

HOSPITALIZATION FOR MENTAL ILLNESS IN A BIRTH COHORT:
PREVALENCE AND RISK FROM CHILDHOOD TO EARLY ADULTHOOD

by

SPOMENKA CALIC NEWMAN

(Under the Direction of Roy P. Martin)

ABSTRACT

The current study focused on prevalence and risk of hospitalization for mental illness in young people. A birth cohort of 6401 subjects born between 1975 and 1976 was followed from birth to age 22, using the Finnish National Registry data. Mental health data were gathered longitudinally, using standardized diagnostic assessment and hospital admission records. Several significant findings emerged as a result of this study: The number of subjects hospitalized for mental illness increased longitudinally from childhood to early adulthood, subjects with chronic manifestations of mental illness showed significantly higher functional impairment than subjects hospitalized in only one developmental period, a significant portion of subjects hospitalized in childhood and early adolescence were hospitalized again before reaching adulthood. Hospitalized subjects were more likely to be male, from low socio-economic status, born to young mothers, and to multiparous mothers. With the exception of male gender, risk factors and functional impairment did not distinguish between subjects hospitalized at different developmental periods.

INDEX WORDS: Child and Adolescent, Mental Disorders, Epidemiology, Hospitalization.

HOSPITALIZATION FOR MENTAL ILLNESS IN A BIRTH COHORT:
PREVALENCE AND RISK FROM CHILDHOOD TO EARLY ADULTHOOD

by

SPOMENKA CALIC NEWMAN

B.S., University of Ljubljana, Slovenia, 1983

M.S., University of Ljubljana, Slovenia, 1989

Ph.D., University of Georgia, 1994

A Dissertation Submitted to the Graduate Faculty of The University of Georgia in Partial
Fulfillment of the Requirements for the Degree

DOCTOR OF PHILOSOPHY

ATHENS, GEORGIA

2004

© 2004

Spomenka Calic Newman

All Rights Reserved

HOSPITALIZATION FOR MENTAL ILLNESS IN A BIRTH COHORT:
PREVALENCE AND RISK FROM CHILDHOOD TO EARLY ADULTHOOD

by

SPOMENKA CALIC NEWMAN

Major Professor: Roy Martin
Committee: Bonnie Cramond
Linda Campbell
Jonathan Campbell

Electronic Version Approved:

Maureen Grasso
Dean of the Graduate School
The University of Georgia
August 2004

DEDICATION

To Bob Newman

The flight to Boston had gone well, much better than I had dared to hope it would, and I had walked off the plane feeling that I had accomplished something important. In the big scheme of things, it probably wasn't much, but in the small scheme of things, in the microscopic place where private battles are won and lost, it counted as a single victory. I felt stronger than I had felt at any time in the past three years. Almost whole, I said to myself, almost ready to become real again.

P. Auster: The Book of Illusions

ACKNOWLEDGEMENTS

This work could not have been accomplished without the guidance of my advisor, Dr. Roy Martin. Thank you for your support and encouragement at those times when it was so needed. I thank my committee members for their guidance in completing this work: Dr. Bonnie Cramond who has been my mentor and friend for almost fifteen years, Dr. Linda Campbell who taught me wonders of therapy, and Dr. Jonathan Campbell who provided many valuable comments to improve this project. Thank you all.

And thank you Bob. You have been my faithful companion on this long journey. Your intellectual curiosity, knowledge, and capabilities continue to amaze me. You inspire my quest for learning. This is dedicated to you.

TABLE OF CONTENTS

	Page
ACKNOWLEDGEMENTS	v
LIST OF TABLES	viii
CHAPTER	
1 INTRODUCTION	1
2 LITERATURE REVIEW	6
Epidemiology and Risk in Child Psychopathology.....	8
Risk Factors for Development of Disorders.....	12
Methodology and Design in Research on Prevalence and Risk.....	26
Implications and Future Research	34
3 METHOD	38
Subjects	38
Risk Factors	40
Diagnoses	42
Definitions	42
Procedure.....	43
Data Analyses.....	44
4 RESULTS	45
Descriptive Statistics	45
Hypotheses Testing	51

5	DISCUSSION.....	75
	Rationale for the Study.....	75
	Summary of Results	76
	Strengths of the Study	81
	Limitations of the Study	81
	Practical Implications and Future Research	83
	Conclusions	85
	REFERENCES	86
	APPENDIX.....	97

LIST OF TABLES

	Page
Table 1: Mental Disorders and ICD-9-CM Codes	39
Table 2: Demographic Data for the Birth Cohort	41
Table 3: Demographic Data for Hospitalized Subjects.....	46
Table 4: Demographic Data for Subjects Hospitalized Before the Age of 15, at Ages 16 To 22, and in Both Periods	47
Table 5: Hospitalization and Diagnoses Data for Subjects Hospitalized Before the Age of 15, at Ages 16 to 22, and in Both Periods.....	48
Table 6: ICD-9-CM Diagnoses for Subjects Hospitalized Before the Age of 15, at Ages 16 to 22, and in Both Periods.....	50
Table 7: Differences in Gender Between the Cohort and the Sample	52
Table 8: Differences in Socio-economic Status Between Cohort and Sample	53
Table 9: Differences in Mother's Age Between Cohort and Sample	54
Table 10: Differences in Father's Age Between Cohort and Sample	56
Table 11: Differences in Mother's Parity Between Cohort and Sample	57
Table 12: Gender Differences in Subjects Hospitalized Before the Age of 15 and at Ages 16 to 22	59
Table 13: SES Differences in Subjects Hospitalized Before the Age of 15 and at Ages 16 to 22	60

Table 14: Differences in Mother’s Age Between Subjects Hospitalized Before the Age of 15 and at Ages 16 to 22.....	62
Table 15: Differences in Father’s Age Between Subjects Hospitalized Before the Age of 15 and at Ages 16 to 22.....	63
Table 16: Differences in Mother’s Parity Between Subjects Hospitalized Before the Age of 15 and at Ages 16 to 22.....	65
Table 17: Differences in Number of Hospitalizations in Subjects Hospitalized Before the Age of 15 and at Ages 16 to 22.....	66
Table 18: Differences in Number of Diagnoses in Subjects Hospitalized Before the Age of 15 and at Ages 16 to 22.....	67
Table 19: Differences in Number of Hospitalizations Between Subjects Hospitalized Before the Age of 15 and Subjects Hospitalized in Both Periods	69
Table 20: Differences in Number of Diagnoses Between Subjects Hospitalized Before the Age of 15 and Subjects Hospitalized in Both Periods	70
Table 21: Differences in Number of Hospitalizations in Subjects Hospitalized at Ages 16 to 22 and Subjects Hospitalized in Both Periods.....	72
Table 22: Differences in Number of Hospitalizations in Subjects Hospitalized at Ages 16 to 22 and Subjects Hospitalized in Both Periods.....	74

CHAPTER 1

INTRODUCTION

According to a report by the Institute of Medicine (1989) psychopathology is increasingly occurring in children. The Global Burden of Disease Study published by the World Health Organization predicted that by the year 2020 neuropsychiatric disorders in children will become the fifth most common cause of disability, morbidity, and mortality among children internationally (Murray & Lopez, 1996). This predicted rate surpasses any other cause of morbidity and mortality in children. In his report “Call to Action for Children’s Mental Health” the Surgeon General focused on a breadth of childhood mental illnesses, pointing to the need for increased national attention (US Department of Health and Human Services Report, 2000).

The review of epidemiology of childhood psychopathology across 20 countries estimates the average prevalence at 16% (Roberts, Attkisson, & Rosenblatt, 1998). According to a recent review of international studies, international psychopathology prevalence rates among children in Australia, Quebec and Finland are similar to those in the United States (Ringeirsen, Oliver & Menvielle, 2002). It is estimated that 9 to 13% of children in the United States experience significant psychopathology that requires treatment (Friedman, Katz-Leavy, & Graetz, 1996). Brandenburg, Friedman, and Silver (1990) have estimated that 14% to 22% of all children can be classified as having developmental, behavioral and emotional disorders. According to Rutter (1989) and Boyle, Offord, Hoffman, Catlin, Byles, Cadman, Crawford, Links, Rae-Grant, and Szatmari (1987), severe forms of diagnosable disorders are estimated to be present in 8% to 10% of children. Kazdin and Kagan (1994) found that 17 to 22% of adolescents had some type of

psychiatric disorder. If a subclinical population at risk for developing problems in the future is included in the estimate, only 34.4% of children in a national sample can be classified as adjusted (McDermott & Weiss, 1995).

The costs associated with the occurrence of mental illness in childhood include long-term risks and consequences to the individuals and families involved, economic cost caused by increasing needs for community resources in schools, mental health facilities, the criminal justice system, and decreased productivity. Symptoms of childhood mental illness negatively impact school performance and peer relationships, two critical areas of a child's functioning (Ringeirsen, Oliver, & Menvielle, 2002). According to Mash and Dozois (1996) a significant proportion of children with early manifested difficulties is likely to express difficulties over time in various forms and levels of severity. For example, aggressiveness in boys is highly predictive of antisocial behavior and other negative outcomes in adolescence and adulthood (Hinshaw, Lahey, & Hart, 1993). Feehan, McGee, and Williams, (1994) reported that two-thirds of adolescents with a disorder at age 15 still have the disorder at age 18. Continuation into adulthood and long term consequences of conduct disorder have been confirmed by numerous studies (Feehan et al., 1994; Kazdin & Kagan, 1994; McGee, Willams & Feehan, 1994). Furthermore, negative effects of early behavioral problems are evident in later adjustment, even when the problems manifested are not severe enough to be diagnosed as mental illness.

It is estimated that in the future, numbers of children affected by mental illness will significantly increase. These estimates are based on increased chronic poverty, family break-up, single parenting, neglect and abuse, immigrant families, HIV, alcohol and drug abuse, prematurity and higher rates of fetal survival, which carry greater risk for behavioral and learning disorders in earlier age (National Commission on Children, 1991). Unfortunately,

empirically validated prevention and intervention programs are not available for many disorders for this age group, and 70% of children needing services do not receive them (Kazdin & Kagan, 1994).

Despite increased research, questions about continuity and discontinuity of disorders over time and various manifestations of behaviors are still core issues in developmental psychopathology (Essau, Pettermann & Feehan, 1997). More information is needed regarding prevalence rates and risk factors as well as the choice of treatments with children and adolescents. According to Steinhausen and Velhurst (1999) major questions related to outcomes of psychiatric disorders that originated in childhood are: To what extent do certain disorders remit? How do they transform to adult disorders? What are the risk factors for stability of disorders from childhood to adulthood? Do processes between child and adult psychopathology more reflect continuity or discontinuity of psychopathology? The major aim of developmental psychopathology is to understand “processes and mechanisms underlying how and why psychopathology in children emerges, how it changes over time, and how it is influenced by the child’s developmental capacities and by the context in which development occurs” (Mash & Dozois, 1996, p. 36). The issue of continuity of disorders and the relationship between childhood and adulthood disorders represents a central focus of the theory and empirical research in child psychopathology (Cicchetti & Cohen, 1995; Rutter & Rutter, 1993; Sroufe & Jacobovitz, 1989).

Investigations of continuity and change of psychopathology may be conducted at several levels of generality, from presence/absence of pathology, to broad subtypes (e.g., internalizing and externalizing problems), to specific syndromes (e.g., phobia). Studies of general psychopathology have confirmed considerable stability from adolescence to young adulthood (Ferdinand & Velhurst, 1995) and continuity of internalizing and externalizing problems

(Achenbach, Howell, McCounaughy, & Stanger, 1995; Ferdinand & Velhurst, 1995). It can be concluded that there is a considerable stability of psychopathology from adolescence to adulthood, and that psychopathology in adolescence increases risk for psychopathology in young adulthood for most syndromes.

Less is known about stability of psychopathology identified before adolescence and the risk it represents for early adulthood. The implications of such findings could lead to prevention and intervention programs targeting younger children, before pathological pathways become stable. Prospective investigations of prevalence, risk, stability, and change are needed when studying psychopathology longitudinally, from early childhood to early adulthood.

This study focuses on three general questions: What are the differences between the general population and hospitalized population? What are the differences between subjects hospitalized in childhood and adolescence and those hospitalized in early adulthood? What are the differences between subjects hospitalized in one developmental period and subjects hospitalized in more than one developmental period? It was hypothesized (hypotheses one to five) that hospitalized subjects are not significantly different from normal population with regard to gender, socio-economic status, mother's age at birth, father's age at birth, and mother's parity (first time mothers versus at least one child). It was further hypothesized that subjects hospitalized before the age of 15 are not significantly different from those hospitalized between ages of 16-22 with regard to gender, socio-economic status, mother's age at birth, father's age at birth, mother's parity (primiparous versus multiparous), a number of hospitalizations, and number of diagnoses (hypotheses six to nine). Finally, it was hypothesized that subjects hospitalized in both periods are not significantly different from those hospitalized either before

the age of 15 or at ages 16-22 with regard to a number of hospitalizations and a number of diagnoses.

CHAPTER 2

LITERATURE REVIEW

Current interest in childhood psychopathology reflects growing awareness that adult psychopathology can often be traced to childhood and that better understanding of developmental aspects of disorders may lead to better prevention of and intervention in mental illness. Rutter (1986) argued that a developmental perspective is not only the desired approach to child psychiatry, but to adult psychiatry as well. Although not all adult psychopathology has childhood origins, processes of development as they occur in childhood and adulthood should nonetheless be considered. What is currently known about child psychopathology is to a large degree extrapolated from empirical findings and theory on adult psychopathology. This has many adverse effects on diagnosis and treatment (Mash & Dozios, 1996). Are the disorders diagnosed in childhood predictive of the same disorders seen in adulthood? Are the disturbances seen in infants and toddlers a risk for disorders manifested in adult life? Does psychopathology seen at different ages represent the same or independent entities? According to Cytryn, McKnew, and Zahn-Waxler, (1986), these are the most important and the most difficult questions in the field of developmental psychopathology.

The current level of knowledge of child psychopathology is characterized as unsystematic and atheoretical, even though the knowledge base of early manifestations of mental illness has increased more over the last decade than in any other time in history (Cicchetti & Cohen, 1995). Absence of studies can be attributed to traditional views suggesting that children are less likely to suffer from mental illness because of neurological immaturity (Donohue,

Hersen, & Ammerman, 1995). For example, empirical research into childhood depression and attempts to develop theoretical models of mood related disorders in children have begun to appear only in the last decade (Ollendick, Mattis, & Neville, 1994). Prior to the 1970s, conventional wisdom assumed that children rarely exhibited depression, followed by the assumption that childhood depression and adulthood depression are etiologically and symptomatically the same. This view ignored the empirical evidence that frequency and manifestations of depressive disorders change with age (Rutter, 1986). Today there is not only awareness of early onset depression, but also of continuity between early onset and adult mood disorders (Lewinsohn, Rhode, Klein, & Seely, 1999). Supporting Rutter's (1986) findings of developmental differences in manifestations of early onset depression, a study by Harrington et al. (1990) reported that postpubertal major depression predicted adult major depression, while prepubertal major depression did not. Unlike several pervasive disorders of childhood, such as mental retardation or autism, many types of child psychopathology appear to be periodic, differing in manifestations over time. Changes may appear in frequency, comorbidity, severity, time of onset, and termination (Loeber & Farrington, 1994).

In general, the empirical evidence seems to point to continuity of externalizing problems over internalizing problems, but also to the differences in continuity within the externalizing group of problems (Mash & Dozois, 1996). For example, conduct disorder in boys is found to be relatively continuous, while some other behavioral problems are found to be more transient (Edelbrock, 1984). However, not enough is known about differences found within groups of internalizing disorders and about comorbid conditions.

Epidemiology and Risk in Child Psychopathology

Epidemiology has been defined traditionally as a study of frequency of pathology in a community, typically involving unselected subjects (Velhulst & Koot, 1992). According to Costello and Angold (1995), epidemiological studies aim to identify prevalence and distribution of disorders in a population of children that may vary in age, gender, socioeconomic status, race, and ethnicity. In addition, epidemiology can include study of the risk of developing a disorder; identifying and describing syndromes; describing natural progression of a disorder including onset, duration, recurrence, complications and disability; identifying factors that influence or predict the course of a disorder; identifying causes of a disorder; and, identifying methods of prevention and control (Von Kroff & Eaton, 1989). Even though a major strength of epidemiology is that it uses samples from the general population and thus contributes to greater generalizability of findings, epidemiology can also focus on clinical samples.

Developmental View of Prevalence and Risk

Interest in epidemiological research in child psychopathology has increased in the last two decades as a result of recognition that findings derived from child epidemiology have relevance for understanding adult psychopathology. According to Velhulst and Koot (1992), epidemiological research focuses on relationships between chosen factors and psychological conditions. Epidemiological research is probabilistic, predicting the occurrence of chosen phenomena in an identified population by quantifying risk for developing psychopathology or malfunction at a later age based on early presence or absence of characteristics and behaviors that are hypothesized to relate to disorders.

