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Mania and intellectual disability: the course of mania symptoms in persons with disability over three years

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MANIA AND INTELLECTUAL DISABILITY: THE COURSE OF MANIA SYMPTOMS IN PERSONS WITH INTELLECTUAL DISABILITY OVER THREE YEARS

A Thesis

Submitted to the Graduate Faculty of the Louisiana State University and Agricultural and Mechanical College
In partial fulfillment of the requirements for the degree of Master of Arts

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The Department of Psychology

By
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ABSTRACT

Although bipolar disorder was one of the earliest described mental illnesses, there is a dearth of research on bipolar disorder in individuals with intellectual deficits. The present study aimed to extend this literature by comparing the presence and variation of manic symptoms over time of persons with intellectual deficits with and without bipolar disorder. Three groups of individuals participated: a bipolar group, a psychopathology group (other than bipolar disorder) and a control group. Two dependent measures of mania were taken from retrospective data, *Mania subscale of the DASH-II* and a *Criterion-referenced subscale*. The presence and consistency of mania symptom endorsements were analyzed over time and across groups. Results indicated that the bipolar group had greater mean endorsements on the Criterion-referenced subscale than the psychopathology and control groups. Further, manic symptom endorsements were more stable over time in the bipolar group than the other two groups. This pattern of serial correlations was inconsistent with hypotheses. These findings are tempered by the fact that the patterns of serial correlations in comparative anchor subscales were also unanticipated. In order to clarify these unexpected findings, research is needed to examine the accuracy of staff to report the frequency of symptomology.
INTRODUCTION

With recent advances in pharmacological treatment, bipolar disorder has come to the forefront of clinical research. Although bipolar disorder is by no means a new disorder, very little is known about how it is manifested in individuals with intellectual disabilities (ID). The present study aims to investigate the presentation of manic symptoms in individuals with ID. A brief overview of the history of ID, dual diagnosis, bipolar disorder, and the comorbidity of bipolar with ID will be discussed.

History of Intellectual Disability

Throughout history there have been individuals deemed less or more capable than others. Over time, the view of the less capable individual has changed with the needs and expectations of the society (Scheerenberger, 1983). Historians suggest that infanticide was a common practice before and during the middle ages. That is, individuals with deficits or physical handicaps preventing them from hunting, gathering, or keeping with expectations of the society were often killed at birth or died at an early age. However, those with mild deficits who were able to contribute to their community probably survived (Scheerenberger, 1983).

During the middle ages, more attention was given to those with physical abnormalities and other medical problems such as hydrocephaly and epilepsy (Ruhrah, 1925). It was not until the sixteenth century that the Swiss physician Paracelsus made a distinction between ID and mental illness. Platter, another Swiss physician, was the first to offer a multi-level description of ID. Although ID was given more attention and recognition by the physicians of the time, the condition was deemed untreatable (Scheerenberger, 1983).

The seventeenth and eighteenth centuries were marked with significant advances to science, medicine and, thus, the understanding of ID. The term idiocy had become the commonly
accepted scientific term of ID. Philosophers of this time, such as John Locke, had a great impact on human thinking and the treatment of individuals with ID. Locke’s empirical theory of knowledge, which stated that the mind is a blank table (tabula rasa) at birth and that ideas come from experience, provided a basis for the treatment and training for individuals with ID (Murphy, 1949). Locke also recognized the difference between idiots (ID) and madmen (mental illness; Doll, 1962).

Considerable progress was made to understand ID and identify associated illnesses in the nineteenth century. Physician W. J. Little was the first to draw critical attention to the potential deleterious effects of premature birth, difficult labor, and mechanical injuries during delivery and hypoxia. Little’s research played a vital role in emphasizing the importance of prenatal and perinatal care. J. Langdon Down also highlighted the importance of the emotional health of childbearing women. Additionally, Down advanced the treatment and the education of individuals with ID (Scheerenberger, 1983).

The works of Jean Etienne Domique Esquirol and Edouard Seguin furthered the concept of the differentiating degrees of ID. Esquirol made a distinct contrast between amentia (ID) and dementia (mental illness). He also divided those with ID into two levels: the imbecile and the idiot (Scheerenberger, 1983). Seguin was the student of Itard, the first to develop a broad educational program for a child who was deaf and mute. Seguin was sensitive to the varying levels of functional behavior among persons with ID. He divided idiocy into four broad categories: idocy, imbecility, backwardness or feeblemindedness, and simpleness or superficial retardation. Seguin’s physiological method, a systematic training of the senses, was an educational approach that had a great impact on the education of those with ID. Many of the ideas and techniques (i.e., simple movements, imitation and generalization) of this method are
still employed in current treatments, such as positive reinforcement procedures and modeling (Scheerenberg, 1983). As Seguin advocated for the education of individuals with ID, he also emphasized the importance of their moral treatment (Talbot, 1964).

At the turn of the century, the interest in ID soared. Many organizations, publications, and programs were established in the interest of those with ID. In 1876, the first meeting of the American Association on Mental Deficiency (AAMD) was held. The following year, 1877, the AAMD officially announced the first definition of ID. By the twentieth century, the three essential components of a definition of ID were well recognized as: 1) early onset; 2) reduced intellectual functioning due to developmental disorder; and 3) an inability to adapt to the full demands of society (Scheerenberger, 1983).

Not only was this a time for professionals to gather to discuss and study the etiology and prevalence of ID, but also a time when the services for these individuals were formulated. In 1911, New Jersey passed the first state law requiring the mandatory special education of children with mild ID. The goal of these classes evolved from simply removing the most difficult or trying from the regular classroom, to raising the moral tone of children, making them more capable in their family life, and later to focusing on academic work and physical education (Scheerenberger, 1983).

Eligibility for special education classes soon came into question. Thus, the need for psychological and intellectual measurement increased. The Binet-Simon Individual Tests of Intelligence, the first intelligence test, appeared in 1905. This version was intended to distinguish between subnormal and normal school-aged children, and it was interpreted in terms of the three levels of ID: idiocy, imbecility, and moronity (Scheerenberger, 1983). Although the early part of the century can be characterized as an increased sensitivity to those with ID, there were also
several restrictive measures taken. Laws were passed that prohibited the marriage among the
intellectually disabled. Sterilization was another measure pursued to prevent reproduction of
those with ID, along with criminals, the physically deformed, and mentally insane. These
negative attitudes toward individuals with ID drove the segregation of these individuals into
institutions away from the rest of society (Scheerenberger, 1983).

In 1924, one of the field’s leaders, Fernald, delivered a speech that acknowledged that no
two individuals with ID were exactly alike: “What is good for one may be bad for another…No
routine procedure will meet the needs of this highly differentiated group” (Fernald, 1924;
pp.217-218). Thus, during this time the special education classes evolved and began to recognize
the student with ID as an individual with differing educational needs (Baker, 1937). In 1930,
President Hoover’s subcommittee on problems involving mental deficiency introduced the first
Bill of Rights for the Handicapped Child (Scheerenberger, 1983).

During the 1940s and 1950s the new version of the Stanford-Binet was introduced,
providing a new classification system for the levels of ID or mental retardation: Borderline (IQ
67-83), Mild (IQ 50-66), Moderate (IQ 33-49), Severe (IQ 16-32), Profound (IQ 16 or below).
This new terminology was introduced to avoid the negativism associated with the earlier
classifications. In 1961, the AAMD distributed their revised definition of ID: ‘Mental retardation
refers to subaverage general intellectual functioning which originates in the developmental period
and is associated with impairment in adaptive behavior’ (Herber, 1961). The revised definition
placed priority on adaptive behavior in the determination of ID. The first measure of adaptive
behavior, introduced in 1936, was the Vineland Social Maturity Scale (Doll, 1935). The rising
interest in adaptive behaviors precipitated the development and funding of learning research.
This research investigated nearly every behavioral aspect of children, youth, and adults with intellectual disabilities (Scheerenberg, 1983).

The second half of the twentieth century was marked by institutional reform and efforts toward deinstitutionalization. The quality of life and physical environment of the institution came under scrutiny; the reformist believed the institution’s residents had a right to live in a more normal environment. Efforts went to establish standards to guide the development and assessment of programs and to petition legislature for funding. By the 1970s the national goal was deinstitutionalization. The primary purpose of the institution changed; the institution was now considered a transitory placement intended to rehabilitate and prepare individuals for life in the community (Scheerenberger, 1983). This community stage in the evolution of service models was characterized by several changes, including a decline in the admission of children and the number of adults living in institutions. Also, there was the creation of community settings of small residential units and the establishment of community-based day programs serving as ‘sheltered workshops’ (Greenspan, 1999).

Greenspan (1999) terms the most recent shift in the service model as the supports stage of ID services. This service model is centered on the belief that individuals with ID have more potential for success than was previously thought. The current view asserts that progress can best be attained when support is given in a ‘normal’ work, school and home setting where services are tailored to meet the needs of the individual. This movement focuses on the strengths and weaknesses of the individual, and thus has changed the concept of ID. As a result, the supports model has generated much controversy and opposition in the field.
Current Definition of Intellectual Disability

ID, or mental retardation, is characterized by three criterion: 1) sub-average intellectual functioning; 2) significant limitations in adaptive skills; and 3) onset before age 18 (American Psychological Association, APA, 2000). Intellectual functioning is defined by the intelligence quotient (IQ). IQ is obtained with the use of one or more standardized, individually administered intelligence tests (i.e., Standford-Binet, Wechsler Intelligence Scales; APA, 2000), which have been noted to have an approximate measurement error of five points. To qualify as having sub-average intellectual functioning, an individual must have an IQ at 70 or below, which is two standard deviations below the mean. Though IQ is a defining factor of intellectual functioning, there is widespread dissatisfaction with the reliance on measures of intelligence because of their co-variation with socioeconomic factors (Flanagan, Genshaft, & Harrison, 1997). Blatt and Kaplan (1966) elucidated the problems and pitfalls of relying too heavily on IQ, thus highlighting the necessary consideration of adaptive behavior.

The second criterion, limitations in adaptive functioning, refers to the inability of an individual to cope with life demands typical of someone of his age, background, and community surroundings (APA, 2000). This criterion requires significant skill impairment in two or more of the following areas: communication, self-care, home living, social/interpersonal skills, use of community resources, self-direction, functional academic skills, work, leisure, health, and safety (APA, 2000). Scales such as the Vineland Adaptive Behavior Scales (VABS) have been designed to assess levels of adaptive functioning (Sparrow, Balla, & Cicchetti, 1984). While impairments in intellectual functioning are fairly stable past the age of six, adaptive functioning skills are likely to improve with training. The VABS has been normalized on the general
population, making it possible to derive overall and sub-scale adaptive behavior scores (Greenspan, 1999).

Onset before age 18 is the final criterion for a diagnosis of ID. APA (2000) states that the age of onset depends on the etiology and level of intellectual impairment. That is, more severe ID tends to be recognized earlier than milder levels of ID. In addition, retardation associated with syndromes such as fragile X syndrome is usually diagnosed at birth, whereas retardation with an unknown etiology tends to be diagnosed later (Greenspan, 1999).

There are currently four degrees of ID that reflect the level of intellectual impairment: **Mild ID** (IQ 50-55 to 70); **Moderate ID** (IQ 35-40 to 50-55); **Severe ID** (IQ 20-25 to 35-40), and **Profound ID** (IQ below 20-25). The first level, Mild ID, accounts for approximately 85% of all individuals with ID (APA, 2000; Greenspan, 1999). These individuals typically develop social and communication skills, have minimal sensorimotor impairment, and are often not easily distinguished from non-disabled individuals. Individuals with Mild ID usually achieve social and vocational skills, but may require supervision, guidance, and assistance during stressful situations.

Moderate ID, the second level of impairment, accounts for approximately 10% of all individuals with ID. These individuals usually acquire communication skills during childhood. With moderate supervision, they profit from vocational training and attend to their own personal care. Generally, individuals with Moderate ID adapt well to life in the community with supervision (APA, 2000).

The third level of impairment, Severe ID, constitutes for 3% to 4% of all persons with ID. These individuals typically do not have communicative speech during early childhood, but may
learn to talk and gain some simple self-care skills and some pre-academic skills during school age. Persons with severe ID usually require supervision in most settings (APA, 2000).

The final and most severe degree of intellectual impairment is Profound ID. This level of impairment accounts for only approximately 1% of all persons with ID. ID in these individuals most often stems from an identified neurological condition. These individuals usually have considerable impairment in sensorimotor functioning, communication, and self-care skills. However, these skill areas may improve with training (APA, 2000).

Etiology and Prevalence of ID

Predisposing factors to ID range widely, and include biological, psychosocial, or a combination of factors. Heredity has been identified as one major predisposing cause. Errors of metabolism, as in Tay-Sachs disease, single gene abnormalities, and chromosomal aberrations are associated with intellectual disabilities. Alterations in early embryonic development due to chromosomal changes and prenatal toxins in syndromes, such as Down syndrome and Fetal Alcohol Syndrome respectively, can also result in intellectual deficits. Environmental factors before, during, and after birth impact the child’s intellect. For example, fetal malnutrition, prematurity, hypoxia, viral infections, and trauma are common etiological factors. Moreover, nutritional deprivation, lack of social and linguistic stimulation, infections, traumas, and poisoning after birth may also account for impairments (APA, 2000; Leonard & Wen, 2002). Although many etiological factors have been identified, approximately 30% to 40% of persons with ID have no identified etiology (APA, 2000).

