, continued to view antisocial personalities from a constitutional perspective, describing such individuals as being deficient in moral sense and motivated by the need to gain immediate gratification of impulses that control them. He claimed that the antisocial personality is unable to inhibit or to postpone action, or to acquire satisfactions in culturally acceptable ways.

Guze (1976), on the other hand, viewed the person with antisocial personality as one that engages in antisocial behaviors. Specifically, he said they must demonstrate at least two of the following: i) a history of excessive fighting; ii) school delinquency; iii) a poor job record; iv) a period of wanderlust; or v) being a runaway, along with a history of police trouble (other than traffic offenses).

Wishnie (1977) included both constitutional and behavioral features in his description of APD emphasizing behaviors which bring the antisocial personality repeatedly into conflict with society. Wishnie added, however, that a mere history of repeated legal or social offenses would not be sufficient to justify this diagnosis. He believed the antisocial personality is incapable of significant loyalty to individuals, or groups, or to social values. He described them as grossly selfish, callous, irresponsible, impulsive, and unable to feel guilt or to learn from experience and punishment. He added that their tolerance for frustration is low and they tend to blame others or to offer plausible

rationalizations for their behavior.

The behavioral perspective has become even more important recently although some reference is made to constitutional factors. Hare (1980), for example, defined psychopaths as individuals who combine deviant personality traits and antisocial behaviors that are often criminal in their severity. Similarly, the Antisocial Personality, as described in the DSM-IV (APA, 1994), is characterized by impulsive-aggressive behavior in childhood including a general disrespect for rules at home and at school with this pattern of behavior continuing throughout adolescence and into adulthood when impulsive-aggressive behaviors tend to negatively impact social relationships and the ability to sustain consistent work. The adult antisocial personality is often a substance abuser and experiences frequent altercations with the law due to serious criminal activity.

Despite changes to the definition of APD over the years, all definitions refer to a disposition for antisocial behavior and social deviance resulting from personal deficiencies or psychological abnormalities (Sutker, Bugg, & West, 1993). Currently, the label antisocial personality is most widely accepted and Antisocial Personality Disorder appears in the DSM III, DSM III-R, and DSM IV. Nonetheless, the terms psychopathic personality, psychopath, sociopathic personality, sociopath, dyssocial personality, and antisocial personality are generally viewed as synonymous and often used interchangeably (Sutker, Bugg, & West, 1993).

Diagnostic Criteria

As with labels and definitions of APD, beliefs about etiology also influenced the development of the DSM. The major change in DSM criteria for Antisocial Personality

Disorder occurred when the DSM II was revised to the DSM III. In the DSM II, diagnosis was derived from attributions regarding personality traits and inferences regarding underlying processes while newer criteria include a lengthy checklist of antisocial behaviors that must be present in certain frequencies before and after age 15 indicating both severity and chronicity of the disorder (Sutker, Bugg, & West, 1993).

These changes to the DSM criteria have been criticized by a number of writers including Millon (1981) who believed that the basic aggressive personality is the same in both antisocial and non-antisocial types and, therefore, he claimed that antisocial behaviors should not be used to describe antisocial personality. The prominent diagnostic criterion features identified by Millon as capturing the essence of antisocial personalities include hostile affectivity, social rebelliousness, vindictiveness, and a fearless attitude. Millon stated that such details as persistent lying, early aggressive sexual behavior, or vandalism should only be listed if there was some value in providing illustrative examples. He felt that the new DSM criteria placed too much emphasis on antisocial behaviors and failed to adequately deal with actual personality characteristics. He believed the criteria had become oriented too much toward the criminal personality and not sufficiently toward those with similar propensities who have avoided criminal involvements. In addition, he believed that the change constituted a shift in focus away from that employed in describing all of the other personality disorders and that the traditional format captured the underlying tone of relevant traits or symptoms.

Antisocial Personality Disorder - The Construct

The criteria for APD in the DSM-III and DSM-IV reflect a polythetic approach to

categorization wherein no one criteria is either necessary or sufficient for a diagnosis. The consequence of this approach is that there are numerous different diagnostic combinations that will fulfill the DSM-IV criteria for APD suggesting that an APD diagnosis is extremely heterogeneous. Heterogeneity at the level of diagnostic criteria, however, does not necessarily imply heterogeneity at the level of the latent construct presumably underlying the APD diagnosis (Lilienfeld, Purcell, & Jones-Alexander, 1997).

Personality Disorder Classification - Categorical

Categorical classifications divide mental disorders into types based on criteria sets with defining features. A categorical approach to classification works best when all members of a diagnostic class are homogeneous, when there are clear boundaries between classes, and when the different classes are mutually exclusive (APA, 1994). The DSM is considered a categorical approach to personality pathology.

DSM

Despite its categorical classification, in the DSM-IV there is no assumption that each category of mental disorder is a discrete entity or that individuals sharing a diagnosis will be a homogeneous group (APA, 1994). In fact, the DSM-IV assumes there will be boundary cases that are difficult to diagnose. With respect to personality disorders, the DSM-IV distinguishes between personality (Axis II) disorders, which generally begin in childhood or adolescence and remain stable into adult life, and other (Axis I) mental disorders, which usually have a later onset and are usually less chronic and stable in their course (Widiger & Trull, 1992). Axis II PDs are also distinct in that they are not only a focus of clinical treatment on their own, but they will often have a significant affect on the

occurrence, expression, course, and/or treatment of many other mental disorders (Widiger, 1997). The effects of Axis I mental disorders tend to be less pervasive (Widiger & Trull, 1992).

Lenzenweger and Clarkin (1996) described the DSM-IV Axis II taxonomy as presenting personality disorders grouped into three clusters, sometimes referred to as dimensions, including: i) the odd-eccentric Cluster A (Paranoid, Schizoid, Schizotypal) PDs; the impulsive-erratic Cluster B (Antisocial, Borderline, Histrionic, Narcissistic) PDs; and the anxious-avoidant Cluster C (Avoidant, Dependent, Obsessive-Compulsive) PDs. The DSM-IV PD clusters may also be viewed as dimensions representing a spectra of personality dysfunction on a continuum with Axis I mental disorders (APA, 1994).

The DSM-IV has received a number of criticisms. Westen and Arkowitz-Westen (1998), for example, believed the range of Axis II should be broadened to encompass the range of personality pathology seen in clinical practice. They pointed out that Axis II is limited to severe personality disturbances, posing difficulty for diagnosing less severe but nonetheless clinically significant personality pathology. They suggested three ways comprehensiveness might be increased: i) by including additional categories to reflect less severe personality disturbances; ii) Axis II could be replaced or supplemented with a functional assessment of personality; or iii) the current categorical system could be replaced with a more dimensional system.

Widiger and Shea (1991) referred to the fact that the DSM does not assume that each mental disorder is a discrete entity discontinuous with other mental disorders. Axis II includes the developmental and personality disorders while all other diagnoses fall under

Axis I. They pointed out that placing PDs on a separate axis has stimulated concern regarding comorbidity of personality and Axis I disorders and that issues regarding differentiation are particularly evident for four pairs of Axis II and Axis I disorders: i) Schizotypal versus Schizophrenic; ii) Borderline versus Mood; iii) Antisocial versus Substance Use; and Avoidant versus Social Phobia diagnoses. They recommended addressing differentiation by adding exclusion criteria, shifting the placement of disorders, deleting overlapping criteria, adding differentiating criteria, and converting to a dimensional format.

Widiger (1997) expressed four main limitations of the diagnostic categories. First, he pointed out that prototypic cases of personality disorder should exhibit all of the defining features yet most cases vary in the extent to which they resemble the prototype. In addition, he said that once categorized, there is a tendency to overlook within-group variablity, to discount disconfirming evidence, and to focus on stereotypical examples of the category. Another limitation noted by Widiger, is that some individuals will exhibit traits associated with more than one PD but they have just one personality. Consequently, most clinicians provide only one diagnosis per patient. Widiger suggested it would be simpler and more meaningful to talk of one PD characterized by varying degrees of borderline, antisocial, and paranoid traits than to state that the patient is suffering from three different, comorbid PDs. Widiger also criticized the DSM which, on the one hand, requires a minimum number of criteria (usually four or five) to indicate a PD and must involve personality traits that result in "clinically significant distress or impairment in social, occupational, or other important areas of functioning". He said that the diagnostic

threshold for certain DSM-IV PDs has no relationship with the point at which one is vulnerable. He has found, for example, individuals with just three of the criteria for Dependent PD can experience significant impairments secondary to their dependent personality traits. Finally, Widiger criticized the diagnostic criteria for being inadequate for the purposes of most clinicians. The DSM-IV contains ten possible PD diagnoses, but the most common diagnosis made by clinicians in general practice is "personality disorder not otherwise specified" because most of their clients exhibiting a PD fail to meet the diagnostic criteria for any one of the ten recognized diagnoses. He added that in addition to not covering the full range of maladaptive personality traits, the DSM-IV also fails to acknowledge the presence of adaptive personality traits which are important for a comprehensive and treatment relevant description of patient's personality

Personality Disorder Classification - Dimensional

A dimensional approach to classification assumes that a small set of continuous trait dimensions can parsimoniously represent all of the character types present in a given population (Strack & Lorr, 1997). Dimensional approaches view PDs as maladaptive variants of personality traits that merge imperceptibly into normality and into one another (APA, 1994). Thus, a dimensional format provides a greater degree of precision in the description of individual patients by indicating the degree to which symptomatology is present and by allowing for alternative cutoff points in clinical decision-making (Widiger & Shea, 1991).

The Big Five Model is one example of a dimensional model wherein Axis II PDs are interpreted in terms of five broad domains including: neuroticism versus emotional

stability, extroversion versus introversion, openness to experience versus closedness to experience, antagonism versus agreeableness, and conscientiousness versus negligence (Costa & McCrae, 1990). APD, for example, can be described in terms of tough-mindedness, aggressiveness, deception, and manipulation, characteristics taken from the domain of antagonism, together with fearlessness, self-assurance, recklessness, and impulsivity taken from the domain of neuroticism, and negligence and hedonism taken from the domain of conscientiousness (Widiger, 1997). Widiger and Trull (1992) explained that Axis II PDs are interpreted as extreme variants of the five factors while Axis I disorders are seen as distinct from but interactive with one or more of the five factors.

An association has been found between the Big Five Model and the DSM-III-R. Results of studies by Wiggins and Pincus (1989) and Costa and McCrae (1990) indicate the capacity of the Big Five to account for a substantial proportion of variance in personality disorder symptomatology. A limitation of these studies is that the samples used were normal college students and community volunteers which minimize the effect of Axis I symptomatology on self-report indices. In addition, it may be questionable whether findings from normal subjects generalize to patients with personality disorders (Widiger & Trull, 1992).

Categorical vs. Dimensional Models

O'Connor and Dyce (1998) examined the degrees to which various models of PD configuration were consistent with primary data sets from clinical and community samples. The DSM three-cluster configuration was tested and findings indicate the DSM, as a

model of the correlational structure of PDs, is statistically significant but somewhat variable and less than perfect. In comparison, fit levels for a four-dimensional model (Torgersen & Alnaes, 1989) were similar to those found for the DSM configuration, while the highest and most consistent levels of fit were obtained for the five-factor and seven-factor models. While levels of fit for these models surpassed those found for the DSM dimensions, O'Connor and Dyce believe extraction of five or more factors from existing PD correlation matrices is probably excessive and state that four factors provide comparable degrees of differentiation between PDs and similar levels of fit to the data. All other models tested (circumplex models and Millon's biosocial model) fell below the standard set by the DSM and were rarely statistically significant.

Yeung, Lyons, Waternaux, Faraone, and Tsuang (1993) also found significant correlations between DSM-III PDs and one or more dimensions of the Big Five personality factors but the correlations were generally low, ranging from -.003 between Dependent PD and factor E to -.39 between paranoid PD and factor A. They suggested a possible explanation for the modest correlations between DSM-III PDs and the five-factor model (FFM) could be that the FFM may not accommodate symptomatologies related to both Axis I and Axis II disorders. APD was associated with low agreeableness and low conscientiousness which include such characteristics as being rude, uncooperative, vengeful, ruthless, irritable, manipulative, aimless, and unreliable. While these characteristics describe the nature of individuals diagnosed with APD, the distinctive maladaptive behaviors described by the DSM-III were not depicted. Yeung et al suggested this may be because of all the PDs described in the DSM, APD is described in

the most behavioral and the least psychological terms.

Schroeder, Wormworth, and Livesley (1992) also examined the convergence of dimensional measures of PDs with Costa and McCrae's Big Five factors. Although multiple regression analyses indicated substantial relationships between the Big Five factors, in particular neuroticism, and many PD scales, ranging from .83 for anxiousness, to .58 for identity problems, the Big Five factors were not strongly related to behavioral aspects of the PDs which ranged from -.01 for stimulus seeking, to .51 for passive-oppositionality. In terms of dimensions, the mean across psychological dimensions was .64, while the mean across behavioral dimensions was only .17. They concluded that despite similarity between the measures, personality disorder measures cannot be entirely subsumed under the Big Five model.

Classification - Future Directions

The DSM PDs and the Big Five dimensions differ in their categorical versus dimensional format, their relative focus on psychopathology, and levels of specificity of personality they describe. Yeung, Lyons, Waternaux, Faraone, and Tsuang (1993) felt it best to treat them as two separate systems with their own applications rather than attempt to substitute one for the other. They did not, however, rule out the possibility that the two systems may be complimentary. They suggested it may be worthwhile, for example, to include the Big Five dimensions as an additional axis for a comprehensive multiaxial psychiatric evaluation which would enable research in the future to explore the utility of these personality dimensions in predicting specific clinical symptoms, course, treatment response, and prognosis of psychiatric disorders.

It is unclear to what extent personality disorder symptoms are continuous extensions of normal traits. Historically, the psychiatric community has adopted a typological approach to personality pathology and has chosen a categorical framework that facilitates communication, is congruent with clinical decision making, and is consistent with the medical tradition (APA, 1994). Psychologists, on the other hand, have advocated a dimensional or continuum approach to the study of personality and other behavioral phenomena. This preference may be because in personality research, using non-clinical samples, there has been a reliance on parametric statistics, and a focus on normative aspects of psychological functioning. More recently however, psychiatry has been showing more interest in dimensional approaches to personality pathology and psychological research has been aimed at detecting discontinuities, that is, types or taxa, in studies of both normal and pathological personality (Lenzenweger & Clarkin, 1996).

Currently, the DSM-IV is the most widely accepted diagnostic nosology and therefore, provides the soundest basis for the definition of APD in this study, and a good starting point for the identification of potential childhood indicators of a developing APD. Research to date has not effectively addressed the goodness of fit between the overall DSM-IV personality disorder taxonomy and the empirically based dimensional structures observed in contemporary personality research. Although findings from personality research on non-clinical samples suggests that somewhere between three and five factors capture the variation in primary descriptors of personality, correspondence between primary factors of personality and personality disorders remains to be explored (Lenzenweger & Clarkin, 1996). Theories of the development of personality disorders

may provide direction for this exploration.

Theories of Personality Disorder Development

A number of theories of personality have been proposed, and some theorists (e.g. Cloninger, 1998; Benjamin, 1996a; Beck, 1997) have tried to explain the development of personality disorders. Antisocial Personality Disorder has been particularly problematic. Theories addressing personality disorder development and APD development in particular, fall into three primary categories: psychobiological, experiential, and interactional.

Psychobiological Theories

Psychobiological theorists consider a number of interrelated factors which could potentially contribute to PD development including genetics and temperament.

Genetics.

Genetics are most commonly researched through prevalence, family, twin, and adoption studies of the relationship between PDs and heritability of traits such as extroversion, neuroticism, psychoticism, conscientiousness, openness, dominance, achievement, impulsiveness, aggressiveness, adjustment, intelligence, and masculinity/femininity and in relation to social attitudes, love styles, and crime (Bouchard, 1997).

Dahl (1993), for example, reviewed family, twin, and adoption studies of the DSM-III-R PDs but he was only able to find 47 genetic studies covering 10 PDs. For 4 of the 10 PDs no family, twin, or adoption studies were found, and 13 of the studies he reviewed dealt exclusively with Borderline PD. Consequently, the findings of Dahl's review are not generalizable. He also found it difficult to draw conclusions regarding

heritability because the study findings were mixed. He also noted methodological difficulties in virtually every study, including an absence of direct interviews with relatives in family studies, lowered cutoff levels for positive criteria, and inadequate diagnostic methods, which made the validity of the study findings questionable. Although Dahl recognized that research can be directed only to a limited extent, he recommended that more research funding be directed toward family and genetic studies so that adequate methodologies can be employed such as using structured interviews with full criteria sets. including information from informants, obtaining adequate samples, and making blind ratings.

For APD, Nigg and Goldsmith (1994) examined genetic research of Axis II disorders. They found that overall, APD was the most well studied of the disorders. Their review revealed that, in the general population, the lifetime prevalence of APD is only 7.3% for males and 1.0% for females but in the criminal population, 40% of males and 18% of females are diagnosed with APD. Conversely, 55% of APD males and 17% of APD females had a criminal record. They also found that first-degree relatives of APD males were at five times greater risk while first-degree relatives of APD females were at ten times greater risk for developing the disorder than the general population. Nigg and Goldsmith pooled results from twin studies of adult criminality and found a concordance rate of 51.5% for identical pairs and 23.1% for fraternal pairs while results from adoption studies indicated a more modest genetic influence with heritability estimates ranging below 30%. Based on results from nonfamilial adoption studies, Nigg and Goldsmith suggested that criminality is often a consequence of alcohol abuse. Given that antisocial behavior

and alcohol abuse both have genetic roots, Nigg and Goldsmith suggested they may be genetically heterogeneous. In studies controlling for alcohol use, however, different genetic and environmental antecedents characterize APD. Specifically, adoptee criminality was found to be associated with criminality in the biological father and with petty crimes against property, but when alcohol was a factor, adoptee criminality was associated with more violent and repetitive crimes. In adoption studies, APD in adult adoptees was associated with APD or antisocial behavior in biological parents, but where biological parents were diagnosed with PDs other than APD, no adoptees were diagnosed with APD (Nigg & Goldsmith, 1994).

The research reviewed by Nigg and Goldsmith (1994) indicated an underlying genetic etiology for APD, but they admitted that genetics alone cannot explain APD development. They explained that APD is a complex outcome of interacting internal and external influences. They found, for example, greater risk (about 50%) for APD adoptees when biological parents were criminal or delinquent and when socioeconomic status of the adoptive home was low. Also found was a possible familial link between APD and Histrionic, Borderline, and Narcissistic PDs with each disorder showing gender variation in prevalence.

Nigg and Goldsmith also suggested it may be possible that certain PDs could be placed on a genetic spectrum with respective Axis I disorders. Schizotypal and Paranoid PDs, for example, may be part of the same genetic spectrum as Schizophrenia. In samples not clinically diagnosed but quantified as antisocial personalities, Nigg and Goldsmith found a high degree of phenotypic and genetic overlap between APD and childhood

antisocial behavior and drug abuse. Substantial genetic but minimal phenotypic overlap of adult APD and alcohol use was also found suggesting that genetic effects for antisocial phenotypes are defined more broadly than criminal behavior.

Nigg and Goldsmith (1994) noted certain limitations of their review including a potential for variability in heritability estimates for the Schizophrenia spectrum depending on how "spectrum" was defined, and possible bias due to subject selection based on practicality rather than random sampling. The distribution of genes and environments and corresponding differences in heritability, gene-environment correlations, and gene-environment interactions may also contribute to variability in samples.

Temperament.

Siever and Davis (1991) proposed a temperament model for PD development that is based on four dimensions: (a) cognitive/perceptual organization, (b) affective regulation, (c) impulse control, and (d) anxiety modulation. In this model, PDs reflect disturbances in particular dimensions. Such disturbances contribute to development of specific defense mechanisms and adaptational strategies that may eventually become pervasive characteristic ways of behaving across occupational and interpersonal situations. Thus, disorders range from extreme, discrete symptoms manifesting as Axis I disorders at one end of the continuum, to milder, persistent, and pervasive disturbances, in one or more dimensions, manifesting as Axis II disorders at the other.

Siever and Davis' (1991) model also related DSM-IV clusters to specified dimensions. Cluster A related to the Cognitive-Perceptual dimension which represents disturbances in cognitive/perceptual organization. These disturbances manifest in thought

disorder, psychotic symptoms, and social isolation. Such distortions can impair social interactions through misunderstanding or suspiciousness of others' motivations. Consequently, a major coping strategy is often social detachment which can amplify cognitive/perceptual distortions by preventing interactions that provide input for reality testing. Cluster B, which includes APD, relates to the Impulsivity-Aggression dimension, and is viewed as a tendency toward action-oriented and aggressive behavioral strategies. These individuals have difficulty anticipating the effects of their behavior. They tend to not learn from the consequences of their behaviors, have trouble inhibiting or delaying action, and are prone to excessive expression of aggression and frustration. They may also be less likely to experience guilt or anxiety. All PDs of the dramatic cluster manifest impulsive-aggressive behaviors, but each PD emphasizes different features of the dimension such as disinhibited rage in Narcissists, low tolerance for frustration in Histrionics, and suicide attempts and substance abuse in Borderlines. The DSM Cluster C was represented by the Anxiety-Inhibition dimension which is described as a low threshold for subjective fear and autonomic arousal in anticipation of aversive consequences. Discrete episodes of anxiety or related symptoms may reflect Axis I disorders, but a pervasively low threshold for anxiety might contribute to the development of a PD. Finally, the Affect Instability dimension was not represented by a specific PD cluster. This dimension is seen as a predisposition that may manifest in, or along with PDs in one or more Axis II Clusters.

For APD, Siever and Davis (1991) explained that individuals with APD experience a diminished capacity to delay or inhibit action, particularly aggressive action. Poor

impulse control becomes chronic and pervasive manifesting in a lack of suppression of aggressive, and antisocial behaviors. Research has yet to determine whether the Impulsivity-Aggression dimension reflects only disinhibited aggression or includes motor disinhibition in relation to disinhibition in the cognitive domain. Their review indicated that impulsive-aggression correlates with lower cortical inhibitory function, reduced arousal, less inhibition of motor responses, weaker sympathetic responsiveness, and more rapid habituation in skin conductance. Findings regarding differences between impulsive and non-impulsive individuals suggest that rather than responding with an evaluative delay, which involves cortical activation, sympathetic arousal, and inhibition of motor output, APD individuals are more likely to activate motor responses to important environmental stimuli.