Risk-factor research contributes to understanding predictors or antecedents that lead to mental disorders, factors involved in relations between antecedents and outcomes, processes by

which they operate, and ways they can be altered (Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997). According to Kraemer, Kazdin, Offord, Kessler, Jensen, and Kupfer (1997), risk can be defined as a specific characteristic of a subject, experience or event in a specific population that precedes the outcome of interest. The strength of the risk factor is defined by the degree of difference in the outcome of a low-risk and high-risk group. The risk factor potency can be represented as an odds ratio, relative risk or attributable risk, and interpreted in terms of clinical and public health significance. According to Kazdin et al. (1997), the risk factor can be understood as a relational concept, characteristic, or experience that relates to an outcome. The risk factor can also be understood as a conditional concept that depends on duration, intensity, timing of exposure, population (age, gender, ethnicity), presence or absence factors, and outcome characteristics (how is outcome defined and measured). Finally, the risk factor can be understood as a process concept that influences a person's life or systems within which a person functions (Kazdin et al., 1997).

Traditionally, risk factor research is used to study antecedent conditions that lead to adverse outcomes. In contrast to traditional approaches to research, the contemporary risk research emphasizes the conditional nature of risk factors. For example, the same antecedent can have different effects on the outcome depending on the level of exposure to a condition or depending on when the exposure to a condition occurs in a person's development (Rutter, 1965). Causal risk factors precede outcome, but they are not the same as a definitive cause which is a necessary and sufficient condition for development of a disorder (Kraemer et al., 1997).

The profile of risk-factors can also differ as a function of different characteristics or variations of the outcome, such as onset and course of the disorder (Kazdin et al., 1997). The same risk-factors may differentially predict the onset, course, maintenance, and remission of the

disorder (Warner, Kessler, Hughes, Anthony, & Nelson, 1995). Finally, the study of resilience in members of high risk groups indicates that the nature of outcomes is probabilistic, meaning that the risk factors influence likelihood rather than certainty of outcomes (Kazdin et al., 1997).

The nature of the subject of study in child psychopathology adds further complexity to the risk factor research. According to Kraemer et al. (1997), inherent in the risk factor research is an awareness that multiple risk factors and multiple paths may lead to an outcome. In studying children, complex organisms embedded in their natural environment during the process of development, one must take into account the presence of multiple influences and interactions that operate in different ways at different points in time. Risk factor research provides the bridge between conceptual complexity of studied phenomena and a need to reliably predict risk for developing a mental disorder under certain circumstances. The implications for clinical practice and policy that result from identifying such systematic sources of influence and antecedent conditions that are likely to lead to adverse mental health outcomes are numerous for individuals and society.

Risk factors can be categorized as organismic and environmental. Genetic factors are currently conceptualized as the major organismic risk factors (Rutter, 1986). Difficult temperament is recognized as one of the earliest risk factors identified in infancy and leading to a number of negative outcomes (Antony, Lonigan, Hooe, & Phillips, 2002; Prior, Smart, Sanson, & Oberklaid, 2001; Rothbart & Adadi; 1994). Most researchers have concluded that temperament variations are strongly influenced by genetic factors. Of those children rated by their mothers to have difficult temperament, 25% were identified to have adjustment problems 4 to 5 years later. When characteristics such as male sex are combined with the report of a difficult temperament, later adjustment problems increase to 44%. According to Werner and

Smith (1992) the most common environmental risk factors are acute environmental stress and chronic adversity, including poverty, parental psychopathology, parental death, family breakup, caregiving deficits, homelessness, community disaster, and perinatal stress.

However, not all children exposed to risk factors develop disorders. Competency and adjustment, as well as conditions, characteristics, and processes that act as protective factors amidst adversity have only recently become an interest of developmental psychopathology (Cicchetti & Garmezy, 1993). Resilience among children exposed to high risk environments shows that children may be resilient to some but vulnerable to other factors, leading to the conclusion that resilience as well as vulnerability may be domain, time, and context specific (Zimmerman & Arunkumar, 1994).

According to Werner and Smith (1982), an accumulation of risk factors can influence the outcome in both additive and synergistic ways. Some risk factors exert stronger influence than others, some risk factors exert a different influence at different developmental periods (Feehan et al., 1994), and some act only in the presence of other factors (Dodge, Pettit, & Bates, 1994). Early adversity and failure to meet a developmental task may set a child on a maladaptive path. However, turning points, including chance and environmental events, may alter the trajectory, leading to either functional development or dysfunction (Rutter, 1986). Because influences of risk factors, continuities and discontinuities, pathways, and processes involved in the onset and course of child psychopathology are interrelated, they require complex conceptual models and research designs (Kazdin & Kagan, 1994).

According to Kraemer et al., (1997) there are several levels in which characteristics, events, or experiences can relate to outcomes. A factor can be characterized as a causal risk factor only when alteration of this factor causes changes in outcome. An example of a causal risk

factor is child rearing practice, where hostility and physical punishment may lead to an increase in a child's aggressive behavior (Dodge et al., 1994). However, causal risk factors in child psychopathology tend to be multiple rather than single and tend to lead to multiple paths toward an outcome. A factor known to be a risk factor but not known to be a causal risk factor is defined as a marker. Fixed markers are nonmalleable risk factors such as sex. Variable markers are malleable or changeable factors, such as age or level of skill. If temporal ordering between factors cannot be defined and causal risk can not be inferred, then two factors associate with one another and the relationship between antecedent and outcome is considered to be correlational. Most risk factor research to date has focused on the causal role risk factors play in a particular outcome (Kraemer et al., 1997). Current research focuses on identifying components of risk factors and their relative contribution to the outcome as well as mechanisms of influence. For example, low SES is a risk factor for childhood onset of psychiatric disorders, but as findings by Dodge et al. (1994) indicate, harsh discipline is a causal risk factor for aggressive and antisocial behavior and it is often found in low SES environments. Identifying harsh discipline rather than poverty as a causal risk factor is more likely to lead to successful intervention.

Risk Factors for Development of Disorders

Age of Onset

Data suggests that 25% of children from preschool to pre-adolescence can be identified as having a significant mental health problem (Offord, Boyle, Racine, Fleming, Cadman, Blum, Byrne, Links, Lipman, MacMillan, Grant, Sanford, Szatmari, Thomas, & Woodward, 1992). Of this group, four in ten children are identified as having problems in more than a single developmental period, thus suggesting a high level of continuity of disorders in childhood and adolescence (Offord et al, 1992, McGee et al., 1994). Two thirds of those who report some kind

of disorder at age 15 are also diagnosed at age 18. When the reported presence of a disorder is used as a predictor, the continuity of disorders is high. Canadian epidemiology data suggest that overall prevalence for severe mental disorders, including both symptoms and impairment is 10-20%, and that the most common disorders affecting children and adolescents are anxiety, attention problems, conduct, and depressive disorders (Waddell, Offord, Shepherd, Hua, & McEwan, 2002).

Studies of prevalence and continuity of mental disorders in preschool years are rare. Studies have reported that one in five children in this age group show problems (Richman, Stevenson, & Graham, 1982), and that some of the problems are persistent (Campbell, 1989). Longitudinal influences of temperamental factors are confirmed by the Australian Temperament Project that followed children from infancy to ages 11-12 (Prior et al., 2001). The results showed that the strongest predictor of behavioral adjustment problems at ages 11-12 are previous behavioral problems, self-regulation capacities, and mother's overall ratings of child's difficulty. A study of 10 to 17 year olds by Anthony, Lonigan, Hooe, and Phillips (2002) demonstrated association between positive and negative temperament and positive and negative affectivity, which are central organizing dimensions of personality and useful in discriminating psychopathologies. The results pointed out that negative temperament is associated with anxiety and depression. According to Hirschfeld and Shea (1992), aspects of personality such as temperament represent developmental antecedents at a subclinical level of mental disturbance, while personality modulates presentation and course of mental illness.

The most commonly noticed problem behavior in early age is hyperactivity (McGee, Feehan, Williams, Patridge, Silva, & Kelly, 1990). Follow-ups of early identified hyperactive children point to significant mental health problems, although not necessarily ADHD, before the

age of 15 in 75% of the sample. This finding suggests that hyperactivity identified prior to entrance to school places children at greater risk for development of subsequent mental health problems. There is also a suggestion that ADHD-like symptoms identified before school entry represent a more persistent and pervasive problem than onset after school entry (McGee et al., 1990).

Another problem behavior commonly identified in preschool children is shyness (Kagan, Resnick, & Snidman, 1994). A study by McGee and Silva (1985) identified 6% of children in a sample of 3 year olds as shy. Shyness was accompanied by having difficulty separating from mother, being withdrawn, being negative, demanding attention, and being non-communicative. Similar behaviors to those identified at age 3 were also exhibited at age 5, thus suggesting a continuity of problems during this period.

Lack of control in the preschool years, including emotional control has also been widely studied. According to Caspi, Henry, McGee, Moffitt, and Silva (1995), lack of control in preschool was predictive of anxiety, depression, conduct problems, oppositional behaviors, and hyperactive behaviors in adolescence. This relationship is predictive for both sexes, but predicts externalizing behaviors in boys more strongly than in girls. However, preschool withdrawal and inhibition is more predictive of internalizing problems in adolescent girls.

According to the Dunedin Multidisciplinary Health and Developmental Study (McGee et al., 1994), during the school age period, 23% of boys and 12% of girls are identified with mental health problems, with 39% persistence over time. Family disadvantage represented a strong risk factor at this age, particularly maternal mental health, parental discord, and history of separation. Children from disadvantaged family backgrounds were 3.5 times more likely to be diagnosed with DSM-III disorders than children from normal backgrounds (McGee et al., 1994).

At the pre-adolescent period, externalizing disorders were twice as prevalent as internalizing disorders, and there were twice as many boys with disorders than girls. Of the 16% of pre-adolescent children with disorders, 44% were previously diagnosed with problems (McGee, Feehan, Williams, & Anderson, 1992). Results suggest that externalizing disorders tend to be more continuous than internalizing disorders, and that continuity tends to be greater for boys than for girls. Also, internalizing disorders for boys at age 11 were predictive of externalizing disorders at age 15, pointing to comorbidity of depression and antisocial behavior in boys (McGee & Williams, 1988). In the pre-adolescent period, family disadvantage was not predictive of continuity of disorder (Rutter, Tizard, Yule, Graham, & Withmore, 1976; McGee & Williams, 1988).

In adolescence, girls for the first time outnumbered boys in prevalence in all diagnostic categories except for ADHD, conduct disorder, and social phobia (McGee et al., 1990). Continuity from pre-adolescence to adolescence was significant; 56% of those diagnosed by the age of 15 already showed disorder at some earlier developmental period. This gender trend continues in early adulthood, with women outnumbering men for all disorders except substance dependence and abuse. In this age group, the most prevalent disorders were depression, social phobia, and alcohol dependence. Of those diagnosed at the age 15, 63% were again diagnosed at the age of 18. Comorbidity of disorders and symptom severity was higher than in earlier periods, as was the case for the pre-adolescent period, during adolescence family disadvantage did not differentiate between those with and without disorders in this age group (Feehan et al., 1994).

Comorbidity

High levels of comorbidity seem to be the most striking finding of child and adolescent epidemiological studies (Kashani, Carlson, Beck, Hooper, Corcoran, McAllister, Fallahi,

Rosenberg, & Reid, 1987). Comorbidity can be defined as manifestation of two or more disorders with greater co-occurrence than could be expected by chance alone. Comorbidity refers to the presence of multiple disorders, as well as mixed patterns of symptomatology within categories of specific disorders. According to Rutter (1989), until recently researchers did not take into account comorbidity in analyzing data. Most commonly found comorbidities in childhood and adolescence are between conduct disorders and attention deficit syndrome (Rutter, 1989), conduct disorders and drug abuse, anxiety and depression (Strauss, Last, Hersen, & Kazdin, 1988), and different types of anxiety disorders (Kashani et al., 1987). Meta analysis of six Canadian epidemiological studies showed overall comorbidity rates between 47% and 68% in children with mental disorders. In addition to having two or more mental disorders, children are also likely to experience more chronic physical health and school related problems (Waddell et al., 2002).

Achenbach (1991) argued that comorbidity is an artifact of methodology used to conceptualize mental disorder, rather than a reflection of mental illness in childhood. He attributed high comorbidity to categorizational approach and recommended using a dimensional approach instead. Verhulst and Van der Ende (1993) agreed that presence of comorbidity may be smaller than generally assumed and artificially produced, but they attributed comorbidity to a sampling bias. Referred individuals are more likely, than-non referred individuals, to exhibit comorbid disorders. For example, children with internalizing disorders who also manifest externalizing symptoms are more likely to be referred, than those children who manifest internalizing symptoms alone.

Multiple manifestations or comorbidity, and multiple pathways observed in child and adolescent psychopathology have great implications not only for the classification of disorders in

this period but also on developing prevention and intervention strategies. In addition to artificial sources of comorbidity, the presence of comorbidity can also be attributed to shared risk factors that can predispose child to more than one disorder, an overlap of risk factors between different disorders, or to increased vulnerability or risk for development of other disorders once disorder is present (Rutter, 1989). It is also possible that manifestations of one disorder may precede manifestation of another. For example, according to Barkley (1996), the presence of impulsivity may precede problems with attention.

Gender

Empirical research strongly supports the notion that gender is a critical factor in understanding development, manifestations, and outcomes of childhood disorders (Kavanagh & Hops, 1994). However, methodological problems weaken the usefulness of its contribution. Gender differences in psychopathology have been historically recognized, but research has primarily focused on descriptive comparisons of frequencies (Marsh & Dozois, 1996). Because girls have received less attention in empirical research (Eme, 1979), and many studies either excluded girls or did not control for gender, the knowledge base in some areas of psychopathology, such as behavioral disorders, is largely built on findings in boys. The base rates of behavioral disorders are inferred from the studies on boys, diagnostic criteria are derived and validated on boys, and research is influenced by referral and sampling bias (Spitzer, Davies, & Barkley, 1990).

With the exception of internalizing disorders, which seem to be more prevalent in girls (Achenbach, 1991), epidemiological research points to a larger prevalence of total disorders in boys, including overcontrolled, undercontrolled, and culture-specific disorders (Weisz & Suwanlert, 1989; Olweus, 1979), and externalizing problems (Achenbach, 1991).

Developmentally, boys show more behavioral problems prior to adolescence, while girls show a larger prevalence of mood disorders from mid-adolescence through adulthood (Boyle et al., 1987). An Australian national survey of mental illness confirmed that boys are more likely to have mental illness than girls, 19% versus 10% (Sawyer, Arney, Baghurst, Clark, Graetz, Kosky, Nurcombe, Patton, Prior, Raphael, Rey, Whaites, & Zubrick, 2001). Boys are at greater risk for mental disorder, but risks for boys are greater in younger ages, while risks for girls are greater later (Waddell et al., 2002). However, because gender differences in externalizing and internalizing problems are studied in referred samples, these findings may be confounded by the failure to control for gender referral bias.

Even though gender differences are a consistent finding in research on the prevalence of psychopathology in children, just like SES, these differences tend to account for a small portion of variance (Mash & Dozois, 1996). According to Hops (1995), pathways from childhood to adulthood psychopathology are age and gender specific, reflecting the different social contexts and expectations that nurture different characteristics of adaptation and maladaptation in boys and girls. Early signs of aggression predict antisocial behavior in boys, but not in girls (Tremblay, Masse, Perron, LeBlanc, Schwartzman, & Ledingham, 1992). These findings are supported by Zahn-Waxler, Cole, Welsh, and Fox (1995) who found that disruptive behavior in girls may be more related to anxiety than in boys. The disruptive girls as compared with boys showed higher electrodermal responding and were highly activated by induction of a sad mood.

Information provided by research on gender is an important contributor to the knowledge base in child psychopathology, but more important than knowing how psychopathology is manifested differentially in boys and girls is to know how similar environments differentially influence developments of adaptive and maladaptive patterns of behaviors. At a minimum,

interpretation of empirical findings must be sensitive to the interaction between biological differences and differential socialization as factors influencing gender effects. Differential effects of environment on development of problems in boys and girls are confirmed in research on development of risk and resilience. Resilient girls are more likely to come from households where risk-taking and independence is supported by a female member, while resilient boys tend to come from households with a male model (Werner et al., 1995). A study by Gelfand and Teti (1990) found that girls are at higher risk than boys for developing internalizing disorders in the presence of maternal depression, while boys are at higher risk for developing internalizing disorders in the presence of paternal avoidant behavior (Katz & Gottman, 1993).