Prevalence rates of intellectual deficits have approximated 1% (Larson, Lakin, Anderson, Kwak, Lee, & Anderson, 2001; Leonard & Wen, 2002). However, rates have varied among studies due to differences in major definitions, classification systems, data collection, and
sampling techniques (Roeleveld, Zielhuis, & Gabreels, 1997). Leonard and Wen’s (2002) review of the literature asserts that the prevalence of ID varies with gender, maternal race, socio-economic, and educational status. Richardson, Katz, & Koller (1986) found that male children have a higher prevalence of ID than females. The gender difference has been often attributed to X-linked conditions, such as Fragile X and unidentified X-linked conditions (Leonard & Wen, 2002). In addition, environmental factors influenced by social class have an impact on the prevalence of ID. Studies have consistently found that the prevalence of Mild ID has been strongly associated with low economic status and a poor educational background of the mother (Decoufle & Boyle, 1995; Drews, Yeargin-Allsopp, Decoufle, & Murphy 1995). Low birth weight is a common indicated risk factor that possibly results from maternal smoking and maternal urinary tract infections (Leonard & Wen, 2002).

Dual Diagnosis

Individuals with ID are estimated to be three to four times more likely to have a psychiatric disorder than those in the general population (APA, 2000; Borthwick-Duffy, 1994). Dual Diagnosis, the coexistence of ID and mental illness, is a relatively new concept that has developed in the last twenty years. Historically, most professionals thought that individuals with ID were incapable of developing psychiatric disorders (Borthwick-Duffy, 1994). Abnormal behaviors were most often attributed to the individual’s ID rather than the presence of a mental illness (Schroeder, Mulick & Schroeder, 1979). More recently, individuals with intellectual deficits were viewed capable of developing mental illness. However, this view holds that a mental disorder of a person with ID is qualitatively different than the mental disorder of an individual with no intellectual impairment (Szymanski & Grossman, 1984). Current views of dual diagnosis are that individuals with ID are likely to have mental disorders similar to those in
the general population. Moreover, it is generally recognized these disabled individuals are more vulnerable to mental illness than their counterparts in the general population (Borthwick-Duffy, 1994).

While most professionals now recognize that individuals with ID are capable and likely to suffer from a mental illness, diagnosing the psychiatric disorder in these individuals is not easily done. Deficits in communication and adaptive skills often make self-report of symptoms impossible, which is frequently the basis of diagnostic interviews and the criteria listed in the Diagnostic and Statistical Manual of Mental Disorders 4th ed.-Text Revision (e.g., flight of ideas, more talkative than usual, inflated self-esteem, feelings of worthlessness or inappropriate guilt; DSM-IV-TR; APA, 2000). Oftentimes these skill deficits are a road block in determining if the abnormal behavior is due to psychiatric disorder, brain injury associated with the intellectual deficits or environmental factors (Reid, 1983).

Graham and Rutter (1968) found that while parents and teachers are able to describe their child’s abnormal behavior, they are less proficient at interpreting the source of the behavior problem. In addition, Costello (1982) asserted that the presence of mental illness in individuals with ID often results in an exaggerated interpretation of intellectual deficits. For example, Reiss, Levitan, and Szyszko (1982) conducted an experiment presenting a single case to many psychologists. The case description was identical except that it varied slightly in the description of the individual; psychologists in condition one were given background information indicating that the individual was intellectually disabled, psychologists in condition two were told the individual was of normal intelligence, and finally, psychologists in condition three were told the individual was an alcoholic. Findings from this study and replication studies indicated that the presence of ID decreased the importance or salience of abnormal behavior. This phenomenon,
commonly called diagnostic overshadowing, is based on the tenet that intellectual deficit is such a salient feature of ID that any co-occurring abnormal behavior is attributed to the deficits in intelligence.

Although currently there is a lack of appropriate diagnostic criteria specifically for persons with ID, several researchers have developed assessment scales to assess psychopathology in this population (Rush, Bowman, Eidman, Toole, & Mortenson, 2004). Scales such as the Aberrant Behavior Checklist (ABC; Aman, Singh, Stewart, & Field, 1985), the Reiss Screen for Maladaptive Behavior (Reiss, 1988), the Assessment of Dual Diagnosis (ADD; Matson, 1997) and the Diagnostic Assessment for the Severely Handicapped-Revised (DASH-II, Matson, 1995) are used to aid in the screening and/or diagnosis of psychopathology in the intellectually disabled population.

Etiology of Dual Diagnosis

Organic, behavioral, developmental, and sociocultural models have been proposed as etiological theories for dual diagnosis (Matson & Sevin, 1994). Organic models of dual diagnosis focus on physiological, biochemical, and genetic factors that may predispose individuals to mental illness. For example, the high occurrence of individuals with Downs Syndrome developing Alzheimer’s dementia is suggestive of an underlying genetic cause of this psychopathology (Sovner & Pary, 1993).

Behavioral models emphasize the interactions between the individual and the environment. These models focus on the principles of classical conditioning, social learning theory and operant psychology (Matson & Sevin, 1994). Pavlovian classical conditioning models have been discussed in the development of anxiety in the general population and in individuals with ID (Ollendick & Ollendick, 1982). Social learning theory suggests that fears and phobias
may be the result of an individual observing another’s reaction to an event or object (Matson & Sevin, 1994). Operant models, on the other hand, are based on the principles of inadequate reinforcement of prosocial behaviors, inappropriate punishment, reinforcements of deviant response sets, and altered stimulus-response functions such as decreased learning abilities (Matson & Sevin, 1994).

Developmental models suggest that development and sequences of cognitive development are fixed and universal. Individuals with ID develop at slower rates than the general population. Thus, their behavior is indicative of their stage of development. Sternlicht (1979), for example, found that patterns of fears of institutionalized individuals with ID were typical of their developmental stage.

The final model, the sociocultural theory of dual diagnosis, highlights the social experience of the individuals and how they may impact psychopathology. Reiss and Benson (1984), for example, have pointed out the numerous negative experiences of individuals with ID such as restrictive placements and social rejection that may impact their mental health. Moreover, Rojahn, and Tasse (1996) asserted that one possible explanation for the increased rates of psychopathology in individuals with intellectual deficits is their lack of coping skills. Not being able to cope with stressful situations may lead to a higher vulnerability to psychopathology and behavior problems. Though there are several promising models for dual diagnosis, investigators should continue to study possible etiological factors of psychopathology (Matson & Sevin, 1994).

Prevalence of Dual Diagnosis

Similar to what was found in the prevalence studies of ID, prevalence rates of dual diagnosis within the disabled population are confounded with issues concerning definitions,
diagnosis, sampling, and data collection (Borthwick-Duffy, 1994). Identifying dual diagnosis requires reliable and valid assessments of both ID and mental illness. Aman’s (1991) review of instruments indicated that the psychometric properties of the scale must be considered for both the overall psychopathology and the specific diagnosis. For example, the Reiss Screen is based on a representative but small standardization sample, which could have implications on the confidence in the cut-off scores. Further, sampling techniques complicate the task of estimating prevalence. Many investigations dealing with prevalence of dual diagnosis are based on samples of institutionalized individuals or service system databases. These samples tend to be skewed toward individuals with greater disability who are in need of services, and thus unrepresentative of the intellectually disabled population. These issues result in inflated estimates of dual diagnosis (Borthwick-Duffy, 1994).

Borthwick-Duffy and Eyman (1990) asked the question, “Who are the dually diagnosed?” After reviewing demographic, adaptive and maladaptive behavior, and diagnostic information for clients receiving state services, the authors concluded that individuals who are referred for psychiatric evaluation and consequently dually diagnosed are likely to have higher cognitive capabilities. That is, individuals with the cognitive capacity to cause disruptions in daily life and lack social skills are more likely to be referred because of their challenging behaviors (e.g., aggression, unacceptable social behavior, and resistance). Secondly, individuals residing in an institution are more likely to have access to qualified professionals to assess for psychopathology, thus increasing the likelihood of dual diagnosis. Finally, diagnostic overshadowing often plays a role in the prevalence rate of dual diagnosis across levels of ID. That is, individuals with profound intellectual deficits are less likely to be identified as mentally
ill because of the severity of their intellectual deficits and the tendency to attribute abnormal behavior to low IQ rather than a psychiatric disorder.

Findings toward the relationship of dual diagnosis with age, gender, residential setting, and intellectual level have been largely inconsistent. Many researchers assert that the prevalence of mental illness is highest among individuals with mild intellectual deficits. Individuals with mild intellectual deficits are more likely to be aware of their limitations than individuals with more severe deficits. That said, Kerker, Owens, Zigler, and Horwitz (2004) hypothesize that individuals with mild intellectual deficits may be at a higher risk of reacting to stressful life events with an affective disorder. These authors also point out that the differences in prevalence estimates may result from the difficulties in diagnosing mental illness in individuals with severe intellectual deficits.

Despite the aforementioned inconsistencies, there is an agreement that certain mental illnesses are more prevalent in persons with ID than in the general population. Kerker et al. (2004) report that adults with ID are more often diagnosed with anxiety and psychotic disorders than individuals without ID. Children with ID are more likely to have an anxiety disorder; however, estimates for affective disorders and ADHD are similar to non-disabled children. Further, Borthwick-Duffy and Eyman (1990) identified eight behaviors that are associated with dual diagnosis: depression, aggression, self-injury, resistive behaviors, temper tantrums, running or wandering away, adjustment changes in social relationships, and socially inappropriate behavior.

Bipolar Disorder

The focus of the present study is the characteristics of bipolar disorder in individuals with ID. An overview of the history, description, prevalence, assessment, and treatment of bipolar
disorder in the general population will be reviewed. Then, the focus will shift to bipolar disorder in individuals with ID.

History of Bipolar Disorder

Bipolar disorder, previously called manic-depressive disorder, is by no means a new illness; it has captured the interest of scholars since the ancient times (Johnson, 2004). Mania and melancholia are two of the earliest described diseases. Hippocrates was the first to systematically describe mania and melancholia; he included them in his first classification of mental disorders: mania, melancholia and paranoia (Angst & Marneros, 2001). However, these classical conceptualizations of mania and melancholia were much broader than they are today.

Aretaeus of Cappadocia, who was strongly influenced by Hippocrates, was the first to conceptualize bipolarity. Though, he did differentiate a biologically and psychologically caused melancholia, the later termed ‘reactive depression’. Aretaeus believed mental disorders are of biological causes. Most importantly, Aretaeus was the first to link mania and melancholia; he viewed them as two different images of the same disease. He asserted that mania and melancholia were of the same etiology. Further, he suggested mania was considered a worsening of melancholia (Angst & Marneros, 2001).

It was not until 1851 that Jean-Pierre Falret first characterized a separate mental disorder as a continuous cycle of depression, mania, and free intervals of varying lengths. This change from mania to melancholia intermixed with intervals free of symptoms, termed by Falret as folie circulaire, was considered to be a disease on its own. The following years were filled with debates between Falret and Jules Baillarger on the importance of the interval between symptomatic episodes of mania or melancholia; that is, whether the interval between episodes is a conceptual aspect of the disease. In 1884, the concept of the disorder had reached general
acceptance with publications in *Brain* and *American Journal of Insanity* (Angst & Marneros, 2001).

As early as 1921, Emil Kraepelin’s description of *manic-depressive insanity* with its variability and patterns of symptoms were similar to descriptions of bipolar disorder in diagnostic manuals today such as the *Diagnostic and Statistical Manual of Mental Disorders* (Johnson, 2004). However, it was Carl Wernicke that pointed out the distinction between manic-depressive illness and recurrent depression or recurrent mania. Later, Wernicke’s colleague, Karl Kleist differentiated between unipolar and bipolar affective disorders. Moreover, Kleist suggested that monopolar mania is separate from manic-depressive disorders. However, this distinction was not recognized by the international psychiatric community for many years (Angst & Marneros, 2001).

In 1966, publications supporting the differentiation between unipolar and bipolar disorders brought about the ‘rebirth’ of bipolar disorder (Angst, 1966 as cited in Angst & Marneros, 2001; Perris, 1966 as cited in Angst & Marneros, 2001). The findings of these publications were similar. One investigation of 326 patients, treated between 1959 and 1963, concluded that: 1) genetics and the environment are significant etiological factors of depression; 2) there is a relationship between female gender and endogenous depression, though, bipolar disorders are equally represented in both males and females; 3) manic-depressive illness is categorically heterogeneous. Unipolar depression differs significantly from bipolar disorders in many characteristics such as genetics, gender, course and premorbid personality; and 4) late-onset depression has a strong link to depression while it has only a weak relationship to bipolar disorder.
Current Definition of Bipolar Disorder

Although mania was the main characteristic of the classic disease, recent literature suggests a spectrum of bipolar disorders including lesser degrees of excitement (Thomas, 2004). There are several different types of mania. Pure mania or euphoric mania is the classically recognized state of euphoria and grandiosity. A presentation of dysphoric mania, accounting for up to 40% of all bipolar episodes, typically includes irritability, crying, hopelessness, and suicidal ideation (Akiskal et al., 1998). Hypomania is characterized by a less severe intensity and duration of mania, oftentimes not resulting in significant impairment in social or occupational functioning (APA, 2000; Thomas, 2004). Further, Johnson (2004) suggests that these symptoms of mania can vary a great deal from person to person.

Currently, there are four major subtypes of this disorder in the Diagnostic and Statistical Manual of Mental Disorders, 4th edition –Text Revision (DSM-IV-TR): bipolar I, bipolar II, cyclothymia, and bipolar not otherwise specified (NOS). The key feature of bipolar I disorder is the presence of a single lifetime manic or mixed episode; depression is not a diagnostic criterion. The DSM-IV-TR (APA, 2000) states that the essential feature of bipolar I disorder is a clinical course that is characterized by the occurrence of one or more manic or mixed episodes. A manic episode is defined as a distinct period of at least one week, during which mood is abnormally and persistently elevated, expansive, or irritable. This mood disturbance must be accompanied by at least three additional symptoms including inflated-self esteem or grandiosity, decreased need for sleep, pressure of speech, flight of ideas, distractibility, increased involvement in goal-directed activities or psychomotor agitation, and excessive involvement in pleasurable activities with a high potential for painful consequences. A mixed episode is defined as a period of one week during which the criteria are met for both a manic episode and a major depressive episode. Mood
disturbances for both a manic and mixed episode must be sufficiently severe to cause marked impairment in occupational or usual social functioning, result in hospitalization, or have psychotic features (APA, 2000).