The reliability and validity of the findings from Siever and Davis' (1991) review are difficult to determine because details of the individual studies reviewed were not reported nor was there any indication as to the total number of studies reviewed. In addition, one might question the validity of the proposed dimensions.

Cloninger (1998) proposed a temperament model based on completely different dimensions. In this model there are four temperament dimensions: (a) reward dependence, (b) novelty seeking, (c) harm avoidance, and (d) persistence. Cloninger believed that about 50% of the variance in temperament can be accounted for by genetics and he associated the temperament dimensions with PDs. Specifically, Cluster A was associated with low reward dependence, Cluster B with high novelty seeking, and Cluster C with high harm avoidance. Persistence was associated with behavioral activation, and

disturbances in persistence were associated with either specific antipersistence effects or obsessive-compulsive behavior in PDs. Cloninger's (1998) research was based on twin and adoption studies of personality in humans, learning abilities in animals, and on neuropharmacological studies of humans and animals, but the validity of his findings may be subject to bias given that the quantitative tests for measuring personality and its disorders (the Tri-dimensional Personality Questionnaire, and the Temperament and Character Inventory) were both developed by Cloninger and their validity was also assessed by Cloninger.

Experiential Theories

A review by Draganic, Lecic-Tosevski, and Calovska-Hertzog's (1997) indicated that constitutional factors interact with childhood experiences that fall into three main psychological risk categories: trauma, early separation or loss, and abnormal parenting.

Trauma

Deviations in a child's personality may be influenced by parental disorders or by traits shared between parents and children (Draganic, Lecic-Tosevski, & Calovska-Hertzog, 1997). Paris (1998b) reviewed the works of researchers who examined the mechanisms of gene-environment interactions and found impulsive or depressed parents were more likely to inflict trauma on their children, and children with difficult temperaments were more likely to receive poor parenting. Impulsive children were also more difficult to calm down and needed more structure from parents. Reports of adults with PDs suggested such predispositions increase the likelihood that negative childhood events will occur. Findings also suggested that abused children are more likely to develop

disturbances in sense of self, in affect and impulse control, and insecurity in relationships.

It has been proposed that intense trauma could evoke a chronic Post Traumatic Stress Disorder (PTSD) that affects personality by producing self-image disturbances, and problems with trust, intimacy, and self-assertion. Supporting this view, Draganic, Lecic-Tosevski, and Calovska-Hertzog (1997) found PTSD frequently co-occurs with PDs, particulary Passive-Aggressive PD, Avoidant PD, and APD. On the other hand, Paris (1998a) reviewed cross-sectional retrospective studies and found the strength of association between traumatic experiences during childhood and adult PDs was only fair (effect size 0.27). He also found a large overlap between the frequency of trauma in PDs and other mental disorders. In addition, the type and severity of trauma reported by PD patients resembled that found in community samples with the majority involving single incidents and a significant number reporting no abuse at all. In fact, research indicates only 25% of adults exposed to severe trauma in childhood develop long-term sequelae Although hypothesized markers for trauma have been tested, including symptoms related to certain PDs, Paris found no empirical evidence supporting a clear relationship between trauma and any of these symptoms.

With respect to research regarding the impact of child abuse, Paris (1998a) found evidence that the effects of child abuse depend on cognitive schemata. When abused children feel stigmatized, self esteem decreases and the outcome is worse while having a social network reduces the likelihood of negative consequences. Only 20% of adults with histories of childhood abuse (sexual or physical) develop psychopathology. Studies indicated that childhood trauma usually occurs in the context of significant family

dysfunction, including high levels of parental psychopathology, emotional neglect, and family breakdown. The long-term effects of sexual abuse were also found to depend on a variety of factors such as severity, frequency, duration, relationship with the abuser, and the nature of the sexual act. Studies also indicated that sensitivity to abuse depends on temperament and social learning. Genetics were found to account for about 40% of the variance in most traits and behavioral genetic research shows that 76% of the variance due to environment is largely unshared. Findings suggested that PDs are not formed by rearing practices but by a multitude of experiences unique to the individual that derive from outside the family (Paris, 1998a).

Implications drawn from Paris' (1998a) review are constrained by several limitations. Specifically, most of the research reviewed by Paris was based on adults retrospective reports of childhood and few studies included the full range of childhood adversities. In addition, only a small number of studies (8) were reviewed regarding gene-environment interactions.

Associations between childhood adversities and adult psychopathology do not necessarily represent environmentally mediated risk processes. Rutter and Maughan (1997) found traumatized children are often those genetically at risk such as when one or both parents have a mental disorder and, therefore, they believe environmental risk hypotheses should be tested using genetically sensitive twin, family, and adoptee designs. Such designs have produced meaningful findings regarding antisocial behavior. Specifically, in the absence of genetic or environmental factors, Rutter and Maughan found the risk of adult crime to be about 3%. Risk increased to 6% when environment

was considered alone, and to 12% when genetics was considered alone. When environmental and genetic risks were considered together, however, risk for crime rose to 40%.

Inferences drawn from associations between childhood trauma and adult PDs are no less problematic. Findings are often based on adults' retrospective reports of childhood experiences but using available prospective data, Rutter and Maughan (1997) found that researchers conducting retrospective studies were more likely to report stronger associations than those looking forward suggesting potential bias in retrospective reporting. In this regard, Draganic, Lecic-Tosevski, and Calovska-Hertzog (1997) found, that traumatized individuals have difficulty integrating trauma-related sensations and perceptions into explicit memories, that childhood memories can be distorted or repressed, and that PDs may be a source of inaccuracy in a patient's retrospective view of their childhood. Rutter and Maughan suggested it is possible that the relatively strong effects that appear to apply, when patient populations are compared with control groups, could be a result of biases in retrospective reporting. Although they found no evidence that adults with psychopathology over-report childhood adversities, they did find evidence that mentally healthy adults tend to under-report childhood adversities which may result in exaggerated differences between cases and controls. Rutter and Maughan stated that one must also taken into account base rates. He explained that it is entirely consistent to have a very big difference between patient groups and controls, while the majority of individuals who experience risk factors are without an adult PD.

Separation and Loss.

Wishnie (1977) believed children need at least one caregiver to provide relative stability in a world of chaos, a person on whom the developing child can focus their hopes for gratification and satisfaction. The nurturant person represents potential relief from painful realities and developmental conflicts. Wishnie found that most behaviorally disordered children have a nurturing caregiver until around age 3 to 7 when this person suddenly changes or is lost. He believed that the pain and rage of this loss creates a sense of panic and hopelessness within the developing child which can lead to a developmental defect in the child's capacity to handle associated emotions. He stated that without consistent attention, and positive responses from parents, children come to believe there is something irrevocably wrong with them, that they are different and separate from others and therefore unlovable, and that their parents are right for not caring about them. Such beliefs often lead to overt acts of deviant behavior which serve to restore lost attention, and allow expression of feelings such as rage and hurt. In addition, the punishment for these acts provides temporary relief from guilt and reduces anxiety by confirming the wisdom of the parent who perceives the traits as inherently bad thereby restoring the child's faith in the myth of parental infallibility that is so necessary for young children who are totally dependent upon their parents for survival. Wishnie believed that individuals have the capacity to observe their own thoughts, memories, feelings, impulses, and behavior and to integrate and draw conclusions from these experiences, but when they experience the panic state, such capacities are immobilized. Wishnie explained that this panic state and the inability to cope with it, represent the central defect in the individual's

emotional makeup that leads to APD.

A criticism of Wishnie's (1977) theory is that he provided no basis for his claims regarding children's responses to parental loss, putting into question the validity of his claims. He suggested that parental loss causes developmental conflicts but he did not explain what these conflicts are. Perhaps he felt that these conflicts prevent the individual from moving through developmental stages. If so, was he implying a connection between unsuccessful resolution of conflicts through stages and development of PDs, and particularly APD? In addition, Wishnie claimed that a child's inability to cope with the "panic state" causes an emotional defect that leads to a particular PD like APD but he does not explain how or why such a defect manifests as APD or why it manifests as APD as opposed to some other Axis I or Axis II disorder. If Wishnie was suggesting that APD is the next logical developmental step for behaviorally disordered children, then how would he explain Paris' finding that only a small percentage of adults, having had adverse experiences in childhood, develop long-term sequelae?

Draganic, Lecic-Tosevski, and Calovska-Hertzog (1997) reviewed the literature on adverse childhood experiences and adult psychopathology and found no evidence of a direct relationship between early loss and adult psychopathology. Rather, they found the effects of early loss interact with such factors as family dysfunction after a loss and buffering factors from outside the family. Children who suffer severe neglect, for example, have a particularly poor long-term prognosis compared with those who had more secure attachment bonds. Findings suggested that the quality of parental bonds is probably the most important determinant of long-term damage. Paris (1998a) supported

this view. He found that the effect of parental separation is determined not by family breakdown, but by personality traits of the affected child, the quality of family life before separation, availability of the noncustodial parent after separation, decreases in financial resources, changes of domicile, continued conflict between parents, and depression in the custodial parent.

Before concluding a specific risk factor has a real effect, Rutter and Maughan (1997) recommended checking whether the effect actually derives from some other associated risk factor. They found that parental loss in the absence of parenting difficulties, for example, was not associated with any appreciable increase in risk of adult psychopathology, but parenting difficulties were associated with psychopathology, even when they arose in the absence of parental loss. Thus, parental loss is an indirect risk factor, because it increases the likelihood of parenting difficulties.

With respect to parental loss or separation and development of APD, Crowell, Waters, Kring, and Riso (1993) reviewed selected studies by researchers investigating proposed connections between early experiences and subsequent development of APD and Borderline PD. Studies reviewed indicated that 90% of APD adults report a history of separations from parents of one month or more, including placement outside the home, hospitalization, death of or desertion by the caretaker, divorce, incarceration, and vacations. About 50% of APD adults also described a history of parental neglect. Crowell et al found that the strongest childhood predictors of antisocial behavior in adult life, were the number of antisocial behaviors the child engaged in, and that behavior in childhood predicted more strongly the development of APD than either family or

environmental factors. They also found that exposure to good discipline by parents was most associated with reduced risk of developing APD. This association was found whether children were placed out of the family home, whether a parent was absent for much of the childhood, and/or whether the parents exhibited antisocial behavior.

Problems associated with the Crowell, Waters, Kring, and Riso's (1993) review included issues affecting validity. Most of the studies reviewed were based on a limited theory-driven perspective and failed to address alternative hypotheses. Interpretations were complicated by the fact that studies often included only small samples, diagnoses from chart reviews, and retrospective data. In many cases the patient was the sole source of diagnostic information. In addition, comorbidity with Axis I diagnoses and other PDs was present in a high percentage of PD patients. Another problem associated with this review has to do with the generalizability of findings. Crowell et al stated that they based their review on selected studies. Whether or not these studies adequately represent the research in the area is unknown.

Parenting.

In their review of research regarding associations between adverse childhood experiences and psychopathology in adult life, Rutter and Maughan (1997) found evidence that chronic adversities were more highly associated with psychiatric disorder than were isolated adverse events. Results distinguished between active negative experiences and a lack of positive experiences. Risks associated with serious neglect, for example, were found to be comparable to those associated with active abuse. Type of abuse, however, may play a role in the development of specific disorders. Norden, Klein, Donaldson,

Pepper, and Klein (1995) examined individuals with prominent Axis II psychopathology and found antisocial traits were associated with reports of significantly poorer maternal and paternal relationships and physical abuse, while Borderline traits were associated with reports of poorer maternal and paternal relationships and sexual abuse. Other PDs did not exhibit similar patterns of associations. Perhaps different children process similar circumstances in different ways. In this regard, Rutter and Maughan (1997) found evidence distinguishing between experiences and the way young people cognitively process those experiences suggesting that long-term effects of experiences are mediated, in large part, by the mental models children develop regarding those experiences. Unfortunately, there has been very little research conducted in this area and cognitive models such as Beck's (1997) Cognitive Model, have only begun to be empirically tested (Dobson, & Pusch, 1993). For this reason, Rutter and Maughan encouraged future researchers to focus on the relative importance of cognitive factors.

Medical Incident History

Medical incident history may also play a role in some PD development. Brain damage due to prenatal and perinatal insults may lead to a continuum of disorders, and central nervous system deficits caused by postnatal insults such as head injuries, poor nutrition, physical abuse, and exposure to toxins which may contribute to antisocial outcomes. Environment, however, can help overcome or heighten the ill effects of early physical or biological insults (Brennan & Mednick, 1997).

Interpersonal Theories

Interpersonal theorists hold that interactions with significant others are most

important in PD development. The main criticism of interpersonal theories is that they are not very well supported by empirical research and therefore, it is difficult to determine their validity.

An underlying assumption of interpersonal theory is that personality is best viewed in terms of recurrent interpersonal dispositions or tendencies to display certain traits, or characteristic patterns of interpersonal behavior (Pincus & Wiggins, 1990). Pincus and Wiggins described PD patterns of interpersonal behavior as disordered thoughts, feelings, and actions in relation to significant others, maladaptive causal loops between interpersonal perception, behavior, and reaction to environment, rigid and extreme use of limited interpersonal actions, regardless of their appropriateness, and adaptive inflexibility or the tendency to perpetuate and foster rather than resolve new problems. They added that reciprocal interpersonal relations may explain how interpersonal behavior could fulfill a self-definitional function and, in the case of PDs, perpetuate maladaptive pathological self-definitions.

Pincus and Wiggins (1990) believed that dysfunctional interpersonal behavior is either the defining feature or a major component of many PDs. They explained that when behaviors are inappropriately inhibited or rigidly enacted they give rise to consistent dysfunctional patterns of interaction which perpetuate dysfunction through their impact on others and the responses they elicit. They proposed that the underlying polarities of personality include reinforcement (pleasure-pain), source of reinforcement (self-other), and instrumental coping style (active-passive) but add that the self-other polarity is of central interest to Interpersonal Theory.

Benjamin (1996b) developed a model she called the "Structural Analysis of Social Behavior (SASB)", an approach that permits operationalized description of interpersonal patterns and their impact on the self-concept. She wrote that the SASB was built on the work of Leary (1957), and Schaefer (1965). Leary used the interpersonal circumplex to propose an interpersonal diagnostic system with points defined by the underlying horizontal (nurturance vs. hostility), and vertical (submission vs. dominance) axes. Schaefer proposed a circumplex model of parental behavior with horizontal (rejection vs acceptance), and vertical (psychological control vs. psychological autonomy-giving) axes. The horizontal axes of both models express a love vs. hate dimension but their vertical axes differ with one expressing dominance vs. submission and the other expressing a dimension of control vs. autonomy-giving. Both of these models have been empirically tested and form the basis of the SASB model which incorporates three orthogonal dimensions (love vs. hate, dominance vs. submission, and control vs autonomy-giving) Benjamin felt that the SASB model offers testable, refutable theory for understanding, on a symptom-by-symptom basis, how DSM PDs are affected by the individual's specific social learning experiences and current social context.

In Benjamin's (1996a) model, the primary characteristics of APD are the need for control of others and autonomy for self, detachment, and lack of remorse. Unlike other Cluster B PDs, Benjamin explained that APD individuals do not fear abandonment, and do not have a sense of entitlement or dependency. She felt that family interactions are the primary contributors to APD development. Children subjected to harsh parenting learn to be uncaring and aggressive. When neglected they learn to self-indulge with no concern for

others. If parents are inconsistent regarding expectations and discipline, particularly if blame or humiliation is used, a child responds by fiercely protecting their autonomy. They learn to not care, to display uninvolved affection or "pseudocare", to con, and to control and blame others. These children may also self-indulge without concern for their own well-being (e.g., drug abuse, prostitution, crime). Finally, where parents allow the child to run the family, the child learns to control without bonding, showing no concern for other family members.

In support of Interpersonal Theory, Benjamin (1996a) studied psychiatric inpatients who completed the SASB Intrex questionnaires. Based on their retrospective reports of childhood, APD individuals experienced excess autonomy and control, and poor internalization of the parent.

Pincus and Wiggins (1990) examined PDs using a structural model of interpersonal dispositions on a two-dimensional circumplex with variables ordered around the orthogonal dimensions of Dominance vs. Submission, and Nurturance vs. Hostility. They found that Histrionic, Dependent, Narcissistic, Antisocial, Paranoid, Schizoid, and Avoidant PDs all had interpretable projections on the interpersonal circumplex, whereas Cumpulsive, Schizotypal, and Borderline PDs did not. Romney and Byner (1997) applied structural equation modeling to test the theory that PDs could be arranged around the interpersonal circumplex. They found the circumplex model provided a good fit for Histrionic, Dependent, Schizoid, Paranoid, and Narcissist PDs but a simplex model proved more appropriate for depicting relationships among Antisocial, Borderline, Avoidant, Passive-Aggressive, and Compulsive Personality Disorders. Pincus and Wiggins (1990)

suggested that some PDs may not fit easily onto the circumplex because the central dysfunction associated with certain PDs may involve cognitive and/or emotional processes rather than interpersonal behavior. They also pointed out that gender may play a role in the applicability of interpersonal approaches. In this regard, male-dominated theories are generally based on models of normal development which emphasize the emergence of the individual and the task of achieving "autonomy" but other theories have been proposed which emphasize "connectedness" as being central to women's development and their ability to develop and maintain relationships (Wastell, 1996).

The empirical validity of interpersonal theory requires further testing. Where interpersonal theories are explained using the interpersonal circle, or circumplex, the validity of these models is at least partially dependent on the reliability of the circumplex. Mixed findings from research investigating circumplex models indicate the need for further development of these models.

Other Theories

Other theories have been proposed to explain PDs but these theories have either not been empirically tested, particularly with regard to APD, or they explain the nature of PDs but not in terms of their development. Such theories include psychodynamic, cognitive-behavioral, learning, cultural deviance, social information processing, and biological theories.

Psychodynamic Theories.

Psychodynamic theorists point to abnormal internalization, differentiation, and integration of object relations, or identity diffusion as the primary cause of personality

disorders (Adler, 1990; Kernberg, 1996). Psychodynamic theories are difficult to test empirically and consequently, the validity of these theories remains questionable.

Cognitive-Behavioral Theories

Cognitive-behavioral models share three fundamental propositions: (a) cognitive activity affects behavior, (b) cognitive activity may be monitored and altered, and (c) desired behavior change may be affected through cognitive change. All models target both cognitive and behavioral change (Dobson & Pusch, 1993). Beck's (1997) model assumes human dysfunctional disorders, psychological or behavioral, are characterized by dysfunctional thinking, and that dysfunctional thinking accounts for the affective and behavioral symptoms. Beck stated that regardless of the intervention used, when patients get better there is an improvement in the way they think, that is, an improvement in their attitudes, beliefs, or automatic thoughts. Dobson and Pusch explained that dysfunctional beliefs (also referred to as schemata or assumptions), which are activated by events that impinge upon those beliefs, are mediated through specific negative appraisals, perceptions, and biased processing of current experience. Beck's model was originally developed with broad applicability in mind and a focus on empirical validation of treatment efficacy as indicated by not only cognitive but also behavioral change in patients.

With respect to PDs, Dobson and Pusch's (1993) model of cognitive-behavioral theory highlights the causal role of deficits in basic areas. It is assumed that a small deficit in a basic area (e.g., autonomy) may result in eccentric behavior or minor life difficulty but as the deficit becomes more pronounced, it may develop into a PD (e.g. a pronounced deficit in autonomy may develop into Dependent PD). It is assumed that basic deficits

develop by way of formation of cognitive schemata or beliefs which are strongly held by an individual and therefore not easily changed. These schemata are believed to be formed in childhood under adaptive circumstances and carried into adulthood without amendment even though no longer adaptive. Basic deficits are assumed to be central to socially inappropriate behaviors exhibited by PDs. For example, with respect to APD, very young children, asserting their independence, may form the belief that no one should stop them from doing what they want. Normally, young children will develop new, more adaptive schema, which accept that sometimes they need to do what they are told. Sufficient reinforcement of initial schema, however, may prevent more adaptive rules being formed and, in adulthood, a dysfunctional schema like "no one should stop me from doing what I want" may result in a diagnosis of APD. Conceptualizing PDs in terms of schematic processes suggest that these chronic difficulties are as amenable to cognitive-behavioral interventions as are depression and anxiety. Altering rigid core beliefs associated with PDs, however, is not easy. Most Axis II patients present with a combination of problems which complicate treatment resulting in the need for therapy of a longer duration involving more frequent sessions and with more severe pathology requiring a greater emphasis on behavioral techniques.

While the cognitive model of PD development holds intuitive appeal, Dobson and Pusch (1993) noted there have been no controlled outcome studies conducted to test the model. Therefore, before any definite conclusions can be drawn, studies dealing with specific PDs need to be conducted. In addition, they note that application of the model has been limited in that not all forms of PD have been targeted. In particular, they are

aware of no study addressing cognitive therapy with antisocial individuals or individuals diagnosed as having an APD.

Learning Theories.

Learning theorists view development of personality as a product of learning. The important elements of a learning situation are drive, cue, response and reward. Drive refers to internal feelings that activate behavior. The cue is the situation precipitating the response, which often has characteristics resembling the original learning situations. Reward is the subsequent positive or negative consequences occurring to the individual making the response. If the response reduces drive, it is experienced as reinforcing but if punishment follows the response, that response will tend not to be repeated and, with successive occurrences of the behavior not being reinforced, the behavior ultimately will be extinguished. In the case of operant learning, drive is not a factor More important is the reaction of the environment to any response displayed by the individual. Responses that are rewarded over time will likely be repeated while those not reinforced tend to be extinguished, particularly where an alternate unpunished or rewarded response replaces the punished response (Azrin & Holz, 1966; Dollard & Miller, 1950; Eron, 1997).

In social learning theory, behavior is viewed in terms of stimuli, reinforcements, and punishments. Behavior is learned and maintained through environmental experiences, either directly or vicariously, and the learning of behaviors is controlled by reinforcement contingencies and punishment. Thus, individuals acquire behaviors that are rewarded or reinforced but avoid behaviors, theirs or a model's, that have been punished or not reinforced (Bandura, 1973; Eron, Walder, Lefkowitz, 1971).

Learning theories have been empirically tested (Bandura, 1973; Dollard & Miller, 1950; Eron, Walder, & Lefkowitz, 1971), but the focus of the research has been normal development. Studies have been conducted regarding the learning of aggression in children but these studies have not included adult outcomes. Thus, more research is needed to investigate learning mechanisms as causal in the development of APD and other PDs.