Adolescence is marked by changes in patterns of disorders, particularly evident in sex distribution. For example, a two fold risk of depression in females and prevalence similar to adults emerge around ages 12-14 (Duffy, 2000). In contrast to the number of boys with disorders in childhood and pre-adolescence, more girls than boys are diagnosed with DSM-III disorders in adolescence. Of those diagnosed, 56% with disorder at age of 15 were identified in earlier developmental periods, twice the rate in the rest of the sample, thus suggesting continuity of disorder. Community sample studies point to continuity between postpubertal depression and adult major depression, but not prepubertal depression. According to McGee et al. (1994), in early adulthood 37% of the sample was diagnosed as having a DSM-III-R disorder, of which 13.3 % was a past major depressive episode, 11.15% social phobia, and 10.4% alcohol dependence. More women were diagnosed, except with alcohol and marijuana dependence. Like in previous periods, continuity was high with 63% of diagnosed disorders at age 18 being diagnosed at age 15. Seven in ten adolescents with comorbid diagnosis at age 15 were identified in early adulthood.

The future task for research on gender and mental illness is to develop an understanding of the processes underlying gender differences. For example, the finding that girls are less likely than boys to manifest disruptive behavior, but more likely to show comorbidity with anxiety, could be explained by interpersonal sensitivity often found in girls. The same characteristic, sensitivity, might be acting as a protective factor from developing disruptive behavior, and as a risk factor for developing internalizing disorders (Zahn-Waxler et al., 1995).

Socio-economic Status

Empirical studies have shown repeatedly that exposure to poverty during development has numerous implications on various aspects of children's development (Huston, 1994; Huston, McLoyd, & Garcia Coll, 1994, Sawyer, Arney, Baghurst et al., 2001, Waddell et al., 2002). The number of economically disadvantaged children has increased in contemporary American society (Luthar, 1997). The United States Bureau of the Census estimated in 1993 that 20% or more of children in the United States live in poverty, while the Institute of Medicine (1989) estimated that 20% of children growing up in inner-city poverty suffer some degree of impairment in social, behavioral, and academic functioning.

The effects of SES on development and functioning stem from numerous potential sources of adverse influence compounded in a complex variable. For example, low SES is associated with adverse factors such as low parental education, low occupational status, limited financial resources, single parenthood, poor nutrition, exposure to violence, and high levels of stress. Because the concept of what it means to be disadvantaged is so broad, empirical research using one or two indexes of SES cannot account for by the complex web of relationships among many factors that are interacting within the concept of SES (Mash & Dozois, 1996).

Despite its conceptual complexity, SES is a widely used variable in developmental research. It is one of the most important predictors of socio-emotional functioning and the best single predictor of cognitive competence (Sameroff, Seifer, & Bartko, 1997). Reports on the relationship in children between low SES and psychopathology date as early as 1958 (Hollingshead & Redlich, 1958). More recently, Achenbach (1991) reported on an increased level of parent-reported behavior problems and syndromes in low SES families as compared to high SES families. However, empirical findings show that SES differences accounted for less than 1% of variance, thus SES might be of questionable clinical significance, despite its statistical significance. An Australian survey of child mental health confirmed a higher proportion of mental health problems in children living in single parent families, blended families, low income families, and with parents who were not employed or had left school at an early age (Sawyer et al., 2001). Low family income also showed association with increased risk for mental illness in children in Canadian epidemiological studies (Waddell et al., 2002). Research on the relationship between child abuse, more prevalent in low SES families, and psychopathology testifies to the importance of controlling for SES. Findings by Mash, Johnston, and Kovitz (1983) show that abused children exhibit a higher level of externalizing problems than non-abused children. However, the effect of social class is largely absent from the relationship between abuse and internalizing disorders (Okun, Parker, & Levendosky, 1994), and family characteristics at age 15 do not differentiate between those who do and do not show disorder at age of 18 (Feehan et al., 1994). According to Duffy (2000), risk for depression associated with social class can largely be attributed to increased risk of severe life events and at least one more vulnerability factor. Bronfenbrenner (1986) has suggested focusing more on interpretation of processes that are connecting low SES and adverse outcomes, rather than SES

itself. For example, adverse events in childhood may alter neuroendocrine functioning in individuals genetically predisposed for depression and reduce their resilience, creating vulnerability to effects of increased stress (Hammen, 1991). Furthermore, individuals with depression not only respond to life stress, but also generate it (Duffy, 2000). Several theoretical models conceptualize how disadvantaged environment influences development and negative psychosocial outcomes (Luthar, 1997).

A model by Post (1992) linked psychosocial and biological factors postulating that life stressors, often associated with low SES environments, may alter gene expression in individuals vulnerable to depression, resulting in permanent neural changes, increased vulnerability to subsequent recurrence of depression, and formation of autonomous mechanisms responding to lesser stress. Other models postulate disruption in biological and psychosocial routines associated with life stress, rather than the psychological threat posed by the event, increased the risk for mental illness (Malkoff-Schwartz, Frank, Anderson, Sherrill, Siegel, & Patterson, 1998).

Cohler, Stott, and Music (1995) conceptualize the effects of SES as multifaceted, ranging from quality of housing and nutrition, to beliefs and attitudes toward self, others, and the future. Guerra, Tolan, Huesmann, Van Acker, and Eron (1995) emphasized the importance of stressful life events and acceptance of aggression. According to Rutter (1979), factors most often associated with socio-economic disadvantage are low parental education and occupation, large family size, single parenthood, and minority status. A model proposed by McLoyd (1989) and supported by numerous empirical studies (Brody, Stonemna, Flor, McCrary, Hastings, & Conyers, 1994; Conger, Conger, Elder, Lorenz, Simons, & Whitback, 1992; Dodge et al., 1994; Harnish, Dodge, & Valente, 1995; Sampson & Laub, 1994) hypothesized that the quality of

parenting, an indirect factor, influences maladaptation in children who are growing up in poor families.

Cumulative models of risk have proposed that a total number of SES related risk factors better predict negative psychosocial outcomes, such as psychiatric disorders, than any single indicator of socio-economic disadvantage (Rutter, 1979). Numerous empirical studies give support to this model. In his study of 10 years olds, Rutter (1979) found that risk of psychiatric disorder rose from 2% in families with no risk factor or a single risk factor, to 20% in families with four or more risk factors. Risk factors included were severe marital distress, low SES, overcrowding, paternal criminality, maternal psychiatric disorder, and admission of the child to foster care. Research supports Rutter's notion that synergistic effects of poverty exceed effects of any one of the single factors. Williams, Anderson, McGee, and Silva (1990) found a strong relation between the number of SES risk factors and behavioral disorders. They concluded that children with less than two disadvantages have 7% increase in risk, but children with eight or more disadvantages have a 40% increase in risk. Their study included risk factors such as residence or school change, low SES, single parenthood, marital separation, young motherhood, low maternal cognitive ability, poor family relationships, search for marriage guidance, and maternal mental health problems. A study by Sameroff, Seifer, Zax, and Barocas (1997) confirmed that a combination of risk factors in contrast to the effects of a single variable had a threefold increase in leading to adverse social-emotional outcomes. Despite overwhelming evidence supporting the association between SES and risk, according to Mash and Dozois (1996) a large number of empirical studies in child psychopathology fail to control for SES, therefore compromising the validity of findings.

Mother's Age and Parenting Experience

Increased risk of adverse outcomes of early pregnancies is well documented (Lee, Suhng, Lu, & Chou, 1998; Hack & Fanaroff, 1999). The disadvantage of early pregnancy continues for years after the child was born and adverse effects are found for both mother and child. Research has identified numerous consequences early pregnancy has on mothers, ranging from adverse social outcomes, partnership breakdowns, large family size, poorer household conditions, and higher risk of psychiatric morbidity (Maughan & Lindelow, 1997). Pregnant women under 20 are more likely to suffer from bleeding during pregnancy, toxemia, hemorrhage, difficult labor, severe anemia and disability (Monroy De Velasco, 1982). The yearly incidence of births to mothers under 20 is approximately 13 million. According to Monroy De Velasco (1982), the percentage of births given by mothers under 20 ranges from 20% in African and Caribbean countries, 10-15% in Latin American countries, and 5-10% in Asia. Survival of immature and low birthweight babies increased in the 1990s as a result of advanced medical technology and methods of care. As a result, neurodevelopmental handicaps and learning problems must be anticipated (Hack & Fanaroff, 1998). Behavioral difficulties, lower intelligence and lower academic performance at ages 5 to 8 are found in several studies (Hack, Breslau, Aram, Weissman, Klein, & Borawski-Clark, 1992).

When the risks of adverse outcomes of pregnancy in first time mothers ranging from ages 15 to 34 were compared, younger mothers had a higher percentage of low-birth babies and preterm births (Lee, Suhng, Lu, & Chou, 1998). Low birthweight is usually defined as 2.500 grams, preterm babies are usually defined as 37 weeks of gestation and less (Hack & Fanaroff, 1998). Younger adolescent mothers in all SES categories had a higher percentage of low weight babies and preterm births than older adolescent mothers. Low birthweight children have a higher

incidence of birth injuries, serious childhood illnesses, and physical and mental disabilities (Monroy De Velasco, 1982).

Research also points to more subtle effects low birthweight has on later cognitive, psychological and social functioning of the child well into adolescence (Fergusson & Woodward, 1999; Hack et al, 1992), and mental health and socioeconomic effects teenage pregnancy has on mothers (Maughan & Lindelow, 1997). Fergusson and Woodward (1999) studied the relationship between maternal age at birth and educational and psychosocial outcomes in children in a cohort of 1025 children in New Zealand. They found that children of teenage mothers have 1.5 to 8.9 times higher risk of adverse outcomes at the age of 18, than children of mothers over 30. Increased maternal age was associated with more nurturing, supportive, stable home environment, and declining risk of educational underachievement, juvenile crime, substance abuse, and mental health problems.

According to Lee et al. (1998), factors that influence adverse effects of early pregnancies are biological immaturity and unfavorable socioeconomic status of adolescent mothers. A review of the literature on consequences of early childbearing by Roosa, Fitzgerald and Carlson (1982) showed that children of adolescent parents exhibit poorer social and intellectual competencies. It is less clear how are these effects related to parenting practices and behavioral interactions between mothers and children. Numerous behavioral and environmental variables, rather than age by itself, are suggested to influence adverse outcomes. Among them are poor SES, family support systems, marital stability, nutrition, prenatal care (Roosa, Fitzgerald & Carlson, 1982).

A study by Maughan and Lindelow (1997) examined social, educational and behavioral precursors of adolescent pregnancies and effects they have on marital, social, and psychiatric

status of adolescent mothers in two cohorts of British teenage mothers. This study confirmed the relationship between socioeconomic and educational disadvantages and teenage pregnancy. The study identified the presence of conduct problems, but an absence of emotional problems prior to pregnancy. Study by Zoccolillo, Meyers, and Assiter (1997) confirmed the presence of psychiatric disorders in a sample of 26 adolescents who were either pregnant or just gave birth. The study found that a diagnosis of conduct disorder is associated with adolescent pregnancy, alcohol and drug dependence and poor pregnancy outcomes. Kessler, Berglund, Foster, Saunders, Stang, and Walters (1997) reported on association of early-onset psychiatric disorders in both males and females and subsequent teenage pregnancy. The diagnosis most predictive of early parenthood was addictive disorders. Anxiety, affective, and conduct disorders were also identified to be predictive of early pregnancy.

This review shows that early pregnancy carries a risk of later mental health problems for both mother and child. It has also identified prior mental health problems in teenage girls and boys as a risk factor for subsequent early pregnancy. Research suggests that numerous behavioral and environmental variables, rather than age itself, influence development of children of teenage mothers. According to Roosa et al (1982), in order to allow for the simultaneous study of organismic, environmental, and behavioral influences on child's development, research should shift from linear to multivariate models.

Methodology and Design in Research on Prevalence and Risk

Establishing boundaries between the normal and abnormal has been a historic task of theory and research in psychopathology; however, the lack of a gold standard to determine what is normal and what is pathological continues to be the major problem in psychiatric epidemiology (Ferdinand & Verhulst, 1999). The two nosological systems used by child and

adolescent psychopathology to classifying individuals with disorders are the dimensional and categorical approach. The empirically based dimensional approach to classification is a result of the use of multivariate statistical techniques and is usually associated with the Child Behavior Checklist and its derivatives (Achenbach, 1991). According to the dimensional approach, psychiatric problems are defined as deviations from normative data based on scores on symptom rating scales by multiple raters including parents, teachers, and self. Categorical approaches, such as the Diagnostic Statistical Manual and the International Statistical Classification of Diseases and Related Health Problems, rely on clinical consensus among professionals in determining criteria for disorders. Historically, categorical approaches have been more influential in psychopathology research than dimensional approaches.

Categorical Approaches to Classification of Disorders

The two dominant categorical approaches used for classification of childhood disorders are the Diagnostic Statistical Manual of Mental Disorders (DSM-IV, Text Revisions) developed by the American Psychiatric Association (2000) and the International Statistical Classification of Diseases and Related Health Problems (ICD-10) developed by the World Health Organization (1992). Following the categorical approach, a person is considered to have a disorder if criteria for diagnosis are fulfilled. The ICD-10 and DSM-IV were developed in close coordination between the World Health Organization and the American Psychiatric Association, resulting in fully compatible terms and codes (American Psychiatric Association, 2000).

The main argument for choosing the categorical over the dimensional approach in research is comparability of findings across studies. The lack of a common assessment approach has been cited as one of the weaknesses in research in developmental psychopathology, and it has hampered comparisons within longitudinal studies and across studies. However, studies

using categorical approaches have weaknesses as well. According to Velhurst (1995), the major problem attributed to categorical classifications is over inclusiveness of criteria resulting in higher prevalence rates. The critical problem, however, is the difficulty of controlling errors in clinical judgment, resulting in inconsistency in interpretation and application of diagnostic procedures. Nonetheless, traditional emphasis on symptoms and diagnosis continues to dominate the field of psychopathology.

According to Adelman (1995), classification systems shape the ways individuals are described, studied, and served; the ways professionals are trained and practice; and the ways policy is developed and practice is funded. Even when dimensional information is available it is still necessary, for practical purposes, to set cut-off scores and make decisions about normalcy versus disorder (Angold & Costello, 1991). Because of the practical significance of ICD and DSM nosologies in public and psychiatric practice, they are the starting point for most developmental epidemiology research. There should be no surprise that the majority of landmark epidemiological studies in developmental psychopathology have used the categorical approach: The Isle of Wight Studies (Rutter, 1976), The Dunedin Multidisciplinary Health and Developmental Study (Silva, 1990; McGee et al., 1994), The New Jersey County Study (Whitaker, Johnson, Shaffer, Rapoport, Kalokow, Walsh, Davies, Braiman, & Dolinsky, 1990).), The Ontario Child Health Study (Offord, Boyle, Racine, Fleming, Cadman, Blum, Byrne, Links, Lipman, MacMillan, Grant, Sanford, Szatmari, Thomas, & Woodward, 1992), The Puerto Rican Child Psychiatry Epidemiological Study (Bird, Canino, Rubio-Stipec, Gould, Ribera, Sesman, Woodbury, Huertas,-Goldman, Pagan, Sanchez-Lacay, & Moscoso, 1988), The Christchurch Health and Developmental Study (Fergusson, Horwood, & Lynskey, 1993), The Northeastern Study (Reinherz, Giaconia, Lefkowitz, Pakiz, & Frost, 1993), The New York Child Longitudinal

Study (Cohen, Cohen, Kasen, Velez, Hartmark, Johnson, Rojas, Brook, & Streuning, 1993), The Oregon Adolescent Depression Project (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993), Yale Family Study (Weissman, Fendrich, Warner, & Wickramaratne, 1992), and The Leckman study (Leckman, Merikangas, Pauls, Prusoff, & Weissman, 1983).

International Classification of Diseases

In the 10th edition of the International Classification of Diseases (World Health Organization, 1992), the disorders with onset in childhood and adolescence are classified in three groups: mental retardation, developmental disorders, and behavioral and emotional disorders with onset specific to childhood and adolescence. In contrast to the ICD-9, in ICD-10 differentiation between neurosis and psychosis is eliminated as the bases for sub-classification and replaced with the term “disorder.” Symptomatically relevant disorders, such as neurotic and psychotic disorder, have been grouped under “affective disorders.” Multiaxial classification is developed for children and adolescents separate from adults, including clinical psychiatric syndromes, specific disorders of psychological development, intellectual level, medical conditions, associated abnormal psychological situations, and global assessment of psychosocial functioning.

Although classifications in ICD-10 and DSM-IV are compatible, there are some differences between them, relating primarily to cross-cultural applicability issues (Essau et al., 1997). Unlike the DSM-IV criteria, criteria used in the ICD-10 does not take into account social consequences of disorders, such as significant impairment in social, occupational, and other areas of functioning. However, it is considered that ICD-10 has greater transcultural applicability than DSM-IV, since it has resulted from field trials and expert consensus world wide, while DSM-IV placed more reliance on US based empirical reviews (Frances, Pincus,

Widiger, First, Davis, Hall, McKinney & Stayna, 1994). Criticism remains for both systems regarding their lack of criteria for defining abnormal behaviors in different cultural backgrounds (Graham & Skuse, 1992), lack of developmental perspective on disorders (Chicchetti & Schneider-Rosen, 1986, Costello & Angold, 1995), and arbitrary thresholds for classifying presence or absence of disorders (Quay, 1986).

