The rate of decline of social skills across dementing and non-dementing individuals with intellectual disabilities: a longitudinal study

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THE RATE OF DECLINE OF SOCIAL SKILLS ACROSS DEMENTING AND NON-DEMENTING INDIVIDUALS WITH INTELLECTUAL DISABILITIES: A LONGITUDINAL STUDY

A Dissertation

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 Doctor of Philosophy

in

The Department of Psychology

by

Julia D. Lott
B.S., Louisiana College, 1993
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August, 2006
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Outside of my family, I owe a debt of gratitude to my mentors Ronald Pryer, Ph.D. and Nancy Grace, Ph.D. Both of these individuals have independently taught me the measure of mentorship. Their concern and support extended beyond professional development to personal growth and enrichment. Across the years, they have given far more of themselves than I deserved and have asked for nothing in return. It is my hope that I carry on the tradition and extend this measure of support to those that follow.

For my friends I can only say thank you, thank you, thank you. To Patrick O’Callaghan thanks for seeing me through from the beginning to the end, through hell and high water. To Steve and Leslie Gruesbeck thanks for showing me there was always something more to life than academics. To Terry Aubin thanks for standing with me, and waiting in the wings.
To the children in my life: Sarah Elizabeth Lott, James “Jimmy” Michael Lott, Emma Gregory Gruesbeck, and Sophia Clay Gruesbeck, remember the world is your oyster and I am always here to help you find the pearls.

Shalom ya’ll!
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ABSTRACT

This study sought to establish rate of decline of adaptive skills in a population of individuals with intellectual disability (ID) and dementia compared to similar persons without dementia, as well as examining the variability of positive and negative social behaviors across diagnostic classes. Among the general population, differential rates of functional decline have been established for normal aging and dementia. This knowledge assists in making differential diagnoses of dementia, establishing prognosis, and long-term planning.

For this study, participants in each group were individually matched for age, gender, Down’s syndrome status, and level of ID. Participants in the matched control group were screened for the presence of dementia with the Early Signs of Dementia Checklist (Visser & Kuilman, 1990). A 2 (groups) X 3 (measures) X 4 (repeated measures) Mixed Multivariate Analysis of Variance was completed to assess rate of decline within groups with the Vineland Adaptive Behavior Scales (Sparrow, Balla, & Cicchetti, 1984) and corresponding changes in positive and negative behaviors as measured by the Matson Evaluation of Social Skills for the Severely Retarded (Matson, 1995).

Hypothesis 1 established that prior to a diagnosis of dementia groups were equivalent (F (1, 40) = 1.086, p = .304). Hypothesis 2 found no significant differences for adaptive behaviors, therefore rate of decline was not established. However, visual analysis of plotted means supports predicted decline in skills for both groups. Furthermore there were significant differences across time for positive social skills as measured with the MESSIER (F (3, 96) = 3.887, p = .011, β = .811). Hypothesis 3
yielded significant correlations between the VABS and the MESSIER Positive domains.
Hypothesis 4 resulted in no significant correlations between the VABS and the MESSIER
Negative skills. The findings of Hypotheses 2, 3, and 4 provide support for the diagnostic
utility of the MESSIER with dementia. However, Hypothesis 4 did not support different
variances of negative behaviors across diagnostic groups. This would suggest that the
measure of negative behaviors is not supported as a diagnostic tool at this time.
INTRODUCTION

The study of dementia among persons with intellectual disabilities* (ID) is a relatively new area of research. Increasing interest in the co-occurrence of intellectual disability and dementia has been reflected by a slowly growing body of investigations over the last eighteen years (Slomaka & Berkey, 1997). Overall, the ID population reflects approximately 3% of the general population (American Association on Mental Retardation, 2005; World Health Organization, 2005). While much research related to studying dementia has been among individuals with Down’s syndrome, this proposed research focuses on dementia among all individuals with ID without respect to the presence of Down’s syndrome. Given the ID population is much broader than the sub-sample of individuals with Down’s syndrome and prevalence studies suggest risks for all individuals with ID, the information below will focus on global features of dementia as related to the ID population (Evenhuis, 1990; Lai & Williams, 1989).

The goal of this research is to use and analyze routine annual assessments to establish rate of decline of adaptive behaviors and variability of social skills for individuals with ID. With a benchmark for rate of decline, future research may be able to estimate standard expectations for decline among sub-classifications of dementia, allowing improved service planning and treatment efforts. While a number of studies suggest that dementia has an early onset among persons with ID, the rate and course of decline for this population remains undefined. As survival rates among persons with ID are gradually approaching that of the general population, age related conditions become

* Note the term intellectual disability will be used to reflect mental retardation throughout the course of this dissertation. A thorough review of the term will be discussed in the Intellectual Disability: Overview section.
increasingly important (Zigman, et al., 2004) and prevalence rate of dementia is increasing among both the general population and those with ID (Hofman, 1991; Jorm, et al., 1987; Rockwood, 1994, c.f. Aguero-Torres, Fratiglioni, & Winblad, 1998). The lack of pre-mortem diagnostic markers for Alzheimer’s and unfeasibility of a cure or prevention further establishes a need to accurately characterize the rate of decline of dementia in measurable terms (Janicki, Heller, Seltzer, & Hogg, 1996). The rate of decline becomes salient information as individuals with more rapid rates of decline require earlier, more immediate assistance (Das & Mishra, 1995; Haveman, et al., 1994). Therefore, predicting rate of decline becomes critical in planning supports and evaluating potential treatments. Moreover, there is a continued need to establish rate of decline, variability of social skills, and course of dementia as these trends may be a crucial piece in differential diagnosis. For example, features of Lewy Body dementia are markedly different from features of Vascular Dementia, as well as Alzheimer’s dementia (more detailed review provided in following sections) all of which result in varied treatment responses (Burt, 1998; Burt & Aylward, 1998; Coffey & Cummings, 1994; Small, et al., 1997).