Cultural Deviance Theories

There are a number of cultural deviance theories but they all have in common an emphasis on the relationship between association with deviant peers and deviant behavior (Thornberry & Krohn, 1997). According to differential association theory, deviant behavior is learned through interacting and communicating with other people and that learning takes place best within intimate primary groups such as peer networks. The effect of peer associations on delinquent behavior is indirect, operating through the learning of definitions (Sutherland, 1939). Social control theorists argue that if any relationship exists, it is because deviant behavior leads to peer association. That is, once delinquency is exhibited, delinquents tend to seek each other out for companionship but the peer group exerts no real causal influence on behavior. Therefore, associating with delinquent peers is seen as a consequence, not a cause, of delinquency (Glueck & Glueck, 1950; Hirshi, 1969). Integrated theorists focus on deviant companions as the primary source of motivation for deviant behaviors but adopt the control theory perspective that weak social bonds increase the odds that adolescents will have delinquent friends, and that these individuals will tend to find one another in the process of seeking and sorting

companionships (Conger, 1976). According to interactional theory, deviant peers model and reinforce deviant behavior and provide a social context rich in normative support for deviance, and therefore, associating with deviant peers is likely to cause both the initiation and the maintenance of deviance. Deviant behavior also exerts a strong causal impact, according to interactional theory, in that it leads to association with deviant peers because people prefer to associate with and have as their friends people who are similar to them. Therefore, the more deviant individuals are, the more likely they are to seek out deviant friends. Given that deviance violates the normative structure of prosocial peer groups, deviant behavior can also cause increased association with deviant peers through exclusionary processes and there are reciprocal causal influences represented by cross-lagged effects from peers at one time to behavior at the next and from behavior at one time to peers at the next (Thornberry, 1987).

Empirical support for the cultural deviance theories is lacking. Like the learning theories, most research in this area has focused on children and adolescents rather than adults. No research, based on these theories, has been conducted to investigate the relationship between association with deviant peers and the development of APD or any other PD.

Social Information Processing Theory

Social Information Processing Theory, as described by Dodge and Swartz (1997), describes a series of sequential mental operations which result in the decision to respond to a particular social stimulus. The first mental operation involves encoding of social cues into short-term memory. In this regard, it has been found that aggressive children attend

to fewer cues, encode a relatively smaller number of cues, and when confronted with ambiguous social situations, seek additional information less frequently than other children. Also aggressive children selectively attend to and recall threatening social cues. The second operation involves the interpretation of the social cues. Aggressive children tend to have a hostile attributional bias which increases the probability of aggressive responding for both ambiguous and benign stimuli. The third operation requires the clarification of goals. There is a need for more research in this area but it has been hypothesized that aggressive children might evaluate goals such as dominance and control more positively than more relational goals. The fourth operation focuses on response access and construction. When faced with a problematic situation, aggressive children generate fewer potential responses. For young children, the number of responses generated is negatively correlated with the number of aggressive acts but with older children, the quality of the response is more important. Aggressive children tend to produce responses that are either hostile or ineffective and irrelevant, particularly if their first response proves to be ineffective. The fifth operation involves response evaluation and a decision to act or not act. In this regard, aggressive children consistently evaluate the potential outcomes of aggressive behavior more positively than do their nonaggressive peers. They believe aggression will result in tangible rewards, peer group approval, reduction of aversive consequences, enhancement of self-esteem and positive feelings, and they expect their hostile behaviors will not lead to suffering in their victims. In fact, there is evidence that aggressive children hold negative outcome expectations for more prosocial behaviors. Aggressive children also value the outcomes of aggression more

highly than do nonaggressive children. They also value control of victims more than nonaggressive children and place less importance on the possibility that their behavior could lead to negative interpersonal relationship outcomes. There is also evidence that aggressive children evaluate their ability to engage in hostile behavior more positively and their ability to enact behavioral strategies that minimize conflict more negatively than less aggressive children. Because of these differences in processing styles, aggressive children have in memory, richer schema and social constructs on aggression than do other children. The final operation involves the behavioral enactment of the selected response strategy Enactment requires certain motor and verbal skills and, if a child's skills for enacting nonaggressive responses are weak, they might resort to more aggressive enactments.

Although considerable empirical research based on social information processing theory has been conducted with aggressive children, there has been little empirical research conducted to investigate the role of social information processing in the development of APD or other PDs in adults.

Biological Theory

Weston and Siever (1993) reviewed the research investigating biologic correlates of PDs. Findings were broken down into DSM clusters. In this regard, they found that few studies have been conducted to investigate biological correlates for the anxiety cluster, Cluster C, although hypotheses of altered noradrenergic, γ-aminobutyric acid (GABA)-minergic, or chemoreceptor function have been suggested for PD-related anxiety symptoms. Active investigation of these factors have not yet been undertaken.

With respect to Cluster A, Weston and Siever (1993) found that abnormalities in cognitive/information processing were related to social introversion and psychoticism, deficits in signal/noise discrimination were linked to social isolation and withdrawal, and deficits in information processing speed were associated with mania and psychotic-like symptoms. They found Indices of catecholamine metabolism, including decreased platelet monoamine oxidase (MAO) and concentrations of CSF Homovanillic Acid and Plasma Homovanillic Acid in Schizotypal PD which suggest neurochemical abnormalities may be linked to psychosis. Given that increasing dopamine and norepinephrine exacerbates psychotic symptoms in schizophrenia, it may be that neurochemical abnormalities amplify underlying cognitive deficits (Weston & Siever, 1993). Dopaminergic activity may also modulate the expression of an underlying genotype for Schizophrenia-related disorders (Siever & Davis, 1991).

Cluster B impulsivity was correlated with serotonergic and noradrenergic system abnormalities. Weston and Siever (1993) found increased serotonergic activity stabilized mood in Borderlines, and an amphetamine challenge (causing a depletion of norepinephrine) reduced psychotic-like symptoms in some PD patients suggesting a biologic basis for affective instability and cognitive disturbances (Weston & Siever, 1993). They also found with respect to APD, that the neurotransmitter serotonin, or central 5-hydroxytryptamine (5-HT), has been found to mediate behavioral inhibition. Specifically, serotonergic deficits were associated with a diminished capacity to translate anticipation of punishment into appropriate behavioral inhibition. In a study reviewed by Coccaro, Astill, Szeeley, and Malkowicz (1990), mice with dysfunctional 5-HT receptors

displayed increased aggression, hyperactivity, and anxiety. In addition, the serotonin metabolite, 5-hydroxyindoleacetic acid (5-HIAA) was inversely associated with impulsive-aggressive behaviors whether self or other-directed (Siever & Davis, 1991). Findings from the Coccaro et al review indicated that 5-HT was associated with stimulus-linked rather than generalized aggression. Serotonergic activity was found to be correlated most closely with an increased "tendency" toward aggressive behavior (mediated by other factors including level of arousal) rather than aggressive behavior itself, and a reduction in 5-HIAA was more closely associated with a history of impulsive, rather than premeditated aggression. In addition, a reduction in 5-HT function was related to irritability while assaultiveness was dependent on increased 5-HT function. They also found evidence that the postsynaptic receptor was less responsive in patients with histories of impulsive aggression. Whether increasing 5-HT activity clinically will diminish impulsive aggressive behaviors in humans is not yet known, but Coccaro et al noted that fluoxetine, a 5-HT reuptake inhibitor, has been tested with borderline patients and results indicated 75% were improved at follow-up. In addition, those suffering the greatest serotonergic dysfunction were found to be most sensitive to pharmacologic enhancement of central 5-HT.

The noradrenergic system may also be involved in control of impulsive-aggression in APD. Siever and Davis (1991) found that higher levels of noradrenergic metabolites were associated with extroversion and sensation-seeking, and increased growth hormone responses to the noradrenergic agonist clonidine were positively correlated with irritable aggression. Given that the noradrenergic system mediates arousal and orientation to the environment, enhanced noradrenergic activity might increase externally directed

aggression while impaired noradrenergic transmission might block other-directed aggression. In APD, both noradrenergic and serotonergic systems may be important determinants of impulsive and aggressive behaviors toward the self and others. Siever and Davis found lesions to the noradrenergic system prevented aggression associated with serotonergic lesions in animals. Findings suggest that pharmacological interventions may help attenuate impulsive/aggressive behaviors in APD.

Research based on biological theories has added much to our understanding of the biological nature of PDs, including APD, but these theories do not clarify the causal relationships between biology and PDs. In particular, if one influences the development of the other, the direction of influence is unclear Future research is needed to determine whether biology is a cause or an outcome of having a PD

Summary and Conclusion

A number of theories have been proposed to explain the development of personality disorders and, in particular, APD development. Findings from empirical research supporting psychobiological theories are promising. Researchers investigating familial aggregation, prevalence rates, and differences due to environmental antecedents suggest a probable underlying genetic substrate for APD that correlates with temperament and behavior (novelty seeking, impulsivity, aggression). No empirical research testing of experiential theories has supported a clear relationship between adverse experiences and the development of personality disorders or their symptoms. In fact, researchers are finding that the effects of adverse experiences interact with such factors as frequency, intensity, duration, and nature of the experience, the quality of parental bonds or family

dysfunction, buffering factors from outside the family, and the way children cognitively process experiences (Paris, 1998a). Although some research findings support an interpersonal perspective, more empirical research is needed to validate these theories. The interpersonal circumplex has been proposed as an interpersonal research tool but studies that have been conducted to investigating circumplex models have produced mixed findings suggesting the need for further development of these models (Pincus & Wiggins, 1990; Romney & Byner, 1997). Although biological research is relatively new, it has contributed greatly to our understanding of the biological nature of PDs and there is considerable potential for future research in this area to inform our understanding of PD development.

Numerous limitations and methodological difficulties potentially affect the reliability and validity of the findings reported herein. Most of the research reviewed is based on adults' retrospective reports of childhood which may be biased due to inaccurate, repressed, or distorted memory, and few researchers have examined the full range of childhood adversities, or considered gene-environment interactions. Many researchers studying PDs and APD development report inconsistent findings making it difficult to draw conclusions. A number of factors contributing to variability of findings include similarities in definitions of Axis II and other disorders, lack of differentiation between construct definitions, small sample sizes, poor selection procedures, variability in samples, inadequate diagnostic methods, potential bias in diagnostic instruments, the absence of direct interviews with relatives, and lowered cutoff levels for positive criteria (Dahl, 1993; Nigg & Goldsmith, 1994). Validity of findings from reviews are also difficult to determine

because details regarding methodology and subject samples are often not reported. In addition, some reviewers only look at selected studies which may not adequately represent research in the area. Finally, little or no research has been conducted with respect to the development of some PDs, and empirical research has only begun to be conducted with respect to some promising theories (e.g., Beck's Cognitive Theory). These areas provide challenges for researchers in the future. In the meantime, one can only imagine the effect such research may have on our understanding of personality disorder development and APD development, in particular.

In summary, research examining early childhood antecedents of antisocial personality has provided contradictory and inconclusive results. Consequently, there is confusion as to which factors contribute to the development of APD and which factors exert the greatest influence. To gain a richer understanding of APD development, the relative importance of specific early childhood indicators of a developing APD need to be assessed. A useful approach to determining which early childhood factors are the strongest indicators of later APD development, is to perform a meta-analysis of existing research.

Meta-analysis (MA) is an objective and quantitative approach to an expert review; a method of systematically identifying and statistically combining the results of studies to arrive at summary conclusions about a body of research in terms of a single estimate of effect or risk (Petitti, 1997; Paddle, 1997). Meta-analytic reviews describe the typical strength of an effect or phenomenon, its variability, its statistical significance, and the nature of the moderator variables from which one can predict the relative strength of the

effect or phenomenon (Rosenthal, 1995).

Meta-analysis resembles original research in that it proceeds through a series of stages which distinguish it as disciplined inquiry (Hedges, Shymansky, & Woodworth, 1989). Similar to those conducting traditional narrative reviews, meta-analysts identify relevant research studies instead of collecting original data (Wolf, 1986) but unlike narrative reviews, which include only selected studies, meta-analyses may include any number of studies (Preiss & Allen, 1995). In fact, the accuracy of estimates improves as the amount of available data increases (Preiss & Allen, 1995). In MA, descriptive features of each study are coded, study data are transformed into a common metric called the effect size, and statistical tests assess which independent variables or study characteristics account for variation in the dependent variables or effect sizes (Durlak, 1995). The procedures followed in MA enable researchers to make meaning out of numerous and often discordant findings in a particular research area. Through the differential weighting of studies and by systematically combining the numerical results of studies with conflicting research methods and findings, researchers can detect regularities, trends, and consistencies among findings to arrive at more accurate and credible conclusions (Hunt, 1997).

If APD in adulthood is associated with inherited biological factors, childhood experiences, interpersonal relationships, or a child's behavior, then the results of this meta-analysis may provide information that will determine which of these factors, alone or in combination, have an effect on the development of APD in adulthood. The results should also provide information about the strength of particular factors as indicators of increased

risk for APD development in adulthood. Accordingly, the following research questions were proposed:

Research Questions.

1. Are there significant effects for all four domains (psychobiological, experiential/external, interpersonal, and behavioral), as early indicators of subsequent APD development?
2. Of the domains with significant effect sizes, which domain is the strongest indicator of APD development?
3. Do all variables within each domain show significant effects as early indicators of APD development?
4. Of the variables with significant effect sizes, which variable is the strongest indicator of APD development?
5. Do particular variables (e.g. year of study, country of study, study type, study design, average age of subjects, diagnostic indices, or methods of assessing APD) have a moderating effect.

CHAPTER III

METHOD

The analysis of data in MA involves five major steps beginning with the formulation of the research question, followed by the retrieval of relevant studies, coding of those studies, calculating the index of effect, and conducting statistical analyses of effects (Durlak, 1995). These are explicated in turn below.

Formulating the Research Question

A review of prior research was conducted to identify important issues needing clarification. The issue of importance that was identified by the literature review was the need to clarify which early childhood influences were the most significant predictors of later APD development. Various sources referred to the same risk factors, but generally speaking, review of the DSM-IV indicated children at risk for developing APD are those suffering from specific disorders or pathology, particularly CD and ADHD, and those who have experienced child abuse, neglect, unstable parenting, or inconsistent parental discipline. A review of personality theories revealed a number of potential contributing factors. Psychobiological theorists point to genetics and temperament as likely contributors to APD development, experiential theorists suggest that children at risk for APD are those who experience trauma, in particular physical or sexual abuse, separation and/or loss, significant medical incidents, or poor parenting, and interpersonal theorists indicate children at risk for APD development are those who are neglected, or those who receive inconsistent discipline or harsh parenting. Finally, a review of research revealed additional factors that may increase a child's risk for APD development including a below

average IQ, poor school achievement or early dropout, emotional abuse, rejection by a caregiver, family dysfunction or breakdown, family socio-economic status, parent delinquency or criminality, parent disorder/pathology, having a delinquent or behavior problem sibling, the child's having or lacking social desirability or peers and/or the child's antisocial behavior.

The risk factors from all sources were identified as best falling into one of four primary domains: i) psychobiological, ii) experiential/external, iii) interpersonal, or iv) behavioral. Based, in part, on the writings of personality theorists, and in part, on the writings of researchers in the area of personality development, the four domains were designed to reflect those influences that may affect personality development, and, in particular, APD development. The psychobiological domain includes innate psychobiological characteristics of the child such as genetics, temperament, intelligence, and certain disorders/pathology that are outlined in the DSM which may occur during childhood and are believed to have a biological basis. The experiential domain includes influential childhood experiences that may affect personality development and, in particular, APD development, including physical abuse, sexual abuse, emotional abuse, separation and loss, poor school achievement, medical incidents, family socio-economic status, and/or family dysfunction or breakdown. The interpersonal domain is intended to reflect those interpersonal interactions with significant others that may influence personality development and/or APD development including neglect, rejection by a caregiver, unstable or erratic parenting, inconsistent discipline, harsh parenting, parental delinquency or criminality, delinquent or behavior problem sibling, parent disorders or

pathology, and social desirability and/or peers. Finally, the behavioral domain reflects the influence of the child on its own development because of its way of behaving in response to biological, experiential or interpersonal stimuli. Within the context of APD, behavioral responses refer more specifically to antisocial behaviors in childhood such as behaviors that violate the basic rights of others, behaviors that are aggressive toward people or animals including bullying, fighting, physical cruelty, and forced sexual activity, behaviors that involve the deliberate destruction of property, including fire setting, and other antisocial behaviors such as deceitfulness or stealing, including break and enter, and behaviors involving serious violation of rules and truancy.

Definition of Terms

For purposes of clarification, the following terms are operationalized below. These operational definitions are not intended as definitive definitions of the constructs. Rather, they are working definitions culled from the relevant research on APD and are used in the present dissertation for purposes of the meta-analysis. For broader theoretical purposes it is clear that some of the following definitions are untenable. The definitions for childhood, adolescence, and adulthood, for example, are not theoretically acceptable. Since they are used in the DSM IV and the relevant research in the present study, however, they are used as operational definitions even though they would not stand up to critical scrutiny as definitions of the constructs.

Psychobiological Domain.

The Psychobiological Domain refers to heritable characteristics that may influence childhood personality development and specifically, APD development, including: i)

genetics, ii) temperament, iii) intelligence, and iv) certain disorders/pathology occurring during childhood.

Genetics.

Genetics refers to an individual's total genetic inheritance in terms of the combined genetic contributions of two parents.

Temperament.

Temperament refers to inherited characteristics that influence personality development, for example, differences in impulse control, anxiety modulation, novelty seeking, or persistence.

Intelligence.

For current purposes of operational definitions, intelligence refers to mental ability, commonly referred to as the intelligence quotient (IQ) as measured by standardized tests of intelligence such as the Wechsler Intelligence Scale for Children.

Disorders/Pathology Occurring During Childhood.

The term disorders/pathology occurring during childhood refers to any disorders or pathology which occur during childhood as outlined in the DSM-IV. In this regard, the most common disorders associated with APD are Conduct Disorder and Attention Deficit/Hyperactivity Disorder.

Conduct Disorder (CD).

In this study, CD is Conduct Disorder as described in the DSM IV. CD is characterized by aggression toward people or animals including bullying, fighting, physical cruelty, and forced sexual activity. Another characteristic of children with CD is the

deliberate destruction of property, including fire setting, deceitfulness or stealing, including break and enter, and serious violation of rules and truancy.

Attention-Deficit/Hyperactivity Disorder (ADHD).

In this study, ADHD is Attention-Deficit/Hyperactivity disorder as described in the DSM IV. Attention-deficit is characterized by a number of symptoms of persistent inattention to a degree that is maladaptive and inconsistent with developmental level including failure to attend to detail, inability to sustain attention, difficulty organizing tasks, and high distractability. Hyperactivity includes both hyperactivity and impulsivity, and is characterized by a number of symptoms including fidgeting, squirming, inability to stay seated, to walk rather than run, to remain quiet, or to wait for their turn when appropriate to do so. ADHD is generally first diagnosed during elementary school years when school adjustment is compromised. In most cases, symptoms attenuate during late adolescence and early adulthood although a minority will continue experiencing the full range of symptoms into mid-adulthood.

Childhood.

In this study, childhood refers to the period up to and including age 15. This age is consistent with the DSM IV criterion for APD which requires that a pervasive pattern of antisocial behaviors be evident since age 15 years and that there be evidence of CD with onset before age 15 years. In addition, age 15 does not violate the DSM IV criterion for ADHD which requires that some hyperactive-impulsive or inattentive and maladaptive symptoms be present before age 7 years, or the DSM IV criterion for CD which requires at least one criterion be present prior to age 10 years and that serious violations of rules

begin before age 13 years.

Adolescence.

In this study, adolescence refers to the period from age 16 years to and including age 17 years. This period extends from the period defined herein as childhood to the period defined herein as adulthood. While most theories of adolescence define it as a much broader age range perhaps from 13 to 20 years (Muuss, 1996; Violato & Travis, 1995), the narrow age-range definition has been adopted in the present study so as to remain consistent with DSM IV criteria of adolescence and adulthood.

Adulthood.

In this study, adulthood is the period beginning at age 18 years and continuing until death. Age 18 is consistent with DSM IV criterion which requires that for individuals over age 18 years, a diagnosis of CD can be given only if the criteria are not also met for APD, and a diagnosis of APD cannot be given to individuals under age 18 years. Age 18 does not violate the DSM IV criterion for ADHD because ADHD can extend from childhood through adolescence, and continue in adulthood.

Experiential/External Domain.

The Experiential/External Domain refers to certain influences that children experience and that are external to them that may affect personality development and specifically, APD development. Such influences may include physical abuse, sexual abuse, emotional abuse, separation and loss, poor school achievement, medical incidents, family socio-economic status, and/or family dysfunction or breakdown.

Separation and Loss.

For the purposes of this study, separation and loss refer to a child's experience of separation from, or the loss of a loved one or caregiver (Farrington, 1979; Gregory, 1965).

Poor School Achievement.

Poor school achievement refers to significantly low scores on measures of attainment based on school tests (Farrington, 1995).

Medical Incident History.

In this study, medical incident history refers to Brennan and Mednick's (1997) definition which includes early physical or biological insults such as brain damage due to prenatal and perinatal insults, or central nervous system deficits caused by postnatal insults such as head injuries, poor nutrition, physical abuse, or exposure to toxins which may contribute to antisocial outcomes.

Family Dysfunction/Breakdown.

In this study, family dysfunction/breakdown refers to ongoing relationship difficulties among family members or individuals in the child's home environment that threaten the stability of the family unit, including but not limited to witnessing abuse between parents or caregivers, parental separation or divorce (Gregory, 1965), or other experiences that impact the child's beliefs regarding the stability of the family unit.

Interpersonal Domain.

The Interpersonal Domain refers to certain experiences that children have in relation to others that may influence childhood personality development and specifically,

APD development, including neglect, rejection by a caregiver, unstable or erratic parenting, inconsistent discipline, harsh parenting, parental delinquency or criminality, delinquent or behavior problem sibling, parent disorders or pathology, and social desirability and peers.

Abuse and Neglect

Abuse and neglect is defined in accordance with the federal Child Abuse Prevention and Treatment Act of 1988, as the physical or mental injury, sexual abuse or exploitation, negligent treatment, or maltreatment of a child by a person who is responsible for the child's welfare, under circumstances which indicate that the child's health or welfare is harmed or threatened (Widom, 1997).