Bipolar II disorder, on the other hand, is diagnosed on the basis of a single lifetime episode of hypomania and at least one episode of major depression. DSM-IV-TR (APA, 2000) states that the essential feature of bipolar II disorder is a clinical course that is characterized by the occurrence of one or more major depressive episodes accompanied by at least one episode of hypomania. A major depressive episode is defined by a period of at least two weeks during which there is either depressed mood or the loss of interest or pleasure in nearly all activities. The individual must also experience four additional symptoms including changes in appetite or weight, sleep, and psychomotor activity; decreased energy; feelings of worthlessness or guilt; difficulty thinking, concentrating, or making decisions; and recurrent thoughts of death or suicidal ideation, plans, or attempts.

A hypomanic episode is defined by a distinct period in which there is an abnormally and persistently elevated, expansive, or irritable mood that lasts at least four days. This period must also be accompanied by at least three additional symptoms including inflated self-esteem or grandiosity (non-delusional), decreased need for sleep, pressure of speech, flight of ideas, distractibility, increased involvement in goal-directed activities or psychomotor agitation, and excessive involvement in pleasurable activities that have high potential for painful consequences. There must be a clear change in functioning that is observable by others and is not characteristic of the individual’s usual functioning. Unlike the criteria for a manic episode, a hypomanic episode is not severe enough to cause marked impairment in social or occupational functioning, or to require hospitalization, in addition, there are no psychotic features (APA, 2000). Johnson
(2004) points out that individuals who have lifetime episodes of hypomania but do not experience depression do not qualify for any diagnosis, as these periods of hypomania may not result in severe impairment in functioning.

Cyclothymic disorder, in comparison to bipolar I and bipolar II, is a milder form of mood disorder. The DSM-IV-TR describes cyclothymia as a chronic, fluctuating mood disturbance involving numerous periods of hypomanic symptoms and numerous periods of depressive symptoms. However, the hypomanic and depressive symptoms are of insufficient number, severity, pervasiveness, or duration to meet the criteria for a mania or major depressive episode. It is not necessary for any of the episodes to meet criteria for a hypomanic episode. These fluctuations of mood must last for at least two years in which there is no period of more than two months that the individual is without hypomanic or depressive symptoms. It is estimated that individuals with cyclothymia have a 15%-50% risk of subsequently developing bipolar I or bipolar II disorder (APA, 2000).

Bipolar Not Otherwise Specified (NOS) includes bipolar features that do not meet criteria for any specific bipolar disorder. Some examples of this situation cited in the DSM-IV-TR include rapid variation between manic and depression symptoms that do not meet criteria for duration; recurrent hypomanic episodes without intercurrent depressive symptoms; manic or mixed episode superimposed on delusional disorder, residual schizophrenia, or psychotic disorder; and hypomanic episodes that are too infrequent to qualify for a diagnosis of cyclothymic disorder. Finally, situations in which the clinician has concluded that a bipolar disorder is appropriate (but unable to determine whether it is primary), due to a general medical condition or substance induced would warrant a diagnosis of bipolar NOS . (APA, 2000).
Recently, new forms of bipolar disorder have been suggested. These include bipolar II-1/2 (depression superimposed on cyclothymic temperament), bipolar III or “pseudo-unipolar” (mania or hypomania associated with the prescription of antidepressants for depressive disorders), and bipolar IV (depression arising from a hyperthymic temperament). However, these new forms or subtypes of bipolar disorder have yet to be formally accepted (Thomas, 2004).

Characteristics

Bipolar disorder is typically a lifelong illness characterized with manic or hypomanic episodes that may or may not be accompanied with depression. Illness onset usually occurs before the age of 25. However, due to the episodic feature of the disorder, an accurate diagnosis is often not obtained immediately. For example, Benazzi and Akiskal (2003) reported that up to 50% of individuals with major depressive episodes have bipolar II disorder. This suggests that a systematic search for hypomanic episodes would result in a re-diagnosis of ‘unipolar’ depressed persons as bipolar II disordered. Bipolar disorder is often misdiagnosed as unipolar depression or schizophrenia, while milder forms of the disorder such as cyclothymia are likely to be misdiagnosed as personality disorders (Akiskal, Hantouche, & Allilaire, 2003; Daniels, Kirkby, Mitchell, Hay, & Bowling, 2003). Moreover, the range of clinical presentation, psychiatric comorbidity, mixed states, and rapid cycling further complicate diagnosis (Thomas, 2004).

Accurate diagnosis and treatment is critical as this illness has a high mortality rate. Studies by Lish, Dime-Meenan, Whybrow, Price and Hirschfeld (1994) and Hantouch, Azorin, Chatenet-Duchene, Lancrenon, Allilaire and Akiskal (2003, as cited in Thomas, 2004) assert that treatment is oftentimes not started until up to 10 years after the onset of the illness. Thomas (2004) found that reported lifetime risk for suicide attempts range from 25% to 50%, and suicide attempts resulting in death occur in 10% to 15% of individuals who are not adequately treated.
Catatonic features, cognitive impairment (e.g., impairment in verbal memory; Quraishi & Frangou, 2002), conduct disorders, and psychoactive substance abuse are also associated features of this disorder (Thomas, 2004).

Comorbid psychopathology among individuals with bipolar disorder appears to be the rule rather than the exception. Estimates of comorbidity show that more than two-thirds of individuals with bipolar disorder have another psychiatric disorder (Thomas, 2004). Most common co-existing psychiatric disorders are panic disorder, generalized anxiety disorder, obsessive-compulsive disorder, bulimia nervosa and substance abuse (Angst, 1998; Freeman, Freeman, McElroy, 2002; Perugi et al., 1998). Goodwin and Hoven (2002) in their community-based household sample from a National Comorbidity Survey found that panic attacks are not uncommon among individuals with bipolar disorder in the general population. Further, the presence of panic attacks in these individuals is associated with significantly elevated rates of comorbid psychopathology (i.e., more likely to meet criteria for comorbid agoraphobia, simple phobia, generalized anxiety disorder, and substance abuse).

Etiology

Current literature suggests that there is a familial nature of bipolar disorder; that is, 1) mood disorders occur at a higher rate in the families of bipolar patients, and 2) a variety of mood syndromes and symptoms that differ both qualitatively and quantitatively from bipolar disorder occurs in the families of bipolar patients (Kelsoe, 2003). Kelsoe’s (2003) review of family studies conducted over the past decades indicated that approximately 7% of people with bipolar disorder have first degree relatives that also have bipolar disorder (compared to 1% general population prevalence) and approximately 10% of people with bipolar disorder have first degree relatives that have unipolar depression (compared to 5% general population prevalence). This
suggests a sevenfold increased risk of having bipolar disorder and a twofold increased risk of having unipolar depression if there is someone with bipolar disorder in the family. Twin studies also lend support to the genetic basis of bipolar disorder. A meta-analysis of the twin studies found that approximately 70% of monozygotic pairs are concordant for the illness, while only 30% of dizygotic pairs are concordant for the illness (Kelsoe, 2003). Adoption studies also found an elevated rate of illness in biological parents and only a population rate in the adoptive parents (Mendlewicz & Rainer, 1977; Wender et al. 1986). Kelsoe’s (2003) conclusions from his review of several studies regarding the genetic basis of bipolar disorder are that 1) genes explain only a portion of the etiology, with environmental factors likely playing a substantial role; and 2) the trait displays variable expressivity, meaning a variety of clinically related presentations can result from the same genes. This last conclusion lends support to the characterization of bipolar as a spectrum of disorders.

As biological variables do not fully explain the individual differences in severity or changes in presentation overtime, the current literature highlights several psychosocial variables that may have an influence on the course of the disorder (Johnson & Meyer, 2004). Expressed emotion, defined as emotionally intrusive or hostile comments from family members toward individuals with the disorder, was one of the first environmental variables identified as impacting the course of the disorder. Although expressed emotion has been shown to be relevant to other disorders such as schizophrenia, the effect size appears to be much larger for individuals with mood disorders than those with schizophrenia (Butzlaff & Hooley, 1998). This confirms the critical importance of psychoeducation for the family members in the treatment of individuals with bipolar disorder.
Social support is another factor that has been suggested. Romans and McPherson (1992) found that individuals with bipolar disorder experience less social support than those without mental disorders. Further, they found that social support is lower for people with a history of more manic episodes. Johnson, Meyer, Winett and Small (2000) have suggested that an absence of emotional support has been related to poorer course of the disorder, including more frequent relapse and less successful lithium treatment.

Presence of negative life events has also been investigated as an impacting factor. As the methodology and the potential confounding variables (i.e., poor coping skills) vary across studies, it is difficult to quantify the impact of these events. Johnson and Meyer (2004) assert that the literature has consistently supported the relationship between life events and increases in symptoms. Further, these findings seem to hold even after excluding events that result from symptoms or deficits in coping. The authors suggest that the best predictors of course within the life-event domain appear to be major negative life events that are associated with loss (i.e., death of a family member).

Johnson and Meyer (2004) assert that individuals who are able to maintain non-critical family relationships, maintain strong social support, and have lower rates of negative life events are likely to have fewer symptoms of bipolar disorder over time. Also, personality traits, such as neuroticism, have also received some attention as mediating factors in the course of the disorder. However, as methodological approaches have varied widely and studies oftentimes do not control for syndromal symptoms, it is difficult to ascertain the impact of personality factors on the course of the disorder.

The literature indicates that psychosocial variables do have an important role in the course of bipolar disorder. Further, these variables have been found to exacerbate symptoms of
depression and mania in the disorder. Symptoms of depression have been associated with
negative life events, low social support and poor self esteem; whereas sleep deprivation, and
behavioral activation have been associated with mania (Johnson & Meyer, 2004). Colombo,
Benedetti, Barbini, Campori and Smeraldi (1999) reported that as many as 10% of patients with
bipolar disorder developed hypomanic or manic symptoms after induced sleep deprivation.
Moreover, Wehr, Turner, Shimada, Lowe, Barker, and Leibenluft (1998) published case studies
that suggest that increasing sleep regulation in individuals with bipolar disorder may have an
impact on symptom reduction. Johnson and Meyer (2004) indicate that recent research suggests
that behavioral activation system sensitivity tends to be higher among individual with bipolar
disorder than comparison samples. Further, the research suggests that higher behavioral
activation system sensitivity predicts a greater risk of mania over time. It has been suggested that
bipolar disorder may reflect dysregulation of the ventral tegmental dopamine-secreting neurons,
which is the hypothesized central tract involved in the behavioral activation system (Depue &

Prevalence

Classic prevalence rates of bipolar have estimated 0.8% to 1.7% for full mania and
appear comparable across cultures (Angst, 1998; Johnson, 2004; Thomas, 2004). Low lifetime
prevalence rates of hypomania have been reported to range between 0.5% and 1.9% (Angst,
1998). Consequently, with the recent shift toward conceptualizing bipolar as a spectrum of
disorders (including hypomania, recurrent brief hypomania, sporadic brief hypomania, and
cyclothymia), current literature suggests that bipolar affects at least 5% of the general population
(Akiskal, Bourgeois, Angst, Post, Moller & Hirschfeld, 2000; Angst, Gamma, Benazzi, Ajdacic,
Eich & Rossler, 2003; Judd & Akiskal, 2003). Of course, as mentioned earlier, misdiagnosis of
bipolar disorder is common (Daniels, Kirkby, Mitchell, Hay & Bowling, 2003). Therefore, caution is warranted when making conclusions based on these reported prevalence rates.

**Assessment**

The most common methods used to diagnose bipolar disorder in the general population cited in the research literature are structured interviews such as the Structured Clinical Interview for DSM (SCID) based on the DSM, or prior to 1980, the Schedule for Affective Disorders and Schizophrenia (SADS based on Research Diagnostic Criteria (RDC; Altman, 2004; Matson, González, Dixon, Cooper & Matson, 2004). Matson et al. (2004) found that 124 out of 164 (75%) published studies in the bipolar literature in the past 20 years used DSM criteria for diagnosis; oftentimes DSM criteria was used in conjunction with a structured interview such as the SCID.

**Schedule for Affective Disorders and Schizophrenia (SADS).** Prior to the advent of DSM-III in 1980, the RDC was the primary instrument for diagnosing psychiatric disorders in the United States. Information required for making diagnosis based on RDC was gathered through a structured interview using the SADS. This interview assessment contains interview questions and guidelines that allow clinicians to obtain information about the presence and severity of symptoms in a standardized manner. Some training is required for reliable administration. There are multiple versions of the SADS; the SADS change version is used to assess outcome in treatment studies, while the lifetime version is used for diagnosing past episodes (Altman, 2004; Endicott & Spitzer, 1978).

**Structured Clinical Interview for DSM (SCID).** With the shift to the use of the DSM-III, the SCID was developed to provide clinicians a standardized diagnostic instrument for deriving DSM-III diagnoses. The SCID’s structured format is similar to that of the SADS’s, but
is intended for diagnosis only; the SCID does not assess the severity of symptoms. It has been modified with revisions of the DSM. The SCID does require some degree of clinical training and practice for reliable administration. It is available in different formats such as versions for Axis I disorders (SCID-I), personality disorders (SCID-PD), and family members or non-patients (SCID-NP; Altman, 2004; Spitzer, Williams, Gibbon, & First, 1992).