Efforts to identifying dementia onset in the ID population have used both caregiver reports and standardized assessments. Although a “gold standard” assessment has yet to be established for this dually diagnosed population, the inclusion of both direct and indirect assessments of dementia has increasingly become standard practice (Burt & Aylward, 1998; 2000). Because floor effects are often found in many direct assessments (i.e. intellectual and adaptive assessments), the inclusion of indirect assessments (i.e. structured interviews) conducted with familiar caregivers increases the reliability of
initial diagnoses of dementia (Burt & Aylward, 1998; Haveman, et al., 1994; Oliver, 1998). Such combined assessments are useful to assist with differential diagnosis, given that cognitive decline due to depression and medical conditions such as hypothyroidism are treatable and reversible (Balis, 1997; Thase, 1982).

The present research identifies rate of decline in adaptive skills among ID population with and without dementia. The following sections provide an overview of intellectual disabilities, dementia, and the unique characteristics of dementia among persons with ID. A general overview of intellectual disabilities establishes parameters of the population considered, along with defining features that impact services/supports for this population. A review of the literature on dementia illustrates varying subtypes and their typical rates of progression, and courses of treatments. Within the last eighteen years, the impact of dementia in individuals with ID has gained attention. A review of the research is provided to highlight noted features that warrant attention and continued research. The specific focus of the present research establishes rate of decline of adaptive behaviors among ID individuals with and without a diagnosis of dementia.
INTELLECTUAL DISABILITY: OVERVIEW

Terminology

“Intellectual disability” is a term that has been predominately used in the United Kingdom for years and has found increasing preferential use in the United States of America (U.S.), being used in place of the terms “mental retardation” and “developmental disabilities” (Center for Disease Control, 2005; Schroeder, Gerry, Gertz, & Velazquez, 2002). Additionally, “intellectual disability” has found international acceptance and has been adopted by the World Health Organization (WHO) and the International Society for the Scientific Study of Intellectual Disabilities. The momentum in changes in terminology is a result of negative connotations and stigmatization found with the term “mental retardation” (Schroeder, et al., 2002). Continuing evolutionary efforts to employ less stigmatizing language has resulted in at least nine such terminology changes over the last 100 years (Schroeder, et al., 2002).

Over the last thirty years, “developmental disability” has become commonly used in the U.S. as a global category that includes mental retardation. The concept of developmental disability was not routinely used until it was adopted in U.S. legislation in the 1970 Developmental Disabilities Services and Facilities Construction Act (PL 91-517). By definition, it was broad and included mental retardation, cerebral palsy, autism, epilepsy and other neurological impairments (Summers, 1986). This term has more recently been seen in the Individuals with Disabilities Education Act (IDEA) of 1997 (IDEA, 1997; Schroeder, et al., 2002; Taylor, 2002). While the term “developmental disability” found widespread use, researchers suggest that the term intellectual disability has a more widespread consensus (Russell, Mammen, & Russell, 2005).
Among the European Agencies instituting the use of the term “intellectual disability” the Law Reform Commission noted that the term “mental” can be pejorative in nature and has an inaccurate association with mental illness. While the term “developmental disability” has commonly been used in place of “mental retardation” it reflects a broader group than individuals with an intellectual insult (Law Reform Commission, 1992; Schroeder, et al., 2002). For instance, “developmental disability” by definition is inclusive of individuals with cerebral palsy, disorders of reading, writing, arithmetic (American Psychiatric Association (APA), 2000), and speech. “Developmental disability” often is overly inclusive, encompassing children who are at risk for mental retardation or cases in which a diagnosis is unclear or unconfirmed (Schroeder, et al., 2002). The use of the more specific term “intellectual disability” has found its way into U.S. legislation and is currently reflected in legislation in 36 of the states (Taylor, 2002).

A growing preference for the use of the term intellectual disability is further illustrated in recommendations from the Center for Disease Control (CDC), and the National Institute of Health (NIH) which suggest elimination of the term “mental retardation” and replacing it with ID (CDC, 2005; NIH, 2005). The term has also shown an increase in professional research literature with an established 8% use from 1992-1997 to 16% use in sampled years from 1997-2002 (Schroeder, et al., 2002). The flagship journal of the American Association on Mental Retardation (AAMR), “Mental Retardation” has published editorials on the change in terminology (Taylor, 2002), and more recently in updating the definition of mental retardation the authors continuously use the term “intellectual disability” (Luckasson, et al., 2002).
Definition

One of the primary roles of the AAMR has been development of a definition and classification system of intellectual disability. Under various names this organization has published manuals on definitions and diagnostic criteria since 1921 and has most recently published revisions in a tenth edition in 2002 (Cuskelley, 2004). Over the years there has been a consistent progression and development of the definition of intellectual disability. During the course of these changes, terms have varied and included: feeble mindedness, idiocy, mental deficiency, mental disability, mental handicap, organic mental disorder, developmental disability, and intellectual disability (Schroeder, et al., 2002).