Emotional Abuse

Emotional abuse occurs when a parent or caregiver verbally interacts with a child or when a parent or caregiver withholds affection from the child so as to cause the child to experience intense feelings of hurt, sorrow, fear, or feelings of worthlessness.

Parental Rejection

Parental rejection herein refers to the psychological rejection of children e.g. interactions appear cold or lacking affection, by one or more parents or by a primary caregiver (Robins, 1966b).

Unstable or Erratic Parenting

In this study, unstable parenting refers to those circumstances that interfere with stable parenting, including: inter-parental conflict (Emery, 1982; Farrington & West, 1971), the absence of a parent due to illness (Farrington & West, 1971), or desertion

(Robins & Hill, 1966).

Inconsistent Parental Discipline.

Inconsistent parental discipline is as described by Patterson (1982) and includes the maintenance of few rules, a lack of discipline exercised when needed, or a lack of supervision.

Harsh Parenting.

Harsh parenting refers to a parenting style that includes a cruel attitude or is overly punitive (Farrington, 1995) or parenting that is excessively overprotective (Howard, 1981).

Parental Delinquency or Criminality

Parental delinquency or criminality refers to behaviors or crimes that go against societal norms, crimes against property, and/or crimes against other people (Cadoret, Troughton, Bagford, & Woodworth, 1990).

Delinquent or Behavior Problem Sibling

The delinquent or behavior problem sibling is one who works erratically, has a poor school record, commits arrestable offenses, drinks, fights, exhibits sexual promiscuity, or has been arrested (Robins, 1966c).

Parent Disorders/Pathology

The reference to parent disorders/pathology refers to any adult disorders or pathology as outlined in the DSM-IV.

Social Desirability/Peers.

Social desirability refers to a child having or not having the support of a social

network (Paris, 1998b) or having difficulties in getting along with contemporaries (Robins, 1966a). Peers refers to those individuals a child associates with, and their influence on the child, socially conforming or delinquent (Robins, 1966a).

Behavioral Domain

The Behavioral Domain includes antisocial behaviors of children (see "Child Antisocial Behavior" below) that may influence childhood personality development and specifically, APD development.

Child Antisocial Behavior

As defined in the DSM IV, child antisocial behavior refers to antisocial behavior in a child or adolescent that is not due to a mental disorder (e.g., Conduct Disorder or an Impulse-Control Disorder) including isolated antisocial acts of children or adolescents (not a pattern of antisocial behavior). Child antisocial behaviors include behaviors that violate the basic rights of others, behaviors that are aggressive toward people or animals including bullying, fighting, physical cruelty, and forced sexual activity, behaviors that involve the deliberate destruction of property, including fire setting, and other antisocial behaviors such as deceitfulness or stealing, including break and enter, and behaviors involving serious violation of rules and truancy.

Antisocial Personality Disorder (APD)

APD herein is Antisocial Personality Disorder as described in the DSM IV. APD is characterized by repeated antisocial behavior in a wide range of personal and social contexts. Generally individuals with APD have had a history of some symptoms of CD before age 15 years. Children with CD tend to express a repetitive and persistent pattern

of impulsive-aggressive behaviors which violate the basic rights of others or which violate major age-appropriate societal norms or rules (DSM IV). In adulthood, the impulsive-aggressive features persist and are associated with failure to sustain consistent work behavior and a general lack of concern for others which impairs social relations. About half of the APD adults have a record including felony convictions or multiple arrests for serious criminal activity other than traffic violations and they are often alcohol or drug abusers. There is also an increased risk of Somatization Disorder in some cases, especially women, (Cloninger, Bayon, & Przybeck, 1997). (For DSM-IV criteria for APD, see Appendix A.)

Retrieval of Relevant Studies

Literature Search

A number of sources were utilized for the literature search including manual and electronic journal searches, reference lists of reviews and other studies, and relevant published studies obtained from persons involved in related research. Computer searches were also conducted of the abstraction and indexing data bases (MEDLINE, PsychINFO, HealthSTAR, ERIC, Social Sciences Abstracts, Sociological Abstracts, Biological Abstracts, Child Abuse and Neglect Documents, Dissertation Abstracts, and Expanded Academic ASAP). The following keywords were used in the search: antisocial personality disorder, antisocial personality, antisocial behavior, personality development, antisocial personality development, antisocial personality etiology, psychopath, psychopathy, sociopath and sociopathy. Along with antisocial personality, the following keywords were searched: heritability, biology, parenting, personality development, and childhood

development.

Inclusion Criteria for Studies.

An initial search of the relevant data-bases using individual and combined keywords or phrases resulted in over 252,000 responses with "antisocial personality etiology" having the fewest responses (8) and "antisocial personality" combined with "biology" having the most responses (108,616).

The studies that fell within the parameters for inclusion, however, were those studies published between 1960 and 2,000 that included 10 or more subjects, that reported statistics such as means, standard deviations, F ratios, t-statistics, X^2, correlations, etc., that could be converted into an effect size (Cohen's d), and which involved the investigation of early childhood indicators of APD. Accordingly, search parameters excluded some articles that were published prior to 1960 (e.g. Carter, 1933; Hirsch, 1958; Kiev, 1936). In addition, some studies were eliminated because they were single case studies or because they had fewer than 10 subjects (e.g. Pam, Inghilterra, & Munson, 1994; Strupp, Schact, Henry, & Binder, 1992), and some studies were eliminated because they did not provide necessary statistics (e.g. Emery, 1982; Mealey, 1995). With respect to APD, many articles referring to antisocial personality were actually studying criminal or violent behavior, (Hare, 1965; Koski, & Mangold, 1993; Lynam, 1996; Rushton, 1996; Kandel, & Mednick, 1988), delinquent behavior (Hill, 1962; Rogers, Johansen, Chang, & Salekin, 1997), or other criteria (e.g. Lahey, Hartdagen, Frick, & McBurnett, 1988; Dastidar, & Kapoor, 1996). In addition, DSM criteria was not used to define APD in all studies (e.g. Bloomingdale & Bloomingdale, 1988; Cloninger, Reich, & Guze, 1975a). Of

those studies using criteria to define APD other than the DSM criteria, studies were only included if the described characteristics of APD were similar to those defined by the DSM and if definitions did not specifically include or exclude important criteria different from that indicated by the DSM. With regard to early childhood indicators, some studies examined influences on childhood development or personality development but not influences on APD development or they did not show a link between factors in childhood and the development of APD in adulthood (e.g. Laporte & Guttman, 1996; Saklofske & Eysenck, 1998). Studies were only included if the variable or variables under investigation appeared to be generally the same as a variable or variables described within the four domains of this meta-analysis as risk factors for APD development. Fifty three studies met the inclusion criteria. These studies included research in areas investigating heritable characteristics, childhood experiences and external influences, interpersonal influences including the influence of parenting, and childhood antisocial behaviors associated with development of an antisocial personality

Coding of Studies

A coding system was developed based on a review of the research literature. The coding system made it possible to translate the features of individual studies into usable quantitative data which could then be analysed to determine why findings vary from one type of study to the next. Each of the 53 studies were coded for 1) characteristics of the studies, 2) characteristics of the subjects, and 3) effect sizes across psychobiological, environmental/external, interpersonal, and behavioral domains. (The coding scheme for all data is presented in Appendix B.)

Calculating Index of Effect

Diverse data from different studies could not be compared or combined to determine an overall effect without first transforming the data into a common metric, in this case, the index of effect (Cohen's d) which indicates relative effectiveness, or magnitude of effect as well as whether effects are significantly different between groups. Each effect to be analyzed was treated as a separate case for statistical purposes. Consequently, in some studies there was only one effect size estimate, but where there are several dependent variables of interest, an effect size estimate was calculated for each. The effect size for each dependent variable was calculated from means and standard deviations, correlations, Chi-squares, t and F ratios, odds-ratios, etc. as is conventional in meta-analysis of d values (see Table 1). Effect sizes for percentages were computed from a table of probit transformations of differences in proportions to effect sizes (see Table 2).

Table 1

Formulae for Converting Study Statistics to Effect Size (d)*

Statistic to be Converted	Formula for Transformation
t	$d = \frac{2t}{\sqrt{df}}$
F	$d = \frac{2\sqrt{F}}{\sqrt{df(error)}}$
r	$d = \frac{2r}{\sqrt{1 - r^2}}$
X^2	$r = \sqrt{X^2/n}$
$\bar{x}$	$d = \frac{\bar{x}_e - \bar{x}_c}{S_c}$

Note * Adapted from Wolf (1986), p.35

Table 2

Effect Size for Percentage of Contribution to APD based on Probit Transformations*

Percent	Effect Size (d)
03-04	0.00
05-07	0.10
08-09	0.20
10-12	0.29
13-14	0.37
15-17	0.45
18-19	0.52
20-22	0.59
23-24	0.65
25-27	0.72
28-29	0.79
30-32	0.85
33-34	0.91
35-37	0.98
38-39	1.04
40-42	1.11
43-44	1.17
45-47	1.29
48-49	1.29
50-52	1.36
53-54	1.43
55-57	1.50
58-59	1.56
60-62	1.64
63-64	1.71
65-67	1.80
68-69	1.88
70-72	1.95
73-74	2.02
75-77	2.09
78-79	2.16
80-82	2.23
83-84	2.30
85-87	2.37
88-89	2.44
90-92	2.51
93-94	2.58
95-97	2.65
98-99	2.72

Note. * Adapted From Glass, McGaw, and Smith (1981), p. 139.

Conducting Statistical Analyses of Effects

Research results were analysed with respect to the calculated effect sizes and statistical significance. Knowing whether a mean effect is significant is not usually as important as its knowing its magnitude and whether effects from different groups of studies differ from each other (Durlak, 1995). Thus, analysis involved comparing and combining studies to determine whether they differed significantly with respect to effect sizes or significance levels. The analysis of the data involved three major components: 1) descriptive statistics for study and subject characteristics; 2) unweighted and weighted effect size analyses of the total aggregate of studies sampled, and each of the psychobiological, experiential/external, interpersonal, and behavioral domains; and 3) analyses of variance for potential moderators of effect size.

Dependent Variable Analysis.

Using the same methods previously described, both unweighted and weighted effect sizes were calculated for the total sample. The dependent variables were combined to form the four domains (i.e., psychobiological, environmental/external, interpersonal, and behavioral) which were then treated as dependent variables and unweighted and weighted mean effect sizes were computed for each domain. The psychobiological domain included measures of effect for genetics, temperament, intelligence, and disorders/pathology occurring during childhood. Measures of effect in the environmental/external domain included effect sizes for physical abuse, sexual abuse, emotional abuse, separation and loss, poor school achievement, medical incidents, family socio-economic status, and family dysfunction. The interpersonal domain included

measures of effect for such variables as neglect, parental rejection, unstable or erratic parenting, inconsistent discipline, harsh parenting, parental delinquency or criminality, delinquent or behavior problem sibling, parent disorders or pathology, and social desirability/peers while the behavioral domain included only one variable, child antisocial behavior, and therefore, only one measure of effect. A summary of coding results for study characteristics and effect sizes within the four domains for each study are presented in Table 3. The full bibliographic details of these studies are presented in the reference section of this dissertation and are identified by an asterisk (*).

Table 3

Study Characteristics and Effect Sizes for Four Domains

Author	Yr	Sample Size	Psycho-Biological Domain	Experiential External Domain	Interpersonal Domain	Behavioral Domain
Rydelius	83	100	1.04	.	0.66	0.70
Crowe	75	104	.	.	0.04	.
Virkkunen	83	23	1.04			
Gottesman	63	136	1.39	.	.	.
Hesselbrock et al	86	321	0.52	.	.	.
Cloninger et al	78	274	0.94	.	.	
Blackburn & Cold	99	164	2.64	.	.	0.04
Rutter et al	94	104	0.85	.	.	
Farrington	95	411	0.10	.	.	
Robins	66c	429	1.57	0.65	0.85	0.79
Robins	66b	872	.	1.70	3.98	
Howard	81	100	2.01	4.48	0.20	0.45
Vaillant	94	912	0.40	.	0.29	.
Lyons et al	95	6452	1.03	.	.	0.37
Cadoret	78	246	0.41	.	0.68	
Reich	86	82	.	0.74	.	
Lewis & Bucholz	91	2572	0.37	.	0.36	.
Biederman et al	97	260	0.29	.	.	.
Barratt et al	97	101	1.42	-0.33	.	.
Brown et al	96	166	1.29	.	.	
Farrington	91	411	.	.	.	1.64
Caudill et al	94	299	.	.	0.65	.
Dinwiddie	97	5520	.		.	0.98
Cloninger et al	75b	1027	2.08	.	.	.

(continues)

Table 3 *(continued)*

Study Characteristics and Effect Sizes for Four Domains

Author	Yr	Sample Size	Psycho-Biological Domain	Experiential External Domain	Interpersonal Domain	Behavioral Domain
Caspi et al	96	1037	.	.	0.20	.
Cadoret et al	95	95	0.10	.	0.43	0.22
Cadoret et al	90	286	.	0.84	0.47	0.98
Robins et al	91	116	0.29	.	.	0.79
Hesselbrock	86	321	1.11	.	.	1.04
Farrington	96	411	0.51	0.91	1.08	0.25
Rey et al	97	145	.	.	0.10	1.43
Norden et al	95	90	.	0.45	.	.
Downey et al	97	78	0.29	.	.	.
Kendler et al	97	5877	0.10	.	.	.
Zanarini et al	90	103	0.45	1.24	0.20	.
Robins	66a	624	1.80	2.73	1.30	0.79
Luntz & Widom	94	699	.	.	0.18	.
Neugebauer et al	99	100543	.	0.29	.	.
Gottesman	94	490	0.09	.	.	.
O'Neal et al	62	325	1.64	2.30	3.64	1.11
Rey et al	95	145	0.10	.	.	.
Waldstein et al	96	106	1.06	0.10	.	0.65
Cloninger et al	75a	387	0.72	2.98	2.48	.
Crowe	74	104	.	1.62	0.10	.
Zoccolillo et al	92	254	1.04	.	.	.
Zanarini et al	89	105	.	6.90	2.01	.
Myers et al	95	30	1.64	.	.	0.85
Vitelli	97	180	.	0.42	0.16	0.19

(continues)

Table 3 *(continued)*

Study Characteristics and Effect Sizes for Four Domains

Author	Yr	Sample Size	Psycho-Biological Domain	Experiential External Domain	Interpersonal Domain	Behavioral Domain
Weiler & Widom	96	1069	.	.	0.41	.
Klintberg et al	90	161	1.08	.	.	0.80
Moss et al	95	73	.	.	0.85	
Mutzell	94	411	.	.	0.37	
Bloomingdale et al	88	182	1.83	.	.	0.91

Note. Abbreviations in column title: Yr is the year of publication of the study.

Independent Variable Analysis

The independent variable analysis assesses the effects of potential moderators. Procedural variables can influence dependent variables, that is, they can moderate effect sizes. A moderator variable is any variable that influences differences in the association between two other variables (Hunter, Schmidt, & Jackson, 1982). In this study, the independent variables consist of seven factors which were classified according to a coding scheme under the following categories: year of study, country of study, study type, study design, average age of subjects, diagnostic indices, and methods of assessing APD. When variables are categorical, group mean differences can be examined and compared by dividing the total sample of studies into two or more subgroups that differ on certain variables believed to be important (Durlak, 1995). Transformation of the coded independent variables thought to be potential moderators resulted in the following categorical divisions:

i) year of study was transformed into two categories, studies published before 1990 vs studies published during the 1990's because of potential differences that may exist due to differences in the nature of populations used in the research, or because of cultural or societal changes that have occurred over the years;

ii) country of study was transformed into two categories, USA vs other countries. The majority of studies in this meta-analysis were conducted in the USA which may influence overall findings. Potential differences in the cultural perspectives or methodologies used by researchers in other

countries may have a moderating effect thereby producing different findings from those obtained in the USA;

iii) study type was transformed into two categories, retrospective vs prospective studies to test the possibility that retrospective studies may provide less accurate findings because they rely on the memory of research subjects;

iv) study design was transformed into two categories, experimental and quasi-experimental vs other designs, because it has been argued that well-designed studies may produce different results than studies using less stringent designs (Glass, 1976). Generally, experimental and quasi-experimental studies exercise more stringent control over possible confounding variables than do correlational or survey type studies.

v) diagnostic indices was transformed into two categories, interview or questionnaire vs other indices. The majority of studies in this meta-analysis based their findings on data collected by way of interview or questionnaire which may produce different results than studies using more standardized procedures, tests, or other indices; and

vi) methods of assessing APD was transformed into two categories, DSM vs not DSM. Methods of assessment may moderate effects through the inclusion or exclusion of subjects. Because of the DSM's emphasis on behavioral characteristics for APD, methods based on the DSM may result in subject samples that are different from those assessed using other

methods that do not adhere to DSM criteria.

Analyses of variance were conducted for each independent variable to investigate potential moderating effects.

CHAPTER IV

RESULTS

The results are presented in four main sections: 1) descriptive analyses of subject and study characteristics, 2) descriptive analysis of dependent variables, 3) effect size analysis of weighted and unweighted indicators of APD development including indicators in the psychobiological, experiential/external, interpersonal, and behavioral domains, as well as the total aggregate, and (4) analysis of the influence of moderating variables on effect size for each domain.

Descriptive Analysis of Subject and Study Characteristics

The present meta-analysis included 53 published articles that met the criteria for inclusion. As described in Table 4, these articles included a total of 135,533 subjects (minimum = 23, maximum = 100,543). Of the total sample, 8,404 were female (6.9%) and 118,451 were male (87.4%). Of the studies wherein age was reported, subjects ranged in age from 8 to 47 years with approximately 17% of subjects being under 18 years of age, and 81% being 18 years of age or over. Of the 53 studies in this meta-analysis, 35 (66%) were conducted in the United States, six (11%) were conducted in England, three (5.7%) were conducted in Sweden, two (3.8%) were conducted in Australia and one (1.9%) study was conducted in each of Canada, Finland, Netherlands, and New Zealand. In those studies where ethnicity was reported, Caucasians accounted for 13.5% of subjects, Blacks accounted for 2.3% of subjects, Hispanics made up 0.4% of subjects, and other ethnicities accounted for 0.6% of the subjects in the total sample. As noted in Table 4, not all studies included reports on every variable. In this regard, gender was not

reported in nine studies, age was not reported in 12 studies, ethnicity was not reported in 24 studies, and the country in which the study was conducted was not reported in three studies. No studies included information regarding family structure or number of siblings.

As illustrated in Table 4, the range of publication dates of the studies was from 1962 to 1999 with the majority of studies being published during the 1990's (n=33)(62.3%). Altogether, the 53 research articles included a total of 97 units of analysis with effect sizes being distributed among the childhood indicator domains. Specifically, there were 35 articles regarding psychobiological influences accounting for 36.1% of effects reported. Seventeen articles referred to childhood experiences or external influences which accounted for 17.5% of effects reported. Twenty five articles were about research on interpersonal influences accounting for 25.8% of effects, and 20 articles referred to research on the effects of behavior on APD development which accounted for 20.6% of the effects reported in all domains.

As elaborated in Table 4, the majority of studies were retrospective (60.4%), and quasi-experimental (84.9%) in design. There were no experimental design studies and only 49.1% of studies included comparison groups. In one study, groups were not reported. Most researchers used either DSM assessment criteria (49.1%) or their own criteria (35.8%) to assess APD. The diagnostic indices most commonly used by researchers were interviews (39.6%) or questionnaires (35.8%).

Table 4

Descriptive Characteristics of the Studies in the Sample

Variable	N	Percent
1. Year of Study		
1962 - 1965	6	11.32
1974 - 1978	6	11.32
1981 - 1989	8	15.1
1990 - 1999	33	62.3
2. Country of Study		
New Zealand	1	1.9
Canada	1	1.9
Finland	1	1.9
Netherlands	1	1.9
Australia	2	3.8
Sweden	3	5.7
England	6	11.3
USA	35	66.0
Not Reported	3	5.7
3. Study Type		
Cross-sectional	6	11.3
Prospective	15	28.3
Retrospective	32	60.4
4. Study Design		
Observation	0	0
Correlation	7	13.2
Unobtrusive	1	1.9
Experimental Between Group	0	0
Experimental Within Subject	0	0
Experimental Complex Interaction	0	0
Quasi-experimental	45	84.9
5. Study Groups		
Clinical	52	98.1
Comparison	26	49.1
Experimental	0	0
Control	0	0
Not Reported	1	1.9

(continues)

Table *(continued)*

Variable	N	Percent
6. Studies Including Average Age of Sample		
8 - 17	6	11.3
18 - 47	35	66.0
Not Reported	12	22.6
7. Studies Including Gender		
Females	39	56.6
Males	44	73.6
Not Reported	9	17.0
8. Gender		
Females	8,404	6.2
Males	118,451	87.4
Not Reported	8,678	6.4
Total Subjects	135,533	
9 Studies Including Ethnicity		
Caucasian	29	54.7
Black	24	45.3
Hispanic	22	41.5
Other	28	52.8
10. Ethnicity		
Caucasian	18,245	13.5
Black	3,189	2.3
Hispanic	505	.4
Other	873	.6
Not Reported	112,721	83.2
11. DSM Assessment Criteria	26	49.1
Researcher Criteria	19	35.8
Psychometric Assessment Criteria	7	13.2
Mixed Assessment Criteria	1	1.9

(continues)

Table *(continued)*

Variable	N	Percent
12. Studies Including Diagnostic Indices		
Observation	1	1.9
Interviews	21	39.6
Questionnaires	19	35.8
Rating Scales	0	0
Standardized Tests	9	17.0
Physiological Measures	1	1.9
Other	1	1.9
Mixed	1	1.9
13. Number of Studies Per Domain		
Psychobiological	35	36.1
Experiential/External Influence	17	17.5
Interpersonal	25	25.8
Behavioral	20	20.6

* Total N is more than 53 and total percent is more than 100 because some studies included data on more than one variable.