A variety of rating scales are available to assess the severity of bipolar disorder; these include observer-rated, clinician-rated, and global mania scales. Newer rating scales have evolved from the early nurse rating scales of the 1970s (i.e., Manic State Rating Scale, MSRS; Beigel, Murphy & Bunney, 1971). Improvement in rating scales over the past years have focused on clear operational definitions of symptoms; well-defined anchor points for assessing severity; more relevant item content (consistent with DSM criteria); guidelines for administration, scoring and interpretation; and reporting of reliability and discriminant and concurrent validity coefficients.

**Manic State Rating Scale (MSRS).** The MSRS was the first observer-rated mania scale; it was designed for use by nursing staff after long-term observation (i.e., 6 to 8 hour shifts) in the hospital ward. It contains 26 items assessing both the frequency and intensity of various signs and symptoms. Limitations of this scale include the long observation period, the specialized training required, inclusion of items not specific to mania (i.e., suspiciousness, depressed affect), and the absence of one key symptom (i.e., sleep disturbance; Altman, 2004; Beigel, Murphy & Bunney, 1971).

**Manchester Nurse Rating Scale for Mania (MNRS-M).** This observer-rating scale, an improved version of the MSRS, is suitable for the daily monitoring of affective states. It contains nine items rated from 0 (not present) to 3 (usually present). It does not require specialized
training to administer reliably. Authors report good inter-rater reliability and concurrent validity with global ratings of mania, $r = .95$ and $r = .65$ respectively. It is a more compact and easier scale to administer when compared to the MSRS; however, it does not include a measure of sleep disturbance (Altman, 2004; Brierley, Szabadi, Rix & Bradshaw, 1988).

**Bech-Rafaelson Mania Scale (BRMS).** The BRMS, a clinician rating scale, consists of 11 items, each is rated from 0 (normal) to 4 (extreme). Four of the 11 items are devoted to activity level alone (e.g., motor activity, verbal, sexual, work and interests), though there is no item to assess distractibility. The authors report high interrater reliability ($r = .80 - .95$). Smolka and Stieglitz (1999) report that discriminant validity of the BRMS is high, $r = .80$. Further, Bech (2002) documents the effectiveness of the BRMS as an outcome measure in clinical trials (Altman, 2004; Bech, Rafaelson, Kramp & Bolwig, 1978).

**Young Mania Rating Scale (YMRS).** The most widely used clinician-administered rating scale for mania is the YMRS (Young, Biggs, Ziegler, & Meyer, 1978). It contains 11 items, four of which are scored 0-4 and the remaining seven are scored from 0-8, based on severity. The YMRS is designed as a 15 to 30 minute interview administered by trained clinicians. It has been used extensively to assess treatment response in clinical trial studies. It is considered to be the gold standard to which scale developers evaluate concurrent validity with newer scales. The authors report good inter-rater reliability for items ($r = .66 - .92$) and total scores ($r = .93$), and good concurrent validity ($r = .71$) with the MSRS (Beigel, Murphy, & Bunney, 1971) and global ratings ($r = .88$). Limitations include the fact that there is no guideline to ensure standardized administration, no report of discriminant validity or test-retest reliability (Altman, 2004). One criticism raised by Altman (2004) is that the YMRS combines symptoms of
mania with psychotic symptoms to yield a total score; he suggests that this is troublesome as these symptoms may respond differently to treatment.

**Clinical Global Impressions Bipolar Scale (CGI; Guy, 1976).** The CGI is one of the earliest general global rating questionnaires used for psychopharmacology treatment studies. Its revised version, the Clinical Global Impressions Bipolar scale (CGI-BP; Spearing, Post, Leverich, Brandt, & Nolen, 1997), contains guidelines clarifying concepts and explanations and rules for ratings. This version includes separate scores for depression and mania; symmetrical scores on a seven-point scale; categories to rate symptoms on admission, during an acute episode, and during prophylactic periods; and a section to rate medication side effects. Inter-rater reliabilities for severity (r = .91), change from last assessment (r = .86), and change from worst phase of illness (r = .76) have been reported (Altman, 2004).

**Mania-Depression Scale (MDS; Mazmanian et al., 1994).** The MDS is a global clinician-rated severity scale with scores ranging from −5 (depressive stupor) to 0 (euthymic) to +5 (manic delirium). Each severity point has defining descriptive behaviors. It is easy to administer, allows for the assessment of both manic and depressive symptoms, and is sensitive to daily variation and treatment effects. Inter-rater reliability is good (r = .84) and concurrent validity is reported as r = .59 with the self-rating Beck Depression Inventory and r = .71 with a visual analogue mood scale (Altman, 2004).

Matson et al.’s (2004) review of treatment studies noted that investigators used various methods to assess bipolar disorder in their studies. A frequency count of the use of these assessment scales indicated that the SCID, Hamilton Depression Rating Scale (HAM-D), SADS, and YMRS were the assessment scales other than DSM most often used as inclusion criteria.
Further, HAM-D, CGI, and YMRS were most often used to assess the efficacy of the treatment in question.

Treatment

Practice guidelines for the treatment of patients with bipolar disorder state that the specific goals of treatment are “establishing and maintaining a therapeutic alliance, monitoring the patient’s psychiatric status, providing education regarding bipolar disorder, enhancing treatment compliance, promoting regular patterns of activity and of sleep, anticipating stressors, identifying new episodes early, and minimizing functional impairments” (American Psychiatric Association, 2002, p.4). Kasper and Attarbaschi (2004) suggest that treatment for mania, specifically, aims to rapidly control the irritability, agitation, impulsivity, aggression, and psychotic symptoms that are often symptomatic of mania. There are several treatment approaches available that seek to help manic individuals to regain and maintain pre-morbid functioning. Medication is the most widely used treatment of bipolar disorder; however, it is sometimes used in conjunction with other psychoeducational treatment methods such as cognitive-behavioral therapy, interpersonal-social rhythm therapy, or family therapy (Michalack, Yatham, Lam, 2004). Alternatively, electroconvulsive therapy (ECT) is also considered to be a treatment option (American Psychiatric Association, 2002).

Pharmacological treatment of bipolar disorder is common. The most often used types of medications are lithium, anticonvulsants, atypical antipsychotics, and antidepressants. Currently, there are only five medications approved by the United States Food and Drug Administration (FDA) for the treatment of bipolar disorder: lithium, chlorpromazine, divalproex, olanzapine, and lamotrigine. Presently, there is very little evidence to link any pharmacological activity to
antimanic or antidepressant effects (Goldberg, 2004). The evidence of the antimanic efficacy of these medications is summarized below.

**Lithium.** Lithium is the most extensively studied agent for relapse prevention in bipolar disorder (Goldberg, 2004). It remains a cornerstone drug for both short and long-term treatment of bipolar disorder. Research by Tondo and Baldessarini (2000) suggest that lithium may be effective in reducing suicide risk. Despite its common use and favorable aspects, lithium is commonly associated with the following side effects: tremor, thirst, urinary frequency, constipation, blurry vision, sedation, and acne (Goldberg, 2004).

**Anticonvulsants.** Anticonvulsants commonly studied for the treatment of bipolar are divalproex, carbamazepine, oxcarbazepine, lamotrigine, gabpentin, and topiramate. Although many anticonvulsants are prescribed to treat bipolar disorder, only divalproex and carbamazepine have found to be effective in placebo-controlled studies (Pope, McElroy, Keck, & Hudson, 1991; Post, Rubinow, & Ballenger, 1986). Lamotrogine is the only anticonvulsant effective as an antidepressant (Calabrese, Bowden, Sachs, Ascher, Monaghan, & Rudd, 1999). Furthermore, the side effects of anticonvulsant medications vary widely (e.g., dizziness, sedation, paresthesias, gait unsteadiness; Goldberg, 2004).

**Antipsychotics.** Atypical antipsychotics commonly prescribed to treat bipolar include: olanzapine, ziprasidone, risperidone, quetiapine, clozapine and aripiprazole. Olanzapine, however, is presently the only atypical antipsychotic recognized by the FDA for the treatment of acute mania. Its efficacy over acute mania (Tohen et al., 1999; Tohen et al. 2000) and mixed states (Tohen et al., 2000) has been demonstrated. Controlled studies support the monotherapeutic use of olanzapine, risperidone, or aripiprazole in acute mania, while olanzapine,
risperidone, quetiapine and clozapine have support for adjunctive use (Goldberg, 2004). Further, placebo-controlled research is needed to investigate the efficacy of these medications.

**Antidepressants.** A central concern over the use of antidepressants for treatment of bipolar depression is its potential to induce mania or accelerate cycle frequency in about 20-40% of individuals (Altshuler et al., 1995; Goldberg & Whiteside, 2002). Altshuler et al. (2001) suggest that there are no controlled data to support the notion that long-term antidepressants help to avoid depression relapse any better than mood stabilizers. Current American Psychiatric Association Practice Guidelines advise against using antidepressants alone in patients with bipolar I disorder (American Psychiatric Association, 2002); however, Goldberg (2004) suggests that antidepressants may prove to be useful when other pharmacotherapies yield less than optimal results.

Though pharmacological agents are the cornerstone of treatment, adherence to treatment and the presence side effects are oftentimes problematic (Goldberg, 2004). Thus, other treatments such as cognitive-behavioral therapy, interpersonal social rhythm therapy, and family therapy are often used in conjunction with medication.

**Cognitive-behavioral therapy.** Cognitive-behavioral therapy is most often used in combination with pharmacotherapy (Leahy, 2004). The medication is used to help stabilize or reduce fluctuations in mood, while cognitive therapy aids the individual in managing his moods when they occur (Leahy, 2004). Leahy (2004) suggests that the content of cognitive schemas differ with mood. As mood changes, these automatic thoughts (e.g., “I’ll get rejected”; “I’ll impress everyone”) and maladaptive assumptions (e.g., “I should be successful at everything”) support the individual’s current mood state (depression or mania). Leahy (2004) describes cognitive-behavioral therapy as a process of evaluating and learning from past episodes,
conducting a cost-benefit analysis of the situation, and finally introducing rational responses (e.g., “No one is successful at everything”) to these automatic thoughts and maladaptive assumptions.

Cognitive-behavioral therapy usually focuses on six targets for treatment: 1) medication adherence, 2) early detection and intervention, 3) stress and lifestyle management, 4) treatment of comorbid conditions, and 5) treatment of bipolar depression (Michalak, Yatham, & Lam, 2004; Otto, Reilly-Harrington, & Sachs, 2003). Several controlled studies have indicated that cognitive-behavioral therapy can positively influence the course of bipolar disorder in aspects such as medication compliance (Cochran, 1984); significantly fewer manic, hypomanic and depressed episodes (Lam et al., 2000); and the reduction in the number of episodes and increasing euthymic periods compared to those in a medication-alone condition (Hirshfeld et al., 1998).

**Interpersonal and social rhythm therapy (IPSRT).** IPSRT is based on the following assumptions: 1) instability is the fundamental dysfunction in bipolar disorder; 2) there is a relationship between psychosocial stressors and changes in biological rhythms (for example, our circadian rhythm is set by zeitgebers such as light, timing of work or meal, etc; Aschoff, 1981); and 3) life events that disrupt our social routines are significantly associated with the onset of mood fluctuations (Malkoff-Schwartz et al., 2000). Individuals that are vulnerable to disruptions may become manic or depressed in response to changes in the regularity of daily routines or social rhythms (Frank & Swartz, 2004). IPSRT focuses on helping patients optimize the regularity of daily routine, resolve social and interpersonal problems, and understand the illness through psychoeducation, social rhythm therapy and interpersonal psychotherapy. Rucci et al. (2002) have found that IPSRT is associated with low rates of episode recurrence, increased
stability of mood between episodes, and significant reductions in suicidal behavior. One major limitation in IPSRT is the challenge of motivating the individual to participate in the therapy and make changes in his daily routine. During mood episodes, non-adherence to pharmacological and social rhythm treatment may be a major problem (Frank & Swartz, 2004).

**Family therapy.** As previously mentioned, the course of bipolar illness over time is strongly influenced by stressful life events (Miklowitz, 2004). Miklowitz, Goldstein, Nuechterlein, Snyder and Mintz (1988) found that individuals with bipolar disorder who are associated with family or marital environments characterized by high expressed emotion (e.g., highly critical, hostile, or overprotective attitudes) have more frequent relapse than individuals with relationships characterized by low expressed emotion. Family-focused treatment (FFT) assumes that increasing the efficiency and emotional tone of the family’s communications and problem solving strategies, and encouraging greater tolerability and acceptance of the illness, will result in a more stable mood over time (Miklowitz, 2004). Just as family therapy has received support in the treatment of other illnesses (i.e., schizophrenia), FFT has received empirical support in one open trial (Miklowitz & Goldstein, 1990) and two randomized trials (Rea, Tompson, Miklowitz, Goldstein, Hwang, & Mintz, 2003; Simoneau, Miklowitz, Richards, Saleem, & George, 1999). The results of these studies suggest that adding FFT to pharmacological treatment increases long-term mood stability among individuals with bipolar disorder by encouraging medication compliance and enhancing the use of positive communication in family relationships (Miklowitz, 2004).