Of the previous definitions features of the definition proposed by Grossman (1983) have been maintained across time. In the AAMR eighth revision of the definition of mental retardation, the disorder was defined as significantly sub-average intellectual functioning with collateral impairments in adaptive behaviors that occur during the course of development prior to the age of 18 (Grossman, 1983). “Significantly sub-average” was defined as two standard deviations below the mean, reflecting the first departure from the previous definition seen in the seventh edition (Cuskelley, 2004). Therefore, mental retardation was defined as an intellectual quotient (IQ) below 70 on a standardized measure of intelligence. The second notable change in the definition was that the diagnosis of mental retardation could be extended upward to an IQ of 75 depending on the reliability of the intelligence test used. These changes altered the IQ ranges of intellectual disability from Mild disability previously listed as lower limits of 52 to upper limits of 67 to lower ranges of 50-55 to an upper range of 70-75, Moderate disability previously listed as a range of 36-51 changed to a range from 35-40 to 50-55,
Severe disability once listed as a range of 20-35 altered to a range from 20-25 to 35-40, and the Profound range of functioning changed from a listing of below 20 to the range of 20-25 or below. Finally an additional category was added called Unspecified which allowed for the classification of individuals who presented with physical limitations that precluded testing or cognitive disabilities presenting at such a level to preclude testing (Grossman, 1983).

The fundamental change across series of previous definitions was the addition of deficits in adaptive behavior. Limitation in adaptive behavior was defined in this context as impairment in effectiveness in meeting standards of maturation, learning, personal independence, or social responsibility that are expected based on age and cultural standards (Grossman, 1983). While this definition has undergone two revisions noted below, the features that remain evident in practice to date include use of adaptive deficits as a primary component of diagnosis as well as utilization of classification ranges (APA, 2000).

Across the five different classification ranges there are differing prevalence rates and presentation of adaptive skills. Mild mental retardation represents 85% of individuals with ID and is characterized as IQ ranging from 50-55 to 70 (APA, 2000; Grossman, 1983). Individuals within this group typically develop social and communication skills within the preschool years, show minimal impairment in sensorimotor functioning, and often are not noticeably different from children without ID until later in life (APA, 2000). As adults, they are not likely to have achieved skills beyond a sixth grade educational level. Individuals functioning within the Moderate range of intellectual disability constitute 10% of the ID population and have an IQ ranging between 35-40 to 50-55
(APA, 2000; Grossman, 1983). These individuals often display social and communication strengths such as being able to initiate and maintain a conversation, but rarely achieve beyond a second grade educational level. An IQ between the levels of 20-25 and 35-40 denotes the Severe range of intellectual disability (Grossman, 1983). This sub-population represents 3-4% of persons with developmental disabilities. These individuals typically acquire little to no communicative speech during early childhood, but may learn to talk during school years and acquire some simple daily living skills (APA, 2000). An IQ below the range of 20-25 characterizes the Profound range of intellectual functioning (Grossman, 1983). This group represents between 1-2% of the population of persons with ID. Commonly, individuals within this group have neurological conditions that account for their ID (APA, 2000). McLaren and Bryson (1987) suggest that in these cases prenatal factors are more common than delivery or postnatal complications. Typically, this group shows considerable impairments in sensorimotor functioning, which further impedes their development of daily living skills.

The ninth revision of the definition of mental retardation made attempts to the remove levels of cognitive functioning (previously detailed) as sub-classifications of mental retardation. The authors maintained the criteria of significantly sub-average intellectual functioning detailed in the 8th revision and deficits in adaptive functioning. They further added ten specific areas of adaptive functioning, which included the following: communication, self-care, home living, social skills, community use, self-direction, health and safety, functional academics, leisure, and work. For a deficit to be noted in the area of adaptive functioning the authors specified a requirement of a limitation in two or more of ten areas identified, thus advancing criteria for diagnosis.
However, the assessment of adaptive behavior was markedly changed and replaced with a 3-step procedure for diagnosing, classifying, and determining needed supports (Luckasson, et al., 1992). By incorporating needed supports, the model of the definition was changed from a deficiency model to a support model of mental retardation (Reiss, 1994; Schalock, et al., 1994). The sub-classifications of Mild, Moderate, Severe, and Profound deficits were thus removed and replaced by intensities of supports needed, which included Intermittent, Limited, Extensive, and Pervasive supports (Luckasson, et al., 1992). Reiss (1994) notes that these changes in interpreting and identifying adaptive functioning were made in an effort to facilitate inclusive education opportunities, supported competitive employment, and supported independent living. Cuskelly (2004) suggests that these assessment measures now create opportunity for the development of appropriate interventions. However, the shift to the support model was not readily embraced and implemented by the clinical community. Criticisms of this definition included that it was theoretical in nature and reflected social concerns and advocacy (Jacobson, 1993; MacMillian, et al., 1995). In a review completed by Conyers, Martin, Martin and Yu (2002), findings yielded that only 10% of articles in primary research journals in the field of intellectual disabilities used the definition from the 9th revision. An additional study noted that the 1983 definition by Grossman was maintained in legislation in 44 of the 51 United States (Denning, Chamberlaine, & Polloway, 2000).

While the AAMR as an organization is principally responsible for defining mental retardation, the definition of mental retardation in the Diagnostic Statistical Manual-Fourth Edition- Text Revision (DSM-IV-TR) reflects a blending of the definitions from the 8th and 9th revision definitions by the AAMR. Features of the definition found in the
DSM-IV TR include sub-average intellectual functioning as well as limitations in adaptive functioning (APA, 2000; Grossman, 1983; Luckasson, et al., 1993). Levels of functioning set forth by Grossman (1983) have been maintained and the ten areas of adaptive functioning established in the 1993 definition (Luckasson et al., 1993) have been incorporated into the definition. Because the disorder is conceptualized as developmental in nature, onset prior to age 18 has been maintained (APA, 2000). A notable departure from both the 8th and 9th revisions is that the DSM-IV definition uses a cut off IQ of 70 for a diagnosis of mental retardation as opposed to the AAMR definition which presents an option for an upper range IQ of 75 (APA, 2000).