Descriptive Analysis of Dependent Variables

As shown in Table 5, certain variables within domains were researched more than others. In the psychobiological domain, for example, only three out of 35 studies included IQ while 10 studies included genetics. In the experiential/external domain, the effects of separation and loss were reported in only 10 out of 17 studies. Sexual abuse and emotional abuse, on the other hand, were each represented by only two studies. Of 25 studies in the interpersonal domain, 15 included the effects of parent disorders while the effects of unstable or erratic parenting on APD development were reported in only one study. The only dependent variable researched in the behavioral domain was child antisocial behavior but 20 studies included this variable accounting for 37.7% of the studies in this meta-analysis

Table 5

Descriptive Analysis of Dependent Variables

Variable	N of Studies	Percent
Psychobiological Domain		
Genetics	10	18.86
Temperament	9	17.0
IQ	3	5.7
Disorders/Pathology during Childhood	19	35.8
Experiential/External Influences Domain		
Abuse		
Physical	5	9.4
Sexual	2	3.8
Emotional	2	3.8
Other Experiential/External Influences		
Separation and Loss	10	18.9
Poor School Attainment or Early Dropout	5	9.4
Medical Incident History	3	5.7
Socio-Economic Status	7	13.2
Family Dysfunction/Breakdown	8	15.1
Interpersonal Domain		
Neglect		
Rejecting	7	13.2
Unstable or Erratic	3	5.7
Inconsistent Discipline	1	1.9
Harsh	3	5.7
Parent Delinquency/Criminality	2	3.8
Delinquent or Behavior Problem Sibling	6	11.3
Parent Disorder/Pathology	2	3.8
Social Desirability/Peers	15	28.3
	7	13.2
Behavioral Domain		
Child Antisocial Behavior	20	37.7

* Total N is more than 53 and total percent is more than 100 because some studies included data on more than one domain.

Descriptive Analysis of Moderator Variables

As illustrated in Table 6, a larger percentage of studies were conducted between 1990 and 1999. Specifically, 57.1% of studies in the psychobiological domain, 47.1% of studies in the experiential/external domain, 56% of studies in the Interpersonal domain, and 65% of studies in the behavioral domain were conducted between 1990 and 1999.

In all domains, the most common study type was retrospective (59.5% - 76.2%) and the most common study design was quasi-experimental (73.8% - 81.0%). Across domains, most researchers chose interviews or questionnaires over other diagnostic indices (65.2% to 76.2%) and chose assessment criteria other than DSM criteria (54.3% - 55.0%)

Table 6

Descriptive Analysis of Moderator Variables

Variable	N	Percent
1. Year of Study		
Psychobiological		
1962 - 1989	15	42.9
1990 - 1999	20	57.1
Experiential/External Influences		
1962 - 1989	9	52.9
1990 - 1999	8	47.1
Interpersonal		
1962 - 1989	11	44.0
1990 - 1999	14	56.0
Behavioral		
1962 - 1989	7	35.0
1990 - 1999	13	65.0
2. Effect Sizes by Study Type		
Psychobiological		
Retrospective	32	76.2
Prospective	9	21.4
Experiential/External Influences		
Retrospective	24	59.5
Prospective	11	26.2
Interpersonal		
Retrospective	29	63.0
Prospective	13	28.3
Behavioral		
Retrospective	13	65.0
Prospective	7	35.0

(continues)

Table 6 *(continued)*

Variable	N	Percent
3. Effect Sizes by Study Design		
Psychobiological		
Quasi-experimental	34	81.0
Other Designs	7	16.7
Experiential/External Influences		
Quasi-experimental	31	73.8
Other Designs	7	16.7
Interpersonal		
Quasi-experimental	34	73.9
Other Designs	8	17.4
Behavioral		
Quasi-experimental	16	80.0
Other Designs	4	20.0
4. Effect Sizes by Diagnostic Indices		
Psychobiological		
Interviews or Questionnaires	30	71.4
Other Diagnostic Indices	11	26.2
Experiential/External Influences		
Interviews or Questionnaires	32	76.2
Other Diagnostic Indices	7	16 7
Interpersonal		
Interviews or Questionnaires	30	65 2
Other Diagnostic Indices	12	26.1
Behavioral		
Interviews or Questionnaires	15	75.0
Other Diagnostic Indices	5	25.0

(continues)

Table 6 *(continued)*

Variable	N	Percent
5. Effect Sizes by Methods of Assessing APD		
Psychobiological		
DSM Criteria	16	42.9
Other Criteria	19	54.8
Experiential/External Influences		
DSM Criteria	8	35.7
Other Criteria	9	54.8
Interpersonal		
DSM Criteria	9	21.7
Other Criteria	16	54.3
Behavioral		
DSM Criteria	9	45.0
Other Criteria	11	55.0

* Total N is more than 53 and total percent is more than 100 where studies included data on more than one domain.

** Total N is less than 53 and total percent is less than 100% where studies did not include all variables.

Effect Size Analysis for Total Aggregate

Unweighted Effect Size Analysis

The effect sizes were analysed across all four domains (psychobiological, experiential/external, interpersonal, and behavioral) and these results are summarized in Table 7. The mean effect sizes across the four domains ranged from a minimum absolute value of 0.75 for the behavioral domain to a maximum of 1.65 for the experiential/experiential domain. Positive effect sizes in this analysis indicate deleterious developmental outcomes. A negative mean effect size d did not emerge within or across the four domains indicating each of the domains and the variables within them are potential risk factors in the development of APD. The overall mean ds for each of the domains were based on large sample sizes (psychobiological n = 29,766, experiential/external n = 111,527, interpersonal n = 20,531, and behavioral n = 17,919). The number of studies for the computation of d for each domain varied from a minimum of 17 for the experiential/external domain to a maximum of 35 for the psychobiological domain.

To facilitate the interpretation of d, Table 7 also contains a presentation of the 95% confidence intervals. The 95% confidence intervals indicate that all of the effect sizes are non-zero (i.e., greater than zero).

Weighted Effect Size Analysis

Studies in a MA differ from one another in both methodological and substantive ways (Shadish & Haddock, 1994). Larger samples, for example, are thought to have a smaller variance and, therefore, are likely to be more precise estimates of effect size.

Thus, it may be that small sample size studies exert more influence on results than warranted, particularly if these studies included results inconsistent with most studies in the meta-analysis. In order to justify combining the 53 studies analyzed in this MA, it was necessary to employ a procedure that would take these differences into account. Hedges (1982) has developed an unbiased estimator of effect size that minimizes variance (see also Rosenthal & Rubin, 1982). Shadish and Haddock referred to Hedges' estimator as a quality rating and added that it is the only standardized weighting scale for studies to date. Accordingly, Hedges' quality-weighted estimate was employed to compute the weighted average effect sizes in the present study. The formula for computing the weighted estimate of effect, which is analogous to Hedges' but uses the standard normal deviate rather than raw data, has been described by Wolf (1986):

$$\text{weighted } Z = \frac{\sum df\,Z}{\sqrt{\sum df^2}}$$

wherein the standard normal deviate (Z) associated with each statistic is weighted by using the degrees of freedom (df) associated with the sample on which it is based. This computation was applied in the present study to derive the weighted effect sizes.

As shown in Table 7, the weighted mean effect sizes across the four domains ranged from a minimum absolute value of 0.32 for the experiential/external domain to a maximum of 0.67 for the behavioral domain. As in the unweighted effect size analysis, the weighted effect size analysis produced only positive mean ds across the four domains. All mean effect sizes decreased for weighted effect size analyses. Specifically, psychobiological decreased from .95 to .65, experiential/external decreased from 1.65 to

.32, interpersonal decreased from .87 to .63 and behavioral decreased from .75 to .67. The 95% confidence intervals indicate non-zero weighted effect sizes for all domains. Compared to unweighted effect size confidence intervals, the weighted effect size 95% confidence intervals are much narrower, as expected given the weighted correction in error of estimate. The results suggest all four domains have an effect on APD development.

Table 7

Unweighted and Weighted Effect Sizes (d) for Total Aggregate of Childhood Indicators of Developing APD

Dependent Variables**	N Studies	N Subjects	Unweighted Effect Size (d)			Weighted Effect Size (d)		
			Mean d	95% CI*		Mean	95% CI*	
				Lower	Upper	d	Lower	Upper
PSYCHOBIOLOGICAL	35	29766	0.95	0.72	1.18	0.65	0.63	0.66
EXPERIENTIAL/EXTERNAL	17	111527	1.65	0.71	2.59	0.32	0.31	0.33
INTERPERSONAL	25	20531	0.87	0.43	1.31	0.63	0.61	0.65
BEHAVIORAL	20	17919	0.75	0.56	0.94	0.67	0.66	0.69

Note. * 95% CI = 95% confidence interval

** Total N of studies is more than 53 because some studies included data on more than one domain.

Effect Size Analysis for Research Aggregates by Domain

The psychobiological domain was represented by 35 published studies which involved 29,766 subjects. The experiential/external domain was represented by 17 published studies involving 111,527 subjects. The interpersonal domain consisted of 25 published studies involving altogether 20,531 subjects while the behavioral domain was made up of 20 studies that included 17,919 subjects overall. The effect size analyses were conducted with the same procedures as were employed for the analysis of the total research aggregate.

Psychobiological Research Aggregate: Unweighted Effect Size Analysis

The results of this analysis are summarized in Table 8. Mean effect sizes ranged from a minimum value of .39 for IQ to a maximum of .98 for temperament. Mean ds for each variable were based on large samples (genetics n = 15,291; temperament n = 4,415, IQ n = 956; disorders/pathology during childhood n = 9,104). The number of studies for the computation of each d varied from a minimum of 3 studies to a maximum of 19 studies. A negative d (indicating positive outcomes for personality development) did not emerge across variables. Positive effect sizes indicate deleterious outcomes for personality development were associated with all variables. Based on the mean ds of variables within the psychobiological domain, IQ had the least effect while temperament had the strongest effect on APD development.

Psychobiological Research Aggregate: Weighted Effect Size Analysis

As shown in Table 8, results of the weighted effect size analysis are similar to results of the unweighted effect size analysis for the Psychobiological domain. Although

the weighted correction in error of estimate resulted in a decrease in mean effect size for all variables except IQ, which increased from an unweighted effect size of .39 to a weighted effect size of .41 the relative contribution of each variable remained consistent with the unweighted results. As indicated by the unweighted effect size analysis, the weighted mean ds also indicate IQ had the least effect on APD development while temperament had the strongest effect.

Experiential/External Research Aggregate: Unweighted Effect Size Analysis

Mean effect sizes in the experiential/external domain, as described in Table 9, ranged from a minimum value of .14 for sexual abuse to a maximum of 1.86 for emotional abuse. Mean ds for each variable were based on samples ranging from 205 for emotional abuse to 101,078 for medical incident history. The number of studies for the computation of each d varied from a minimum of 2 studies to a maximum of 10 studies. A negative d (indicating positive outcomes for personality development) did not emerge across variables indicating all variables were associated with deleterious outcomes for personality development. Based on the mean ds of variables within the experiential/external domain, sexual abuse had the least effect on APD development while emotional abuse had the strongest effect.

Experiential/External Research Aggregate: Weighted Effect Size Analysis

Results of the weighted effect size analysis are shown in Table 9. As expected, results of the weighted effect size analysis for the experiential/external domain resulted in a decrease in the values of the mean ds for most variables compared to their respective unweighted effect size values. The mean ds increased, however, for poor school

achievement (from .47 to .75) and medical incident history (from .28 to .29) while sexual abuse remained the same at .14 after correcting for error of estimate. In addition, the weighted correction in error of estimate resulted in a change in relative contribution of each variable compared to their relative contribution as indicated by the unweighted results. The unweighted effect size indicated emotional abuse had the strongest effect followed by separation and loss, family dysfunction, and family socio-economic status. The weighted effect size analysis also found emotional abuse had the strongest effect but the next strongest effect was family dysfunction followed by separation and loss, and poor school achievement.

Interpersonal Research Aggregate: Unweighted Effect Size Analysis

As illustrated in Table 10, effects sizes in the interpersonal domain ranged from .30 for unstable or erratic parenting to .59 for parental rejection and for inconsistent discipline. Mean ds for each variable were based on samples ranging from 411 for unstable or erratic parenting to 7,098 for parent disorders/pathology. The number of studies for the computation of each d varied from one (unstable or erratic parenting) to 15 (parent disorders/pathology) studies. A negative d (indicating positive outcomes for personality development) did not emerge across any of the variables indicating all variables were associated with negative outcomes for personality development. Based on the mean ds of variables within this domain, unstable or erratic parenting had the least effect on APD development while parental rejection and inconsistent discipline had the strongest and equal effects.

Interpersonal Research Aggregate: Weighted Effect Size Analysis

As shown in Table 10, results of the weighted effect size analysis were the same as the results of the unweighted effect size analysis for neglect, unstable or erratic parenting, harsh parenting, and parent delinquency or criminality. The weighted correction in error of estimate resulted in an increase in effect size from .40 to .47 for delinquent sibling, and from .45 to .53 for social desirability. For all other variables, the weighted correction in error of estimated resulted in a decrease in mean effect size. The relative contribution of effect for the variables differed between weighted and unweighted results. The unweighted results suggested that parental rejection (59) and inconsistent discipline (59) had the most effect on APD development followed by parental disorders/pathology (58), while the weighted mean ds indicated parental rejection (53) and social desirability (53) had the most effect on APD development followed by parent disorders/pathology (.49). The unweighted and weighted analyses were in agreement with respect to the contributors having the least effect, that is, unstable or erratic parenting (.30), harsh parenting (.40), and parent delinquency or criminality (.44).

Behavioral Research Aggregate: Unweighted Effect Size Analysis

The results of this analysis are summarized in Table 11. The mean d for child antisocial behavior was based on a large sample of 17,919 subjects and computation of d was based on 20 studies. The mean d for child antisocial behavior was positive (.75) indicating deleterious outcomes for personality development associated with this variable

Behavioral Research Aggregate: Weighted Effect Size Analysis

As shown in Table 11, the weighted correction in error of estimate for the

behavioral domain resulted in a decrease in effect size for child antisocial behavior from .75 to .67. With only one variable in this domain, the relative contribution of variables is not relevant. Both the unweighted effect size analysis and the weighted effect size analysis produce a positive mean d suggesting that child antisocial behavior is associated with development of APD in adulthood.

Table 8

Unweighted and Weighted Effect Sizes (d) for Psychobiological Indicators of Developing APD

Dependent Variables**	N Studies	N Subjects	Unweighted Effect Size (d)			Weighted Effect Size (d)		
			Mean d	95% CI*		Mean d	95% CI*	
				Lower	Upper		Lower	Upper
PSYCHOBIOLOGICAL	35	29766	0.95	0.72	1.18	0.65	0.63	0.66
Genetics	10	15291	0.64	0.3	0.98	0.58	0.56	0.6
Temperament	9	4415	0.98	0.53	1.42	0.88	0.84	0.93
IQ	3	956	0.39	-4.03	0.82	0.41	0.35	0.48
Disorders/Pathology during Childhood	19	9104	0.89	0.57	1.21	0.58	0.55	0.61

Note. * 95% CI = 95% confidence interval

** Total N of studies is more than 35 because some studies included data on more than one domain.

Table 9

Unweighted and Weighted Effect Sizes (d) for Experiential/External Indicators of Developing APD

Dependent Variables**	N Studies	N Subjects	Unweighted Effect Size (d)			Weighted Effect Size (d)		
			Mean d	95% CI*		Mean d	95% CI*	
				Lower	Upper		Lower	Upper
EXPERIENTIAL/EXTERNAL	17	111527	1.65	0.71	2.59	0.32	0.31	0.33
Physical Abuse	5	1087	0.41	0.12	0.7	0.36	0.29	0.43
Sexual Abuse	2	285	0.14	0.31	0.58	0.14	0.01	0.13
Emotional Abuse	2	205	1.86	-10.97	14.69	1.53	1.37	1.7
Separation and Loss	10	3288	0.95	0.42	1.49	0.78	0.74	0.82
Poor School	5	1358	0.47	-0.86	1.8	0.75	0.68	0.82
Medical Incidents	3	101078	0.28	-0.65	1.21	0.29	0.28	0.3
Family SES	7	1958	0.69	0.18	1.19	0.48	0.43	0.53
Family Dysfunction	8	2271	0.93	0.43	1.43	0.83	0.78	0.88

Note. * 95% CI = 95% confidence interval

** Total N of studies is more than 17 because some studies included data on more than one domain.

Table 10

Unweighted and Weighted Effect Sizes (d) for Interpersonal Indicators of Developing APD

Dependent Variables**	N Studies	N Subjects	Unweighted Effect Size (d)			Weighted Effect Size (d)		
			Mean d	95% CI*		Mean d	95% CI*	
				Lower	Upper		Lower	Upper
INTERPERSONAL	25	20531	0.87	0.43	1.31	0.63	0.61	0.65
Neglect	7	3240	0.48	0.25	0.7	0.48	0.45	0.52
Rejecting	3	1259	0.59	-1.13	2.3	0.53	0.47	0.59
Unstable or Erratic	1	411	0.3	0.3	0.3	0.3	0.2	0.4
Inconsistent Discipline	3	2234	0.59	-0.26	1.44	0.48	0.43	0.52
Harsh Parenting	2	736	0.4	-2.78	3.58	0.4	0.32	0.48
Parent Delinquency	6	2136	0.44	0.02	0.87	0.44	0.4	0.49
Delinquent Sibling	2	1283	0.4	-2.01	2.81	0.47	0.41	0.52
Parent Disorders/Pathology	15	7098	0.58	0.34	0.82	0.49	0.46	0.52
Social Desirability	7	2134	0.45	0.16	0.74	0.53	0.47	0.58

Note. * 95% CI = 95% confidence interval

** Total N of studies is more than 25 because some studies included data on more than one domain

Table 11

Unweighted and Weighted Effect Sizes (d) for Behavioral Indicators of Developing APD

Dependent Variables	N Studies	N Subjects	Unweighted Effect Size (d)			Weighted Effect Size (d)		
			Mean d	95% CI*		Mean	95% CI*	
				Lower	Upper	d	Lower	Upper
BEHAVIORAL	20	17919	0.75	0.56	0.94	0.67	0.66	0.69
Child Antisocial Behavior	20	17919	0.75	0.56	0.94	0.67	0.66	0.69

Note. * 95% CI = 95% confidence interval

Study Characteristics and Effect Size

Analysis of Moderating Variables

To determine whether the identified independent variables potentially moderate the effect sizes of different domains, the independent variables were analysed one at a time using univariate analyses[1]. Accordingly, analyses of variance were conducted for five independent variables with risk factors as dependent variables within the four domains. It was not necessary to use post hoc multiple range tests to test for significant differences between levels of the independent variables as each independent variable had only two levels. See Table 12 for a summary of results for each analysis of variance.

Psychobiological Domain

Year of study had a moderating effect on the unweighted effect size in the psychobiological domain. Studies published prior to 1990 produced a significantly higher effect size than studies published in 1990 and thereafter $F(1, 33) = 4.44$, $p < .05$. In addition, methods of assessing APD also had a moderating effect on the unweighted effect size in this domain. Specifically, studies involving methods other than those based on the DSM produced a significantly higher effect size than studies involving methods based on the DSM $F(1, 33) = 3.88$, $p < .05$.

[1] Ideally, the moderator effects on dependent variables should be analyzed with multivariate analyses of variance (MANOVA) where multiple dependent variables are used. Such analyses require larger sample sizes than the present ones, however. Accordingly, it was not possible in the present study to utilize MANOVA procedures as there were 22 dependent variables overall and sometimes with only 2 or 3 studies.

Experiential/External Domain

In the experiential/external domain, the unweighted effect size was moderated by year of study and no other variable, with studies published prior to 1990 producing a significantly higher effect size than those published in the 1990's $F(1, 15) = 9.22$, $p < .01$.

Interpersonal Domain

Similarly, in the interpersonal domain, year of study was the only variable having a moderating effect on the unweighted effect size. Again, studies published before 1990 produced significantly higher effect sizes than studies published during or after 1990 $F(1, 23) = 7.44$, $p < .05$.

Behavioral Domain

In the behavioral domain, none of the seven independent variables had a moderating effect on the unweighted effect size for this domain.

Table 12

Analysis of Variance of Variables Potentially Affecting Effect Sizes in the Four Domains

Independent Variable	Psychobiological		Experiential/External		Interpersonal		Behavioral	
Clusters***	A	B	A	B	A	B	A	B
Year of Study	1.21*	0.75	2.68**	0.49	1.45*	0.41	0.83	0.71
Study Type	0.7	1.02	1.26	1.86	1.15	0.78	0.93	0.65
Study Design	0.93	1.1	1.7	1.39	1.03	2.1	0.74	0.79
Diagnostic Indices	0.94	0.99	1.77	1.09	0.72	1.64	0.75	0.74
Methods Assessing APD	0.72	1.15*	1.38	1.89	0.61	1.47	0.8	0.7

Note. * $p < .05$
** $p < .01$
*** For Year of Study, A = 1962 - 1989 and B = 1990 - 1999.
For Study Type, A = Retrospective and B = Prospective.
For Study Design, A = Quasi-Experimental and B = Other Designs.
For Diagnostic Indices, A = Interview or Questionnaire and B = Other Indices.
For Methods of Assessing APD, A = DSM and B = Other Methods

CHAPTER V

DISCUSSION

The major findings of the present study may be summarized as follows: 1) for the total aggregate of childhood indicators of developing APD, there were negative outcomes in all four domains across both the unweighted and weighted effect size analyses, 2) results indicate two moderators had a significant impact on the psychobiological domain (year of study and methods of assessment), one moderator (year of study) had a significant impact on the experiential/external and interpersonal domains, and no moderator variable had a significant impact on the behavioral domain, 3) in the psychobiological domain, both unweighted and weighted effect sizes indicate that IQ contributed least to development of APD and that temperament contributed most, 4) in the experiential/external domain, unweighted and weighted effect sizes attribute the least influence on APD development to medical incident history and the most influence to emotional abuse, 5) for the interpersonal domain, unstable or erratic parenting contributed least and social desirability and rejection by the caregiver contributed most to APD development as indicated by both the unweighted and weighted effect sizes, and, 6) in the behavioral domain, the unweighted and weighted effect size analyses indicate that child antisocial behavior contributes significantly to development of APD. Comparing effect sizes across the most significant contributors from each domain, the greatest contributor to APD development is emotional abuse by the child's caregiver (1.53). The next highest contributors were child's temperament (.88), child's antisocial behavior (.67), and both social desirability (.53) and rejection by the caregiver (.53). Initially, these findings suggest that interpersonal

experiences, and in particular, with caregivers, likely have more impact on a child's later development of APD, than do characteristics of the child, the child's behavior, or external influences, including the influence of peers.