**Electroconvulsive therapy (ECT).** Electroconvulsive Therapy is a procedure used in treating individuals with severe depression, acute mania, and certain schizophrenic syndromes. Oftentimes ECT is used with suicidal patients who cannot wait for pharmacological agents to
take effect. This treatment involves a brief application of an electric stimulus that is used to produce a generalized seizure. However, it remains unknown how or why this treatment works (National Mental Health Association, 2004). Controlled studies have demonstrated its efficacy in acute mania and bipolar depression (American Psychiatric Association, 2002). These studies indicate that individuals treated with ECT have superior clinical outcomes than individuals on medication alone (Sikdar, Kulhara, Avasthi, & Singh, 1994; Small et al., 1988; Mukherjee, Sackeim, & Schnur, 1994; Zornberg & Pope, 1993). Although, there are several studies indicating the treatment’s efficacy, ECT remains a controversial treatment option due concerns over side effects such as memory loss and confusion (National Mental Health Association, 2004).

**Bipolar Disorder and Intellectual Disability**

Until recently, the majority of research on bipolar disorder in individuals with ID has comprised of studies that examine either descriptive case reports, prevalence of behavioral features associated with mood states (e.g., SIB, aggression), and/or the efficacy of pharmacological treatments (Aman, Collier-Crespin, & Lindsay, 2000; Berney & Jones, 1988; Hellings, 1999; Lowry & Sovner, 1992; Vanstraelen & Tyrer, 1999). However, more studies are beginning to focus on psychopathology in people with ID and mania. Cain et al. (2003) reported a range of challenging behaviors and functional impairments had been associated with mania in individuals with ID. For example, Cain et al. (2003) in a retrospective chart review reported that intellectually disabled individuals diagnosed with bipolar disorder were more likely to show more mood symptoms, such as irritability, elevated mood, and euphoric mood than individuals diagnosed with non-psychotic depression, major depression (with psychosis), or schizophrenia. Further, persons with bipolar and ID were also reported to be more likely to exhibit non-mood
symptoms, such as increased self-esteem, disturbed speech, increased energy, decreased sleep, distractibility, and increased engaging in pleasurable activity. In one retrospective case series, King (2000) contrasted the phenomenology, outcome, treatment response, and clinical characteristics of individuals with rapid and non-rapid cycling and found that those who cycle rapidly have a poorer outcome than those who do not. Another study by Wieseler, Campbell, and Sonis (1988) reports a case study in which a rating scale, derived from two mania-rating scales used in the general population (i.e., Young Mania Rating Scale and Manic State Rating Scale), was used to track the rapid cyclic pattern of an intellectually disabled resident with bipolar disorder and found periods of behavioral fluctuations in symptoms associated with bipolar disorder (i.e., activity/inactivity).

Prevalence

Estimates of individuals being dually diagnosed with bipolar disorder and ID have ranged from 0.9% to 4.8% of the intellectually disabled population (Reid, 1972; Ruedrich, 1993a). These estimates vary, in part, due to the difficulties in diagnosing bipolar disorder in this population. An evaluation of rates of psychopathology in persons with severe and profound ID by Kirkpatrick-Sanchez, Williams, Matson, Anderson, and Gardner (1996) indicate that bipolar disorder was more often identified in individuals with severe ID in comparison to those with profound ID (no cases were identified). Further, Pary, Strauss, and White (1996) in their population survey of bipolar disorder in persons with ID found no individuals who had bipolar disorder and Down Syndrome. This finding is consistent with other studies, which suggest that the prevalence of bipolar disorder is much lower in those with Down Syndrome in comparison with individuals with other developmental disabilities. Perhaps this is due to a disturbance in
genetics leading to a predisposition to depression and a relative protection from mania (Cooper & Collacott, 1993; Craddock & Owen, 1994).

Assessment

Cain et al. (2003) suggest that the use of DSM-IV criteria can be useful in the diagnosis of bipolar disorder in intellectually disabled persons. However, diagnosing bipolar disorder in this population remains challenging because of difficulties in communication, atypical presentation, and lack of clear diagnostic criteria (Arumainayagam & Kumar, 1990). Individuals with severe intellectual deficits are oftentimes non-verbal, thus the use of methods that rely on self-report of feelings is not possible. Hasan and Mooney (1979) indicate that misdiagnosis may be more common in this population due to the under-reporting of depressive and manic symptoms by their caregivers. Evans, Byerly, and Greer (1995) raise the point that affective symptoms may be difficult to distinguish from those related to developmental disabilities.

Further, it has been suggested that the appearance or presentation of bipolar disorder in this population may be distinctly different from the general population (Ruedrich, 1993a). That is, mood disorders in the intellectually disabled population are more often atypical, chronic, or rapid cycling (Sovner, 1989).

Ruedrich (1993a) presented three major approaches to assessing bipolar disorder in individuals with ID: 1) direct application of current diagnostic systems (i.e., DSM, International Classification of Diseases, ICD); 2) an extrapolation and/or minor revisions of current diagnostic schemes for application to person with ID (i.e., behavioral equivalents); and 3) development of derived diagnostic systems for specific application to persons with ID (i.e., DASH-II). Current literature states that clinicians should focus on core DSM or ICD symptoms when diagnosing bipolar disorder in individuals with intellectual deficits. In cases when a patient is non-verbal or
minimally verbal, it is suggested that attention be focused on the use of observable signs and validated psychiatric measures documented by knowledgeable caregivers and the clinician (Hellings, 1999; Lowry & Sovner, 1992; Sturmey, Laud, Cooper, & Matson, 2004).

Treatment

**Pharmacotherapy.** A review of pharmacotherapy of bipolar disorder persons with ID by Aman, Collier-Crespin, and Lindsay (2000) indicates that the main pharmacologic treatment in this population is lithium alone or in combination with carbamazapine or valproic acid. Several case studies describing individuals with ID and bipolar disorder suggest that lithium was effective in the management and prevention of manic episodes (Arumainayagam & Kumar, 1990; Kadambari, 1986; Ruedrich, 1993b). A double-blind long-term trial of lithium indicated that treatment with lithium resulted in fewer weeks of illness (Naylor, Donald, LePoidevin, & Reid, 1974). Although the majority of the reports are from uncontrolled treatment studies and case reports, they consistently suggest that treatment with lithium resulted in improvement in symptoms (i.e., remissions of symptoms, Rivinus & Harmatz, 1979; lithium alone or with carbamazepine resulted in partial or complete improvement, Glue, 1989). Pary (1991) noted that 10 out of 15 (67%) individuals with ID had side effects of lithium such as tremor, GI irritation, rash, sedation, thirst, and polyuria leading to the discontinuation of their medication.

Other pharmacotherapies, such as carbamazepine and valproic acid, also have support as effective treatments. Sovner’s (1991) comprehensive review on the use of anticonvulsant agents in developmentally disabled individuals concluded that carbamazepine and valproic acid may be considered a primary therapy choice with individuals with ID due to lithium’s unreliable response and side effects. In this article, Sovner concluded that carbamazepine is useful alone or in combination with lithium with atypical or rapid-cycling forms of bipolar. Ruedrich, Swales,
Fossaeea, Toliver, and Rutkowski (1999) report a retrospective chart review of 28 individuals on divalproex treatment (valproic acid) with ID that showed significant scale improvement on the CGI. Similarly, Buzan, Dubovsky, Firestone, and Pozzo (1998) review data from 10 individuals treated with clozapine; nine of the 10 patients had improved ratings on the CGI. The authors concluded that clozapine was well tolerated and efficacious for psychosis and mania in individuals with ID. Controlled studies are needed to further investigate the efficacy of pharmacological agents in the treatment of bipolar in individuals with ID.

**Electroconvulsive therapy.** Though pharmacological treatments are the most common forms of treatment for bipolar disorder, Aziz, Maixner, DeQuardo, Aldridge, and Tandon (2001) suggest that individuals with ID tend to be sensitive to medications and vulnerable to developing pronounced side effects. Thus, these authors suggest that electroconvulsive therapy (ECT) may be the treatment of choice. Aziz et al. (2001) presented two cases in which previous treatment with anti-psychotics and lorazepam yielded no response, while, use of ECT resulted in marked improvement in symptoms. Kessler (2004) describes four patients with ID and affective disorders (rapid cycling bipolar disorder; bipolar disorder, manic phase; major depression with psychotic features; and schizoaffective disorder) who had not responded to prior pharmacological treatment. All four patients were reported as responding dramatically to ECT. These findings from case studies, along with a review of other reports in the literature, lead these authors to conclude that ECT is safe and effective in individuals with ID who have comorbid psychiatric disorders (Aziz et al. 2001; Kessler, 2004).

As mentioned earlier, identifying bipolar disorder in individuals with ID is difficult. Sovner (1989) among others have suggested that bipolar disorder may manifest differently in individuals with ID than in the general population (e.g., rapid cycling, chronic). Much of the
research on bipolar disorder in individuals with ID involves the examination of case reports, behavioral features associated with the illness, and pharmacological treatments. Although the hallmark feature of bipolar disorder is the waxing and waning of symptoms, the variation of symptoms in bipolar disorder in persons with intellectual disabilities has yet to be fully investigated. The present study sought to further develop the understanding of mania in individuals with ID by contrasting the presence and variation of manic symptoms in individuals with ID with comorbid bipolar disorder and those without bipolar disorder.
RATIONALE

The purpose of this study was to characterize symptoms of mania over time in persons with bipolar disorder and ID. Given the limited data available on bipolar symptoms with individuals with ID, this was a preliminary descriptive study. Currently, the literature lacks a systematic investigation into what characterizes a change in mood among individuals with ID. In addition, little is understood in how this variation in behavior differs from individuals with ID without bipolar disorder. The present study aimed to extend the investigation of bipolar disorder in the intellectually disabled population in two ways. First, this research assessed the presence of manic symptoms in participants identified with and without bipolar disorder. Secondly, this study examined the consistency of manic symptoms in these participants over time.
METHOD

Participants

Participants included residents from Pinecrest Developmental Center (PDC) in Pineville, Louisiana. PDC is a state-run facility that provides 24-hour supervision to over 550 individuals with varying levels of intellectual disability and adaptive functioning. All diagnoses were provided by licensed clinical psychologists and based on DSM-IV-TR criteria.

Three groups of individuals with ID participated in this study: individuals with bipolar disorder, individuals with psychopathology other than bipolar disorder, and individuals with no psychopathology. The psychopathology group served as a validity control group to ensure that differences in the symptom variation between groups were due to the presence of bipolar disorder, and not the presence of psychopathology in general. The following criteria were used to select participants into their respective groups: 1) presence of a bipolar disorder diagnosis given by a licensed psychologist, and 2) criteria on DSM-IV checklist for the past occurrence of a manic episode. These criteria were chosen based on the inclusion criteria cited in the bipolar literature of the general population.

There were 15 individuals with a diagnosis of bipolar disorder who met the inclusion criteria for this study (e.g., bipolar diagnosis confirmed by licensed psychologist and staff endorsement of symptoms on DSM-IV-TR checklist). However, one participant in the bipolar group had insufficient data in the archive (less than 75% or 27 monthly data points) and was dropped from the study. Therefore, the bipolar group comprised 14 individuals, the psychopathology group comprised 14 individuals, and the control group comprised 14 individuals. Thus, a total of 42 residents were included in this study.
Participants’ ages ranged from 30 to 78 years, with the average age of 53 years. Nine participants (64.3%) in each group had limited verbal capacity. Results of a chi-square analysis indicated that the groups did not significantly differ on variables of age, ethnicity, gender, level of ID, verbal ability, or ambulation. Further, there were no significant differences between groups on the presence of hearing or visual impairments. Demographic information is presented in Table 1.

Table 1
Demographic Characteristics of Groups (N=42)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Bipolar (n=14)</th>
<th>Psychopathology (n=14)</th>
<th>Control (n=14)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-21</td>
<td>0 (0 %)</td>
<td>0 (0 %)</td>
<td>0 (0 %)</td>
</tr>
<tr>
<td>22-45</td>
<td>4 (28.6 %)</td>
<td>6 (42.9 %)</td>
<td>5 (35.7%)</td>
</tr>
<tr>
<td>46-65</td>
<td>7 (50.0 %)</td>
<td>6 (42.9 %)</td>
<td>6 (42.9 %)</td>
</tr>
<tr>
<td>66+</td>
<td>3 (21.4 %)</td>
<td>2 (14.2 %)</td>
<td>3 (21.4 %)</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>9 (64.3 %)</td>
<td>6 (42.9 %)</td>
<td>9 (64.3 %)</td>
</tr>
<tr>
<td>Male</td>
<td>5 (35.7 %)</td>
<td>8 (57.1 %)</td>
<td>5 (35.7 %)</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>2 (14.3 %)</td>
<td>3 (21.4 %)</td>
<td>2 (14.3 %)</td>
</tr>
<tr>
<td>Caucasian</td>
<td>12 (85.7 %)</td>
<td>11 (78.6 %)</td>
<td>12 (85.7 %)</td>
</tr>
</tbody>
</table>

(table cont.)
Many participants had multiple axis I diagnoses. Thirteen of the bipolar participants had at least two axis I diagnoses, while the psychopathology groups had seven participants with multiple axis I diagnoses. These diagnoses were noted during the chart review and are listed in Table 2. Further, the most recent mood episode was noted during the chart review for participants in the bipolar group. Six of the bipolar participants had a most recent episode of
mania, three participants had a most recent episode of hypomania, and five participants had a
diagnosis of bipolar NOS.