In 2002, the AAMR released a 10th revision of the definition for mental retardation. In this version, “mental retardation” is routinely referred to as “intellectual disability”. The disability maintains the characterizations of significant limitations in both intellectual and adaptive functioning evinced prior to the age 18. Functioning has to be established as more than two standard deviations below the mean for both intellectual and adaptive functioning. The authors of this definition have become more prescriptive in terms of adaptive functioning requiring that measures of adaptive functioning must be normed on general population including individuals from the general population both with and without disabilities. The definition is further developed noting that intellectual disability is a multidimensional construct. The definition reflects five dimensions for consideration which include: intellectual abilities, adaptive behavior, behavior problems, social roles, and health concerns (Luckasson, et al., 2002). While authors of this definition have made efforts to move beyond diagnosis and establish treatments, they
have also incorporated features of Grossman (1983) definition that were previously removed (Cuskelly, 2004).

**Prevalence and Etiology of ID**

The WHO reports prevalence rates of ID ranging from 2 to 85 per 1000 individuals (Roeleveld, Zielhuis, & Gabreels, 1997). Prevalence as noted in the DSM-IV TR estimates that 1% of the general population function within some range of ID (APA, 2000). More recent studies suggest the prevalence of ID may be has high as 3% of the general population (AAMR, 2003; WHO, 2005). By definition, ID is limited to individuals functioning 2 standard deviations below the standard mean of an IQ of 100, yielding a prevalence rate of 2.5% (Grossman, 1983).

While thirty to forty percent of cases of ID show no clear etiology (APA, 2000), there has been increasing knowledge of causes of many intellectual disabilities. At this time, there are over 700 known genetic syndromes linked with intellectual disability (Schroeder, et al., 2002). Many additional factors may account for ID, including organic, developmental, and behavioral factors as well as individual levels of social adaptive skills (Matson, Anderson, & Bamburg, 2000). Other factors include genetic abnormalities, disturbances in embryonic development, complications with pregnancy and/or delivery, medical conditions acquired during infancy/early childhood, and environmental influences (APA, 2000; Hodapp & Dykens, 1996).

**Adaptive Behavior Skills**

Adaptive behavior is commonly defined as the completion of daily living skills that are required for social competency. Competency is determined based on age-related expectations, social/cultural standards, and the performance of the skill rather than the
ability to complete the skill (Sparrow, Balla, & Cicchetti, 1984). As previously detailed adaptive behaviors include: communication, self-care, home living, social skills, community use, self-direction, health and safety, functional academics, leisure, and work (Luckasson, et al., 1992); and comprise an essential component in the diagnosis of ID (APA, 2000; Grossman, 1983; Luckasson, et al., 2002).

Doll (1947) initially coined the term “social maturity” which led to the development of instruments which assessed adaptive behavior, with his Vineland Social Maturity Scale (1947) as one of the first widely adopted tools. The most commonly used assessment measures of adaptive behaviors include, its current derivative, the Vineland Adaptive Behavior Scale (VABS; Sparrow, Balla, & Cicchetti, 1984) and the AAMR Adaptive Behavior Scale (AAMR-ABS; Nihira, Leland, & Lambert, 1993). Reliability and validity are well established for both scales (Nihira, et al., 1993; Sparrow et al., 1984). The VABS assesses three primary domains that include: communication, socialization, and daily living skills. This scale was normed with 3000 individuals inclusive of individuals with and without disabilities. Given that the VABS has been normed with individuals with and without ID, it becomes an ideal measure of adaptive behavior based on the AAMR standards set forth in 2002. Supplementary norms are provided for groups of individuals that present with ID and physical disabilities. Both reliability and validity are well established (Sparrow, et al., 1984). The AAMR-ABS consists of two parts that evaluate coping skills relevant to personal independence and responsibility as related to daily living. The scale was normed on 4000 individuals with developmental disabilities residing in community settings (Nihira et al, 1993).
Social Skills

Social skills are a primary component of adaptive behavior (Grossman, 1983), and as such are reflective as a primary component of adaptive behavior scales (Bielecki, & Swender, 2004; Guralnick, 1986; Hazinski, & Matson, 1985; Kelly, Furman, Phillips, Hathorn & Wilson, 1979; Marchetti, & Campbell, 1990; Matson, & Andrasik, 1982; Meyers, Nihira, & Zetlin, 1979; Oswald & Ollendick, 1989; Sparrow, et al., 1984). Social behaviors are shown in a variety of manners to include interacting effectively with others, identifying and responding to social cues, avoiding conflicts, and adjusting to complex situations (Matson & Swiezy, 1994). These are all features that are necessary for individuals to perform successfully in social situations (Guralnick, 1986; McFall, 1982).

Individuals with learning disabilities are more likely to show deficits in social skills than individuals among the general population (Lovett & Harris, 1987; Singh & Winton, 1983). Identified causes for social skills deficits include lack of opportunities to practice appropriate social behaviors, inadequate feedback or reinforcement, and adverse effects of problem behaviors (Elliot & Gresham, 1993; Njardvik, Matson, & Cherry, 1999). Among individuals with ID and a diagnosis of psychopathology there are even greater deficits in social skills (Duncan, Matson, Bamburg, Cherry, & Buckley, 1999; Matson, Smiroldo, & Bamburg, 1998). These deficits in social skills often result in social rejection and impairments to social inclusion (Fee, Matson, & Manikam, 1990; Guralnick, 1986; Kazdin, Matson, Esveldt-Dawson, 1984; Kelly, et al. 1979; Kennedy, 1988; Raymond & Matson, 1989).