Interpreting the Data

The magnitude of effect size was used as a basis for comparison (Dilk & Bond, 1996; Durlak, 1995). The magnitude of an effect size is not necessarily related to its practical importance. Relatively small effects may have considerable practical significance under certain circumstances while large effects may have little practical significance. Given that only 2% of the general population develop APD (DSM IV, 1994), one might question the importance of even moderate effect sizes when interpreting the results of the analyses in this study.

Interpretation of the Total Aggregate Effect Size Analysis

As seen in Table 7, effect sizes demonstrate a consistent direction across unweighted and weighted effect size analyses for all domains (psychobiological, experiential/external, interpersonal, and behavioral). Magnitude of effect was also consistent across unweighted and weighted effect size analyses for the psychobiological (0.95 vs 0.65), interpersonal (0.87 vs 0.63), and behavioral (0.75 vs 0.67) domains but the unweighted and weighted effect sizes for the experiential/external domain differed significantly in terms of the magnitude of effect (1.65 vs .32). Also in the experiential/external domain, the magnitude of the unweighted effect size was more and the magnitude of the weighted effect size was less than the effect sizes in the other domains. The large difference in unweighted and weighted effect sizes in the

experiential/external domain may be accounted for by the large sample (111,527) that makes up this domain.

Interpretation of the Effect Size Analysis for the Four Domains

As indicated in Table 7, the magnitude of effects for the psychobiological, interpersonal, and behavioral domains showed weighted effect sizes ranging from .63 to 67 while the magnitude of effect for the experiential/external domain showed a weighted effect size of (.32).

Psychobiological Domain

The psychobiological domain looks at specific heritable biological factors including genetics, temperament, IQ, and disorders/pathology occurring during childhood. As shown in Table 8, the effect sizes for all of these variables were non-zero. The weighted effect sizes for each variable in this domain can be ordered in terms of their relative importance. The smallest effect size was .41 for IQ, for genetics the effect size was .58, for disorders/pathology occurring during childhood the effect size was .58, and the largest effect size in this domain was .88 for temperament. The overall magnitude of effect for the psychobiological domain was .65. Results for this domain suggest that inherent characteristics of children contribute significantly to development of APD, in particular, a child's temperament. These findings support Siever and Davis' (1991) temperament model of PD development which holds that temperament consists of four dimensions (cognitive/perceptual organization, affective regulation, impulse control, and anxiety modulation) and that PDs reflect disturbances in particular dimensions. With respect to APD development in particular, disturbances are said to be in the impulse control

dimension.

Experiential/External Domain.

The experiential/external domain attempts to assess the impact of constitutional factors interacting with childhood experiences to produce psychological risk for APD development. As described in Table 9, the effect sizes for all of the variables in this domain were non-zero. Based on the weighted effect size analysis, the effect sizes for each variable in this domain are ordered in terms of their relative importance so that the variables having the least effect on APD development were sexual abuse (.14) and medical incident history (.29). Although the effect of physical abuse was only .36, this effect was more than double that of sexual abuse. This finding supports Norden, Klein, Donaldson, Pepper, and Klein's (1995) findings that physical abuse is more often associated with APD development than is sexual abuse which they found to be associated more with Borderline Personality development. The magnitude of effect for socio-economic status of a child's caregiver was .48. The magnitude of effects for poor school achievement (.75), separation and loss (.78), and family dysfunction/breakdown (.83) were somewhat higher for APD development but the variable displaying the highest contribution to APD development was emotional abuse which had a very high magnitude of effect (1.53). This finding suggests that the impact of emotional abuse is vastly more significant than any other form of abuse in the development of APD and supports Livesley's (1998) view that emotional dysregulation is an important higher-order dimension in personality disorders.

Interpersonal Domain.

The interpersonal domain includes a number of interpersonal variables and assesses

the relative importance of interactions with significant others in the development of APD. As shown in Table 10, the effect sizes for all of these variables were non-zero. Based on the weighted effect size analyses, the effect of harsh parenting was .40, the effect of parent delinquency was .44, the effect of having a delinquent sibling was .47, the effects of being neglected by a caregiver or receiving inconsistent discipline were both .48, and the magnitude of effect for parent disorders/pathology was .49. The variables having the highest magnitude of effect in this domain were social desirability and rejection both showing effects of .53 It might be noted that the primary difference between neglect (exploitation, negligent treatment, or maltreatment) and rejection (cold or lacking affection) is that while neglect may be viewed as a form of abuse as defined herein, rejection specifically involves emotional abuse directed toward the child by the parent or caregiver. Thus, scores indicating a higher magnitude of effect for rejection compared to neglect are consistent with the finding of a high magnitude of effect for emotional abuse. With respect to social desirability (the support or lack of support of a social network), a lack of social support might be viewed as a form of rejection, that is, by peers In this regard, cultural deviance theories have in common the importance of reinforcement or a lack of reinforcement from peers in the development of deviance but these theories do not specifically refer to reinforcement in the form of emotional support. Kiesler, Schmidt, and Wagner (1997) state, however, that the impact of messages between interacting individuals includes an emotional component.

Behavioral Domain.

The behavioral domain looks at childhood antisocial behavior and assesses the

magnitude of effect such behavior has on the development of APD. According to the DSM IV, antisocial behavior is a primary characteristic of APD. As shown in Table 11, results from the effect size analysis in this study indicate magnitude of effect for child antisocial behavior of .67. This finding suggests that antisocial behavior is also a characteristic of children who develop APD and that antisocial behavior is, in fact, a significant contributor in the development of APD.

Interpretation of the Analysis of Study Characteristics on Effect Size

It may be possible that the magnitude of effects for each domain are moderated by differences occurring due to changes, over the years, in theoretical views held by researchers. Changes in the DSM, and its revisions, for example, reflect a shift away from a psychodynamic perspective toward a more biological orientation during and after the 1960's (Grob, 1991). During the past decade, for example, researchers have shown a heightened interest in studying the role of neurotransmitters in human behavior (Weston & Siever, 1993; Siever & Davis, 1991). Similarly, there has been increased interested in genetic research aimed at identifying etiological alleles, or DNA variations, that may help to explain personality development (Carey & Goldman, 1997; Dahl, 1993; Nigg & Goldsmith, 1994). Given that theoretical shifts have occurred over time, and that notable changes have occurred during the past decade, it is possible that studies conducted prior to 1990 may be systematically different from studies conducted during the 1990's. Therefore, the study characteristic, year of study publication, may act as a moderator of the overall magnitude of effect found for any or all of the four domains.

In addition to changes in theoretical perspective and research interests, more and

more sophisticated methods and methodologies are being applied in an attempt to unravel the complex nature of personality and its disorders (Cloninger, 1998). Therefore, the magnitude of effect for each domain in this study may also be moderated by certain differences in researchers' choice of methods for assessing APD (non-DSM vs. DSM), the diagnostic indices they select (interview or questionnaire vs. other indices), and the type of study (retrospective vs. other types) and study design (experimental/quasi-experimental vs. other designs) they feel are appropriate for their investigations.

The study characteristics most likely to act as moderators in this MA are, therefore, the year of study publication, study type, study design, diagnostic indices, and methods of assessing APD

Psychobiological Domain.

Given that genetic research has increased during the last decade, and given that more of the studies published in the psychobiological domain were published during the 1990's, it might be expected that the year of study publication may moderate this domain. In fact, studies published prior to 1990 were found to be significantly different (1.21) from those published during the 1990's (.75). Specifically, the strongest effects were reported in studies published prior to 1990. A possible explanation for this finding could be that in more recent years, researchers may have followed stricter methods of assessment of APD thereby arriving at more conservative findings. In this regard, methods of assessing APD was also found to have a moderating effect on this domain. In particular, studies using methods of assessing APD not based on the DSM reported significantly higher results (1.15) than those reported in studies using assessment methods based on the DSM (.72).

Experiential/External and Interpersonal Domains.

The experiential/external domain showed a significant difference between studies published prior to the 1990's (2.68) and those published during the 1990's (0.49). Similarly, in the interpersonal domain, the effects reported in studies published prior to 1990 (1.45) were stronger than those reported in studies published during the 1990's (0.41). A closer examination of the studies included in the experiential/external and interpersonal domains revealed similar sampling of subjects in the studies conducted during both time periods with subjects being derived from the general population, as well as subjects from clinical and criminal populations. The only obvious difference between the samples was the number of studies included in each time period and the number of studies representing each contributing variable. Approximately 53% of the studies in the experiential/external domain were published prior to 1990 while only 47% were published during the 1990's. Higher effects for separation and loss and poor school attainment were reported in studies published prior to 1990. In this domain, separation and loss was researched in 59% of the studies while poor school attainment was researched in only 29% of the studies. The number of interpersonal studies included in each time period was similar, with 56% being conducted during the 1990's and 44% being conducted prior to the 1990's. The variables associated with the differences in this domain were parent disorders/pathology and neglect. Parent disorders/pathology was researched in 60% of the studies in this domain while only 28% of studies in this domain were researching neglect. Thus, the differences between time periods in the experiential/external and interpersonal domains may be merely a statistical artifact

Other possible explanations are less easily explained in view of the findings. One might wonder if children had fewer resources for coping with loss or for getting help in school in years prior to 1990. Luecken (2,000) studied attachment and loss experiences during childhood and found parental loss in childhood was associated with hostility in adulthood only if the quality of the surviving family relations was poor. It seems more likely, however, that family relations in the 1990's would be less supportive given that many more families are headed by two breadwinners, both working outside the home and, therefore, potentially having less time and energy to devote to their children. Research has been conducted to assess the effects on child development of both parents working outside of the home. Violato and Russell (2,000) studied the effects of nonmaternal care on child development and found that extensive nonmaternal care of infants and children resulted in insecure attachment to the mother. In addition, they found that insecure attachment resulted in negative sequelae in both social-emotional and behavioral child development domains.

Perhaps a larger family size contributed to child neglect in families prior to 1990. In this regard, Nelson, Saunders, and Landsman (1993) compared families recently referred to child welfare for child neglect, families known for chronically neglecting their children, and families with no reports of neglect. They found that recently referred families appeared to have faced a crisis because of illness, injury, or family dissolution but chronically neglecting families had significantly more members and extremely low incomes.

It may be that recent medical advances in assessment and treatment of mental disorders has improved the functioning of mentally disordered parents. The introduction of clozapine treatment in the US in 1990, for example, is said to have "opened the era of 'atypical' antipsychotic drugs". These treatments have shown reduced potential to induce extrapyramidal symptoms, an increased efficacy for the negative symptoms of schizophrenia, as well as effectiveness in some patients previously regarded as treatment-refractory (Shen, 1,999). Berk (2,000) stated that selective serotonin reuptake inhibitors (SSRIs) have, more than any other development, bridged the gap in terms of efficacy in the treatment of a number of disorders including the treatment of mixed Anxiety-Depression, Panic Disorder, Social and Generalized Anxiety Disorder, and PTSD

Further research is needed to determine whether the differences found in these domains between studies published prior to 1990 and studies published during the 1990's merely reflect a statistical artifact or whether there may be some other cause or causes related to societal changes or developments.

Behavioral Domain

No moderating effects were found for this domain.

Theoretical Implications

A number of theories have been proposed to explain the development of APD Although research in this area is relatively new, there is considerable potential for psychobiological research to inform our understanding of APD development. No empirical research testing experiential theories has supported a clear relationship between adverse experiences and APD, or APD symptoms. Rather, the effects of adverse

experiences interact with such factors as frequency, intensity, duration, and nature of the experience, the quality of parental bonds or family dysfunction, buffering factors from outside the family, and the way children cognitively process experiences (Paris, 1998a). Although some research findings support an interpersonal perspective, more empirical research is needed to validate these theories and while childhood antisocial behavior has been shown to be a precursor of APD, there is no evidence that APD develops from antisocial behavior alone. Hypotheses that APD depends on specific genotypes or physiological characteristics, or that APD is a natural outcome for children who display antisocial behaviors, or that APD develops from certain traumatic childhood experiences, or from particular interpersonal problems with parents or peers have a certain appeal but data do not point to such specificity. Crowell, Waters, Kring, and Riso (1993) believed that etiological variables co-occur in association with socio-economic status, particular family constellations, parental psychopathology, and even the effects of the child on its own environment. At the same time, psychosocial variables tend to be intercorrelated. In other words, APD develops within a complex milieu of interacting factors. In support of this view, all four domains in this MA showed significant effects on APD development. In fact, every one of the twenty two variables in this study were found to have a significant effect on APD development. Consequently, a theory of distinct etiologies is problematic and a new approach to the study of APD development is needed. In this regard, Brantley and Garrett (1993) believe an integration of findings from these areas may provide the ultimate contribution to our understanding. If APD is caused by a number of interacting variables, then a new theoretical model for examining these interactions is needed.

Other Implications of the MA

In terms of development of APD, the DSM IV points to a history of CD or CD along with ADHD as necessary for APD development. The results of this study do not support the view that disorders/pathology occurring in childhood play a primary role in APD development. In fact, the contribution of genetics (.58) was found to be equal to that of disorders/pathology occurring during childhood. In addition, other early childhood indicators of APD development were found to be much more important than disorders/pathology occurring during childhood childhood disorders/pathology. Specifically, poor school achievement (75), separation and loss (.78), family dysfunction (83), and temperament (88) had a greater impact on APD development and the magnitude of effect of emotional abuse (1 53) was more that twice that of disorders/pathology occurring during childhood.

These findings raise questions regarding the validity of the DSM-IV and its diagnostic criteria for APD DSM criteria have changed over the years along with changes in beliefs about the etiology of the disorder Currently, the DSM-IV takes a behavior-based approach which places primary emphasis on overt antisocial behaviors as the primary contributing factor as well as the defining characteristic of APD. As a result, other contributing factors, are not taken into consideration. Findings from this research suggest there may be more important contributors to APD development than CD, CD combined with ADHD, or any other disorders.

Delimitations of the Data Base Analysed

The results of this study cannot be generalized to all children exhibiting behaviors

associated with the variables in any of the four domains herein. For example, with respect to childhood disorders/pathology, in the psychobiological domain, a history of some symptoms of CD before age 15 years is required for a diagnosis of APD in the DSM IV, but one cannot conclude that all individuals with a history of CD will develop APD. Similarly, in the interpersonal domain, some children who have been neglected or rejected by their parents or who have received inconsistent discipline, may develop APD in adulthood while other children with similar histories will not develop APD

This MA did not examine all studies related to APD development but was delimited to those studies involving the examination of specific psychobiological, experiential/external, interpersonal, and behavioral variables chosen by the author, and which included statistics that could be converted into an effect size. The selection of studies was further limited to accessible research published from 1960 to the present. It was decided that studies prior to 1960 would be excluded partly because the period prior to 1960 was heavily influenced by psychodynamic and psychoanalytic concepts and partly because the earlier nosologies, used for the identification of personality disorders, were heavily criticized for being confusing because of conflicting nomenclatures. As a result, the findings reported in this study do not reflect earlier views of APD development.

Another delimitation of the present MA is that the sample was restricted to published studies, primarily because the time frame of this research was not long enough to make locating and obtaining the unpublished works feasible. It has been argued that a comprehensive meta-analysis requires complete inclusion of both published and unpublished studies. A counter argument is that only published studies should be used as

these have undergone peer review and thus a quality check while unpublished studies lack any systematic review process (Hedges, Shymansky, & Woodworth, 1989). In any case, one way to address this criticism is to conduct a file drawer analysis. The file drawer analysis ("Fail Safe N") is an estimate of the number of studies that may not have been included in the MA ("fugitive literature") that would be necessary to include so as to reverse the present findings and produce an effect size equal to zero. The file drawer analysis estimates the degree to which bias favoring significant findings, due to a lack of representation of the fugitive literature such as unpublished studies, threatens the validity of the overall conclusions of the MA (Rosenthal, 1979).

Fail Safe N

Validity of findings can be estimated by calculating the "fail-safe *N*", that is, the number of studies with reports of nonsignificant findings that would be required to reverse a conclusion drawn from the MA (Kurtzweil, Scogin, & Rosen 1996; Wolf, 1986). The estimate can be calculated using the following formula (Wolf, 1986, p 38) where:

ES = effect sizes in the present study per domain

N = number of studies in the meta-analysis

$N_{fs.05}$ = number of studies required to nullify present mean ES at the .05 level of significance

$$N_{fs.05} = \left(\frac{\text{sum of ES}}{1.645} \right)^2 - N$$

According to computations for each of the four domains, the following number of published studies with negative, or zero effect sizes are required to nullify the present findings: 1) the psychobiological domain, 568 unpublished studies, 2) the

experiential/external domain 599 unpublished studies, the interpersonal domain 729 unpublished studies, and the behavioral domain, 95 unpublished studies. These results may be explained by differences between domains in the number of effect sizes. The number of effect sizes represented by the psychobiological domain (41), the experiential/external domain (42), and the interpersonal domain (46) is approximately double that of the behavioral domain (20). The probability that such large numbers of contradictory studies exist but were not included in the present MA is very remote. Accordingly, we can place confidence in the generalizability and stability of the present findings.

Limitations of the Data Base Analysed

Bangert-Drowns (1995) pointed out that MA is a quantitative form of integrative review that is most usefully applied to the examination of domains with large groups of studies. He added that other circumstances may be better addressed by a literature review than an integrative review. This study was limited by the number of studies published in this area and meeting the inclusion criteria. The number of cases, however, was large enough to make a narrative review unmanageable. A larger sample size may have provided a richer database but it is unknown whether a larger sample would have made any difference to the overall findings. The number of studies included in the present MA, however, is consistent with meta-analytic work in general (Hunt, 1997). Indeed the number of studies exceeds the average of typical MAs.

This study was also limited by the nature of the sample studies included. Although the number of male and female subjects was reported in a number of studies, none of the sample studies included sex differences in the development of APD. Consequently, gender

could not be coded in this MA as a potential moderator. In addition, none of the studies included ethnicity as a risk factor for APD development. For this reason, ethnicity could not be included in the effect size analyses. Another potential limitation of the sample has to do with quality of design. The overall quality of design across studies may be questioned given that no studies were conducted using an experimental design and half of the studies did not include comparison groups. Internal validity may also be questioned given that APD was operationalized in this MA based on the DSM-IV, while DSM assessment criteria was used to assess APD in less than half of the studies in this sample.

A possible criticism of this MA is that in certain cases, more than one effect size was reported in the same study and therefore the results are not independent and may appear more reliable than they really are. In cases where there is more than one unit of analysis in the same study, Durlak (1995) stated that while it may be untrue, it is most practical to regard each finding as independent of the others. He added, however, that a second option is to use each study as a unit of analysis by averaging across all effect sizes within each study. This approach has its own problems. Different types of dependent measures may yield effects of different magnitudes so that averaging across measures may obscure important differences (Durlak, 1995). Accordingly, the first option was employed in the present study.

A question may be raised regarding the importance of a variable such as "medical incident history" which was represented by an exceptionally large number (101,078) of subjects. It may be said that medical incident history should be considered a benchmark for outcomes in this meta-analysis but this dependent measure is only one out of eight

dependent measures in the experiential domain and it is only one out of 22 dependent measures across all domains that act as potential risk factors for APD development. Also, medical incident history was made up of only three out of 17 studies in the experiential domain and 53 studies across all for domains in this meta-analysis. In addition, only one of the three studies accounted for most of the subjects (100,543) represented by this dependent measure. To place a great deal of importance on one study just because it has a large sample size does not take into consideration other factors, such as the possibility that the quality of the study may be poor which would mitigate the importance of that study relative to the overall findings within the experiential domain and the overall findings across domains in this meta-analysis.

Strengths and Limitations of Study Methodology

Strengths

A major strength of using MA, compared to a narrative review, as the method for this study is that it resolves the problem of selective inclusion of studies because it is not limited to a manageable number of studies. In fact, unlike narrative reviews, the accuracy of estimates improves as the amount of available data increases (Preiss & Allen, 1995).

Another advantage of using MA is that study characteristics could be coded and examined to reveal patterns of underlying relations that might explain how and why researchers come up with different results, and which variables contribute most to overall effects. In addition, by using MA it is possible to draw generalized conclusions from numerous studies with qualifications arising from moderating factors (Lytton, 1994).

With respect to interpreting findings, a MA makes it less likely that interpretations of findings will be misleading because previous studies are integrated by statistically pooling their results into a single quantitative analysis (Lytton, 1994).

Limitations.

A weakness of MA is that it is restricted by the limitations of the existing literature from which the study samples are drawn. In addition, not all studies on a topic are published and many published studies do not include statistics that can be converted into an effect size. Consequently, MAs are limited by the availability and suitability of published data, and therefore, the studies included may not be a representative sample of relevant research (Rosenthal, 1995).

Some critics believe that poorly designed studies include results significantly at variance with those of the best designed studies and therefore, poorly designed studies should not be included in a meta-analysis (Glass, 1976). In this MA, studies were not excluded on the basis of quality, in part to preserve sample size, and in part because 85% of the studies used the same design so that quality of design was similar across most studies. Glass, McGaw, and Smith (1981) examined this problem and found no significant difference between the findings of well-designed and poorly-designed studies. Olson, Wilkerson, and Kaufman (1997) examined high-validity and low-validity experiments and found that average effects seldom differed by more than one-tenth standard deviation. Based on their findings, they believe that including all studies in a review alleviates bias. Glass (1976) added that exclusion of studies on methodological grounds involves arbitrary decisions, often not supported by evidence.

It could be argued that the mixing of constructs in this MA could jeopardize external validity. It is true that the data used for this MA was derived from studies where several different theories were tested. In this regard, Wortman (1983) believed the important issue is construct validity. He felt that if the same hypothesis is being tested across studies, in this case the development of APD, then aggregation is appropriate but if one finds heterogeneous results, it may be that the theoretical construct being investigated is too broad. In such cases, he said it may be necessary to exclude studies. The results in the present study, however, were quite homogeneous across all four domains.

With respect to MA results, conclusions may be limited by differences in effect size due to variation in study characteristics (Lytton, 1994; Preiss & Allen, 1995). It has been argued that results from heterogeneous studies may not be reliable (Paddle, 1997). Even where studies are tested and found homogeneous, however, application of results to specific individuals assumes that subjects included in the original studies are an appropriate reference group for the individuals in question, an assumption that can be misleading (Smith & Egger, 1998).