Research Design and Data Analysis

In general, the choice of research methodology depends on the aim and focus of the study, hypothesis, expected prevalence of disorders, and availability of data, time, money, and personnel (Essau, Peterman & Feehan, 1997). The central aim of epidemiological research is to develop an understanding of emergence and course of mental disorders (McGee, Feehan & Williams, 1995). According to Verhulst (1995) there is a growing awareness that epidemiological research can provide useful data on the distribution and co-occurrence of disorders and maladaptive behaviors in various populations. This information can then be used to develop selection criteria for distinguishing normal from clinical range, and to develop distinguishing criteria for disorders. The major tasks for future epidemiological research is to extend studies of children and adolescents into adulthood, and study referral pathways that lead to psychiatric care (Verhulst & Koot, 1992).

Three basic types of epidemiological studies are descriptive, analytic, and experimental (Boyle, 1995). Because of ethical and practical limitations, experimental epidemiological studies are rare (Verhulst, 1995). Most epidemiological studies are observational studies occurring in a natural setting without manipulation of factors by investigators. Descriptive observational studies are used when little is known about the phenomenon under investigation. Descriptive observational studies are used to provide estimates or base line of disorders in a defined

population. Analytical observational studies are employed when enough knowledge already exists to form a hypothesis to be tested. According to Verhulst (1995) analytical observational studies are used to test or generate a hypothesis about etiological factors and causal mechanisms. The aim of analytical observational epidemiological research is to identify groups at risk for development of a disorder by providing differential rates of disorder in different groups in the population.

The main research designs used in epidemiology are case-control design, cross-sectional design, and longitudinal designs. In case-control design subjects with the disorder are compared to those without the disorder, while matched for other relevant characteristics (Verhulst & Koot, 1992). Because this design relies on retrospective recollections and is vulnerable to bias, the use of secondary sources, such as medical charts, is recommended as a complement to the subjects' recollections. Cross sectional studies use assessment at one point in time, leading to conclusions about correlates rather than causes of disorder. The absence of temporal sequence between etiological factors and onset of psychopathology is the main weakness of cross sectional design. According to Loeber and Farrington (1994) and Rutter (1994a), findings of cross-sectional studies are limited because the design does not allow for control of developmental factors and can not establish causality of relationships between factors and outcomes. This design is useful in testing a hypothesis prior to engaging in longitudinal prospective studies (Rutter, 1994a).

In the longitudinal designs, groups of individuals with and without the disorder are followed for extended periods of time, or a group of individuals is selected and then followed. Longitudinal prospective studies provide multiple points of measurement and can establish causal relationships between risk factors and outcomes. A commonly used type of longitudinal prospective design is prospective cohort design. In this design a sample of subjects is assessed at

one point in time and reassessed after certain periods. If a cohort is represented by a community sample, this design allows one to study prevalence and continuity of disorders in the chosen population. If assessment includes etiological factors, prospective cohort longitudinal design makes it possible to identify causative mechanisms or risk factors (Verhulst, 1995).

The prospective longitudinal design is the strategy of choice for examining continuity of disorders from childhood to adulthood because it provides information on frequency, type of disorder, comorbidity, severity, and age of onset. Because the main interest in child psychopathology is an understanding of developmental processes and temporal sequences in which disorders develop, it is crucial to study variations over time between and within individuals. In addition to studying prevalence and persistence of psychopathology in the community at different periods, the longitudinal prospective design helps identify etiological factors and causative mechanisms. The longitudinal prospective design can help clarify the temporal sequence between etiological factors and the onset of psychopathology as well as factors involved in maintenance of disorders. Another advantage of longitudinal design over cross sectional is that it provides information on variation over time within and between individuals, whereas cross sectional design is limited to changes between individuals (Verhulst, 1995).

However, prospective longitudinal cohort studies are rare, and among the existing longitudinal cohort studies of child psychopathology very few span into adulthood (McGee et al., 1995). Such studies require coordination of child and adult methodology and assessment procedures appropriate for a range of age groups (Verhulst, 1995). Limitations of longitudinal studies include long delay from start to availability of first results, cost, selective attrition, and logistical difficulties and problems with comparing results of different assessments and

diagnostic systems (Rutter, 1994). According to Boyle et al. (1995) in studies of childhood psychopathology, attrition rates range from 25% to 30%. In studies of adolescent substance abuse losses may be up to 55%. Other problems with longitudinal research are controlling for informant bias and sampling problems, assuring the length of study to capture onset of disorders, distinguishing aging effects from cohort effects, and determining which correlates are the risk factors causing the development of disorder (Loeber & Farrington, 1994). Ideally, prospective longitudinal studies should involve children from birth to early adulthood, large enough samples to provide prevalence estimates, samples selected from the general population, and use of comparable assessments to allow comparisons over time (McGee et al., 1995).

Measures of risk are commonly used in studies based on categorical data. Data analyses of choice are measures of frequency and measures of risk. In cohort longitudinal studies using categorical data, the measures of risk used are the risk ratio and attributable risk. In case-control studies the commonly used risk measure is odds ratio, inferring to the likelihood of developing disorder in a group with specific etiological factors versus a group without the same factor. Prevalence measures identify frequency or proportion of population with disorder in the chosen population, while incidence measures refer to the rate of new cases of disorder during a specific time period (Verhulst, 1995). Attributable risk is the proportion of the risk for developing the disorder in the presence of a factor as a result of exposure to the risk factor (Verhulst & Koot, 1992). Prevalence can be conceptualized as probability of risk that an individual in a particular population will have the disorder (Verhulst, 1995). Incidence refers to a number of new cases developed in a population during a defined period. Incidence measures are not used in child psychopathology because childhood disorders do not have well defined onsets (Verhulst, 1995).

Implications and Future Research

Research shows that the prevalence of mental disorders in childhood and adolescence is high, but rates vary for disorders, age, gender, SES and ethnicity. Continuity of disorders from childhood to adulthood, lasting effects of disorders on individuals, family, and community, and increased risks caused by societal and family changes point to growing needs for services as early as indicated.

According to Verhulst and Koot (1991) longitudinal research should be the approach of preference for collecting information about prevalence of disorders, but to also inform prevention and treatment in child and adolescence psychopathology. Being aware of the number of children in need of services at a given point should aid communities in planning for mental health and special education services, as well as working on prevention programs with high risk populations (Verhulst, 1995). Kazdin and Kagan (1994) reported that 70% of children in need of treatment do not receive services, and empirically validated treatments for many disorders of childhood and adolescence are unavailable. Similar findings were confirmed by McGee et al. (1995). The National Plan for Research on Child and Adolescent Mental Disorders report points to the need for risk factor research in general and focusing on sequencing risk factors and interaction among different factors (Jensen, Koretz, Locke, Schneider, Radke-Yarrow, Richters, & Rumsey, 1993). Unavailability of empirically validated therapies for children and adolescents creates an even more urgent need to engage in research on developmental aspects of psychopathology to inform future prevention and intervention strategies. However, examples of direct influence of studies of prevalence and risk on planning and policy in the mental health area are rare (Verhulst, 1995, Waddell et al., 2002). Lack of clear standards and categories to distinguish normal children from disordered, different manifestation of disorders over time, high

comorbidity rates, and difficulty separating effects of development from effects of continuity and discontinuity of disorders, makes not only research but also policy and decision making difficult to implement.

The ultimate aim of epidemiological research is to inform clinical practice. Future research should try to unify methodology, standardize assessment, and introduce systematic methods of data collection (Waddell et al., 2002). According to Verhulst (1995) studies should extend to following subjects into adulthood to accumulate data that can be used to test outcomes of early identification of groups at risk for development of disorders, as well as effects of early prevention and intervention practices.

The purpose of the current study is to determine what are the differences between subjects who were hospitalized for mental illness from birth to age of 22 and normal population, what are the differences between subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22, and what are the differences in mental health between subjects hospitalized in both periods and subjects hospitalized in either period. Prospective longitudinal design is used in identifying mechanisms of risk and examining continuity of disorders from childhood to adolescence and adulthood. More specifically, the study is examining the relationship between gender, socio-economic status, mother's and father's age at birth, and mother's parity, and mental health outcomes. Mental health outcomes are assessed at ages 0-15 and 16-22 and defined as a number of hospitalizations for mental illness and number of mental health diagnoses given to subjects at the time of hospitalization. Based on the literature review it was hypothesized that being born to an adolescent mother or mother with no prior child rearing experience, being from low SES background, and being male is associated with higher risk for mental disorder.

Hypothesis I: There is no significant difference in gender distribution between the cohort and the hospitalized sample.

Hypothesis II: There is no significant difference in socio-economic status (SES) distribution between the cohort and the hospitalized sample.

Hypothesis III: There is no significant difference in mother's age at birth between the cohort and the hospitalized sample.

Hypothesis IV: There is no significant difference in father's age at birth between the cohort and the hospitalized sample.

Hypothesis V: There is no significant difference in mother's parity (primiparous or first time mothers versus multiparous mothers) between the cohort and the hospitalized sample.

Hypothesis VI: There is no significant difference in gender distribution between subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22.

Hypothesis VII: There is no significant difference in SES distribution between subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22.

Hypothesis VIII: There is no significant difference in mother's age at birth between subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22.

Hypothesis IX: There is no significant difference in father's age at birth between subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22.

Hypothesis X: There is no significant difference in mother's parity between subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22.

Hypothesis XI: There is no significant difference in a number of hospitalizations between subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22.

Hypothesis XII: There is no significant difference in a number of diagnoses between subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22.

Hypothesis XIII: There is no significant difference in number of hospitalizations between subjects hospitalized before the age of 15 and subject hospitalized both before the age of 15 and at ages 16 to 22.

Hypothesis XIV: There is no significant difference in number of diagnoses between subjects hospitalized before the age of 15 and subject hospitalized both before the age of 15 and at ages 16 to 22.

Hypothesis XV: There is no significant difference in number of hospitalizations between subjects hospitalized at ages 16 to 22 and subject hospitalized both before the age of 15 and at ages 16 to 22.

Hypothesis XVI: There is no significant difference in number of diagnoses between subjects hospitalized at ages 16 to 22 and subject hospitalized both before the age of 15 and at ages 16 to 22.

CHAPTER 3

METHOD

Subjects

The population consisted of 6401 subjects from the Helsinki Longitudinal Temperament Project (Martin, Bridger & Huttunen, 2000), 372 of whom were hospitalized with diagnoses of mental disorder during the study. The Helsinki Longitudinal Temperament Project was a longitudinal study of a cohort of 6401 participants who were born in Finland's Helsinki region between July 1, 1975 and June 30, 1976. Hospitalization data were collected from birth until 1991 and 1992 until December 1998, when the subjects were between 22 and 23 years old, depending on their date of birth. Hospitalization records of admissions were obtained from the Hospitalization Registry of Finland and included every hospital in Finland. Hospitalization records were accompanied with up to four diagnoses given to each subject at admission. Table 1 shows mental health diagnoses and ICD-9-CM codes included in this study (see Appendix for the complete description of ICD-9-CM codes).

Finland has a population of 5.2 million people. According to the Finnish Foreign Ministry (www.finland.org) about 60% of Finns reside in urban areas. Fertility rate in Finland is 1.7 children per women, but the number of births has been declining and is projected to decline further in the future. Due to the decreases in fertility rates and increases in life expectancy (74 years for males and 81 for females), the Finnish population of people ages 65 and older is increasing, while the population ages 15 and under is decreasing. In 1998, 2.5 million people or 41% of Finns were employed, 47.5% of which were women. High educational level reflects societal development and economic viability of Finland. In 1996, 67% of population

Table 1

Mental Disorders and ICD-9-CM Codes

Mental Disorders	ICD-9-CM Codes						
Addictive and substance abuse disorders	305.0	305.2	305.5	305.9	304.6		
Adjustment disorders and reactions	308.9	309	309.2	309.3	309.4	309.8	309.9
Anxiety disorders	300.0	300.1	300.3	300.4	300.8	300.9	300.20
Depressive and mood disorders	296.2	311					
Disorders of childhood and adolescence	312.0	312.3	312.8	313.9	314.1	314.9	
Personality Disorders	301.3	301.8					
Psychosis	298.9	299.8					

ages 25-64 completed at least secondary education, ranking Finland among the top five countries in the European Union. According to the Organization of Economic Cooperation and Development (OECD), in 1998, 80% of Finns ages 25-34 attained upper secondary education. Based on the OECD review Finnish schools are ranked the world's best in literacy and in top five in math and science.

Table 2 shows the demographic data of the cohort. A cohort of 6401 subjects consisted of 52.71% males and 47.29% of females. Primiparous, or first time mothers, made 56.73% of the cohort, while 43.27% mothers were multiparous at the time the subjects were born. Socio-economic status of families was determined based on father occupation and defined as 3 categories, starting with the lowest SES. With regard to socio-economic status, 46.88% of children were low-middle SES, 39.44 were middle, and 13.68 were high-middle SES. One third or 35.59% of mothers and 32% of fathers were in ages 25 to 28 when the child was born. Adolescent mothers made up 5.7% of the cohort, while only 1.85 of fathers were younger than 20 at the time the child was born.

Risk Factors

The purpose of the study was to determine which set of factors is associated with hospitalization for mental illness. The risk factors included gender, SES, mother's age, father's age, mother's parity, number of hospitalizations, and number of diagnoses. It was hypothesized that male gender, low SES, adolescent pregnancy, and being born to primiparous mothers are risk factors for hospitalization for mental illness.

Table 2

Demographic Data for the Birth Cohort

Demographic Characteristics		Frequency	Percentage
Gender			
	Male	3027	47
	Female	3374	53
	Total	6401	
Socio-economic Status			
	Low	1470	34
	Middle	2084	49
	High	723	17
	Total	4277	
Mother's Age			
	15-20	316	6
	21-24	1187	21
	25-28	1957	36
	29-32	1397	25
	33-40	642	12
	Total	5499	
Father's Age			
	16-20	111	2
	21-24	885	14
	25-28	2024	33
	29-32	1857	30
	33-40	1063	17
	41-65	226	4
	Total	6166	
Mother's Parity			
	Primiparous	3624	57
	Multiparous	2764	43
	Total	6388	

Diagnoses

Participants were assigned ICD-9-CM diagnoses. The list of diagnoses assigned to study participants consists of numeric codes and descriptions (Whiteman, 2001). The ICD-9-CM codes included in the study are classified by mental disorders in Table 1 (see Appendix for complete description of ICD-9-CM codes).

Definitions

Addictive and substance abuse disorders: Include maladaptive effects of abusing addictive substances or dependency on them, to the detriment of health or social functioning.

Adjustment disorders and reactions: Mild in severity and transient in duration, these disorders are longer lasting than acute stress reactions. They occur in persons without preexisting mental disorders and may be diagnosed at any age. They are generally considered reversible and relatively situation specific, related in time and content to the specific stressful events. In children, such disorders are not associated with significant and lasting impact on development.

Anxiety disorders: Neurotic disorder characterized by various physical and mental manifestations of apprehension, tension, or feelings of uneasiness related to anticipation of danger which source is either unknown or unrecognized.

Depressive and mood disorders: Neurotic disorders characterized by disproportionate mood changes that do not appear to be directly associated with stressful events. The distinction between depressive neurosis and psychosis is made by the degree of depression, presence of neurotic or psychotic features, and the degree of disturbance of the individual's behavior. Depressive reaction is characterized as adjustment reaction to stressful event.

Disorders of childhood and adolescence: Includes disturbances of conduct and hyperkinetic syndromes first diagnosed in childhood and adolescence. Conduct disorder involves aggressive and destructive behaviors characterized by frequency, severity, association with other symptoms, and abnormality in their social context. Conduct disorder is distinguishable from adjustment reaction by longer duration and lack of clear stressors preceding change in behavior. Conduct disorder is distinguishable from personality disorders by its lack of ingrained maladaptive patterns of behavior. Hyperkinetic syndrome of childhood is primarily characterized by short attention span and distractibility, as well as significant disturbance of conduct, delay in specific skills, marked mood fluctuations, and aggression. In the absence of conduct and mood related features symptoms of short attention span, distractibility, and overactivity are characteristics of attention deficit hyperactivity disorder.

Personality disorders: Maladaptive and ingrained patterns of behavior recognizable in adolescence or childhood, and present throughout most of the adult life. The personality may be abnormal in the balance of components or quality of expression, which has an adverse effect on an individual's functioning in society.

Psychosis: Mental disorders in which mental functioning is impaired to a degree that significantly interferes with individual's insight, everyday functioning, and adequate contact with reality.

Procedure

The demographic data were collected at birth and consisted of gender, socio-economic status defined by father's occupation, mother's age at the time of birth, father's age at the time of birth, and mother's parity. The diagnostic data included information on time of hospitalization, frequency of hospitalizations, and the number and type of diagnoses given to each subject at

hospital admission. Diagnostic data were collected in two periods, from 1976-1991, and 1992-1998. The period from 1976-1991 is referred as the early period, including subjects from birth to 15 years of age. The period from 1992-1998 is referred to as the late period, including subjects ages 16 to 22.