Assessment of social skills has typically occurred in one of three formats to include: behavioral observations, role playing, and behavioral checklists (Bielecki, &
Swender, 2004; Castles, & Glass, 1986; Platt & Spivack, 1975; Shapiro & Browder, 1990). In the past, assessment of social skills has predominately been limited to individuals with Mild and Moderate mental retardation (Singh & Winton, 1983). However, individuals functioning within the Severe and Profound ranges of ID are capable of social skills. Examples of social interactions for this subgroup include verbal skills (salutary greetings), motor skills (eye contact, reaching for familiar people), and interaction skills (interest in others around them) (Kuhn, 2004).

Behavioral checklist and rating scales are the most direct and commonly used formats of assessment for individuals functioning within the Severe and Profound ranges of ID (Browder & West, 1992). The Matson Evaluation of Social Skills for Individuals with sEvere Retardation (MESSIER) was developed to assess social skills excesses and deficits for individuals functioning within the Severe and Profound ranges of ID (Matson, 1995). The MESSIER was normed with 1236 individuals within Severe and Profound ranges of ID residing in developmental centers in Louisiana and Texas (Matson, 1995; Matson, Carlisle & Bamburg, 1998). Convergent validity for the MESSIER has been established with the VABS and sociometric ratings (LeBlanc, Matson, Cherry, & Bamburg, 1999; Matson, et al., 1998; Matson, LeBlanc, & Weinheimer, 1999). Test-retest and inter-rater reliability have been established (Matson et al., 1999). Specific strengths of the MESSIER compared to the VABS include a larger range of items, assessment of social skill deficits, and provision of greater specificity in discriminating social skills for individuals functioning within the Severe and Profound ranges of ID (Bielecki, & Swender, 2004; Matson, et al., 1998).
DEMENTIA: OVERVIEW

Dementia reflects a group of symptoms, rather than a specific disease process. It is characterized by multiple cognitive deficits, as well as one of the following: aphasia, apraxia, agnosia, or disturbance of executive functioning (APA, 2000). These problems reflect overall reductions from previous levels of functioning and are of sufficient severity to cause impairment in social and/or occupational functioning (APA, 2000). In making a diagnosis of dementia one is required to assess current level of functioning while at the same time document a higher level of previous functioning. Once a diagnosis of dementia is made, determining domains of affected cognitive functioning enables an examiner to quantify the breadth and severity of dementia as well as assist in establishing the specific underlying disease process (Lanska & Schoenberg, 1993). Differential diagnosis of sub-classifications of dementia is relevant as there are various forms of dementia that occur at different frequencies and require different treatment interventions (Lanska & Schoenberg, 1993). Effective management of dementia requires early identification and good communication across care givers and service providers (Ford, Bryant, Mangoni, & Jackson, 2003). Early identification facilitates treatment planning to include decisions for disease modifying drugs as well as long range planning for decline in skills and potential development of problem behaviors (Berg, 2003; Ralph, Patterson, Graham, Dawson, & Hodges, 2003). Late identification can result in lost opportunities for treatment gains or potential inadequate treatment (Kuslansky, Buschke, Katz, Sliwinski, & Lipton, 2002). It should be noted that while there are many available treatments that slow or arrest the development of dementia dependent on disease pathology, there is no one drug available or treatment that will change the long term
outcome of dementia (Cacabelos, 2002; Klag, 1999; Mace, Whitehouse, Smyth, 1993). The presence of dementia often results in an overall decrease in life expectancy, with men showing a shorter duration dementia and life expectancy than women (Aguerro-Torres, Fratiglioni, Gou, Viitanen, & Winblad, 1998; Keene, Hope, Fairburn, & Jacoby, 2001). Overall, there are concerns that dementia is under-diagnosed in younger people in general, and men more specifically (Newens, Forster, & Kay, 1993).

Clinicians need to be familiar with the concept of normal aging and typically expected cognitive decline when diagnosing dementia (APA, 2000; c.f. Lanska & Schoenberg, 1993). Eighty percent of the population never experience significant memory loss, thus reflecting healthy aging (APA, 2000). Slight forgetfulness is common with aging, but is not of sufficient severity to interfere with social and occupational functioning (APA, 2000). The presence of significant cognitive decline ensures change is not a result of testing error, and assists in distinguishing minor changes that occur in typical aging (Lanska & Schienberg, 1993).

A concept separate from both normal aging and dementia is that of minimal cognitive decline or mild cognitive impairment (MCI). MCI is often seen prior to the diagnosis of dementia and is often considered the earliest stage of Alzheimer’s disease (AD) (Galvin, et al., 2005; Ralph et al., 2003). Some researchers suggest that features of MCI can be seen as early as the late 20’s or as early as 20 years before a diagnosis of AD is made, with evidence of premature neuronal decay (Cacabelos, 2002; Ralph et al., 2003). Additionally, there is evidence that AD has a latent period of 10, 15, or 30 years before a diagnosis is made (Klag, 1999). A study completed by Ralph et al. (2003) examined 55 subjects, 38 of whom were diagnosed with AD and 18 of whom were
diagnosed with MCI. Within a two year time frame, 14 of the 18 individuals identified with MCI had converted to a diagnosis of AD, thus supporting the concept of a prodromal phase of dementia. At this time, there are concerns over consistent standards to discriminate MCI from normal aging; therefore it becomes difficult to differentiate cognitive decline in these situations as well as minimal cognitive decline associated with depression (Ritchie, Artero, & Touchon, 2001).