Another limitation of this MA is that the results may not provide a sufficient basis for accurate causal inferences because of problems of covariation among potential moderators of effect size estimates (Knight, Fabes, & Higgins, 1996). Currently, there is no logical, systematic procedure for identifying study characteristics that may act as moderators (Wolf, 1986). In addition, strong causal inferences cannot be made by investigating the moderating effects of nonexperimental study factors (e.g., year of

publication) because nonexperimental study factors are related to other factors which may be the causal agents (Knight, Fabes, & Higgins, 1996).

Summary and Conclusions

The purpose of the present study was to seek a better understanding of the development of APD by identifying which early childhood factors are indicators of developing antisocial personality. Previously, it has been suggested that development of APD in adulthood may be related to psychobiological factors, childhood experiences or external influences, interpersonal relationships during childhood, and/or a child's antisocial behavior. In the present study, research was examined that had been conducted in each of these domains to determine which childhood factors constitute a risk for developing APD and which risk factors are the strongest indicators of APD development. Variables in all four domains (psychobiological, experiential/external, interpersonal, and behavioral) were found to have a significant effect on APD development. Within the domains, separation and loss (.78), family dysfunction (.83), temperament (.88) and emotional abuse (1.53) were the greatest risk factors for APD development.

Some independent variables were found to have a moderating effect on these findings. In particular, year of study moderated findings in the psychobiological, experiential/external, and interpersonal domains with studies conducted prior to 1990 having a stronger effect than studies conducted during the 1990's. In the psychobiological domain, methods of assessment also had a moderating effect. Magnitudes of effect reported in studies involving methods of assessment based on the DSM were lower than those reported in studies involving other methods.

Future Directions

For APD investigators, more research is needed involving women with APD. Lilienfeld, Purcell, and Jones-Alexander (1997) point out that the DSM-III-R's emphasis on aggressive conduct disorder symptoms may lead to an under-diagnosis of APD in females. Aggressive conduct disorder items tend to have low or even negative correlations in females, but not in males suggesting the presence of gender bias.

In the present MA, over 60 per cent of studies conducted during the 1990's used DSM-based methods for assessing APD as compared to 30% of the studies conducted prior to the 1990's. Therefore, it would seem that methods based on the DSM are either more conservative or the DSM criteria that underlies these assessment methodologies may not be getting at the true construct of APD. In this regard, findings from this study do not suggest the primary contributors to APD development, as outlined in the DSM-IV, are as important as a number of other contributors. The DSM-IV requires a history of CD prior to age 15 or CD along with ADHD prior to age 10. Although disorders/pathology occurring during childhood, in this MA, was found to have a significant effect on APD development, the magnitude of effect (.58) was considerably lower than a number of other contributors. Future researchers may want to examine the comparative validity between DSM-based methods of assessment and other methods of assessment.

Problems associated with differential diagnoses also complicate the assessment of APD. For the comorbidity of APD, it is important to note that there are a number of APD characteristics that are associated with other disorders. For example, Zanarini and Gunderson (1997) noted that APD and Borderline Personality Disorder (BPD) have

similar criteria sets both identifying their impulsivity and unstable interpersonal relationships. A BPD diagnosis, however, includes identity problems, lapses of reality testing, and intense affects which are not features of APD. Similarly, Tyrer (1993) reports that individuals with recurrent behavioral disturbance, unstable interpersonal relationships and periodic affective symptoms due to Bipolar Affective Disorder are often diagnosed using current DSM criteria, as having a cluster B personality disturbance such as APD. To improve the reliability of the DSM criteria, problems associated with differential diagnoses need to be addressed.

There is some question as to whether assessment criteria for APD should place so much emphasis on childhood disorders that manifest in antisocial behaviors. Results herein suggest an emotional component to APD etiology that is not being taken into consideration by DSM-IV criteria. The behavioral approach taken in the DSM-IV emphasizes aggressive conduct disorder symptoms, an approach that may explain the under-diagnosis of APD in females. Given that the most profound risk factor identified in this MA was emotional abuse by a caregiver, results suggest that assessing practitioners need to go beyond behavioral indices of the DSM-IV criteria to include assessment of their clients' affective and interpersonal styles. In this regard, it may be that future research should explore the utility of personality dimensions in PD assessment. Specifically, they might examine the possibility of combining the DSM categorical format with a dimensional format, as suggested by Yeung, Lyons, Waternaux, Faraone, and Tsuang (1993) who thought it may be worthwhile to include personality dimensions, such

as those outlined in Costa and McCrae's (1990) Five Factor Model, as an additional axis in the DSM.

In this study, all of the variables within all four domains were found to have a significant effect on APD development. These findings support the view that APD develops by way of a very complex interaction of multiple factors. Thus, it would seem that a theory of distinct etiologies is problematic and that a new approach to the study of APD development is needed based on a model that integrates and examines contributing sources of influence. Cummings, Davies, and Campbell (2000) describe a general model for developmental psychpathology wherein family and other social support influence child psychological functioning (cognitions, emotions, physiological responses, and social interactions) which, in turn, determines the child's adjustment or maladjustment over time. Applying this general model to the study of parenting, Cummings et al (2000) propose a developmental framework wherein the relationship between parenting and child adjustment may be moderated by the ecological context which also influences the child. This view suggests a more complex relationship wherein the effect parents have on their child's development is neither simple nor powerful but rather myriad factors in each of three categories, parent, child, and context, influence a child's development as it unfolds over time. They added that no single characteristic of parenting can be assigned primacy because parenting involves multiple dimensions, the effects of which are influenced by and dependent upon other factors including family, ecological, and other contextual variables.

If one considers only those variables in this study having large magnitudes of effect (> .75) for APD development, however, only one variable (temperament $\underline{d}$ = .88) showed

a large magnitude of effect for child and no contextual variables showed a large magnitude of effect. Other variables having large magnitudes of effect suggest that parents and family stability are the primary influences contributing to a child's development of APD. Specifically, emotional abuse by a caregiver (1.53) was, by far, the strongest indicator of APD development. Family dysfunction (.83) and separation and loss (.78) were also strong indicators.

Given that parents and family stability appear to have strong effects on the development of personality psychopathology, it may be there is a relationship between attachment and APD development. Thus, future investigators may find it advantageous to use attachment theory as a framework for testing potential causal models for the development of APD. Violato and Genuis (1997), for example, proposed a structural latent variable path model of adolescent adjustment and psychopathology. According to their model, secure attachment results in emotional stability and positive psychological outcomes, while insecure attachment results in psychological disturbances and pathology. Attachment in childhood is affected by the relationship between the child and caregiver. Over the course of development, isolation and abuse are seen as distinct but correlated latent variables which lead to insecure attachment. Insecure attachment reciprocally affects abuse and isolation which in turn further affect attachment so that attachment, abuse, and isolation are considered mutually influential and interdependent in this model.

Violato and Genuis (1997) designed a study to test the latent variable path model and to investigate the link between early attachments and psychopathology in adolescence. Using structural equation modeling to fit the model to the data, they found the overall fit

of the model to be high, accounting for 98.4% of the variance and covariance in the data. All three latent variables, attachment, abuse, and isolation, were clearly identified and found to be intercorrelated. Also identified was one direct path from childhood attachments to diagnosis of psychopathology which was found to be significant (path coefficient = - 48; $p < 001$). Overall, the results of the study indicate that childhood attachments play a role in the development of psychopathology. Application of this model to specific disorders, such as APD, however, has not been undertaken. Therefore, it may be useful to design a prospective, longitudinal study that proposes a causal link between childhood attachments and APD Using a causal model such as the latent variable path model suggested by Violato and Genuis (1997), research may reveal a causal link to APD that is more parsimonious than other multifactorial models such as the one proposed by Cummings, Davies, and Campbell (2000). The results of the present meta-analysis with the largest effect sizes on family and caregiver variables, suggest that family and experiential variables do have prepotency over other variables contrary to the Cummings et al (2000) suggestions.

References[1]

Adams-Webber, J. R. (1992). Some fundamental asymmetries in the structure of personal constructs. In G. J. Neimeyer, R. A. et al (Eds.), Advances in personal construct psychology: A research annual (pp. 49-85). Greenwich, CT: Jai Press.

Adler, D. A. (1990). Personality disorders: Theory and psychotherapy. New Directions for Mental Health Services, 47, 17-42.

American Psychiatric Association (1994). Personality Disorders. In Diagnostic and statistical manual of mental disorders (4th ed., pp. 629-673). Washington, DC: American Psychiatric Association.

Azrin, N H., & Holz, W C. (1966). Punishment. In W. K. Honig (Ed.), Operant behavior: Areas of research and application, (pp. 380-447). New York: Appleton-Century-Crofts

Bandura, A. (1973) Aggression: A social learning analysis. Englewood Cliffs, NJ: Prentice Hall.

Bangert-Drowns, R. L. (1995). Misunderstanding meta-analysis. Evaluation and the Health Professions, 18(3), 304-314.

*Barratt, E. S., Stanford, M. S., Kent, T. A., Felthous, A. (1997). Neuropsychological and cognitive psychophysiological substrates of impulsive aggression. Biological Psychiatry, 41, 1045-1061.

[1] References with asterisks (*) indicate studies used in the meta-analysis.

Beck, A. T. (1997). The past and future of cognitive therapy. Journal of Psychotherapy Practice & Research, 6(4), 276-284.

Benjamin, L. S. (1996a). Antisocial Personality Disorder. In A. Frances (Ed.), Interpersonal diagnosis and treatment of personality disorders (2nd ed., pp. 191-220). New York: Guilford Press.

Benjamin, L. S. (1996b). History and assumptions of the approach. In A. Frances (Ed.), Interpersonal diagnosis and treatment of personality disorders (2nd ed., pp. 15-25). New York: Guilford Press.

Berk, M. (2,000). Selective serotonin reuptake inhibitors in mixed anxiety-depression. International Clinical Psychopharmacology, 15 (Suppl. 2), S41 - S45.

*Biederman, J., Faraone, S. V., Hatch, M., Mennin, D., Taylor, A., & George, P. (1997). Conduct disorder with and without mania in a referred sample of ADHD children. Journal of Affective Disorders, 44, 177-188.

*Blackburn, R., & Coid, J. W. (1999). Empirical clusters of DSM-III personality disorders in violent offenders. Journal of Personality Disorders, 13(1), 18-34.

*Bloomingdale, L. M., & Bloomingdale, E. C. (1988). Childhood identification and prophylaxis of Antisocial Personality Disorder. Journal of Forensic Sciences, 33(1), 187-100.

Bouchard, T. J. (1997). The genetics of personality. In K. Blum & E. P. Noble (Eds.), Handbook of psychiatric genetics (pp. 273-296). Boca Raton, FL: Crc Press.

Brantley, P. J., & Garrett, V. D. (1993). Psychobiological approaches to health and disease. In P. B. Sutker & H. E. Adams (Eds.), Comprehensive handbook of psychopathology (2nd ed.). New York: Plenum Press.

Brennan, P.A., & Mednick, S.A. (1997). Medical histories of antisocial individuals. In D. M. Stoff, J. Breiling, & J. D. Maser (Eds.), Handbook of antisocial behavior (pp. 269-279). New York: John Wiley & Sons.

*Brown, S. A., Gleghorn, A., Schuckit, M. A., Myers, M. G., & Mott, M. A. (1996). Conduct disorder among adolescent alcohol and drug abusers. Journal of Studies on Alcohol, 57, 314-324.

*Cadoret, R. J. (1978). Psychopathology in adopted-away offspring of biologic parents with antisocial behavior. Archives of General Psychiatry, 35, 176-184.

*Cadoret, R. J., Troughton, E., Bagford, J., & Woodworth, G. (1990). Genetic and environmental factors in adoptee antisocial personality. European Archives of Psychiatry and Neurological Sciences, 239, 231-240.

*Cadoret, R. J., Yates, W. R., Troughton, E., Woodworth, G., Stewart, M. A. (1995). Adoption study demonstrating two genetic pathways to drug abuse. Archives of General Psychiatry, 52, (42-52).

Carey, G., & Goldman, D. (1997). The genetics of antisocial behavior. In D. M. Stoff, J. Breiling, & J. D. Maser (Eds.), Handbook of antisocial behavior, (243-254). New York: John Wiley & Sons.

Carter, H. D. (1933). Twin similarities in personality traits. Character and Personality, 4(61).

*Caspi, A., Moffitt, T. E., Newman, D. L., & Silva, P. A. (1996). Behavioral observations at age 3 years predict adult psychiatric disorders: Longitudinal evidence from a birth cohort. Archives of General Psychiatry, 53, 1033-1039.

*Caudill, B. D., Hoffman, J. A., Hubbard, R. L., Flynn, P. M., Luckey, J. W. (1994). American Journal of Drug and Alcohol Abuse, 20(3), 341-354.

Clarkin, J. F., & Lensenweger, M. F. (Eds.) (1996). Major theories of personality disorder. New York: Guilford Press.

Cloninger, C. R. (1998). The genetics and psychobiology of the seven-factor model of personality. In K. R. Silk (Ed.), Biology of personality disorders (pp. 63-92). Washington, DC: American Psychiatric Press.

Cloninger, C. R., Bayon, C., & Przybeck, T. R. (1997). Epidemiology and Axis I comorbidity of antisocial personality. In D. M. Stoff, J. Breiling, & J. D. Maser (Eds.), Handbook of antisocial behavior (pp. 12-21). New York: John Wiley & Sons.

*Cloninger, C. R., Christiansen, K. O., Reich, T., & Gottesman, I. I. (1978). Implications of sex differences in the prevalences of antisocial personality, alcoholism, and criminality for familial transmission. Archives of General Psychiatry, 35, 941-952.

*Cloninger, C. R., Reich, T., & Guze, S. B. (1975a). The multifactorial model of disease transmission: II. Familial relationship between Sociopathy and Hysteria (Briquet's Syndrome). British Journal of Psychiatry, 11-22.

*Cloninger, C. R., Reich, T., & Guze, S. B. (1975b). The multifactorial model of disease transmission: III. Familial relationship between Sociopathy and Hysteria (Briquet's Syndrome). British Journal of Psychiatry, 23-32.

Coccaro, E. F., Astill, J. L., Szeeley, P. J., Malkowicz, D. E. (1990). Serotonin in personality disorder. Psychiatric Annals, 20(10), 587-592.

Conger, R. (1976). Social control and social learning models of delinquent behavior: A synthesis. Criminology, 14, 17-40.

Costa, P. T., & McCrae, R. R. (1990). Personality disorders and the five-factor model of personality. Journal of Personality Disorders, 4(4), 362-371

*Crowe, R. R. (1974). An adoption study of antisocial personality. Archives of General Psychiatry, 31, 785-791.

*Crowe, R. R. (1975). An adoptive study of psychopathy: Preliminary results from arrest records and psychiatric hospital records. In R. R. Fieve, H Brill, & D Rosenthal (Eds.), Genetic Research in Psychiatry (pp. 95-193). Baltimore: John Hopkins University Press.

Crowell, J. A., Waters, E., Kring, A., & Riso, L. P. (1993). The psychosocial etiologies of personality disorders: What is the answer like? Journal of Personality Disorders, 1, 118-128.

Cummings, E. M., Davies, P. T., & Campbell, S. B. (2000). Developmental psychopathology and family process: Theory, Research, and Clinical Implications (pp. 89-161). New York: Guilford Press.

Dahl, A. A. (1993). The personality disorders: A critical review of family, twin, and adoption studies. Journal of Personality Disorders, 1, 86-99.

Dastidar, P. G., & Kapoor, S. (1996). Fractured vs. intact families: Differential impact on children. Journal of the Indian Academy of Applied Psychology, 22(1-2), 35-42.

Dilk, M. N., & Bond, G. R. (1996). Meta-analytic evaluation of skills training research for individuals with severe mental illness. Journal of Consulting and Clinical Psychology, 64(6), 1337-1346.

*Dinwiddie, S. H. (1997). Characteristics of injection drug users derived from a large family study of alcoholism. Comprehensive Psychiatry, 38(4), 218-229

Dobson, K. S., & Pusch, D. (1993). Towards a definition of the conceptual and empirical boundaries of cognitive therapy. Australian Psychologist, 28(3), 137-144.

Dodge, K. A., & Swartz, D. (1997). Social information processing mechanisms in aggressive behavior In D M. Stoff, J. Breiling, & J. D Maser (Eds.), Handbook of Antisocial Behavior (pp. 171-180). New York: John Wiley & Sons.

Dollard, J., & Miller, N. E. (1950) Frustration and aggression. New Haven, CT. Yale University Press.

*Downey, K. K., Stelson, F W., Pomerleau, O. F., & Giordani, B. (1997). Adult Attention Deficit Hyperactivity Disorder: Psychological test profiles in a clinical population. The Journal of Nervous and Mental Disease, 185(1), 32-38.

Draganic, S., Lecic-Tosevski, D., & Calovska-Hertzog, N. (1997). Trauma: The meeting point between posttraumatic stress disorder and borderline personality disorder. Psihijatrija Danas, 29(1-2), 31-47.

Durlak, J. A. (1995). Understanding meta-analysis. In L. G. Grim & P. R. Yarnold (Eds.), Reading and understanding multivariate statistics (pp. 319-352). Washington, DC: American Psychological Association.

Emery, R. E. (1982). Interparental conflict and the children of discord and divorce. Psychological Bulletin, 92, 310-330.

Engler, B. (1995). Personality theories: An Introduction (4th ed., pp. 152-189). Boston: Houghton Mifflin.

Eron, L. D. (1997). The development of antisocial behavior from a learning perspective. In D. M. Stoff, J. Breiling, & J. D. Maser (Eds.), Handbook of antisocial behavior (pp. 140-147). New York: John Wiley & Sons.

Eron, L. D., Walder, L. O., & Lefkowitz, M. M. (1971). Learning of aggression in children. Boston, MA: Little Brown.

Farrington, D. P., & West, D. J. (1971). A comparison between early delinquents and young aggressives. British Journal of Criminology, 11, 341-358.

Farrington, D. P. (1979). Environmental stress, delinquent behavior and conviction. In I. G. Sarason and C. D. Spielberger (Eds.), Stress and anxiety, (Vol.6). Washington, DC: Hemisphere.

*Farrington, D. P. (1991). Antisocial personality from childhood to adulthood. The British Psychological Society, 4, 389-394.

*Farrington, D. P. (1995). The Twelfth Jack Tizard Memorial Lecture. The development of offending and antisocial behavior from childhood: Key findings from the Cambridge study in delinquent development. The Journal of Child Psychology and Psychiatry, 360(6), 929-964.

*Farrington, D. P. (1996). Psychosocial influences on the development of antisocial personality. In G. Davies, S. Lloyd-Bostock, M. McMurran, & C. Wilson (Eds.), Psychology, Law, and Criminal Justice: International Developments in Research and Practice (pp. 424-444). New York: Walter de Gruyter.

Glass, G. V. (1976). Primary, secondary, and meta-analysis of research. Educational Researcher, 5(10), 3-8.

Glass, G. V., McGaw, B., & Smith, M. L. (1981). Meta-Analysis in social research. Beverly Hills: Sage.

Glueck, S., & Glueck, E.T. (1950). Unraveling juvenile delinquency. New York: Commonwealth Fund.

*Gottesman, I. I. (1963). Heritability of personality: A demonstration. Psychological Monographs: General and Applied, 77(9), 1-21.

*Gottesman, I. I. (1994). Personality and natural selection. In Charles A. Nelson (Ed.), Threats to optimal development: Integrating biological, psychological, and social risk factors (pp. 63-74). Hillsdale, NJ: Erlbaum.

Gregory, I. (1965). Introspective data following childhood loss of a parent: I. Delinquency and high school drop out. Archives of General Psychiatry, 13, 99-109.

Grob, G. N. (1991). Origins of DSM-I: A study in appearance and reality. American Journal of Psychiatry, 148(4), 421-431.

Guze, S. B. (1976). Conclusions and discussions. In Criminality and psychiatric disorders (pp. 124-146). New York: Oxford University Press.

Hare, R. D. (1965). A conflict and learning theory analysis of psychopathic behavior. Journal of Research in Crime and Delinquency, 2(1), 12-19.

Hare, R. D. (1980). The assessment of psychopathy in criminal populations. Personality and Individual Differences, 1, 111 -119.

Hedges, L. V. (1982). Estimation of effect size from a series of independent experiments. Psychological Bulletin, 92(2), 490-499.

Hedges, L. V., Shymansky, J. A., & Woodworth, G. (1989). A practical guide to modern methods of meta-analysis. Washington, DC: National Science Teachers Association.

*Hesselbrock, M. N. (1986). Childhood behavior problems and adult Antisocial Personality Disorder in alcoholism. In Roger E. Meyer (Ed.), Psychopathology and addictive disorders (pp. 78-94). New York: Guilford Press.

*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.

Hill, H. (1962). The social deviant and initial addiction to narcotics and alcohol. Quarterly Journal of Studies on Alcohol, 23(4), 562-582.

Hirsch, J. (1958). Recent developments in behavior genetics and differential psychology. Disorders of the Nervous System, 19(7) Monograph Supplement.

Hirschi, T. (1969). Causes of delinquency. Berkeley, CA: University of California Press.

*Howard, J. (1981). The expression and possible origins of depression in male adolescent delinquents. Australian and New Zealand Journal of Psychiatry, 15, 311-318.

Hunt, M. (1997). How science takes stock: The story of meta-analysis. New York: Russell Sage Foundation.

Hunter, J. E., Schmidt, F. L., & Jackson, G. B. (1982). Meta-Analysis: Cumulating research findings across studies. Beverly Hills: Sage.

Kandel, E., & Mednick, S. A. (1988) IQ as a protective factor for subjects at high risk for antisocial behavior. Journal of Consulting and Clinical Psychology, 56(2), 224-226

*Kendler, K. S., Davis, C. G., & Kessler, R. C. (1997). The familial aggregation of common psychiatric and substance use disorders in the National Comorbidity Survey: A family history study. British Journal of Psychiatry, 170, 541-548.

Kernberg, O. F. (1996). A psychoanalytic theory of personality disorders. In J. F. Clarkin & M. F. Lenzenweger (Eds.), Major theories of personality disorder (pp. 106-140). New York: Guilford Press.

Kiesler, D. J, Schmidt, J. A., & Wagner, C. C. (1997). A circumplex inventory of impact messages: An operational bridge between emotion and interpersonal behavior. In R. Plutchik & H. R. Conte (Eds.), Circumplex models of personality and emotions (pp. 221-244). Washington, DC: American Psychological Association.