Table 2
Axis I Diagnoses across Groups

<table>
<thead>
<tr>
<th>Axis I diagnosis</th>
<th>Bipolar</th>
<th>Psychopathology</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety Disorder</td>
<td>1</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Autistic Disorder</td>
<td>1</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>Bipolar I Disorder</td>
<td>9</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Bipolar II Disorder</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Bipolar NOS</td>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Dementia NOS</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Dysthyemic Disorder</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Enopresis</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Enuresis</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Major Depressive Disorder</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Obsessive Compulsive Disorder</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>PDD NOS</td>
<td>5</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Pica</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Schizophrenia</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Stereotypic Movement Disorder (with SIB)</td>
<td>5</td>
<td>5</td>
<td>0</td>
</tr>
</tbody>
</table>
As medication may have an impact on the presence and severity of symptoms, current medications were noted during the participant’s chart review. All participants in the bipolar and psychopathology groups were on psychotropic medications. Approximately 71% of the bipolar group participants were prescribed multiple medications, whereas approximately 50% of the psychopathology group were prescribed multiple medications. Overall, the type of medications prescribed across the bipolar and psychopathology groups did not differ substantially. Only one participant in the control group was prescribed psychotropic medication (e.g., Zyprexa); this medication was being titrated at the time of the chart review. Table 3 lists the psychotropic medication prescribed to the participants at the time of the chart review.

Table 3
Psychotropic Medication Usage of Groups

<table>
<thead>
<tr>
<th>Medication Class</th>
<th>Frequency Count</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bipolar</td>
</tr>
<tr>
<td>Anti-depressants</td>
<td></td>
</tr>
<tr>
<td>Desyrel</td>
<td>1</td>
</tr>
<tr>
<td>Remeron</td>
<td>0</td>
</tr>
<tr>
<td>Paxil</td>
<td>1</td>
</tr>
<tr>
<td>Anti-obsessional</td>
<td></td>
</tr>
<tr>
<td>Luvox</td>
<td>0</td>
</tr>
<tr>
<td>Anti-psychotics</td>
<td></td>
</tr>
<tr>
<td>Abilify</td>
<td>1</td>
</tr>
<tr>
<td>Risperdal</td>
<td>2</td>
</tr>
</tbody>
</table>

(table cont.)
<p>| | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Serequel</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Thorazine</td>
<td>1</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Zyprexa</td>
<td>6</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

**Anxiolitics**

<p>| | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Ativan</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Buspar</td>
<td>1</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Inderal</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

**Mood Stabilizer**

<p>| | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Depakote/Dapakene</td>
<td>6</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Limictal</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Lithium</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Tegretol</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

**Non-Stimulants**

<p>| | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Strattera</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Behaviors that were targeted in the participants’ behavior support plans were also noted. It was observed that the target behaviors largely focused on non-specific symptom control (e.g., ‘bipolar symptoms’) in addition to other possibly unrelated maladaptive behaviors such as physical aggression, self-injury, and temper tantrums. All participants in the bipolar and psychopathology groups had target behaviors, while only six participants (approximately 43%) in the control group had a target behavior listed in their support plan. Specific behavioral targets by group are listed in Table 4.
<table>
<thead>
<tr>
<th>Behavioral Target</th>
<th>Bipolar</th>
<th>Psychopathology</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anger Management</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Biting others</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Chewing</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Disruptive Behavior (chain of disruptive behaviors)</td>
<td>3</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Elopement</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Food Theft</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Inappropriate Sexual Behavior</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Inappropriate Toileting</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Motoric Behavior</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Noncompliance</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Pica</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Refusing to walk/crawling</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Rumination</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>SIB</td>
<td>6</td>
<td>8</td>
<td>2</td>
</tr>
<tr>
<td>Sleep Disturbance</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Stereotypies</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

(table cont.)
Stripping 0 1 0
Tantrums 1 6 3
Threatening Others 0 0 1
Physical Aggression 4 6 2
Verbal Aggression 2 1 0

Note. Many participants had multiple target behaviors. Only specific behavioral targets are listed in Table 4, as the non-specific symptoms of disorders are redundant with Axis I information listed in Table 2.

Participant Selection

Residents were assigned to the bipolar group if they met the following criteria: 1) a chart review revealed that the individual was given a diagnosis of bipolar disorder by a licensed psychologist; and, 2) ratings on the DSM-IV checklist indicated the individual met criteria for the past occurrence of one or more manic episodes. Further, all diagnoses of bipolar disorder were re-confirmed by the licensed psychologist currently assigned to the participants' case.

Individuals selected into the psychopathology group were those that met the following criteria: 1) a chart review revealed that the individual had an Axis I diagnosis other than bipolar disorder; 2) ratings on the DSM-IV checklist indicated that the individual did not meet criteria for the past occurrence of manic episodes; and, 3) demographic variables including age, gender, level of ID, verbal ability, and ambulation were similar to individuals in the bipolar group. Control group participants were selected based on: 1) a chart review that revealed that the individual had no Axis I diagnosis; 2) ratings on the DSM-IV checklist that indicated that the individual did not meet criteria for the past occurrence of a manic episode; and, 3) demographic variables including age, gender, level of ID, verbal ability, and ambulation were similar to individuals in the bipolar group.
**DSM-IV Checklist.** Since the focus of this investigation was the presence and variability of manic symptoms over time, the DSM-IV-TR checklist was structured to focus on the past occurrence of a manic episode. As defined in the DSM-IV-TR (APA, 2000), to meet criteria on the checklist, the direct care informant had to endorse the presence of a distinct period of abnormally and persistently elevated symptoms of expansiveness or irritable mood lasting at least one week. The informant must have also indicated that during this mood disturbance there were specific symptoms, including at least three of the following: inflated self-esteem or grandiosity; decreased need for sleep; more talkative than usual or pressure to keep talking; flight of ideas or subjective experience that thoughts are racing; distractibility; increase in goal-directed activity or psychomotor agitation; and excessive involvement in pleasurable activities that have a high potential for painful consequences. Additionally, the informant had to endorse that during this mood disturbance the individual was difficult to manage or harder to care for than when not in a disturbed mood. The DSM-IV checklist is included as an appendix.

**Measures**

The present study used two measures to identify items as dependent measures of mania: 1) Parent Version of the Young Mania Rating Scale (P-YMRS); and 2) the Diagnostic Assessment for the Severely Handicapped-II (DASH-II).

**Parent Version of Young Mania Rating Scale (P-YMRS)**

The Young Mania Rating Scale (YMRS) is a well-known, commonly used, valid and reliable measure of mania recognized in the general population literature. This 11-item scale is typically used to assess the severity of mania in patients with bipolar disorder. The items are: elevated mood, increased motor activity energy, sexual interest, sleep, irritability, speech (rate and amount), language (thought disorder), content, disruptive-aggressive behavior, appearance,
and insight. It was designed to be a clinician-rating scale for persons without ID (Young, Biggs, Ziegler, & Meyer, 1978).

Recently, a parent version of the YMRS (P-YMRS) has been developed to assess bipolar disorder in youths. The P-YMRS is based on the original 11-item YMRS, but is in an adapted questionnaire format. The P-YMRS has been shown to accurately discriminate between individuals with bipolar disorder, unipolar disorder, and other diagnoses (Gracious, Youngstrom, Findling, & Calabrese, 2002). Gracious, Youngstrom, Findling, and Calabrese (2002) report acceptable internal consistency (.72) and good diagnostic efficiency (classification rates exceeding 78%), and high correlations with the original YMRS (r = .97). The total score range from 0-60. The average scores in the P-YMRS validity study were approximately 25 for mania and 20 for hypomania. The authors note that any score above 13 indicated a potential case of mania or hypomania, whereas any score above 21 was a probable case (Gracious, Youngstrom, Findling, & Calabrese, 2002). The P-YMRS was chosen for the present study because the YMRS is widely recognized in the literature, it has been shown to be reliable and valid, it can be used as an informant-based measure, it offers items that are scored on a Likert scale, and many items target observable behavior (Youngstrom, Gracious, Danielson, Findling, & Calabrese, 2003). The P-YMRS was administered to direct-care staff as an informant-based mania rating scale.

Diagnostic Assessment Severely Handicapped (DASH-II)

The DASH-II is an informant-based screening tool designed to evaluate psychopathology of individuals with severe and profound ID (Matson, 1995). This measure has been designed and validated for use with individuals with intellectual deficits. It is based on observable behaviors that can be reported by an informant who is familiar with the individual with ID.
This rating method consists of 84 items across 13 subscales representing major psychiatric disorders: 1) Anxiety, 2) Depression, 3) Mania, 4) PDD/Autism, 5) Schizophrenia, 6) Stereotypies, 7) Self-injury, 8) Elimination, 9) Eating, 10) Sleep, 11) Sexual, 12) Organic, and 13) Impulse. The DASH-II has good psychometric properties, inter-rater reliability is .86 and test-retest reliability is .84 (Matson, 1995). Many of the subscales of the DASH-II have been validated in previous research with a severe and profound ID population (e.g., PDD/Autism, Depression, and Mania subscales; Matson & Smiroldo, 1997; Matson, Smiroldo, & Hastings, 1998; Matson, et al. 1999).

Mania Subscale of the DASH-II

The Mania subscale is one of the 13 subscales of the DASH-II. Items on this subscale include: is restless or agitated, has a decreased need for sleep, is cranky or irritable, is easily distracted, is extremely happy or cheerful for no obvious reason, talks loudly, and talks quickly. Matson and Smiroldo’s (1997) validation study on this subscale reported rates of internal consistency correlations ranging from .42 to .76. Further, a discriminate analysis correctly classified 90.9 % of the manic individuals and 100 % of the controls using DSM-IV diagnoses as the criteria for group assignments. Additionally, the individual items of the Mania subscale (r = .43 to .91) and the total subscale score (r = .94) were significantly correlated with DSM-IV diagnosis. Thus, the DASH-II was administered to direct-care staff as an informant-based rating scale.

As identifying mania in this population remains a challenge for researchers and clinicians, two measures were referenced as indicators of mania for this study: the P-YMRS and the Mania subscale of the DASH-II. The P-YMRS was used as a criterion measure. There were five participants whose P-YMRS score reached 21 or above (e. g. ‘probable case of mania’;
These participants’ total P-YMRS score was correlated with their DASH-II item scores. Items of the DASH-II that were correlated with the criterion measure (P-YMRS) at .35 or higher were included in a derived subscale of the DASH-II. This P-YMRS derived subscale was called the **Criterion-referenced subscale** for the purpose of this study. The Mania subscale of the DASH-II was the alternative dependent measure of mania. In addition, two other subscales, the Pervasive Developmental Disorder (PDD) and Schizophrenia subscales of the DASH-II, were included in the analysis to serve as comparative anchor points for the examination of consistency of the mania measures. PDD symptoms are assumed to be stable over time. However, the course of schizophrenia symptoms commonly fluctuates with stress induced by the environment (APA, 2000; Reid, 1989). As the presence or absence of symptom fluctuation of PDD and schizophrenia have been recognized (e.g., DSM-IV-TR, APA, 2000) and should not significantly differ between the case groups, the serial correlation coefficients of these subscales served as indicators to whether the variability in the Mania and Criterion-referenced subscales are due to actual vacillation in symptoms or assessment error.

**Hypothesis**

Patterns of means and serial correlations across subscales were hypothesized for each group. It was assumed that assessing the patterns of means and serial correlations across the subscales would provide a validity estimate of subscale mean endorsements and serial correlations (Campbell & Fiske, 1959). A higher mean endorsement of manic symptoms on the Mania and Criteria-referenced subscales was anticipated for the bipolar group over the other two groups. Further, it was expected that the bipolar and psychopathology group would have comparable endorsements on the PDD and Schizophrenia subscale, while the control group
would have fewer endorsements on these subscales because these individuals have no Axis I diagnoses.

Moreover, it was expected that the bipolar group’s serial correlation on the Mania and Criterion-referenced subscales would diverge with the other two groups because their manic symptoms would wax and wane over time, and thus yield a low serial correlation. The other groups were hypothesized to consistently have few manic symptoms and thus, have a high serial correlation on these subscales. For the Schizophrenia subscale, it was assumed that the bipolar and psychopathology groups would converge with low serial correlations since both groups may have some schizophrenia-like symptoms that vacillate over time. Moreover, it was expected that the control group would consistently have few symptoms endorsements on this subscale and yield a high serial correlation. Finally, it was hypothesized that all three groups would have consistent symptoms endorsements, or a high serial correlation, on the PDD subscale since symptoms of PDD should be stable over time. Table 5 displays the hypothesized pattern of serial correlations across subscales.

Table 5
Hypothesized Pattern of Serial Correlations across Subscales

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Case Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bipolar</td>
</tr>
<tr>
<td></td>
<td>Psychopathology</td>
</tr>
<tr>
<td></td>
<td>Control</td>
</tr>
<tr>
<td>Mania</td>
<td>Low Score</td>
</tr>
<tr>
<td></td>
<td>High Score</td>
</tr>
<tr>
<td></td>
<td>High Score</td>
</tr>
<tr>
<td></td>
<td>(Less consistent)</td>
</tr>
<tr>
<td></td>
<td>(Highly consistent)</td>
</tr>
<tr>
<td>Criterion-referenced</td>
<td>Low Score</td>
</tr>
<tr>
<td></td>
<td>High Score</td>
</tr>
<tr>
<td></td>
<td>High Score</td>
</tr>
<tr>
<td></td>
<td>(Less consistent)</td>
</tr>
<tr>
<td></td>
<td>(Highly consistent)</td>
</tr>
</tbody>
</table>

(table cont.)
Procedure

The DSM-IV Checklist, DASH-II, and P-YMRS data were collected from direct-care staff familiar with the participants for a minimum of six months prior to the study. All instruments were administered to the same staff person for each resident to control for inter-rater error between instruments. The bipolar group and psychopathology group were selected based on the aforementioned criteria. Data for controls were collected shortly after individuals in the bipolar group had been selected. The author, who was trained in the administration of these assessment measures as outlined in their respective manuals, collected this data within a period of a month. Data collection and storage were conducted in accordance with accepted procedures to secure patient confidentiality. Retrospective DASH-II data was obtained from a main database for the designated three year time period (January 2002 - January 2005).
EXPERIMENTAL DESIGN

Maximum likelihood estimation was employed to test the null hypothesis that there is no significant difference between the variance/covariance structures of the case groups. First, a model, termed the restricted model, was fit to the data of all the groups. Then, a second model, termed the general model, was fit to each group separately. *Restricted Maximum Likelihood* was used to estimate these models. The difference of the restricted model from the general model was then compared to the $X^2$ table as an omnibus test. In the case that the omnibus comparison was not significant, no further analyses were conducted.