Data Analyses

Research questions were addressed using multiple chi-square test. The chi square test of independence is a nonparametric tests used to test association between two categorical variables and whether or not the proportions in the categories of one variable differ depending upon the level of the other variable (Harris, 1995). The null hypotheses were tested by comparing obtained frequencies with expected frequencies, based on a theory that the variables are independent. The data for chi-square goodness-of-fit test and chi-square test of independence were organized in the form of contingency tables. The hypothesized differences in proportions between variables were further tested and reported as odds ratios for their difference from the null hypothesis. The ratio of the two odds was used to compare the differences in the outcomes between variables (Sokal & Rohlf, 1995).

CHAPTER 4

RESULTS

There were three major questions addressed in the study: What are the differences between normal population and a sample of subjects hospitalized for mental illness, what are the differences between the subjects who were hospitalized before the age of 15 and those who were hospitalized at ages 16 to 22, and what are the differences between the subjects hospitalized either before the age of 15 or at ages 16 to 22 and the subjects hospitalized in both periods. The first question was addressed through hypotheses one to five, the second question was addressed through hypotheses six through twelve, and the third question was addressed through hypotheses thirteen to sixteen.

Descriptive Statistics

The study identified 372 subjects who were hospitalized for mental illness in the period between 1975-1991 and 1992-1998. The demographic data for the sample is shown in Table 3. Unlike the even gender distribution in the cohort (53% of females and 47% of males), the sample of hospitalized subjects was dominated by males (81% of males), low SES children (45% in hospitalized sample versus 34% in cohort), children of adolescent mothers (11% in hospitalized sample versus 6% in cohort), and children of multiparous mothers (48% in hospitalized sample versus 43% in cohort).

The study identified 71 subjects hospitalized between 1975-1991 and 278 subjects hospitalized between 1992-1998. The demographic data is shown in Table 4. There were 23 subjects who were hospitalized before the age of 15 (1975-1991) and again at ages 16

Table 3

Demographic Data for Hospitalized Subjects

Demographic Characteristics		Frequency	Percentage
Gender			
	Male	302	81
	Female	69	19
	Total	371	
Socio-economic Status			
	Low	138	45
	Middle	123	40
	High	47	15
	Total	308	
Mother's Age			
	15-20	33	11
	21-24	64	21
	25-28	96	31
	29-32	76	25
	33-40	36	12
	Total	305	
Father's Age			
	16-20	8	2
	21-24	64	18
	25-28	102	30
	29-32	106	30
	33-40	57	16
	41-65	15	4
	Total	352	
Mother's Parity			
	Primiparous	190	52
	Multiparous	177	48
	Total	367	

Table 4

Demographic Data for Subjects Hospitalized Before the Age of 15, at Ages 16 to 22, and in Both Periods

Demographic Characteristics	Age 0-15		Age 16-22		Age 0-22	
	Frequency	Percentage	Frequency	Percentage	Frequency	Percentage
Gender						
Female	20	28	45	16	4	18
Male	51	72	233	84	18	82
Total	71		278		22	
Socio-economic Status						
Low	26	46	106	46	6	33
Middle	22	38	89	38	12	67
High	9	16	38	16	0	0
Total	57		233		18	
Mother's Age						
15-20	5	9	27	11	1	6
21-24	12	23	44	19	8	47
25-28	20	38	70	30	6	35
29-32	10	19	64	27	2	12
33-40	6	11	30	13	0	0
Total	53		235		17	
Father's Age						
16-20	1	2	7	3	0	0
21-24	11	16	49	18	4	21
25-28	19	28	74	28	9	47
29-32	23	34	81	30	2	11
33-40	12	18	42	16	3	16
41-65	1	2	13	5	1	5
Total	67		266		19	
Mother's Parity						
Primiparous	40	58	138	50	12	57
Multiparous	29	42	139	50	9	43
Total	69		277		21	

Table 5

Hospitalization and Diagnoses Data for Subjects Hospitalized Before the Age of 15, at Ages 16 to 22, and in Both Periods

	Age 0-15		Age 16 to 22		Age 0-22	
	Frequency	Percentage	Frequency	Percentage	Frequency	Percentage
Hospitalization						
Single	34	48	111	40	1	4
Multiple	37	52	167	60	22	96
Total	71		278		23	
Diagnosis						
Single	51	72	177	64	6	26
Multiple	20	28	101	36	17	74
Total	71		278		23	

to 22 (1992-1998). A number of hospitalizations and diagnoses is shown in Table 5. Multiple hospitalizations rose to 60% at ages 16 to 22, compared to 52% before the age of 15. Number of multiple diagnoses rose at ages 16 to 22 to 36% from 28% before the age of 15. Among subjects who were hospitalized in both periods only 1 subject (4%) had a single hospitalization in each period, while 96% of subjects were hospitalized multiple times. In this group a number of subjects with multiple diagnoses rose to 74%.

The types of diagnoses at different hospitalization periods are displayed in Table 6. The largest percentage of assigned diagnoses were from adjustment disorders and reactions family, regardless of the time of hospitalization (49% among subjects hospitalized before the age of 15, 49% of subjects hospitalized at ages 16 to 22, and 36% of subjects hospitalized at both periods). Distribution of addictive and substance abuse disorders, adjustment disorders and reactions, anxiety disorders, and personality disorders between subjects hospitalized before the age of 15 and at ages 16 to 22 is similar. There was an increase in hospitalization for psychosis (from 1% before the age of 15 to 9% at ages 16 to 22) and a decrease in disorders of childhood and adolescence (from 12% before the age of 15 to 4% at ages 16 to 22). Among subjects hospitalized in both periods there was more psychoses (13%), more addictive and substance abuse disorders (11%), and more depressive and mood disorders diagnoses (6%) than in either period. In this group there were fewer personality disorders (9%) and fewer adjustment disorders and reactions (36%).

Table 6

ICD-9-CM Diagnoses for Subjects Hospitalized Before the Age of 15, Subjects Hospitalized at Ages 16 to 22, and in Both Periods

Mental Disorders	Age 0-15		Age 16-22		Age 0-22	
	Frequency	Percentage	Frequency	Percentage	Frequency	Percentage
Addictive and Substance Abuse Disorders	6	6	18	6	6	11
Adjustment Disorders and Reactions	45	45	150	49	20	36
Anxiety Disorders	21	21	63	21	9	16
Depressive and Mood Disorders	0	0	2	0	3	6
Disorders of Childhood and Adolescence	12	12	13	4	5	9
Personality Disorders	15	15	34	11	5	9
Psychosis	1	1	27	9	7	13
Total	100		307		55	

Hypotheses Testing

The first hypothesis stated that there was no significant difference in gender distribution between the cohort and the hospitalized sample. Data relevant to this hypothesis are presented in Table 7. For the cohort, there were 53% females and 47% males. For the sample, there were 19% of females and 81% males. The data were submitted to a chi-square goodness-of-fit test in which expected values were calculated based on cohort values. The chi-square indicated that there was a significant difference in gender distribution between cohort and sample (chi-square=176.26; $df=1$; $n=371$; $p<.001$). There were more male subjects in the hospitalized sample than expected, based on cohort values. Males were almost five times more likely than females to be hospitalized for mental illness (odds ratio=4.86).

The second hypothesis stated that there was no significant difference in socio-economic status (SES) distribution between the cohort and the hospitalized sample. Data relevant to this hypothesis are presented in Table 8. For the cohort, there were 34% low SES subjects, 49% middle SES subjects, and 17% high SES subjects. For the sample, there were 45% low SES subjects, 40% middle SES subjects, and 15% high SES subjects. The data were submitted to a chi-square goodness-of-fit test in which expected values were calculated based on cohort values. The chi-square indicated that there was a significant difference in SES between cohort and sample (chi-square= 16.28; $df=2$; $n=308$; $p<.001$). There were more low SES subjects in the hospitalized sample than expected.

The third hypothesis stated that there was no significant difference in mother's age at birth between the cohort and the hospitalized sample. Data relevant to this hypothesis are presented in Table 9. For the cohort, 6% of the mothers were younger than 20, 21% of mothers were 21-24, 36% of mothers were 25-28, 25% of mothers were 29-32, and 12% of mothers were

Table 7

Differences in Gender Between the Cohort and the Sample

Gender	Observed Frequency	Expected Frequency
Male	302	174.37
Female	69	196.63
Total	371	

Table 8

Differences in Socio-economic Status Between Cohort and Sample

Socio-economic Status	Observed Frequency	Expected Frequency
1	138	104.72
2	123	150.92
3	47	52.36
N	308	

Table 9

Differences in Mother's Age Between Cohort and Sample

Mother's Age	Observed Frequency	Expected Frequency
15-20	33	18.3
21-24	64	64.05
25-28	96	109.8
29-32	76	76.25
33-40	36	36.6
Total	305	

33-40. For the sample, there 11% of mothers were younger than 20, 21% of mothers were 21-24, 31% of mothers were 25-28, 25% of mothers were 29-32, and 12% of mothers were 33-40. The data were submitted to a chi-square goodness-of-fit test in which expected values were calculated based on cohort values. The chi-square indicated that there was a significant difference in mother's age between cohort and sample (chi-square= 13.54; $df=5$; $n=305$; $p<.02$). There were more adolescent mothers in the hospitalized sample than expected based on cohort values.

The fourth hypothesis stated that there was no significant difference in father's age at birth between the cohort and the hospitalized sample. Data relevant to this hypothesis are presented in Table 10. For the cohort, 2% of the fathers were younger than 20, 14% of fathers were 21-24, 33% of fathers were 25-28, 30% of fathers were 29-32, 17% of fathers were 33-40, and 4% of fathers were older than 41. For the sample, 2% of fathers were younger than 20, 18% of fathers were 21-24, 30% of fathers were 25-28, 30% of fathers were 29-32, 16% of fathers were 33-40, and 4% of fathers were older than 41. The data were submitted to a chi-square goodness-of-fit test in which expected values were calculated based on cohort values. The chi-square indicated that there was no significant difference in father's age at birth between cohort and hospitalized sample (chi-square= 7.96; $df=5$; $n=352$; $p=ns$).

The fifth hypothesis stated that there was no significant difference in mother's parity (first time mothers, primiparous, versus at least one child at the time of subject's birth, multiparous) between the cohort and the hospitalized sample. Data relevant to this hypothesis are presented in Table 11. For the cohort, 57% of the mothers were primiparous and 43% of the mothers were multiparous. For the sample, 52% of mothers were primiparous and 48% of mothers were multiparous. The data were submitted to a chi-square goodness-of-fit test

Table 10

Differences in Father's Age Between Cohort and Sample

Father's Age	Observed Frequency	Expected Frequency
16-20	8	7.04
21-24	64	49.28
25-28	102	116.16
29-32	106	105.6
33-40	57	59.84
41-65	15	14.08
Total	352	

Table 11

Differences in Mother's Parity Between Cohort and Sample

Mother's Parity	Observed Frequency	Expected Frequency
Primiparous	190	209.19
Multiparous	177	157.81
Total	367	

in which expected values were calculated based on cohort values. The chi-square indicated that there was a significant difference in mother's parity between cohort and hospitalized sample (chi-square= 4.09; $df=1$; $n=367$; $p<.05$). There were more multiparous mothers in hospitalized sample than expected based on cohort values.

The sixth hypothesis stated that there was no significant difference in gender between subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22. Data relevant to this hypothesis are presented in Table 12. For the subjects hospitalized before the age of 15 there were 28% females and 72% males. For the subjects hospitalized between 16 to 22, 16% were females and 84% males. The data were submitted to a chi-square test of independence. The chi-square indicated that there were significant gender differences between subjects hospitalized before the age of 15 and those hospitalized at ages 16 to 22 (chi-square=5.37; $df=1$; $n=349$; $p<.05$). There were more hospitalized males in the later period than expected.

The seventh hypothesis stated that there was no significant difference in socio-economic status (SES) between subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22. Data relevant to this hypothesis are presented in Table 13. For the group of subjects hospitalized before the age of 15, 46% were low SES, 38% middle SES, and 16% high SES subjects. For the subjects hospitalized at ages 16 to 22, 46% were low SES, 38% middle SES, and 16% high SES. The data were submitted to a chi-square test of independence. The chi-square indicated that there were no significant SES differences between subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22 (chi-square=.00; $df=2$; $n=290$; $p=ns$).

Table 12

Gender Differences in Subjects Hospitalized Before the Age of 15 and at Ages 16 to 22

Gender	0-15	16-22	Total
Female	20	45	65
Male	51	233	284
Total	71	278	349

Table 13

SES Differences in Subjects Hospitalized Before The Age of 15 and at Ages 16 to 22

Socio-economic Status	0-15	16-22	Total
1	26	106	132
2	22	89	111
3	9	38	47
Total	57	233	290

The eighth hypothesis stated that there was no significant difference in mother's age at birth between the subjects hospitalized before the age of 16 and subjects hospitalized at ages 16 to 22. Data relevant to this hypothesis are presented in Table 14. For the subjects hospitalized before the age of 15, 9% of the mothers were younger than 20, 23% of mothers were 21-24, 38% of mothers were 25-28, 19% of mothers were 29-32, and 11% of mothers were 33-40. For the subjects hospitalized at ages 16 to 22, there 11% of mothers were younger than 20, 19% of mothers were 21-24, 30% of mothers were 25-28, 27% of mothers were 29-32, and 13% of mothers were 33-40. The data were submitted to a chi-square test of independence. The chi-square indicated that there was no significant difference in mother's age between subjects hospitalized before the age of 15 and subject hospitalized at ages 16 to 22 (chi-square=2.62; $df=4$; $n=288$; $p=ns$).

The ninth hypothesis stated that there was no significant difference in father's age at birth between the subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22. Data relevant to this hypothesis are presented in Table 15. For the subjects hospitalized before the age of 15, 2% of the fathers were younger than 20, 16% of fathers were 21-24, 28% of fathers were 25-28, 34% of fathers were 29-32, 18% of fathers were 33-40, and 2 % of fathers were older than 41. For the subjects hospitalized at ages 16 to 22, 3% of fathers were younger than 20, 18% of fathers were 21-24, 28% of fathers were 25-28, 30% of fathers were 29-32, 16% of fathers were 33-40, and 5% of fathers were older than 41. The data were submitted to a chi-square test of independence. The chi-square indicated that there was no significant difference in father's age at birth between subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22 (chi-square= 2.18; $df=5$; $n=333$; $p=ns$).

Table 14

Differences in Mother's Age Between Subjects Hospitalized Before the Age of 15 and at Ages 16 to 22

Mother's Age	0-15	16-22	Total
15-20	5	27	32
21-24	12	44	56
25-28	20	70	90
29-32	10	64	74
33-40	6	30	36
Total	53	235	288

Table 15

Differences in Father's Age Between Subjects Hospitalized Before the Age of 15 and Subjects Hospitalized At Ages 16 to 22

Father's Age	0-15	16-22	Total
16-20	1	7	8
21-24	11	49	60
25-28	19	74	93
29-32	23	81	104
33-40	12	42	54
41-65	1	13	14
Total	67	266	333

The tenth hypothesis stated that there was no significant difference in mother's parity (primiparous versus multiparous mothers) between the subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22. Data relevant to this hypothesis are presented in Table 16. For the subjects hospitalized before the age of 15, there were 58% primiparous mothers and 42% multiparous mothers. For the subjects hospitalized at ages 16-22, there were 50% primiparous mothers and 50% multiparous mothers. The data were submitted to a chi-square test of independence. The chi-square indicated that there was no significant difference in mother's parity between subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22 (chi-square= 1.46; $df=1$; $n=346$; $p=ns$).

The eleventh hypothesis stated that there was no significant difference in the number of hospitalizations between the subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22. Data relevant to this hypothesis are presented in Table 17. For the subjects hospitalized before the age of 15, 48% of subjects were hospitalized once and 52% of subjects were hospitalized more than once. For the subjects hospitalized at ages 16 to 22, 40% of subjects were hospitalized once and 60% of subjects were hospitalized more than once. The data were submitted to a chi-square test of independence. The chi-square indicated that there was no significant difference in the number of hospitalizations between subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22 (chi-square=1.48; $df=1$; $n=349$; $p=ns$).

The twelfth hypothesis stated that there was no significant difference in the number of diagnoses between subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22. Data relevant to this hypothesis are presented in Table 18. For the subjects hospitalized before the age of 15, 72% received one diagnosis and 28% received multiple diagnoses. For the subjects hospitalized at ages 16 to 22, 64% received one diagnosis and 36% received multiple

Table 16

Differences in Mother's Parity Between Subjects Hospitalized Before The Age of 15 and at Ages 16 to 22

Mother's Parity	0-15	16-22	Total
Primiparous	40	138	178
Multiparous	29	139	168
Total	69	277	346

Table 17

Differences in Number of Hospitalizations in Subjects Hospitalized Before the Age of 15 and at Ages 16 to 22

Hospitalization	0-15	16-22	Total
Single	34	111	145
Multiple	37	167	204
Total	71	278	349

Table 18

Differences in Number of Diagnoses in Subjects Hospitalized Before the Age of 15 and at Ages 16 to 22

Diagnosis	0-15	16-22	Total
Single	51	177	228
Multiple	20	101	121
Total	71	278	349

diagnoses. The data were submitted to a chi-square test of independence. The chi-square indicated that there was no significant difference in the number of diagnoses between subjects hospitalized before the age of 15 and subjects hospitalized at ages 16 to 22 (chi-square=1.67; $df=1$; $n=349$; $p=ns$).