**Prevalence**

At present, the overall prevalence of dementia is between 2-4% for individuals over the age of 65 (APA, 2000). Overall, Kaplan, Saddock, and Grebb, (1994) suggest prevalence rates for individuals over 65 years of age range from 5-15%. Prevalence rates increase with age from 1.4% to 1.6% for individuals between 65-69 years of age and range from 16% to 25% for individuals over 85-years of age (APA, 2000). More recent estimates by Insel and Badger (2002) indicate that 10% of the population over 65 years old and as many as 50% of individuals over 85 years old have some form of dementia.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Prevalence of Dementia in the General Population</th>
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<tbody>
<tr>
<td></td>
<td>65 and older</td>
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<tr>
<td>3-11%</td>
<td>25-47%</td>
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<td>1%</td>
<td>------</td>
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<tr>
<td>10%</td>
<td>50%</td>
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<td>2-4%</td>
<td>16-25%</td>
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<tr>
<td>2%*</td>
<td>32%*</td>
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<tr>
<td>5-15%</td>
<td>------</td>
</tr>
<tr>
<td>3.7-8.3%</td>
<td>30.5-37.5%</td>
</tr>
</tbody>
</table>

*Prevalence rate specific to AD.
Average life expectancy has shown increases from age 47 in 1900 to 75 years of age in 1999 (Klag, 1999). It is estimated that with aging baby boomers and improvements in health care the overall number of individuals with dementia will continue to increase across time. Current estimates suggest that 50% of the population will live until 75 years of age and 25% of the population will live beyond 80 years, which increases overall prevalence considerations (Harper, 1993).

**Risk Factors for Dementia**

Schultz, et al. (2004) suggest that age is the strongest risk factor for dementia, thus underscoring the importance of differentiating normal aging from pathological aging across time (i.e., sensitivity to change creating opportunities for earlier intervention). Additional risk factors include: family history, low educational level, previous head trauma, cardiovascular disease, stroke, diabetes, apolipoproteins E-3 and E-4 alleles, and previous major depressive episodes (Bush & Beail, 2004; Ravaglia, 2002; Sliwinski, Buschke, Stewart, & Masur, 1997; Torpy, 2004). Henderson and Jorm (1997) suggest risks related to environmental exposures are accelerant considerations rather than causative factors of dementia. Risk factors also include social support, depressive states, family history among first-degree relatives, Down’s syndrome among first degree relatives, and under-activity as a behavioral trait in the past. Van Duijm and Hoffman (1991) suggest a history of Parkinson disease; advanced maternal age, head trauma, and hypothyroidism are considered risk factors for dementia. Henderson and Jorm (1997) further differentiated risk factors for early and late-onset dementia. Early-onset was associated with physical under activity, history of nervous breakdown, starvation, malnutrition, and head injury in the distant past. Late-onset was positively associated
with starvation and malnutrition and negatively associated with long-term use of analgesics.

**Longitudinal Studies**

There have been numerous studies assessing the longitudinal effects of aging creating a broad base of research. The Nun study is an example of one such study group. This group reflects 678 nuns between the ages of 75 and 106. Effects of aging among this group have been studied since the mid-1980’s, establishing differences between healthy and pathological aging, as well as stressing the importance of early identification of preclinical stages of AD (Mortimer, Borenstein, Gosche, Snowdon, 2005; Snowdon, 2003).

The Consortium to Establish A Registry for Alzheimer’s Disease (CERAD) reflects a database that was established by the National Institute on Aging, to assist in the standardization of procedures for evaluation and diagnosis for individuals with AD. In the course of the CERAD study 1094 individuals with AD and 463 non-demented controls were screened, some for a period of seven years. AD has been confirmed by autopsy for 87% of cases studied. While the primary focuses of the CERAD study has been evaluation and diagnosis, supplemental information on the natural history of AD, family histories, behavioral changes, personality changes, neuropsychological, and neuropathological findings have been established (CERAD, 1986).

The Kungsholmen project is a community based longitudinal study located in Stockholm, Sweden. The project was initiated in 1987 and consists of individuals over the age of 75. At the beginning of the study in 1987, 1800 subjects were enrolled and by the 4th follow-up in 2000 there were 265 participants still enrolled. This group was
studied for a period of 13 years with assessments being administered five times throughout the study. The goal of the study was to acquire general knowledge of the aging process from medical, psychological and social perspectives. Findings of the study included documentation of age-related cognitive decline, differences across demographic groups and varying health related concerns; similarities between cognitive decline in AD and Vascular Dementia (VD); and findings supporting a long preclinical period of dementia where decline can be identified (Backman, et al., 2004).

**Rates of Decline**

Existing assessments using the Mini Mental Status Exam (MMSE), Blessed Information Memory Concentration, and cognitive sections of the Cambridge Examination for Mental Disorders of the Elderly (CAMDEX) have been used in establishing rates of decline among dementing and non-dementing groups. Longitudinal investigations such as the CERAD study, showed a progression to severe stage dementia for 54% of cases studied across 3 years. The Kungsholem project showed a progression of severe cases increased from 19% to 58% in 3 years to 78% after 7 years. More specific changes that have been noted across individuals with dementia include a 3-4 point decline on the MMSE each year (APA, 2000). A prospective study with 91 individuals across 11 years by Keene et al., (2001), that life expectancy was limited to 8.5 years after the onset of dementia.