In the case that this maximum likelihood estimation procedure yielded a significant $X^2$ statistic, group estimated marginal means and serial correlations were then compared post-hoc. The post hoc comparison for the serial correlation was calculated by taking the difference of the group parameters and dividing it by the standard error of the parameters (Ullman, 2001). These pair-wise comparisons were then compared to a z-table for significance.
RESULTS

Annual Analyses

The annual data included three data points for each participant, one from each year 2002, 2003, and 2004 (including data collected at the time of study in January 2005). Annual data were data points that were collected during the same month and year for participants matched across groups. This approach was used to ensure that the time of assessment did not differ between groups. When three common data points could not be found in the archival database, data from January 2005 was used, since most participants had data collected during this month. There was one case when data across the matched triad did not coincide for month and year and in this instance, data from the prior month was used. Thus, all annual data points were collected within a two-month period for participants matched across groups. The time interval between annual data points ranged from 9 months to 22 months. One participant in the control group had only two annual data points due to missing data in the archive.

The maximum likelihood estimation procedure was used with each subscale. The restricted models testing the null hypothesis, that there were no significant differences between groups, was rejected for the Mania, \(X^2(8, N=42) = 27.913, p<.001\); Criterion referenced, \(X^2 = (8, N=42) = 47.25, p<.001\); and PDD subscales, \(X^2 (8, N=42) = 24.41, p<.005\). Model convergence could not be achieved for the Schizophrenia subscale. Further inspection of the data revealed that there was not enough variability in the Schizophrenia subscale for the control group during the second year. All symptom endorsements on this subscale for the control group in the year 2003 were zero.

Post hoc pair-wise comparisons were conducted with the estimated marginal group means for the three significant subscales. A Bonferroni correction procedure was used to protect
against inflation of family-wise error rate. Bonferroni type adjustments are made by assigning alpha for each DV, so that overall alpha for the total set of dependent variables does not exceed some critical value (Tabachnick & Fidell, 2001). Significant mean differences were found on the Mania subscale, $F(2, 45) = 9.26, p<.001$. The bipolar and psychopathology groups had significantly higher mean endorsements on the Mania subscale than the control group, $p<.001$ and $p=.02$, respectively. The difference between the bipolar and psychopathology groups were non-significant ($p=.07$). Significant mean differences were also found for the Criterion-referenced subscale, $F(2, 42) = 11.22, p<.001$. The bipolar group had a significantly higher mean endorsement than the psychopathology group, $p=.03$; and the control group, $p<.001$. The difference between the psychopathology and control groups was non-significant. There was also a significant mean difference found for the PDD subscale, $F(2, 51) = 8.86, p<.001$. As expected, both the bipolar and psychopathology groups had a higher mean endorsement on the PDD subscale than the control group, $p<.001$ and $p=.02$, respectively. The mean difference between the bipolar and psychopathology groups was non-significant ($p=.60$). Estimated marginal group means and standard deviations for these subscales are displayed in Table 6.

Table 6
Estimated Marginal Subscale Means by Group

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Case Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bipolar</td>
</tr>
<tr>
<td></td>
<td>$M$</td>
</tr>
<tr>
<td>Mania</td>
<td>3.36 &lt;sub&gt;a&lt;/sub&gt;</td>
</tr>
<tr>
<td>Criterion-referenced</td>
<td>7.61 &lt;sub&gt;a, b&lt;/sub&gt;</td>
</tr>
</tbody>
</table>

(table cont.)
Schizophrenia\textsuperscript{a} - - - - - -

Note. Means in a row sharing subscripts are significantly different at $p<.05$ using a Bonferroni correction. For all subscales, higher means indicate higher symptom endorsements. \textsuperscript{a} Model convergence criteria could not be achieved for the Schizophrenia subscale. Estimated likelihood ratios could not be reliably calculated.

Post hoc pair-wise comparisons were also conducted with the serial correlations of the groups for all four subscales. Again, Bonferroni procedures were used to protect against inflation of family wise error rate due to multiple comparisons. As these calculations were done by hand, alpha was adjusted to a significance level of $p=.02$. No significant differences were found between the serial correlations between groups on the Mania subscale: $p=.25$ (bipolar compared to psychopathology); $p=.22$ (psychopathology compared to control); and $p=.08$ (bipolar compared to control). Further, no significant differences were found between the serial correlations between groups on the Criterion-referenced subscale: $p=.26$ (bipolar compared to psychopathology); $p=.14$ (psychopathology compared to control); and $p=.04$ (bipolar compared to control). However, a difference was found on the PDD subscale. The control group had a significantly higher serial correlation than the psychopathology group $z=2.59$, $p=.005$. The serial correlations for each group are displayed in Table 7.

Table 7. Serial Correlations for Each Subscale by Group

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Bipolar</th>
<th>Psychopathology</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rho</td>
<td>SD</td>
<td>Rho</td>
<td>SD</td>
</tr>
<tr>
<td>Mania</td>
<td>.42</td>
<td>.17</td>
<td>.26</td>
</tr>
</tbody>
</table>

(table cont.)
<table>
<thead>
<tr>
<th>Subscale</th>
<th>Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Criterion-referenced</td>
<td>.48</td>
</tr>
<tr>
<td>PDD</td>
<td>.09</td>
</tr>
<tr>
<td>Schizophrenia(^a)</td>
<td>-</td>
</tr>
</tbody>
</table>

Note. Correlations in a row sharing subscripts are significantly different at p<.02. For all subscales, higher correlations indicate higher serial correlations.

\(^a\) Model convergence criteria were not achieved for the Schizophrenia subscale. Estimated likelihood ratios could not be calculated reliably.

As the serial correlations of both the Mania and Criterion-referenced subscales for the control group were extremely low (.04), a floor effect was suspected. The descriptive statistics and frequency data of the two subscales for each group were computed. For the control group, the Mania subscale scores ranged from 0 to 6, while the other groups’ scores ranged from 0 to 10 (maximum possible score of 14). Of the 41 annual data points (1 missing) for the control group, 48.8% had a Mania subscale score of 0. Further, 80.5% of the control group’s data points had a total Mania subscale of 2 or below.

The Criterion-referenced subscale frequency and descriptive statistics data indicated similar findings. The control group’s scores on the Criterion-referenced subscale ranged from 0-14, while the psychopathology and bipolar groups’ scores ranged from 0 to 17 and 0 to 26, respectively. Of the control group’s 41 data points (1 missing), 53.7% had a criterion-referenced subscale score of 0. Further, 75.6% of these data points had a subscale score of 2 or below. These descriptive statistics indicate restricted range of scores in the mania and Criterion-referenced subscales for the control group.

In order to get a better estimate of the means and serial correlations for the Schizophrenia subscale, the estimated likelihood procedure and pair-wise comparisons were re-calculated without data from year 2 for all subscales. Convergence criteria were achieved for all subscales. The restricted models testing the null hypothesis, that there were no significant differences...
between groups, was rejected for the Criterion-referenced, $X^2(8, N=42) = 26.90, p<.001$; and Schizophrenia subscales, $X^2(8, N=42) = 38.80, p<.001$. The null hypothesis was not rejected for the Mania, $X^2(8, N=42) = 14.42, n. s.;$ and the PDD subscales, $X^2(8, N=42) = 8.51, n. s.$

Pair-wise comparisons of estimated marginal means were conducted for the Criterion-referenced and Schizophrenia subscales. A Bonferroni correction procedure was used. A significant mean differences was found on the Criterion-referenced subscale, $F(2, 31) = 5.84$, $p=.005$. The bipolar group had a significantly higher mean endorsement than the control group on this subscale, $p<.01$. Mean differences between the bipolar and psychopathology groups, and psychopathology and control groups, were non-significant, $p=.10$ and $p=.77$, respectively. No significant mean differences were found between groups on the Schizophrenia subscale.

Estimated marginal subscale means excluding data from year 2 are displayed in Table 8.

Table 8
Estimated Marginal Subscale Means by Group (excluding 2003)

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Case Group</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Bipolar</td>
<td>Psychopathology</td>
<td>Control</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Mania</td>
<td></td>
<td>3.56</td>
<td>.52</td>
<td>2.65</td>
<td>.52</td>
</tr>
<tr>
<td>Criterion-referenced</td>
<td>7.54a</td>
<td>1.20</td>
<td>3.80</td>
<td>1.20</td>
<td>1.86a</td>
</tr>
<tr>
<td>PDD</td>
<td></td>
<td>3.39</td>
<td>.50</td>
<td>2.86</td>
<td>.50</td>
</tr>
<tr>
<td>Schizophrenia</td>
<td></td>
<td>1.86</td>
<td>.39</td>
<td>.66</td>
<td>.39</td>
</tr>
</tbody>
</table>

Note. Correlations in a row sharing subscripts are significantly different at $p<.005$. For all subscales, higher correlations indicate higher serial correlations.

Pair-wise comparisons were also conducted with the serial correlations of the Criterion-referenced and Schizophrenia subscales. Alpha was adjusted to $p=.02$ to protect against inflation.
of family-wise error rate (Tabachnick & Fidell, 2001). Significant differences were found between the serial correlations on the Criterion-referenced subscale. The bipolar group had a significantly higher serial correlation than the control group, \( p=.01 \). The difference between the bipolar and psychopathology group was non-significant (\( p=.03 \)). No significant differences were found on the Schizophrenia subscale: \( p=.14 \) (bipolar compared to psychopathology); \( p=.43 \) (psychopathology compared to control); \( p=.06 \) (bipolar compared to control). As the serial correlations of the Schizophrenia subscale for the control group were low (-.12), a floor effect was suspected. The descriptive statistics and frequency data of this subscale for each group were computed. For the control group, the Schizophrenia subscale ranged from 0 to 9. Of the control group’s 41 total annual data points (1 missing), 82.9\% had a Schizophrenia subscale score of 0. Further, with the exclusion of year 2003 (28 data points), 75.0\% of the control group’s of the data had a total Schizophrenia subscale endorsement of zero. The serial correlations for each group excluding data from 2003 are displayed in Table 9.

Table 9
Serial Correlations for Each Subscale by Group (excluding 2003)

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Case Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bipolar</td>
</tr>
<tr>
<td></td>
<td>Rho</td>
</tr>
<tr>
<td>Mania</td>
<td>.53</td>
</tr>
<tr>
<td>Criterion-referenced(^a)</td>
<td>.79(^a)</td>
</tr>
<tr>
<td>PDD</td>
<td>.18</td>
</tr>
<tr>
<td>Schizophrenia(^a)</td>
<td>.44</td>
</tr>
</tbody>
</table>

Note. Correlations in a row sharing subscripts are significantly different at \( p<.02 \). For all subscales, higher correlations indicate higher serial correlations.\(^a\)Significant omnibus test.
Monthly Analysis

The monthly analysis included data from each month for three years, January 2002 – January 2005. Although some participants did have missing data, all had at least 75% or 27 data points out of the maximum 37 monthly data points. The maximum likelihood estimation procedure was used with each subscale. The restricted model testing the null hypothesis that there were no significant differences between the groups was rejected for the Criterion-referenced, $\chi^2 (38, N=42) = 120.45, p<.001$, and Schizophrenia subscales, $\chi^2 (38, N=42) = 142.45, p<.001$. The null hypothesis was not rejected for the Mania, $\chi^2 (38, N=42) = 52.17, n. s.$, and the PDD subscales, $\chi^2 (38, N=42) = 36.01, n. s.$

Post hoc pair-wise comparisons were conducted with the estimated marginal group means for the Criterion-referenced and Schizophrenia subscales. A significant mean difference was found on the Criterion-referenced subscale, $F(1, 156) = 51.07, p<.001$. The bipolar group had a significantly higher mean endorsement on the Criterion-referenced subscale than the psychopathology group. A significant mean difference was also found on the Schizophrenia subscale, $F(1, 6) = 10.08, p=.02$. The bipolar group had a significantly higher mean endorsement on the Schizophrenia subscale than the psychopathology group. Estimated marginal group means and standard deviations are displayed in Table 10.

### Table 10
**Estimated Marginal Subscale Means by Group**

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Bipolar M</th>
<th>Bipolar SD</th>
<th>Psychopathology M</th>
<th>Psychopathology SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>(table cont.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Post hoc pair-wise comparisons for the serial correlations were also conducted for the Criterion-referenced and Schizophrenia subscales. A significant difference was found between the bipolar and psychopathology group on the Criterion-referenced subscale, $z=2.09$, $p=.02$. The bipolar group had a significantly higher serial correlation than the psychopathology group. The difference between the bipolar group and the psychopathology group on the Schizophrenia subscale was non-significant. The serial correlations for each group are displayed in Table 11.