The thirteenth hypothesis stated that there was no significant difference in the number of hospitalizations between subjects hospitalized before the age of 15 and subjects hospitalized both before the age of 15 and at ages 16 to 22. Data relevant to this hypothesis are presented in Table 19. For the subjects hospitalized before the age of 15, 48% were hospitalized once and 52% were hospitalized multiple times. There were 23 subjects hospitalized before the age of 15 and again at ages 16 to 22. Among them, only 1 subject, or 4%, was hospitalized once in each period, and 96% were hospitalized more than one time in each period. The data were submitted to a chi-square test of independence. The chi-square indicated that there was a significant difference in the number of hospitalizations between subjects hospitalized before the age of 15 and subjects hospitalized in both periods (chi-square=14.08; $df=1$; $N=94$; $p<.001$). Because of the small cell size the rejection of null hypothesis should be interpreted with caution. Subjects hospitalized in both periods had more hospitalizations than expected. Subjects hospitalized in both periods were two times more likely to have multiple hospitalizations than subjects hospitalized before the age of 15 (odds ratio=2.22).

The fourteenth hypothesis stated that there was no significant difference in the number of diagnoses between subjects hospitalized before the age of 15 and subjects hospitalized before the age of 15 and at ages 16 to 22. Data relevant to this hypothesis are presented in Table 20. For the subjects hospitalized before the age of 15, 72% received one diagnosis and 28% received

Table 19

Differences in Number of Hospitalizations Between Subjects Hospitalized Before the Age of 15 and Subjects Hospitalized in Both Periods

Hospitalization	0-15	0-22	Total
Single	34	1	35
Multiple	37	22	59
Total	71	23	94

Table 20

Differences in Number of Diagnoses Between Subjects Hospitalized Before the Age of 15 and Subjects Hospitalized in Both Periods

Diagnoses	0-15	0-22	Total
Single	51	6	57
Multiple	20	17	37
Total	71	23	94

more than one diagnoses. There were 23 subjects hospitalized before the age of 15 and again at ages 16 to 22. Among them, 26% had one diagnosis in each period and 74% had more than one diagnosis. The data were submitted to a chi-square test of independence. The chi-square indicated that there was a significant difference in the number of diagnoses between subjects hospitalized before the age of 15 and those hospitalized in both periods (chi-square=15.24; $df=1$; $n=94$; $p < .001$). Subjects hospitalized in both periods had more diagnoses than expected. Subjects hospitalized in both periods were more than seven times more likely to have multiple diagnoses than subjects who were hospitalized before the age of 15 (odds ratio=7.5).

The fifteenth hypothesis stated that there was no significant difference in the number of hospitalizations between subjects hospitalized at ages 16 to 22 and subjects hospitalized both before the age of 15 and at ages 16 to 22. Data relevant to this hypothesis are presented in Table 21. For the subjects hospitalized at ages 16 to 22, 40% were hospitalized once and 60% were hospitalized multiple times. There were 23 subjects hospitalized in both periods. Among them, 1 subject or 4% was hospitalized once in each period and 22 subjects or 96% were hospitalized multiple times. The data were submitted to a chi-square test of independence. The chi-square indicated that there was a significant difference in the number of hospitalizations between subjects hospitalized at ages 16 to 22 and subjects hospitalized in both periods (chi-square=11.52; $df=1$; $N=301$; $p < .001$). However, because of the small cell size the rejection of null hypothesis should be interpreted with caution. Subjects hospitalized in both periods had more multiple hospitalizations than expected. Subjects hospitalized in both periods were almost two times more likely to have multiple hospitalizations than subjects hospitalized at ages 16 to 22 (odds ratio=1.6).

Table 21

Differences in Number of Hospitalizations in Subjects Hospitalized at Ages 16 to 22 and
Subjects Hospitalized in Both Periods

Hospitalizations	16-22	0-22	Total
Single	111	1	112
Multiple	167	22	189
Total	278	23	301

The sixteenth hypothesis stated that there was no significant difference in the number of diagnoses between subjects hospitalized at ages 16 to 22 and subjects hospitalized both before the age of 15 and at ages 16 to 22. Data relevant to this hypothesis are presented in Table 22. For the subjects hospitalized at ages 16 to 22, 64% had one diagnosis and 36% had multiple diagnoses. There were 23 subjects hospitalized in both periods. Among them, 26% had one diagnosis and 74% had multiple diagnoses. The data were submitted to a chi-square test of independence. There was a significant difference in the number of diagnoses between subjects hospitalized at ages 16 to 22 and subjects hospitalized in both periods (chi-square=12.58; df=1; $n=23$; $p < .001$). Subjects hospitalized in both periods had more multiple diagnoses than expected. Subjects hospitalized in both periods were five times more likely to have multiple diagnoses than subjects hospitalized at ages 16 to 22 (odds ratio=5.08).

Table 22

Differences in Number of Diagnoses Between Subjects Hospitalized at Ages 16 to 22 and Subjects Hospitalized in Both Periods

Diagnoses	16-22	0-22	Total
Single	177	6	183
Multiple	101	17	118
Total	278	23	301

CHAPTER 5

DISCUSSION

Rationale for the Study

The current study focused on a critical aspect of mental health, hospitalization for mental illness. Even though studies have been closing gaps in existing knowledge in the epidemiology of mental illness among children and adolescents, numerous questions regarding prevalence and risk remain unanswered. A review of the literature shows a particular absence of studies dealing with severe mental illness and hospitalization among children and adolescents. Because the epidemiological studies often do not control for severity of symptoms and functional impairment, the findings pertaining specifically to prevalence and risk of severely mentally ill children and adolescents are hard to come by.

The absence of American hospitalization data can be attributed to factors such as current intervention practices favoring outpatient treatments, diversity of institutions and standards for care of mentally ill children and adolescents, the financial and human costs of hospitalization, trends favoring closing of government run mental health institutions and a greater reliance on privately run facilities, the lack of a centralized health care system, the lack of a central data bank of hospitalization records, and the lack of uniform data collection and analysis methods. In addition, the cost of conducting longitudinal studies and mobility of the population in the United States coupled with high attrition rates, make longitudinal epidemiological studies all that more difficult to complete. Thus, leading research in epidemiology of mental illness often relies on

data collected in countries with centralized healthcare systems, uniform standards of care, and national data registries.

In the current study, a birth cohort of 6401 subjects was followed longitudinally from birth to age 22 using the Finnish National Registry data. The demographic data available for the entire cohort and the prospective diagnostic assessment data available for the sample of hospitalized subjects were used to identify prevalence, risk factors, and functional impairments associated with hospitalization for mental illness. The study focused on three general questions. What are the differences between the general population and subjects hospitalized for mental illness with regard to risk factors? What are the differences between subjects hospitalized in childhood and early adolescence and those hospitalized in late adolescence and early adulthood with regard to risk factors and functional impairment? What are the differences between subjects hospitalized in either period and subjects hospitalized in both periods with regard to functional impairment?

It was hypothesized that hospitalized subjects do not differ from the normal population with regard to gender, socio-economic status, mother's age at birth, father's age at birth, and mother's parity; that subjects hospitalized before the age of 15 do not differ from those hospitalized between the ages of 16 and 22 with regard to gender, socio-economic status, mother's age at birth, father's age at birth, mother's parity, number of hospitalizations and number of diagnoses; and that subjects hospitalized in both periods do not differ from subjects hospitalized in either period with regard to the number of hospitalizations and diagnoses.

Summary of Results

Several significant findings emerged as a result of this study. The number of subjects hospitalized for mental illness increased from childhood to early adulthood. Male subjects, low

SES subjects, subjects born to adolescent mothers, and subjects born to multiparous mothers were at a significantly higher risk for hospitalization for mental illness. Subjects with chronic manifestations of mental illness showed significantly higher functional impairment than subjects hospitalized in only one developmental period. And finally, a significant portion of children hospitalized in childhood and early adolescence were hospitalized again before reaching adulthood.

Of a total of 6401 subjects in the population of children born in Finland between 1975 and 1976, 372 children or 5.8% were hospitalized for mental illness by the age of 22. According to Offord et al. (1992), 25% of children from preschool to pre-adolescence have significant mental health problems, while the overall prevalence for severe mental disorder is between 10-20% (Wadell et al., 2002).

Developmental Changes in Prevalence of Hospitalization

The study found that rates of hospitalization increased longitudinally from childhood and early adolescence to late adolescence and early adulthood, and that a significant number of children who received treatment for mental health problems before the age of 15 continued needing mental care in an inpatient setting before they reached early adulthood. Rates of hospitalization increased from 1.4 % in childhood and early adolescence, to 4.3% in late adolescence and early adulthood. The study did not find developmental changes with regard to risks factors such as low SES, mother's age at birth, father's age at birth, and mother's parity. However, risk of hospitalization for male subjects increased with age.

Findings on prevalence of hospitalization follows trends in prevalence of mental health problems in the general population, showing an increase from childhood to adulthood. Research also suggests that a majority of young adults have a chronic history of mental health issues.

Newman, Moffitt, Caspi, Magdol, Silva, and Stanton (1996) found that rates of mental disorders steadily increased from late childhood to late adolescence. They identified 18% of diagnosed children at age 11 and 40% at age 21. Three fourths of the cases identified at age 21 had a previous history of mental disorder.

Transition from childhood and adolescence to adulthood is a developmentally critical period with potentially serious implications for later mental health. The effects of mental health problems on general functioning are wide ranging, from interference in daily activities, developing a reputation for disturbed behavior, and higher incidence of criminal convictions. These effects present obstacles in completing education, finding employment, and forming satisfactory relationships (Newman et al., 1996). Because of major life choices made in this period, emerging mental health problems may have serious and long lasting consequences for later life stages.

Comorbidity and Continuity of Mental Illness

The current study addressed the question of continuity of mental illness by identifying subjects who were hospitalized in both developmental periods. These subjects represented only 0.3% of the general population and only 8% of all hospitalized children. However, nearly 25%, or every fourth child who was hospitalized before the age of 15, was re-hospitalized later at ages 16 to 22. This finding confirms theoretical assumptions in developmental psychopathology suggesting that mental disorders tend to be continuous, chronic, or episodically remitting, and that disorders identified in early adulthood tend to have preexisting diathesis (Cicchetti & Cohen, 1995). Research on continuity of disorders in early age shows that four in ten children have problems in more than one developmental period and two thirds of those who have a disorder at age 15 are also diagnosed at age 18 (Offord et al., 1992).

In addition to a relatively high recidivism rate found among hospitalized subjects, when subjects hospitalized in both periods were compared to subjects hospitalized in one period, they showed significantly higher levels of functional impairment measured by number of hospitalizations and number of diagnoses. High comorbidity is a constant finding in studies of developmental psychopathology, affecting severity of impairment and the likelihood of repeated hospitalizations. Newman et al., (1996) found that subjects with comorbid diagnoses not only suffered significantly more functional impairments than subjects with single diagnoses, but they also discovered a strong linear relationship between the number of disorders and level of impairment. In addition to reflecting the developmental nature of mental illness, increased comorbidity among chronic cases may also reflect referral bias. Comorbidity rates in clinic based samples tend to be overestimated, leading to a so-called “Berkson’s bias” caused by selective treatment seeking among comorbid cases (Berkson, 1946).

Risk Factors for Hospitalization

The study identified gender, SES, mother’s age at birth, and mother’s parity as significant factors associated with higher risk for hospitalization for mental illness. Male gender emerged as a risk factor that can strongly predict likelihood of hospitalization. This finding confirms findings by numerous studies showing that, with the exception of internalizing disorders, the prevalence of total mental disorders is greater in boys (Weisz & Suwanlert, 1989; Olewus, 1979; Achenbach, 1991). An Australian national survey found that 19% of boys and 10% of girls are likely to have mental illness (Sawyer et al., 2001), but risk in girls increases in adolescence and early adulthood (Duffy, 2000; Waddell et al., 2002).

Higher hospitalization rates for male subjects can be attributed to several factors. According to Achenbach (1991), research on gender differences in referred samples is

confounded by referral bias. Males are more likely to be referred for intervention. The larger prevalence of males among hospitalized subjects, than among subjects identified to have mental health problems, may reflect not only a difference in gender distribution at different levels of severity and functional impairment, but also a methodological issue. The prevalence of mental health problems is often studied in non-referred samples, whereas studies of hospitalization use clinical samples, which are suspect to referral bias. However, it is also possible that males are more likely to develop severe forms of mental illness and manifest greater functional impairment, leading to higher rates of hospitalization.

The current study identified significant differences in mother's age at birth between the hospitalized subjects and the normal population. Children hospitalized for mental illness are more likely to be born to mothers under 20. Research reports numerous behavioral and environmental variables associated with early pregnancy and the risk they pose for mental illness in children (Fergusson & Woodward, 1999; Roosa et al., 1982). Studies also found an association between early onset of psychiatric disorders and subsequent teenage pregnancies for both sexes, thus adding possible genetic risk factors in confluence with environmental risks posed by early pregnancy (Kessler et al., 1997).

The results of the current study show that SES, defined by the father's occupation, distinguished between hospitalized subjects and the normal population. Low educational and occupational levels among parents are the SES factors most often found to be associated with various disadvantages for children (Rutter, 1979). Both SES and early pregnancy are complex factors whose effects range from partnership breakdowns, large family size, poorer household conditions, and higher risk of psychiatric problems (Maugham & Lindelow, 1997). The current

study adds one more mental health outcome affected by low SES and early pregnancy: higher risk for hospitalization for mental illness.

Strengths of the Study

The current study addressed research questions through a longitudinal approach, focusing on prevalence and risk factors associated with hospitalization at critical developmental stages. The record of developmental changes in prevalence can be obtained only by prospective assessments of the same age cohort in a longitudinal design. Despite limitations of a single cohort longitudinal design arising from potential exposure effects or historical period effects inherent in such a design, single cohort design is the preferred approach in demonstrating age-related developmental changes. In contrast, cross-sectional design is limited to comparisons between different age cohorts, which leads to confounding of age effects with cohort effects (Nesselroade & Baltes, 1979).

The advantage of the current study is that it used prospectively collected longitudinal data in a large birth cohort, which provided a reliable comparison of non-referred population and clinical samples. This methodology allows one not only to draw conclusions on prevalence of hospitalization in the population, but also to imply causal relationships between risk factors and mental health outcomes. By using public record information on hospitalizations, instead of subjective reporting by study members or elected informants, the current study provides objective information. By limiting the inquiry to hospitalized subjects the study provides information on prevalence and risk in a severely affected population of children and adolescents.

Limitations of the Study

Because of their low base rates and time constraints, this study excluded hospitalizations for a number of mental disorders such as cognitive impairments, organic mental disorders,

somatoform disorders, learning disorders, and eating disorders. Also, the longitudinally collected data were incomplete, missing information for a year-long period between 1991 and 1992. Therefore, the hospitalization records for subjects between age 16 and 17 were not included.

By utilizing already collected public record data, rather than multiple informants' reports on mental health diagnoses, the study did not control for reliability of diagnoses. Using already available public record data also prevented expanding the information to include multiple indicators of functional impairment. Demographic information was collected at birth, thus excluding information on demographic changes over time. In order to develop a more complete picture of severe mental illness in childhood and adolescence, future studies should expand information to include indications of the clinical course of disorders that lead to hospitalization. The patterns of remission, relapse, and the motivation to seek intervention, should be explored.

Because the study used Finnish data, the results of this study may not be directly applicable to the population of the United States. Finnish society provides health care to all of its citizens, thus making access to mental health intervention accessible regardless of SES. Even though one could reasonably assume that hospitalization for mental illness primarily reflects a level of functional impairment and need for intervention, it is likely that rates of hospitalization depend on availability and affordability of treatment. According to the Institute of Medicine (1994), only 10% to 30% of affected cases receive any treatment. Epidemiological studies consistently find that rates of mental illness exceed rates of services (Newman et al., 1996). Thus it is reasonable to assume that rates of treatment partially reflect needs for services, and partially availability of care.

Practical Implications and Future Research

Epidemiology of mental illness relies on cumulative evidence from multiple studies in order to establish rates of prevalence and determine risks. The current study adds one more element to the puzzle of mental illness in childhood and adolescence. The high prevalence of mental disorders in children and adolescents and the lasting effects functional impairment has on the individual, families, and community, points to the importance of a better understanding of developmental aspects of severe psychopathology. A review of literature on mental illness indicates sparse knowledge about severely affected children and adolescents. Prevalence, risk factors, diagnoses, comorbidity, and recidivism among hospitalized children have not been adequately explored.