**Reversible/Non-reversible Dementias**

Subtyping the specific form of dementia proves to be critical as some variants of dementia are reversible and underscoring the need for thorough assessment. Reversibility is often a function of pathology, early identification, and application of effective
interventions (APA 2000). Often, an individual may present with an acute confusional state characterized by disturbances of attention, impaired concentration, clouding of consciousness, and disorientation. When these symptoms have developed over a short time, a clinician should search for an organic cause. Frequently, with immediate treatment of the problem, the individual is able to return to previous levels of functioning (APA, 2000). In the absence of immediate treatment, an initially reversible delirium may develop into an irreversible dementia. Current data reflects that this occurs at a rate of 9-25% annually for individuals presenting with acute confusional states (Balis, 1997).

Reversible forms of dementia include endocrinopathies (e.g. hypothyroidism, Addison’s disease), hypercalcemia, metabolic disorders (e.g. vitamin deficiency, diseases of thyroid, parathyroid, and adrenal glands), encephalopathies, electrolyte/water imbalance, hypoxia due to cardiac or pulmonary disease, normal pressure hydrocephalus, carotid artery occlusive, temporal arteritis, cerebral abscess, neurosyphilis, Lyme disease, nutritional disorders, intoxications, infections, and neoplastic diseases, (Balis, 1997; Klag, 1999), and effective treatments are available for all the above listed problems. Disorders of thyroid metabolism are among the more frequently presented reversible dementias. Furthermore, marked indications of normal pressure hydrocephalus are identified by the presence of a clinical triad of symptoms, which include gait disturbance, urinary incontinence, and dementia (Jarvik, 1989).

Drug intoxication may produce signs or symptoms consistent with delirium. The more frequent causes of drug-related dementias include psychotropics, sedatives, and hypnotics. However, late onset of psychotic symptoms may be a sign of dementia, and in
these cases the client may actually benefit from treatment with antipsychotic medication (Jarvik, 1989).

Another form of reversible dementia is sometimes referred to as pseudodementia (Kales & Mellow, 2003; Wells, 1979). Typically this refers to situations in which depression mimics organic forms of dementia. High concordance rate of depression and dementia, make assessment among the elderly difficult. Depression in the general population with dementia ranges from 34 to 80% (Eveinhuis, 1996). Symptoms of depression-induced dementia include: apathy, psychomotor retardation, impaired concentration, occasional confusion, and complaints of memory loss (Jarvik, 1989).

Pseudodementia is typically noted after treatment when decline remits in response to treatment of depression. In some instances, the term pseudodementia may refer to decline secondary to a major psychiatric disorder. Similarly, the decline remits with treatment of the psychiatric disorder (Harper, 1993).

Specific treatments are needed for dementia related to: AD, Huntington's disease, multiple sclerosis, encephalopathies, and multi infarct dementias (Jarvik, 1989). For example, features that indicate the likelihood of multi infarct dementia include: hypertension, previous strokes, abrupt onset, stepwise deterioration, focal neurological symptoms, fluctuating course, and nocturnal confusion. This form of dementia often has an earlier onset and is more common among males, and persons of African American descent (Harper, 1993).

**Dementia Diagnosis/Subtypes**

Because treatment is often hinged on the appropriate diagnosis of dementia, the following section will illustrate identifying and differential features of the commonly
diagnosed forms of dementia. Among the different subtypes of dementia there are
varying rates of decline, requiring different levels of supports and future planning. For
instance, progressive dementia secondary to Alzheimer’s, Vascular disease, or Diffuse
Lewy Bodies determines which interventions are recommended. However, static
dementia due to head trauma or successfully treated Vascular dementia do not require
supporting reductive interventions as, by definition, the uncomplicated course predicts no
further decline (NIH Consensus Development Conference, 1987). Prevalence studies
suggest that Alzheimer’s is the leading cause of dementia, followed by Vascular
dementia as the second most prevalent cause (APA, 2000; Berg, 2003), and both of these
subtypes sometimes are in need of supportive reductive interventions.

Alzheimer’s dementia (AD) was first described by Alois Alzheimer in 1907 and is
currently considered the most prevalent form of dementia. Prevalence rates of AD as a
subtype of dementia have shown a great deal of variability with rates ranging from 50%
to 85% of individuals with dementia diagnosed with this form (Berg, 2003; Klag, 1999).
Alzheimer’s is considered an age-related disorder that is not a typical part of normal
aging. Furthermore, it is considered a leading health problem in developed countries
(Cacabelos, 2002). Current estimates suggest that 25 million individuals worldwide
present with AD, with another 75 million individuals at risk in the next 25 years
(Cacabelos, 1999). Onset is generally gradual with progressive cognitive decline and is
noted after 60 years of age (APA, 2000), but cases have been reported as early as 30
years of age. Early onset (65 year old or younger) is seen in 5-10% of cases (Cummings
& Cole, 2002). Duration of illness may vary from 3 to 20 years until death (Roggia &
Lippa, 2002). Caregiver reports of changes in personality provide predictive value in that
subjects with personality changes (e.g., changes in mood, impulsivity, and loss of inhibition) had twice the likelihood of developing dementia (Smith-Gamble et al., 2002). People that present with late onset decline may have a genetic component, and there is evidence that suggests an increase in prevalence of AD among first degree relatives of greater than 80% (Cacabelos, 1999). Chromosomal links have been noted on chromosomes 14, 19, and 21 (APA, 2000). Postmortem studies reveal three lesions characteristic of AD: neuritic plaques, neurofibrillary tangles, and granuvocular degeneration of neurons (Zigman et al., 2004). Ultimately, the diagnosis can only be confirmed postmortem. Because of the uncertainty in diagnosis, this subtype is to be given only once all other forms of dementia have been ruled out (APA, 2000). While postmortem exams are the expected standard to confirm a diagnosis of AD, it should be noted that established clinical criteria in making the diagnosis of AD has shown an accuracy of 80% when compared to postmortem exams (c.f. Lanska & Schoenberg, 1993). Once dementia with prominent amnestic features are seen, there is little difficulty in making an accurate diagnosis. Diagnoses are made based on the type of symptoms, the progression of symptoms over time, and the absence of other causative factors (Salmon & Hodges, 2001). Standard rule-out diagnoses include central nervous system disorders (e.g., cerebrovascular disease, Parkinson’s disease, Huntington’s disease), systemic conditions known to cause dementia (e.g., hypothyroidism, vitamin deficiencies, HIV infection), or persisting effects of a substance (e.g., Korsakoff’s syndrome) (APA, 2000).