Table 11
Serial Correlations for Each Subscale by Group

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Bipolar Rho</th>
<th>Bipolar SD</th>
<th>Psychopathology Rho</th>
<th>Psychopathology SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mania</td>
<td>.14</td>
<td>.05</td>
<td>.04</td>
<td>.06</td>
</tr>
<tr>
<td>Criterion-referenced $^{a,b}$</td>
<td>.30</td>
<td>.05</td>
<td>.14</td>
<td>.06</td>
</tr>
<tr>
<td>PDD</td>
<td>.04</td>
<td>.05</td>
<td>.01</td>
<td>.05</td>
</tr>
<tr>
<td>Schizophrenia $^a$</td>
<td>.42</td>
<td>.05</td>
<td>.35</td>
<td>.06</td>
</tr>
</tbody>
</table>

Note. For all subscales, higher correlations indicate higher serial correlations. $^a$ Significant omnibus test. $^b$ Significant group difference.
DISCUSSION

Manic symptoms were evaluated over time in three groups of individuals with intellectual disabilities: a group diagnosed with bipolar disorder, a group diagnosed with psychopathology other than bipolar disorder, and a group with no Axis I diagnosis. The Mania subscale of the DASH-II, and a subscale derived from the P-YMRS were the two dependent measures of mania. The derived subscale from the P-YMRS was called the Criterion-referenced subscale for the purpose of this study. Manic symptoms were evaluated in two ways: 1) symptom presence (mean score), and 2) consistency over time (serial correlation).

Manic symptoms were first evaluated on an annual basis over a period of three years. The estimated marginal means across groups were consistent with the hypotheses despite a few exceptions. It was expected that the bipolar group would have significantly greater symptoms endorsements on both the Mania and Criterion-referenced subscales than the other two groups. However for the Mania subscale, the bipolar group had significantly more symptoms endorsements than the control group but not the psychopathology group. Further, the psychopathology group had significantly more symptom endorsements than the control group. However, there was no significant difference between the bipolar and psychopathology groups. For the Criterion-referenced, the bipolar group had significantly more symptoms endorsements than both the psychopathology and control groups. One reason why the results on the Criterion-referenced subscale were as expected, while the Mania subscale was not, may be the fact that the Criterion-referenced subscale had significantly more items than the Mania subscale. Perhaps it was more sensitive to symptoms distinct to mania rather than symptoms exhibited with general psychopathology.
The mean scores on the PDD subscale were as expected. There were more symptom endorsements for the bipolar and psychopathology groups than the control group. This was not surprising as both the bipolar and psychopathology groups had various Axis I diagnoses (including those in the PDD spectrum), while the control group had no Axis I diagnoses.

As far as the Schizophrenia subscale, convergence was not achieved for the model. Further inspection of the data revealed that there were no symptoms endorsements in the annual data for 2003 for control participants. This artifact in the data resulted in limited variability in the cell, and thus convergence was not achieved. To better examine this data, the annual data were re-analyzed without data from year 2003 for all groups and subscales. A similar pattern to what was observed in the full analysis resulted from this analysis of group means. Convergence was achieved for all subscales. However, only the Criterion-referenced and Schizophrenia subscales resulted in significant omnibus tests. It is somewhat surprising that there was not a significant difference between the bipolar and the psychopathology groups on the Criterion-referenced subscale. Although not significant, it may be noted that the mean of the bipolar group was quite larger than the psychopathology group on this subscale. There was no significant difference in estimated marginal group means on the Schizophrenia subscale.

Next, the serial correlations of the annual data points were evaluated across groups. The results of this analysis were quite different than what was hypothesized. First, the patterns of the serial correlations for the Mania and Criterion-referenced subscales were unexpected. Contrary to the hypotheses, the bipolar group had higher, though not significant, serial correlations than the other two groups on these subscales. Further, the control group, which was expected to have highly consistent endorsements over time, had very low serial correlations.
An analysis of the descriptive statistics indicated a floor effect for the control group on these two subscales. Approximately half of the annual data points on the Mania subscale, and over half of the annual data points for the Criterion-referenced subscale for the control group, had no symptom endorsements. Though this descriptive information does explain the low serial correlation coefficient for the control groups, it does not clarify the relatively higher serial correlation coefficients for the bipolar group over the psychopathology group.

Moreover, the comparative anchor scales (PDD and Schizophrenia) yielded an unexpected pattern of results. For the PDD subscale, the bipolar and psychopathology groups had very low serial correlations. The control group, on the other hand, had a high serial correlation, significantly higher than the psychopathology group. All three groups were hypothesized to have high serial correlations on the PDD subscale, as symptoms of PDD should be stable over time (DSM-IV-TR, APA, 2000). However, the pattern of serial correlations suggests poor convergent validity across groups. It is difficult to explain why symptoms that should be stable over time have low serial correlations for two groups but high ones for another group.

The Schizophrenia subscale, as previously mentioned, did not achieve model convergence when the full data set was analyzed. Thus, the serial correlation data were examined without the data from year 2003. This pattern of correlations was also unexpected. The bipolar group had a relatively high serial correlation on this subscale when compared to the low serial correlations of the psychopathology and control groups, although this difference was not significant. The bipolar and psychopathology groups were hypothesized to have low serial correlations for the Schizophrenia subscale. These groups were expected to illustrate convergent validity on this subscale because fluctuation of symptoms was anticipated in these two groups.
Further, it was expected that the control group would be a measure of divergent validity for this subscale. It was anticipated that the control group would have a high serial correlation or highly consistent symptom endorsement on this subscale; however, due to a lack of symptoms, a floor effect was observed. As with the Mania and Criterion-referenced subscales, a descriptive analysis of the data revealed a floor effect for the control group on the Schizophrenia subscale. Approximately 83% of the annual data points (including year 2003) for the control group had no schizophrenia symptom endorsements, and 75% of the control group’s annual data excluding year 2003 had no schizophrenia symptom endorsements. Again, while this descriptive information clarifies the unexpected low coefficient for the control group, it does not explain the other discrepant findings.

Although descriptive statistics elucidate the unexpected findings of the control group on three of the four subscales, the patterns of serial correlations for the bipolar and psychopathology groups are difficult to explain. It remains unclear why there are low correlation coefficients for both the bipolar and psychopathology groups on the PDD subscale, which characterizes symptoms of PDD that should remain stable over time. Also, it is unclear why the bipolar group has relatively high correlation coefficients on subscales that characterize symptoms that should fluctuate over time, such as the Mania, Criterion-referenced, and Schizophrenia subscales.

One possible explanation for these unexpected patterns of serial correlation is that evaluation of annual data points (one assessment per year) may not be sensitive to the changes in symptoms over time. It may be necessary to have assessments that are more proximal in time to detect fluctuation in manic symptoms. It is not inconceivable that many of the participants were exhibiting comparable frequency of symptoms of mania at the three points in time. Thus, evaluation of these patterns with more frequent monitoring of manic symptoms was warranted.
Therefore, to examine this possibility further, monthly symptom assessments were evaluated for the two groups that had mania symptom endorsements: bipolar and psychopathology groups.

Monthly assessments across the four subscales were evaluated. The Criterion-referenced and Schizophrenia subscales resulted in significant omnibus tests. The estimated marginal means and serial correlations of these subscales were further evaluated with pair-wise comparisons.

Similar to what was observed with the annual data, the bipolar group had a significantly higher estimated marginal mean than the psychopathology group on the Criterion-referenced subscale. This difference was quite large. This further supports the notion that the Criterion-referenced subscale may be more sensitive to symptoms of mania than the Mania subscale, which resulted in a non-significant omnibus test. Again, the Criterion-referenced subscale included more items than the Mania subscale and appears to better differentiate the bipolar group from the psychopathology group.

Statistically significant differences were also found on the Schizophrenia subscale. However, it would be difficult to argue a true clinical difference on this subscale between the bipolar and psychopathology groups as the means were 1.40 and 1.00, respectively. There were extremely low standard deviations for these means, therefore statistically they were significantly different. However, clinically there is no true difference between these groups on this Schizophrenia subscale.

The pattern of serial correlations across the subscales was examined. Like the annual data, the pattern of serial correlations was higher for the bipolar group than the psychopathology group across the Mania and Criterion-referenced subscales. These patterns are inconsistent with the original hypotheses, but echo the annual findings. There is relatively high symptoms
consistency in manic symptoms for the bipolar group, which were expected to fluctuate over time.

Serial correlations are similar across groups for the PDD and Schizophrenia subscales. That is, the serial correlations on the PDD subscale were low for both groups. Further, both the bipolar and psychopathology groups had moderate serial correlations on the Schizophrenia subscales, .42 and .35 respectively (Osborne, 2003). The relatively high serial correlation on Schizophrenia subscale is one difference between the monthly and annual serial correlation analyses. Similar scores across groups on these subscales were expected; however, the coefficients are in the opposite direction than was anticipated. Both groups had high coefficients on the Schizophrenia subscale, and low coefficients on the PDD subscale. Thus, it appears that there was low consistency in symptom endorsement for PDD symptoms, which were assumed to be stable over time. There was also high consistency in symptoms endorsement for schizophrenia symptoms, which were assumed to fluctuate over time.

Pair-wise comparisons were only conducted for the Criterion-referenced and Schizophrenia subscales. The bipolar group had a significantly higher serial correlation than the psychopathology group on the Criterion-referenced subscale. That is, the bipolar group had a significantly more consistent endorsement of mania items than the psychopathology group. This finding is inconsistent with the original hypothesis, but is in-line with the results of the annual data serial correlations. There was no significant difference between the groups’ serial correlations on the Schizophrenia subscale.

Overall, similar patterns of means and serial correlations were observed across the annual and monthly data analyses. As hypothesized, the bipolar group had higher mean endorsements of manic symptoms on the Criterion-referenced subscale than the other groups. Further, the bipolar
group had a high serial correlation or consistent symptom endorsements for manic symptoms, which were expected to fluctuate over time. This finding was unexpected. There are several possible explanations for these unanticipated results.

First, perhaps symptoms of mania in the ID population are more chronic than in the general population. Therefore, staff endorsed symptoms of mania at each assessment because they are always present or were present in the last two weeks (e.g., similar to cyclothymia but do not meet criteria for manic episode). This explanation would support previous suggestions by Sovner (1989) that mood disorders in the ID population may be more chronic or rapid cycling.

A second, and perhaps more plausible explanation, is that staff perceive symptoms of mania as consistently present and are unlikely to accurately report when the symptoms were last observed. A common criticism of indirect assessment is that informants may not be motivated, trained, or competent enough to respond accurately (Lalli, Browder, Mace, & Brown, 1993; Sturmey, 1996). The staff’s work schedule often changes to compensate the needs of the home. Therefore, staff may not work the same time on the same day every week or even with the same individuals. Thus, they may know that the participant exhibits these symptoms but is unable to reliably report when it has last occurred.

A third possible explanation for the unexpected pattern of serial correlations is that the DASH-II may not be sensitive to symptom fluctuation over time. The DASH-II was intended to be a screening measure of psychopathology for individuals with ID. That is, it was originally designed to screen individuals for psychopathology so that a clinician could further evaluate individuals who had symptom endorsements one standard deviation above the mean (Matson, 1995). As the DASH-II was not originally designed to track symptoms over time, perhaps it is
not sensitive to change in symptoms and thus unable to detect true vacillation in manic symptoms.

Further, the Criterion-referenced subscale was based on the data of only five participants (those with a P-YMRS score of 21 or above). While Gracious, Youngstrom, Findling, & Calabrese (2002) state that any score above 21 indicates a probable case of mania, this cut-off may be somewhat conservative for a descriptive study such as this. In addition, some of the items of the P-YMRS, particularly those relying on verbal and cognitive abilities, were not endorsed for these individuals (i.e. Has your child shown changes in his/her thought patterns?; Is your child talking more quickly or more than usual?; Is your child talking about different things than usual?). As a result, there may have been an under-identification of current mania for these participants. Moreover, as few current mania cases were identified with the P-YMRS, the coefficients of the criterion-referenced subscale may be less stable than desired. These limitations should be taken into consideration when reflecting on the present findings and pursuing further research in this area.

While diagnosing bipolar disorder in individuals with intellectual disabilities is often a challenge, the results of the present evaluation of manic symptoms indicated that the bipolar group significantly differed from other groups on observable symptoms of the Criterion-referenced subscale, such as head banging, sleep difficulties, public masturbation and verbal abusiveness. This finding is in line with previous findings suggesting that clinicians can focus on observable symptoms in cases when DSM-IV criteria are not applicable (Sturmey, Laud, Cooper & Matson, 2004). Moreover, results of the current investigation lend support to previous suggestions that mood disorder symptoms, specifically symptoms of mania, are more chronic or rapid cycling in the ID population than what is observed in the general population (Sovner,
However, this conclusion should be taken cautiously as the explanation for the present findings are called into question by the unanticipated results on the comparative subscale (PPD and schizophrenia). In order to clarify these findings, research is needed to examine the ability of staff to accurately report the frequency of symptom presentation during specified periods of time. Future research should continue to examine mania in the intellectually disabled population in hopes of deriving tools that aid clinicians and researchers in the diagnosis and treatment of bipolar disorder in this population.
REFERENCES


APPENDIX
DSM-IV BIPOLAR CHECKLIST

Please indicate “yes” or “no” if these statements have ever applied to the individual in question. If a statement is not applicable to an individual, please indicate “n/a”.

Y or N

_____ 1. A distinct period of abnormally and persistently elevated, expansive or irritable mood lasting at least one week?

_____ 2. Is the individual difficult to manage or harder to care for during this mood disturbance than when not in a disturbed mood?

During this period of abnormal mood, have you observed the individual with the following symptoms:

_____ 1. Inflated self-esteem or grandiosity

_____ 2. Decreased need for sleep (seems rested after only 3 hours of sleep)

_____ 3. More talkative than usual or pressure to keep talking

_____ 4. Flight of ideas or subjective experience that thoughts are racing

_____ 5. Distractibility (attention too easily drawn to unimportant or irrelevant external stimuli)

_____ 6. Increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation (restlessness, pacing or holding multiple conversations at once)

_____ 7. Excessive involvement in pleasurable activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments)
VITA

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