Prevalence of severe mental disorder, functional impairment, and continuity from childhood to adulthood, suggest the need for prevention and primary intervention with younger children, before manifestations of illness take a chronic path. The current study found that a number of affected young adults had a history of mental problems prior to reaching adulthood. Newman et al., (1996) reported that young adulthood has the highest prevalence of mental disorders, but the lowest incidence of new cases in the same developmental period. Intervention targeting young adults and currently favored brief therapies may be too little and too late to be effective in dealing with potentially chronic problems. Targeting the population at highest risk for developing disorders with intense prevention and intervention early, may reduce human suffering and the financial cost of fighting mental illness later in life. However, this can not be achieved without declaring prevention of mental illness in young people an issue of national priority.

High comorbidity found in children and adolescents has been associated with greater impairment, increased duration of illness, and a greater complexity required of assessment and intervention. Because of the small sample of children who were hospitalized in more than one developmental period and the low base rate of some disorders in early childhood, this study was not able to identify comorbidity patterns and changes over time. Future studies should explore how comorbidity affects functional impairment, choices of intervention, and remission among hospitalized subjects.

Future research should expand on the finding that one fourth of children hospitalized in childhood are at risk to be hospitalized again before they reach adulthood. It is likely that these children will continue on the chronic pathway into adult age. Longitudinal studies should follow subjects into adulthood to identify factors that lead to adult hospitalizations. Research should also explore motivation to seek treatment as one of the crucial factors affecting low intervention rates. Newman et al., (1996) pointed out that only one in four diagnosed subjects seek intervention even when health services are widely available. To increase motivation to seek treatment and decrease stigma attached to mental illness, prevention programs should be offered to all children. By limiting intervention to referred children, we lose the opportunity to prevent mental illness or change its course in a large portion of those affected who do not seek services.

Longitudinal, epidemiological studies of mental health, such as the current study, should help guide policy and intervention. Unfortunately, direct influence of studies on prevalence and risk on planning and policy has been rare (Verhulst, 1995; Waddell et al., 2002). Being able to identify those at highest risk of hospitalization could aid communities in developing prevention and intervention programs. If accumulated epidemiological research is going to have any real effect, the task to integrate theory and research with policy and service delivery is crucial.

Conclusions

The current study attempted to provide answers to some of the most elemental questions regarding hospitalized young people, such as: What is the prevalence of hospitalization, what are the risk factors for hospitalization, do risk factors differ at different developmental periods, and do subjects hospitalized in more than one period differ from other hospitalized populations. The most significant findings of this study are that being male, being born to a young mother, to a multiparous mother, and into a low SES family increases risk of hospitalization. Furthermore, one in four children hospitalized in childhood and early adolescence are at risk to be hospitalized again before reaching adulthood. These children are likely to be more functionally impaired as measured by comorbidity and the number of hospitalizations.

The findings of the current study contribute to the cumulative knowledge of epidemiology of mental illness by providing information on the little explored population of children hospitalized for mental illness. Prevalence of severe mental problems, longitudinally increased rates of hospitalization, and chronic cases affected by functional impairment suggest that efforts to educate the public about mental health in young people should focus on the widespread nature of mental problems, rather than their rarity.

REFERENCES

- Achenbach, T.M. (1991). *Manual for the Child behavior Checklist/4-18 and 1991 Profile*. Burlington, VT: University of Vermont Department of Psychiatry.
- Achenbach, T.M., Howell, C.T., McCounaughy, S.H., & Stanger, C. (1995). Six-year predictors of problems in a national sample: III. Transitions to early adult syndromes. *Journal of the American Academy of Child and Adolescent Psychiatry*, 34, 658-669.
- Adelman, H.S. (1995). Clinical psychology: Beyond psychopathology and clinical interventions. *Clinical Psychology: Science and Practice*, 2, 28-44.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed. Text Revision). Washington, DC: Author.
- Angold, A. & Costello, E (1991). Developing a Developmental Epidemiology. In D. Cicchetti & S.L. Toth (Eds.) *Rochester Symposium on Developmental Psychopathology: Models and Integrations*. Vol. 3. Rochester, N.Y.: University of Rochester Press.
- Anthony, J.L., Lonigan, C.J., Hooe, E.S., & Phillips, B.M. (2002). An affect-based, hierarchical model of temperament and its relations with internalizing symptomatology. *Journal of Clinical Child and Adolescent Psychology*, 31 (4), 480-491.
- Barkley, R.A. (1996). Attention-Deficit/Hyperactivity Disorder. In Mash, E.J. & Barkley, R.A. (Eds.), *Child Psychopathology* (pp.63-113). New York: Guilford Press.
- Berkson, J. (1946). Limitations of the application of the fourfold table analysis to hospital data. *Biometrics*, 2, 47-53.
- Bird, H., Canino, G., Rubio-Stipec, M., Gould, M.S., Ribera, J., Sesman, M., Woodbury, M., Huertas, Goldman, S., Pagan, A., Sanchez-lacay, A., & Moscoso, M. (1988). Estimates of prevalence of childhood maladjustment in a community survey in Puerto Rico. *Archives of General Psychiatry*, 45, 1120-1126.
- Boyle, M.H. (1995). Sampling in epidemiological studies. In F.C. Verhulst & H.M. Koot (Eds.), *The epidemiology of child and adolescent psychopathology* (pp. 66-85). Oxford: Oxford University Press.
- Boyle, M.H., Offord, D.R., Hoffman, H.G., Catlin., G.P., Byles, J.A., Cadman, D.T., Crawford, J,W., Links, P.S., Rae-Grant, N.I., & Szatmari, P. (1987). Ontario Child Health Study: I. Methodology. *Archives of General Psychiatry*, 44, 826-831.

Brandenburg, N.A., Friedman, R.M., & Silver, S.E. (1990). The epidemiology of childhood psychiatric disorders: Prevalence findings from recent studies. *Journal of the American Academy of Child and Adolescent Psychiatry*, 29, 76-83.

Brody, G.H., Stonemna, Z., Flor, D., McCrary, C., Hastings, L., & Conyers, O. (1994). Financial resources, parent psychological functioning, parent co-caregiving, and early adolescent competence in rural two parent African-American families. *Child Development*, 65, 590-605.

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

Campbell, S.B. (1989). Developmental perspectives. In T.H. Ollendick & M. Hersen (Eds.), *Handbook of child psychopathology* (2nd ed., pp. 5-28). New York: Plenum Press.

Caspi, A., Henry, B., McGee, R., Moffitt, T.E., and Silva, P.A., (1995). Temperamental origins of child and adolescent behavior problems from age 3 to age 15. *Child Development*, 66, 55-68.

Cicchetti, F., & Cohen, D.J. (1995). Perspectives on developmental psychopathology. In D. Cicchetti, & D.J. Cohen (Eds.), *Developmental Psychopathology: Vol. I: Theory and method* (pp.3-20). New York: Wiley.

Cicchetti, D. & Garmezy, N. (1993). Prospects and promises in the study of resilience. *Development and Psychopathology*, 4, 497-502.

Cicchetti, D., & Schneider-Rosen, K. (1986). An organizational approach to childhood depression. In M. Rutter, C. Izard, & P. Read. (Eds.), *Depression in young people: Clinical and developmental perspectives*, (pp. 71-134). New York: Guilford.

Cohen, P., Cohen, J., Kasen, S., Velez, C.N., Hartmark, C., Johnson, J., Rojas, M., Brook, J., & Streuning, E.L. (1993). An epidemiological study of disorders in late childhood and adolescence- 1. Age and gender specific prevalence. *Journal of Child Psychology and Psychiatry*, 34, 851-866.

Cohler, B.J., Stott, F.M., & Musick, J.S. (1995). Adversity, vulnerability, and resilience: Cultural and developmental perspectives. In D. Cicchetti & D.J. Cohen (Eds.), *Developmental psychopathology*, Vol. 2: *Risk, disorder, and adaptation* (pp.753-800). New York: Wiley.

Costello, E.J. & Angold, A. (1995). Developmental epidemiology. In D. Cicchetti & D.J. Cohen (Eds.), *Developmental psychopathology*, Vol. 1: *Theory and methods* (pp.7523-56). New York: Wiley.

Cytryn, L., McKnew D.H., & Zahn-Waxler, C. (1986). Developmental issues in risk research: The offspring of affectively ill parents. In M. Rutter, C.E. Izard, & Read, P.B. (Eds.), *Depression in Young People*, (pp. 166-187). New York: The Guilford Press.

Dodge, K.A., Pettit, G.S., & Bates, J.E. (1994). Socialization mediators of the relation between socioeconomic status and child conduct problems. *Child Development*, 65, 649-665.

Donohue, B., Hersen, M., & Ammerman, R.T. (1995). Historical overview. In M. Hersen & R.T. Ammerman (eds.), *Advanced abnormal child psychology* (pp. 3-19). Hillsdale, NJ: Erlbaum.

Duffy, A. (2000). Toward effective early intervention and prevention strategies for major affective disorders: A review of antecedents and risk factors. *Canadian Journal of Psychiatry*, 45 (4), 340-349.

Edelbrock, C. (1984). Developmental considerations. In T.H. Ollendick & M. Hersen (Eds.), *Child behavioral assessment: Principles and procedures* (pp. 20-37). New York: Pergamon Press.

Eme, R.F. (1979). Sex differences in childhood psychopathology: A review. *Psychological Bulletin*, 86, 574-595.

Essau, C.A., Feehan, M., Ustun, B. (1997). Classification and assessment strategies. In C.A. Essau & F. Petermann (Eds.) *Developmental Psychopathology: Epidemiology, Diagnostics and Treatment* (pp. 19-62). Amsterdam, Netherlands: Harwood Academic Publishers.

Essau, C.A., Petermann, F., & Feehan, M. (1997). Research methods and designs. In C.A. Essau & F. Petermann (Eds.) *Developmental psychopathology: Epidemiology, diagnostics and treatment* (pp. 63-97). Amsterdam, Netherlands: Harwood Academic Publishers.

Feehan, M., McGee, R., & Williams, S. (1994). Mental health disorders from age 15 to age 18 years. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 1118-1126.

Ferdinand, R.F., & Verhulst, F.C. (1995). Psychopathology from adolescence into young adulthood: an 8-year follow-up study. *American Journal of Psychiatry*, 152, 1586-1594.

Ferdinand, R.F., & Verhulst, F.C. (1999). Children from the community at risk for psychopathology in adulthood. In Steinhausen, H.C. & Verhulst, (Eds.) *Risk and outcomes in developmental psychopathology* (pp. 11-25). New York: Oxford University Press.

Fergusson, D.M., Horwood, L.J., & Lynskey, M.T. (1993). Prevalence and comorbidity of DSM-III R diagnosis in a birth cohort of 15 year olds. *Journal of the American Academy of Child and Adolescent Psychiatry*, 98, 307-313.

Fergusson, D.M., & Woodward, L.J. (1999). Maternal age and educational and psychosocial outcomes in early adulthood. *Journal of Child Psychology and Psychiatry, and allied disciplines*. 40(3), 479-489.

Frances, A., Pincus, H., Widiger, T., First, M., Davis, W., Hall, W., McKinney, K., & Stayna, H. (1994). DSM-IV and international communication in psychiatric diagnosis. In J.E. Mezzich Y Honda, & M.C. Kastrup (Eds.), *Psychiatric diagnosis: A world perspective* (pp.11-22). New York: Springer.

Friedman, R., Katz-Leavy, J., & Graetz, B.W. (1996). Prevalence in serious emotional disturbance in children and adolescents' mental health. *Washington, DC: US Government Printing Office, Department of Health and Human Services Publication, (SMA) 96-3098.*

Gelfand, D.M., & Teti, D.M. (1990). The effects of maternal depression on children. *Clinical Psychology Review, 10*, 329-353.

Graham, P. & Skuse, D. (1992). The developmental perspective in classification. In H. Remschmidt & M.H. Schmidt (eds.), *Developmental psychopathology* (pp. 1-6). Gottingen, Hogrefe and Huber Publisher.

Guerra, N.G., Tolan, P.H., Huesmann, L.R., Van Acker, R., & Eron, L.D. (1995). Stressful events and individual beliefs as correlates of economic disadvantage and aggression among urban children. *Journal of Consulting and Clinical Psychology, 63*, 518-528.

Hack, M., Breslau, N., Aram, D., Weissman, B., Klein, N., & Borawski-Clark, E. (1992). The Effect of Very Low Birth Weight and Social Risk on Neurocognitive Abilities at School Age. *Developmental and Behavioral Pediatrics, 13*(6), 412-420.

Hack, M. & Fanaroff, A.A. (1998). Outcomes of children of extremely low birthweight and gestational age in the 1990's. *Early Human Development, 53*(3), 193-218.

Hammen, C. (1991). Generation of stress in the course of unipolar depression. *Journal of Abnormal Psychology, 100*, 555-561.

Harnish, J.D., Dodge, K.A., & Valente, E. (1995). Mother-child interaction quality as a partial mediator of the roles of maternal depressive symptomatology and socioeconomic status in the development of child behavior problems. *Child Development, 66*, 739-753.

Harrington, R., Fudge, H., Rutter, M., Pickles, A., & Hill, J. (1990). Adult outcomes of childhood and adolescent depression. *Archives of General Psychiatry, 47*, 465-472.

Harris M.B. (1995). *Basic statistics for behavioral science research*. Needham Heights, Mass: Allyn and Bacon.

Hinshaw, S.P., Lahey, B.B., & Hart, E.L. (1993). Issues of taxonomy and comorbidity in the development of conduct disorder. *Development and Psychopathology, 5*, 31-49.

Hirschfeld, R.M. & Shea M.T. (1992). In Paykel E.S. (Ed) *Handbook of affective disorders*. 2nd ed. (pp 185-194). New York: Guilford Press.

Hollingshead, A.B., & Redlich, F.C. (1958). *Social class and mental illness*. New York: Wiley.

Hops, H. (1995). Age- and gender-specific effects of parental depression: A commentary. *Developmental Psychology, 31*, 428-431.

Huston, A.C. (1994). *Children in poverty*. New York: Cambridge University Press.

Huston, A.C., McLoyd, V.C., & Garcia Coll, C. (1994). Children in poverty: Issues in contemporary research. *Child Development, 65*, 275-282.

Institute of Medicine. (1989). *Research on children and adolescents with mental, behavioral, and developmental disorders*. Washington, DC: National Academy Press.

Institute of Medicine (1994). *Reducing risks for mental disorders: Frontiers for preventive intervention research*. Washington, DC: National Academy Press.

Jensen, P.S., Koretz, D., Locke, B.Z., Schneider, S., Radke-Yarrow, Richters, M., & Rumsey, J.M. (1993). Child and adolescent psychopathology research: Problems and prospects for the 1990s. *Journal of Abnormal Psychology, 21*, 551-580.

Kagan, J., Resnick, J.S., & Snidman, N. (1984). Biological bases of childhood shyness. *Science, 240*, 167-171.

Kashani, J.H., Carlson, G.A., Beck, N.C., Hooper, E.W., Corcoran, C.M., McAllister, J.A., Fallahi, C., Rosenberg, T.K., & Reid, J.C., (1987). Depression, depressive symptoms, and depressed mood among a community sample of adolescents. *American Journal of Psychiatry, 144*, 931-934.

Katz, L.F. & Gottman, J.M. (1993). Patterns of marital conflict predict children's internalizing and externalizing behaviors. *Developmental Psychology, 29*, 940-950.

Kavanagh, K., & Hops, H. (1994). Good girls? Bad boys?: Gender and development as contexts for diagnosis and treatment. In T.H. Ollendick & R.J. Prinz (Eds.), *Advances in clinical child psychology* (Vol. 16, pp. 45-79). New York: Plenum Press.

Kazdin, A.E., Kraemer, H.C., Kessler, R.C., Kupfer, D.J., & Offord, D.R. (1997). Contributions of risk-factor research to developmental psychology. *Clinical Psychology Review, 17*, 4, 375-406.

Kazdin, A.E., & Kagan, J. (1994). Models of dysfunction in developmental psychoapthology. *Clinical Psychology: Science and Practice, 1*, 35-52.

Kessler, R.C., McGonagle, K.A., Zhao, S., Nelson, C.B., Hughes, M., Eshleman, S., Wittchen, H.U., & Kendler, K.S. (1994). Lifetime and 12-month prevalence of DSM-III-R

psychiatric disorders in the United States: Results from the National Comorbidity Study. *Archives of General Psychiatry*, 51, 8-19.

Kessler, R.C., Berglund, P.A., Foster, C.L., Saunders, W.B., Stang, P.E., & Walters, E.E. (1997). Social consequences of psychiatric disorders: teenage parenthood. *American Journal of Psychiatry*, 154(10), 1405-1411.

Kraemer, H.C., Kazdin, A.E., Offord, D., Kessler, R., Jensen, R., & Kupfer, D.J. (1997). Coming to terms with the terms of risk. *Archives of General Psychiatry*, 54, 337-343.