Kiev, M. (1936). Temperamental differences in twins. British Journal of Psychology, 27, 51.

*Klinteberg, A. A., Schalling, D., & Magnusson, D. (1990). Childhood behaviour and adult personality in male and female subjects. European Journal of Personality, 4, 57-71.

Knight, G. P., Fabes, R. A., & Higgins, D. A. (1996). Concerns about drawing causal inferences from meta-analyses: An example in the study of gender differences in aggression. Psychological Bulletin, 119(3), 410-421.

Kogan, N. (1990). Personality and aging. In J. E. Birren and K. W. Schaie (Eds.), The handbook of aging (pp. 330-346). San Diego, CA: Academic Press.

Koski, P. R., & Mangold, W. D. (1993). A sociological perspective: Antisocial behavior and violence. In A. S. Bellack and M. Hersen (Eds.), Psychopathology in adulthood (pp. 104-120). Boston, MA: Allyn & Bacon.

Kurtzweil, P. L., Scogin, F., & Rosen, G. M. (1996). A test of the fail-safe N for self-help programs. Professional Psychology: Research and Practice, 27(6), 629-630.

Lahey, B. B., Hartdagen, S. E., Frick, P. J., & McBurnett, K. (1988). Conduct disorder: Parsing the confounded relation to parental divorce and antisocial personality. Journal of Abnormal Psychology, 97(3), 334-337.

Laporte, L., & Guttman, H. (1996). Traumatic childhood experiences as risk factors for Borderline and other personality disorders. Journal of Personality Disorders, 10(3), 247-259.

Leary, T. (1957). Interpersonal diagnosis of personality: A functional theory and methodology for personality evaluation. New York: Ronald Press.

Lenzenweger, M. F., & Clarkin, J. F. (1996). The personality disorders: History, classification, and research issues. In J. F Clarkin and M. F. Lenzenweger (Eds.), Major theories of personality disorder (pp. 1-35). New York: Guilford Press.

*Lewis, C. E., & Bucholz, K. K. (1991). Alcoholism, antisocial behavior and family history. British Journal of Addiction, 86, 177-194.

Lilienfeld, S. O., Purcell, C., & Jones-Alexander, J. (1997). Assessment of antisocial behavior in adults. In D. M. Stoff, J. Breiling, & J. D. Maser (Eds.), Handbook of Antisocial Behavior (pp 60-74). New York: John Wiley & Sons.

Livesley, W. J. (1998). Suggestions for a framework for an empirically based classification of personality disorder. Canadian Journal of Psychiatry - Revue Canadienne de Psychiatrie, 43(2), 137-147.

Luecken, L. J. (2000). Attachment and loss experiences during childhood are associated with adult hostility, depression, and social support. Journal of Psychosomatic Research, 49(1), 85-91.

*Luntz, B. K., & Widom, C. S. (1994). Antisocial Personality Disorder in abused and neglected children grown up. American Journal of Psychiatry, 151, 670-674.

Lynam, D. R. (1996). Early identification of chronic offenders: Who is the fledgling psychopath? Psychological Bulletin, 120(2), 209-234

*Lyons, M. J., True, W. R., Eisen, S. A., Goldberg, J., Meyer, J. M., Faraone, S. V., Eaves, L. J., & Tsuang, M. T. (1995). Differential heritability of adult and juvenile antisocial traits. Archives of General Psychiatry, 52, 906-915.

Lytton, H. (1994). Replication and meta-analysis: The story of a meta-analysis of parents' socialization practices. In R. Vandorveen, M. Van Ijzendoorn, & J. Vaalsiner (Eds.), Reconstructing the mind: Replicability in research on human development (pp. 117-149) Norwood, NJ: Aggx.

Mealey, L. (1995). The sociobiology of sociopathy: An integrated evolutionary model. Behavioral and Brain Sciences, 18(3), 523-541.

Millon, T. (1981). Antisocial Personality: The Aggressive Pattern. In Disorders of personality: DSM-III: Axis II, (pp. 181-215). New York: John Wiley & Sons.

Mischel, W., & Shoda, Y. (1995). A cognitive affective system theory of personality: Reconceptualizing situations, dispositions, dynamics, and invariance in personality structure. Psychological Review, 102(2), 246-268.

*Moss, H. B., Mezzich, A., Yao, J. K., Gavaler, J., & Martin, C. S. (1995). Aggressivity among sons of substance-abusing fathers: Association with psychiatric disorder in the father and son, paternal personality, pubertal development, and socioeconomic status. American Journal of Drug and Alcohol Abuse, 21(2), 195-208.

*Mutzell, S. (1994). Mortality, suicide, social maladjustment and criminality among male alcoholic parents and men from the general population and their offspring. International Journal of Adolescence and Youth, 4, 305-328.

Muuss, R. E. (1996). Theories of adolescence (6th ed.). New York: McGraw-Hill.

*Myers, W. C., Burket, R. C., & Harris, H. E. (1995). Adolescent psychopathy in relation to delinquent behaviors, conduct disorder, and personality disorders. Journal of Forensic Sciences, 40(3), 435-439.

Nelson, K. E., Saunders, E. J., & Landsman, M. J. (1993). Chronic child neglect in perspective. Social Work, 38(6), 661-671.

*Neugebauer, R., Hoek, H. W., & Susser, E.(1999). Prenatal exposure to wartime famine and development of Antisocial Personality Disorder in early adulthood. JAMA, 282(5), 455-462.

Nigg, J. T., & Goldsmith, H. H. (1994). Genetics of personality disorders: Perspectives from personality and psychopathology research. Psychological Bulletin, 115(3), 346-380.

*Norden, K. A., Klein, D. N., Donaldson, S. K., Pepper, C. M., & Klein, L. M. (1995). Reports of the early home environment in DSM-III-R personality disorders. Journal of Personality Disorders, 9(3), 213-223.

O'Connor, B. P., & Dyce, J. A. (1998). A test of models of personality disorder configuration. Journal of Abnormal Psychology, 107(1), 3-16.

Olson, M. R., Wilkerson, M., & Kaufman, J. J. (1997). Meta-analysis: A procedure for integrating research results. Journal of Adult Education, 25(1), 17-26.

*O'Neal, P., Robins, L. N., King, L. J., & Schaefer, J. (1962). Parental deviance and the genesis of sociopathic personality. American Journal of Psychiatry, 118, 1114-1124.

Paddle, G. M. (1997). Meta-analysis as an epidemiological tool and its application to studies of chromium. Regulatory Toxicology & Pharmacology, 26 (1, pt.2), S42-S50.

Pam, A., Inghilterra, K., & Munson, C. (1994). Agoraphobia: The interface between anxiety and personality disorder. Bulletin of the Menninger Clinic, 58(2), 242-261.

Paris J. (1996). Antisocial personality disorder: A biopsychosocial model. Canadian Journal of Psychiatry - Revue Canadienne de Psychiatrie, 41(2), 75-80.

Paris, J. (1998a). Does childhood trauma cause personality disorders in adults? Canadian Journal of Psychiatry, 43, 148-153.

Paris, J. (1998b). Significance of biological research for a biopsychosocial model of the personality disorders. In K. R. Silk (Ed.), Biology of Personality Disorders (pp. 129-148). Washington, DC: American Psychiatric Press.

Patterson, G. R. (1982). A social learning approach: Coercive family process (Vol.3). Eugene, OR: Castalia Publishing.

Petitti, D. B. (1997). Meta-analysis and endocrinology. Endocrinology & Metabolism Clinics of North America, 26(1), 31-44.

Pincus, A. L., & Wiggins, J. S. (1990). Interpersonal problems and conceptions of personality disorders. Journal of Personality Disorders, 4(4), 342-352.

Preiss, R. W., & Allen, M. (1995). Understanding and using meta-analysis. Evaluation and the Health Professions, 18(3), 315-335.

*Reich, J. (1986). The relationship between early life events and DSM III personality disorders. Hillside Journal of Clinical Psychiatry, 8(2), 164-173.

*Rey, J. M., Singh, M., Morris-Yates, A., & Andrews, G. (1997). Referred adolescents as young adults: The relationship between psychosocial functioning and personality disorder. Australian and New Zealand Journal of Psychiatry, 31,219-226.

*Rey, J. M., Morris-Yates, A., Singh, M., Andrews, G., & Stewart, G. W. (1995). Continuities between psychiatric disorders in adolescents and personality disorders in young adults. American Journal of Psychiatry, 152(6), 895-900.

*Robins, L. N. (1966c). Childhood behavior predicting later diagnosis. In Deviant children grown up: A sociological and psychiatric study of Sociopathic Personality (pp. 135-158). Baltimore: Williams & Wilkins.

*Robins, L. N. (1966b). The family setting of the young sociopath. In Deviant children grown up: A sociological and psychiatric study of Sociopathic Personality (pp. 159-180). Baltimore: Williams & Wilkins.

*Robins, L. N. (1966a). Social status in childhood and the diagnosis of Sociopathic Personality. In Deviant children grown up: A sociological and psychiatric study of Sociopathic Personality (pp. 181-201). Baltimore: Williams & Wilkins.

Robins, L. N., & Hill, S. Y. (1966). Assessing the contribution of family structure, class and peer groups to juvenile delinquency. Journal of Criminal Law, Criminology, and Police Science, 57, 325-334.

*Robins, L. N., Tipp, Y., & Przybeck, T. (1991). Antisocial personality. In Lee N. Robins & Darrel A. Regier (Eds.), The epidemiologic catchment area study (pp. 258-290). New York: Free Press.

Rogers, R., Johansen, J., Chang, J. J., & Salekin, R. T. (1997). Predictors of adolescent psychopathy: Oppositional and conduct-disordered symptoms. Bulletin of the American Academy of Psychiatry & the Law, 25(3), 261-271.

Romney, D. M., & Bynner, J. M. (1997). Evaluating a circumplex model of personality disorders with structural equation modeling. In R. Plutchik & H. R. Conte (Eds.), Circumplex models of personality and emotions (pp. 327-346). Washington, DC American Psychological Association.

Rosenthal, R. (1979). The "file drawer problem" and tolerance for null results. Psychological Bulletin, 86(3), 638-641.

Rosenthal, R. (1995). Writing meta-analytic reviews. Psychological Bulletin, 118(2), 183-192.

Rosenthal, R. & Rubin, d. B. (1982). Comparing effect sizes of independent studies. Psychological Bulletin, 92(2), 500-504.

Rushton, J. P. (1996). Self-report delinquency and violence in adult twins. Psychiatric Genetics, 6(2), 87-89.

*Rutter, M., Harrington, R., Quinton, D., & Pickles, A. (1994). Adult outcome of Conduct Disorder in childhood: Implications for concepts and definitions of patterns of Psychopathology. Adolescent Problem Behaviors: Issues and Research, 57-80.

Rutter, M., & Maughan, B. (1997). Psychosocial adversities in childhood and adult psychopathology. Journal of Personality Disorders, 11(1), 4-18.

*Rydelius, P. A. (1983). Alcohol-abusing teenage boys: Testing a hypothesis on alcohol abuse and personality factors, using a personality inventory. Acta Psychiatrica Scandinavia, 68, 381-385.

Saklofske, D. H., & Eysenck, S. B. G. (Eds.) (1998). Individual differences in children and adolescents. New Brunswick, NJ: Transaction Publishers.

Sanderson, C. A., & Cantor, N. (1999). A life task perspective on personality coherence: Stability versus change in tasks, goals, strategies, and outcomes. In D Cervone and Y. Shoda (Eds.), The coherence of personality: Social-cognitive bases of consistency (pp. 372-392). New York: Guilford Press.

Schaefer, E. S. (1965). Configurational analysis of children's reports of parent behavior. Journal of consulting Psychology, 29, 552-557.

Schroeder, M. L., Wormworth, J. A., & Livesley, W. J. (1992). Dimensions of personality disorder and their relationships to the Big Five dimensions of personality. Psychological Assessment, 4(1), 47-53.

Shadish, W. R., & Haddock, C. K. (1994). Combining estimates of effect size. H. Cooper and L. V. Hedges (Eds.). The Handbook of Research Synthesis (261-281). New York: Russell Sage Foundation.

Shen, W. W. 1999). A history of antipsychotic drug development. Comprehensive Psychiatry, 40(6), 407-414.

Siever, L. J., & Davis, K. L. (1991). A psychobiological perspective on the personality disorders. American Journal of Psychiatry, 148(12), 1647-1658.

Smith G. D., & Egger, M. (1998). Incommunicable knowledge? Interpreting and applying the results of clinical trials and meta-analyses. Journal of Clinical Epidemiology, 51(4), 289-295.

Stoff, D. M., Breiling, J., & Maser, J. D. (Eds.). (1997). Handbook of antisocial behavior. New York: John Wiley & Sons.

Strack, S., & Lorr, M. (1997). The challenge of differentiating normal and disordered personality. Journal of Personality Disorders, 11(2), 105-122.

Strupp, H. H., Schact, T. E., Henry, W. P., & Binder, J. L. (1992). Jack M.: A case of premature termination. Psychotherapy, 29(2), 191-205.

Sutherland, E. H. (1939). Principles of Criminology (3rd ed.) Philadelphia: Lippincott.

Sutker, P. B., Bugg, F., & West, J. A. (1993). Antisocial Personality Disorder In P B. Sutker, & H. E. Adams (Eds.), Comprehensive handbook of psychopathology (2nd ed., pp. 305-313). New York: Plenum Press.

Szurek, S.A. (1969). Childhood origins of psychopathic personality trends. The antisocial child, his family and his community (S. A. Szurek & I. N. Berlin, Eds.). Palo Alto, CA: Science and Behavior Books.

Thornberry, T.P. (1987). Toward an interactional theory of delinquency. Criminology, 25, 863-891.

Thornberry, T. P., & Krohn, M. D. (1997). Peers, drug use and delinquency. In D. M. Stoff, J. Breiling, & J. D. Maser (Eds.), Handbook of antisocial behavior (pp. 218-233). New York: John Wiley & Sons.

Torgersen, S., & Alnaes, R. (1989). Localizing DSM-III personality disorders in three-dimensional structural space. Journal of Personality Disorders, 3, 274-281.

Tyrer, S. P. (1993). Misdiagnosis of bipolar affective disorder as personality disorder. Canadian Journal of Psychiatry, 38 (9), 587-589.

*Vaillant, G. E. (1994). Evidence that the Type 1/Type 2 dichotomy in alcoholism must be re-examined. Addiction, 89, 1049-1057

*Virkkunen, M. (1983). Insulin secretion during the glucose tolerance test in antisocial personality. British Journal of Psychiatry, 142, 598-604.

Violato, C., & Genuis, M. (1997). Origins of psychopathology: A developmental model. Medicine, Mind and Adolescence, 12(1-2), 51-65.

Violato, C., & Russell, C. (2,000). Effects of nonmaternal care on child development: A meta-analysis of published research. In C. Violato, E. Oddone-Paolucci, and M. Genuis (Eds.), The changing family and child development (pp. 268-301). Aldershot, England: Ashgate Publishing.

Violato, C., & Travis, L. (1995). Advances in Adolescent Psychology. Calgary, Canada: Del Selig.

*Vitelli, R. (1997). Comparison of early and late start models of delinquency in adult offenders. International Journal of Offender Therapy and Comparative Criminology, 41(4), 351-357.

*Waldstein, S. R., Malloy, P. R., Stout, R., & Longabaugh, R. (1996). Predictors of neuropsychological impairment in alcoholics: Antisocial versus nonantisocial subtypes. Addictive Behaviors, 21(1), 21-27.

Wastell, C. A. (1996). Feminist developmental theory: Implications for counseling. Journal of Counseling & Development, 74(6), 575-581.

*Weiler, B. L., & Widom, C. S. (1996). Psychopathy and violent behaviour in abused and neglected young adults. Criminal Behaviour and Mental Health, 6, 253-271.

Westen, D., & Arkowitz-Westen, L. (1998). Limitations of Axis II in diagnosing personality pathology in clinical practice. American Journal of Psychiatry, 155(12) 1767-1771.

Weston, S. C., & Siever, L. J. (1993). Biologic correlates of personality disorders. Journal of Personality Disorders, 1, 129-148.

Widom, C. S. (1997). Does violence beget violence? A critical examination of the literature. Psychological Bulletin, 106 (1), 3-28.

Widiger, T. A. (1997). Personality disorders as maladaptive variants of common personality traits: Implications for treatment. Journal of Contemporary Psychotherapy, 27(4), 265-282.

Widiger, T. A., & Shea, T. (1991). Differentiation of Axis I and Axis II disorders. Journal of Abnormal Psychology, 100(3), 399-406.

Widiger, T. A., & Trull, T. J. (1992). Personality and psychopathology: An application of the five-factor model. Journal of Personality, 60(2), 363-393.

Wiggins, J. S., & Pincus, A. L. (1989). Conceptions of personality disorders and dimensions of personality. Psychological Assessment: A Journal of Consulting and Clinical Psychology, 1, 305-316.

Wishnie, H. (1977). The impulsive personality: Understanding people with destructive character disorders. New York: Plenum Press.

Wolf, F. M. (1986). Meta-analysis: Quantitative methods for research synthesis. Beverly Hills: Sage.

Wortman, P. M. (1983). Evaluation research: A methodological perspective. Annual Review of Psychology, 34, 223-260.

Yeung, A. S., Lyons, M. J., Waternaux, C. M., Faraone, S. V., & Tsuang, M. T. (1993). The relationship between DSM III personality disorders and the five-factor model of personality. Comprehensive Psychiatry, 34(4), 227-234.

Zanarini, M. C., & Gunderson, J. G. (1997). Differential diagnosis of antisocial and borderline personality disorders. In D. M. Stoff, J. Breiling, & J. D. Maser (Eds.), Handbook of antisocial behavior (pp. 83-91). New York: John Wiley & Sons.

*Zanarini, M. C., Gunderson, J. G., Marino, M. F., Schwartz, E. O., & Frankenburt, F. R. (1989). Childhood experiences of borderline patients. Comprehensive Psychiatry, 30(1), 18-25.

*Zanarini, M. C., Gunderson, J. G., Marino, M. F., Schwartz, E. O., & Frankenburt, F. R. (1990). Psychiatric disorders in the families of borderline outpatients. In P. S. Links (Ed.), Family environment and Borderline Personality Disorder. Progress in psychiatry series (pp. 69-84). Washington, DC: American Psychiatric Press.

*Zoccolillo, M., Pickles, A., Quinton, D., & Rutter, M. (1992). The outcome of childhood Conduct Disorder: Implications for defining adult personality disorder and Conduct Disorder. Psychological Medicine, 22, 971-986.

Appendix A

Diagnostic Criteria for Antisocial Personality Disorder

A. There is a pervasive pattern of disregard for and violation of the rights of others occurring since age 15 years, as indicated by three (or more) of the following:

(1) failure to conform to social norms with respect to lawful behaviors as indicated by repeatedly performing acts that are grounds for arrest

(2) deceitfulness, as indicated by repeated lying, use of aliases, or conning others for personal profit or pleasure

(3) impulsivity or failure to plan ahead

(4) irritability and aggressiveness, as indicated by repeated physical fights or assaults

(5) reckless disregard for safety of self or others

(6) consistent irresponsibility, as indicated by repeated failure to sustain consistent work behavior or honor financial obligations

(7) lack of remorse, as indicated by being indifferent to or rationalizing having hurt, mistreated, or stolen from another

B. The individual is at least 18 years.

C. There is evidence of Conduct Disorder with onset before age 15 years.

The occurrence of antisocial behavior is not exclusively during the course of Schizophrenia or a Manic Episode.

Appendix B

Coding Scheme

Variable	Column	Data Representation
ID Number of Study	36893	1 - 53
Year of Study	36988	1 = 1962 - 1989 2 = 1990 - 1999
Country of Study	37144	1 = USA 2 = Britain 3 = New Zealand 4 = Canada 5 = Sweden 6 = Finland 7 = Australia 8 = Netherlands 99 = Not Reported
Average Age	37238	1 = 8 - 17 2 = 8 - 47 99 = Not Reported
Total Subjects	15-21	10 - 200,000
Total Subjects	22-24	99 = Not Reported
Males	25-31	10 - 200,000
Females	32-38	10 - 200,000
Ethnicity - Caucasian	39-45	10 - 200,000
Ethnicity - Black	46-52	10 - 200,000
Ethnicity - Hispanic	53-59	10 - 200,000
Ethnicity - Other	60-66	10 - 200,000
Ethnicity - Not Reported	67-73	10 - 200,000

(continues)

Coding Scheme *(continued)*

Study Groups	74-76	1 = Clinical 2 = Comparison 3 = Experimental 4 = Control 99 = Not Reported
Study Design	77-79	1 = Observational 2 = Correlational 3 = Unobtrusive 4 = Experimental - Between Groups 5 = Experimental - Within Subjects 6 = Experimental - Complex Interaction 7 = Quasi-experimental 99 = Not Reported
Diagnostic Indices	80-82	1 = Observation 2 = Interview 3 = Questionnaire 4 = Standardized Tests 5 = Physiological Measures 6 = Other 7 = Mixed 99 = Not Reported
Method of Assessment	83-85	1 = Psychometric Assessment Criteria 2 = DSM Assessment Criteria 3 = Researcher's Criteria 4 = Mixed Criteria 99 = Not Reported
Study Type	86-88	1 = Retrospective 2 = Prospective 3 = Cross-Sectional 4 = Cross-Sequential 99 = Not Reported
Psychobiological Domain	89-90	1 = Genetics 2 = Temperment 3 = IQ 4 = Childhood Disorders/Pathology

(continues)

Coding Scheme *(continued)*

Experiential/External Domain	91-92	1 = Physical Abuse 2 = Sexual Abuse 3 = Emotional Abuse 4 = Separation and Loss 5 = Poor School Achievement 6 = Medical Incident History 7 = Socio-economic Status 8 = Family Dysfunction/Breakdown
Interpersonal Domain	93-94	1 = Neglect 2 = Parental Rejection 3 = Unstable Parenting 4 = Inconsistent Discipline 5 = Harsh Parenting 6 = Parent Delinquency/Criminality 7 = Delinquent or Behavior Problem Sibling 8 = Parent Disorder/Pathology 9 = Social Desirability
Behavioral Domain	95-96	1 = Child Antisocial Behavior