Leckman, J.F., Merikangas, K.R., Pauls, D.L., Prusoff, B.A., & Weissman, M.M. (1983). Anxiety disorders and depression: Contradictions between family study data and DSM-III conventions. *American Journal of Psychiatry*, 140, 809-818.

Lee, M.C., Shung, L.A., Lu T.H., & Chou, M.C. (1998). Association of parental characteristics with adverse outcomes of adolescent pregnancy. *Family Practice*, 15(4), 336-342.

Lewinsohn, P.M., Hops, H., Roberts, R.E., Seeley, J.R., & Andrews, J.A. (1993). Adolescent psychopathology: I. Prevalence and incidence of depression and other DSM-III R disorders in high school students. *Journal of Abnormal Psychology*, 102, 133-144.

Lewinsohn, P.M., Rhode, P., Klein, D.N., & Seely, J.R. (1999). Natural course of adolescent major depressive disorder: Continuity into young adulthood. *Journal of American Academy of Child and Adolescent Psychiatry*, 38, 56-63.

Loeber, R., & Farrington, D.P. (1994). Problems and solutions in longitudinal and experimental treatment studies of child psychopathology and delinquency. *Journal of Consulting and Clinical Psychology*, 62, 5, 887-900.

Luthar, S.S. (1997). Sociodemographic disadvantage and psychosocial adjustment: Perspectives from developmental psychopathology. In S.S., Luthar, J.A., Burack, D. Cicchetti & J.R., Weisz, (Eds.), *Developmental psychopathology: Perspectives on adjustment, risk, and disorder* (pp. 459-485). New York: Cambridge University Press.

Malkoff-Schwartz, S., Frank, E., Anderson, B., Sherrill, J.T., Siegel, L., & Patterson, D. (1998). Stressful life events and social rhythm disruption in the onset of manic depressive bipolar episodes: A preliminary investigation. *Archives of General Psychiatry*, 144, 55, 702-707.

Martin, R.P., Bridger, R., Huttunen, M. (2000). Prediction of mental health and somatic health hospital admissions from early childhood temperament types. Paper presented at the International Congress of Psychology, Stockholm, Sweden, July 2000.

Mash, E.J. & Dozois, A.A.J. (1996). Child psychopathology: A developmental systems perspective. In E.J. Mash & R.A. Barkley (Eds.), *Child psychopathology* (pp. 3-62). New York: Guilford Press.

Mash, E.J., Johnston, C., & Kovitz, K. (1983). A comparison of the mother-child interactions of physically abused and non-abused children during play and task situations. *Journal of Clinical Child Psychology, 12*, 337-346.

Maughan, B. & Lindelow, M. (1997). Secular change in psychosocial risks: The case of teenage motherhood. *Psychological Medicine, 27*(5), 1129-1144.

McGee, R. & Silva, P.A. (1985). Non co-operation of preschoolers. *American Journal of Diseases in Children, 140*, 8-9.

McGee, R., & Williams, S. (1988). A longitudinal study of depression in nine-year-old children. *Journal of the American Academy of Child and Adolescent Psychiatry, 27*, 342-348.

McGee, R., Feehan, M., Williams, S., Patridge, F., Silva, P.A., & Kelly, J., (1990). DSM-III disorders in a large sample of adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 29*, 611-619.

McGee, R., Feehan, M., Williams, S., & Anderson, J. (1992). DSM-III disorders from age 11 to age 15 years. *Journal of the American Academy of Child and Adolescent Psychiatry, 31*, 50-59.

McGee, R., Williams, S., & Feehan, M. (1994). Behaviour problems in New Zealand children. In P.R. Joice, R.T. Mulder, M.A. Oakley-Browne, J.D. Sellman, and W.G. A. Watkins (Eds.), *Development, personality, and psychopathology* (pp.15-22). Christchurch School of Medicine, Department of Psychological Medicine, Christchurch, New Zealand.

McGee, R., Feehan, M., & Williams, S. (1995). Long-term follow-up of a birth cohort. In C.A. Essau & F. Petermann (Eds.), *Developmental psychopathology: epidemiology, diagnostics and treatment* (pp. 366-385). Amsterdam, Netherlands: Harwood Academic Publishers.

McDermott, P.A., & Weiss, R.V. (1995). A normative typology of healthy, subclinical, and clinical behavioral styles among African American children and adolescents. *Psychological Assessment, 7*, 162-170.

McLoyd, V.C. (1989). Socialization and development in changing economy. *American Psychologist, 44*, 293-302.

Monroy De Velasco, A. (1982). Consequences of early childbearing. *Draper Fund Report, Dec.(11)*, 26-27.

Murray, C.J.L., & Lopez, A.D. (1996). Evidence-based health policy: Lessons from the Global Burden of Disease Study. *Science, 247*, 740-743.

National Commission on Children. (1991). *Beyond rhetoric: A new American agenda for children and families: The final report on National Commission on Children*. Washington, DC: U.S. Government Printing Office.

Nesselroade, J.R. & Baltes, P.B. (1979). *Longitudinal research in the study of behavior and development*. San Diego, CA: Academic Press.

Newman, D.L., Moffitt, T.E., Caspi, A., Magdol, L., Silva, P.A., & Stanton, W.R. (1996). Psychiatric disorder in a birth cohort of young adults: Prevalence, comorbidity, clinical significance, and new case incidence from ages 11 to 21. *Journal of Consulting and Clinical Psychology, 64*(3), 552-562.

Offord, D.R., Boyle, M.H., Racine, Y.A., Fleming, J.E., Cadman, D.T., Blum, H.M., Byrne, C., Links, P.S., Lipman, E.L., MacMillan, H.L., Grant, N.I.R., Sanford, M.N., Szatmari, P., Thomas, H., & Woodward, C.A. (1992). Outcome, prognosis, and risk in a longitudinal follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry, 31*, 916-23.

Okun, A., Parker, J.G., Levendosky, A.A. (1994). Distinct and interactive contributions of physical abuse, socioeconomic disadvantage, and negative life events to children's social, cognitive, and affective adjustment. *Development and Psychopathology, 6*, 77-98.

Ollendick, T.H., Mattis, S.G., & Neville, J.K. (1994). Panic in children and adolescents: A review. *Journal of Child Psychology and Psychiatry, 35*, 113-134.

Olweus, D. (1979). Stability of aggressive reaction patterns in males: A review. *Psychological Bulletin, 86*, 852-875.

Post, R.M. (1992). Transduction of psychological stress into neurobiology of recurrent affective disorder. *American Journal of Psychiatry, 149*, 999-1010.

Prior, M., Smart, D., Sanson, A., & Oberklaid, F. (2001). Longitudinal predictors of behavioural adjustment in pre-adolescent children. *Australian and New Zealand Journal of Psychiatry, 35* (3), 297-308.

Quay, H.C. (1986). Classification. In H.C. Quay & J.S. Werry (Eds.) *Psychopathological disorders in childhood* (3rd ed., pp.1-34). New York:Wiley.

Reinherz, H.Z., Giaconia, R.M., Lefkowitz, E.S., Pakiz, B., & Frost, A.K. (1993). Prevalence of psychiatric disorders in a community population of older adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 32*, 369-377.

Richman, N., Stevenson, J., & Graham, P.J. (1982). *Preschool to school: a behavioural study*. Academic Press, London.

- Ringeisen, H., Oliver, K.A., & Menvielle, E. (2002). Recognition and treatment of mental disorders in children: Considerations for pediatric health systems. *Pediatric Drugs*, 4 (11), 697-703.
- Roberts, R.E., Attkisson, C.C., & Rosenblatt A. (1998). Prevalence of psychopathology among children and adolescents. *American Journal of Psychiatry*, 155, (6), 715-725.
- Roosa, M.W., Fitzgerald, H.E., & Carlson, N.A. (1982). Teenage parenting and child development: a literature review. *Infant Mental Health Journal*, 3(1), 4-18.
- Rothbart, M.K. & Ahadi, S.A. (1994). Temperament and the development of personality. *Journal of Abnormal Psychology*, 103, 55-66.
- Rutter, M. (1965). Classification and categorization in child psychiatry. *Journal of Child Psychology and Psychiatry*, 6, 71-83.
- Rutter, M. (1979). Protective factors in children's responses to stress and disadvantage. In M.W. Kent & J.E. Rolf (Eds.), *Primary prevention in psychopathology* (pp.49-74). Hanover, NH: University Press of New England.
- Rutter, M. (1986). The developmental psychopathology of depression: Issues and Perspectives. In M. Rutter, C.Z. Izard, & P.B. Read (Eds.), *Depression in young people: Developmental and Clinical Perspectives* (pp.3-32). New York: Guilford Press.
- Rutter, M. (1989). Isle of Wight revisited: Twenty-five years of child psychiatric epidemiology. *Journal of the American Academy of Child and Adolescent Psychiatry*, 28, 633.
- Rutter, M., Tizard, J., Yule, W., Graham, P., & Withmore, K. (1976). Isle of Wight studies, 1964-1974. *Psychological Medicine*, 6, 313-32.
- Rutter, M., & Rutter, M. (1993). *Developing minds: Challenge and continuity across the life span*. New York: basic Books.
- Samerof, A.J., Seifer, R., & Bartko T.W. (1997). Environmental perspectives on adaptation during childhood and adolescence. In S.S., Lutar, J.A., Burack, D. Cicchetti & J.R., Weisz, (Eds.), *Developmental psychopathology: Perspectives on adjustment, risk, and disorder* (pp. 507-536). New York: Cambridge University Press.
- Sampson, R.J., & Laub, J.H. (1994). Urban family and the family context of delinquency: A new look on a structure and process in a classic study. *Child Development*, 65, 523-540.
- Sawyer, M.G., Arney, F.M., Baghurst, P.A., Clark, J.J., Graetz, B.W., Kosky, R.J., Nurcombe, B., Patton, G.C., Prior, M.R., Raphael, B., Rey, L.C., Whaites, L.C., & Zubrick, S.R. (2001). The mental health of young people in Australia: Key findings from the child and

adolescent component of the national survey of mental health and well-being. *Australian and New Zealand Journal of Psychiatry*, 35, 806-814.

Silva, P.A. (1990). The Dunedin Multidisciplinary Health and Development Study: a fifteen year longitudinal study. *Perinatal and Pediatric Epidemiology*, 4, 76-107.

Sokal, R.R. & Rohlf, F.J. (1995). *Biometry: The principles and practice of statistics in biological research* (3rd ed). N.Y.: W.H. Freeman and Company.

Spitzer, R.L., Davies, M., & Barkley, R.A. (1990). The DSM-III R field trial of disruptive behavior disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 29, 690-697.

Sroufe, L.A., & Jacobovitz, D. (1989). Diverging pathways, developmental transformations, multiple etiologies and problem of continuity in development. *Human Development*, 32, 196-203.

Sroufe, L.A. & Rutter, M. (1984). The domain of developmental psychopathology. *Child Development*, 55, 17-29.

Steinhausen H.C., & Verhulst, F.C. (1999). Introduction. In H.S. Steinhausen and F.C. Verhulst (Eds.), *Risks and outcomes in developmental psychopathology*. New York: Oxford University Press.

Strauss, C.C., Last, C.G., Hersen, M., & Kazdin, A.E. (1988). Association between anxiety and depression in children and adolescents with anxiety disorders. *Journal of Abnormal Child Psychology*, 16, 57-68.

Tremblay, R.A., Masse, B., Perron, B., LeBlanc, M., Schwartzman, A., & Ledingham, J.E. (1992). Early disruptive behavior, poor school achievement, delinquent behavior, and delinquent personality: Longitudinal analysis. *Journal of Consulting and Clinical Psychology*, 60, 64-72.

U.S. Bureau of the Census. (1993). *Poverty in the United States: 1992* (Current Population Reports, Series P-60, No.185). Washington, DC: U.S. Government Printing Office.

U.S. Department of Health and Human Services. (2000). Report of the Surgeon General's conference on Children's mental health: a national action agenda. Washington, DC: Department of Health and Human Services.

Verhulst, F.C. (1995). The epidemiology of child and adolescent psychopathology: Strengths and limitations. In F.C. Verhulst & H.M. Koot (Eds.), *The epidemiology of child and adolescent psychopathology* (pp.1-21). Oxford: Oxford University Press.

Verhulst, F.C. & Koot, H.M. (1992). *Child psychiatric epidemiology: Concepts, methods, and findings*. Newbury Park, CA: Sage.

Verhulst, F.C. & van der Ende, J. (1993). "Comorbidity" in an epidemiological sample: A longitudinal perspective. *Journal of Child Psychology and Psychiatry*, 34, 767-783.

Von Kroff, M. & Eaton, W.W. (1989). Epidemiologic findings on panic. In R. Baker (Ed.), *Panic disorder: Theory, research and therapy* (pp.35-50). New York: Wiley.

Waddell, C., Offord, D.R., Shepherd, C.A., Hua, J.M., & McEwan, K. (2002). Child psychiatric epidemiology and Canadian policy-making: The state of the science and the art of the possible. *Canadian Journal of Psychiatry*, 47, 825-833.

Warner, L.A., Kessler, R.C., Hughes, M., Anthony, J.C., & Nelson, C.B. (1995). Prevalence and correlates of drug use and dependence in the United States. *Archives of General Psychiatry*, 52, 219-229.

Weissman, M.M., Fendrich, M., Warner, V., & Wickramaratne, P., (1992). Incidence of psychiatric disorder in offspring at high and low risk for depression. *Journal of the American Academy of Child and Adolescent Psychiatry*, 31, 640-648.

Weisz, J.R., & Suwanlert, S. (1989). Over- and under-controlled referral problems among children and adolescents from Thailand and the United States: The wat and wai of cultural differences. *Journal of Consulting and Clinical Psychology*, 55, 719-726.

Werner, E.E. & Smith, R.S. (1992). *Overcoming the odds: High risk children from birth to adulthood*. Ithaca, N.Y.: Cornell University Press.

Whitaker, A., Johnson, J., Shaffer, D., Rapoport, J.L., Kalokow, K., Walsh, B.T., Davies, M., Braiman, S., & Dolinsky, A. (1990). Uncommon troubles in young people: Prevalence estimates of selected psychiatric disorders in nonreferred population. *Archives of General Psychiatry*, 47, 487-496.

Williams, S., Anderson, J., McGee, R., & Silva, P.A. (1990). Risk factors for behavioral and emotional disorder in preadolescent children. *Journal of the American Academy of Child and Adolescent Psychiatry*, 29, 413-419.

World Health Organization (1993). *The ICD-10 classification of mental and behavioral disorders*. Geneva: World health Organization.

Zahn-Waxler, C., Cole, C.M., Welsh, J.D., & Fox, N.A. (1995). Psychophysiological correlates of empathy and prosocial behaviors in preschool children with behavior problems. *Development and Psychopathology*, 7, 27-48.

Zimmerman, M.A. & Arunkumar, R. (1994). *Resiliency research: Implications for schools and policy*. Social Policy Report, 8(4), 1-17.

Zoccolillo, M, Meyers, J., & Assiter, S. (1997). Conduct disorder, substance dependence, and adolescent motherhood. *American Journal of Orthopsychiatry*, 67(1), 152-157.

APPENDIX

ICD-9-CM Codes and DiagnosesAddictive and substance abuse disorders:

Alcohol abuse (305.0)

Cannabis abuse (305.2)

Opioid abuse (305.5)

Other, mixed, or unspecified drug abuse (305.9)

Other specified drug dependence (304.6)

Adjustment disorders and reactions:

Unspecified acute reaction to stress (308.9)

Adjustment reaction (309)

Adjustment reaction-brief depressive reaction (309.0)

Adjustment reaction with primary disturbance of other emotions (309.2)

Adjustment reaction with predominant disturbance of conduct (309.3)

Adjustment reaction with mixed disturbance of emotions and conduct (309.4)

Other specified adjustment reactions (309.8)

Unspecified adjustment reaction (309.9)

Anxiety Disorders:

Neurotic Depression (300.4)

Other Neurotic Disorders (300.8)

Unspecified Neurotic disorder (300.9)

Anxiety states (300.0)

Hysteria (300.1)

Phobic disorders (300.20)

Obsessive-compulsive disorder (300.3)

Depressive/Mood Disorders:

Major depressive disorder (296.2)

Depressive disorders not elsewhere specified (311)

Disorders of childhood and adolescence:

Disturbance of conduct, not elsewhere classified (312)

Undersocialized conduct disorder, aggressive type (312.)

Disorders of impulse control, not elsewhere classified (312.3)

Other or mixed emotional disturbances of childhood or adolescence (313.8)

Hyperkinetic syndrome of childhood (313.9)

Hyperkinesis with developmental delay (314.1)

Unspecified hyperkinetic syndrome (314.9)

Personality Disorders:

Explosive personality disorder (301.3)

Other personality disorder (301.8)

Psychosis:

Unspecified psychosis (298.9)

Other specified early childhood psychosis (299.8)