Vascular dementia (VD) is considered the second leading cause of dementia, accounting for 20-30% of all cases (Berg, 2003; Ford et al., 2003). VD typically affects people between ages of 60-75 years of age (Alexoportious, 2003; Klag, 1999). When
considering dementia due to a vascular disorder, there must be evidence of a cerebrovascular disease that is judged to be related to the dementia. Evidence of VD is often demonstrated with CT and MRI tests showing lesions in cerebral and cortical structures or EEG findings reflecting focal lesions (APA, 2000). Typically, one year after a stroke 25% of patients develop dementia. For those who do not develop dementia, their relative risk of developing dementia in the future is 5.5 times more likely compared to individuals without a history of stroke (Madureira, Guerreiro, & Ferro, 2001). Focal neurological signs include extensor plantar response, pseudobulbar palsy, gait abnormalities, exaggeration of deep tendon reflexes, or weakness of an extremity (Nussbaum, 1997). Vascular dementia is differentiated from AD by a more sudden onset compared to the gradual onset associated with AD (Jarvik, 1989). In VD, sudden onset is followed by a fluctuating course of decline characterized by rapid changes, often referred to as a stepwise progression (APA, 2000). Another distinguishing feature of VD as opposed to AD is that risk of VD can be prevented with early treatment of vascular disease and ongoing treatment may minimize or prevent ongoing decline secondary to VD. This condition is associated with focal neurological signs, and commonly hypertension is associated. Early treatment of hypertension and vascular disease may prevent further progression (APA, 2000). Genetic components further contributing to vascular dementias include hyperlipidemia and hypertension (Jarvik, 1989). Overall, men show greater risk for VD than women (Klag, 1999).

The final broad category is dementia due to a general medical condition. For this diagnosis to be made there must be evidence from history, physical examination, or laboratory findings to support a related diagnosis of dementia. Specific subtypes of this
area include: HIV infection, traumatic brain injury, Parkinson’s disease, Huntington’s
disease, Pick’s disease, Creutzfeldt-Jakob disease, normal pressure hydrocephalus,
hypothyroidism, brain tumor, or vitamin B12 deficiency (APA, 2000). When considering
diagnosis of dementia secondary to head trauma, degree and type of cognitive
impairments or behavioral disturbances are directly related to the location and the extent
of injury.

Pick’s disease (commonly listed as Frontal Lobe Dementia) is a degeneration of
the brain that specifically affects the frontal and temporal lobes, and typically affects
people between 40-65 years of age (Riley, 1999). This disorder was first described by
Ludwig Pick in the early part of the twentieth century. Previously, researchers estimated
that Pick’s disease represented 5% of all dementia cases; however, more recent studies
report an 8% prevalence rate across dementias (Ford et al., 2003). Due to the influence of
the frontal lobes, there are changes in personality and social skills early in the course of
the disease (Kertesz, 2000). One may expect deterioration of social skills, emotional
blunting, behavioral disinhibition, and prominent language abnormalities. As the illness
progresses, difficulties with memory may be noted as well as apraxia. Eventually, the
primitive reflexes (snout, suck, grasp) may be released and readily elicited. Throughout
the course of the disease, apathy and/or agitation may be noted. (APA, 2000) Pick’s
disease is differentiated from Alzheimer’s disease by an autosomal dominant mode of
inheritance. In addition, morphological postmortem studies show a presence of abnormal
cell inclusions and an absence of the neurofibrillary tangles and plaques seen in
Alzheimer’s disease (Jarvik, 1989).
Creutzfeldt-Jakob (C-J) is another subtype of dementia disease of particular recent interest because of its association with Bovine Spongiform Encephalopathy (also known as BSE; and Mad-Cow disease) (Colinge, 2005). The disease follows a quick progression and death may occur within a year. Also, there is concern of infecting caregivers or other family members as this is a sub-acute spongiform encephalopathy caused by abnormal protein particles, once called a “slow virus”, but now recognized as self reproducing particles even simpler than viruses, and called prions. The presentation of this disorder is a dementia accompanied by involuntary movement and periodic EEG activity. This disease may develop at any age in adults, but is more commonly noted between the ages of 40-60 years. Five to 15% of cases have a familial component consistent with autosomal dominant mode of inheritance (APA, 2000). Familial cases may be due to an infection superimposed on a preexisting genetically determined dysfunction (Jarvik, 1989). Prodromal symptoms include fatigue, anxiety, change in appetite, sleep disturbance, and poor concentration. These symptoms may be followed (within several weeks) with incoordination, altered vision, or abnormal gait. C-J is a rapidly progressive dementia that can progress to death in as little as several months (APA, 2000). Other associated symptoms include myoclonus, cerebellar ataxia, and seizures. Interestingly, C-J often is considered a human variant of mad cow disease (APA, 2000), and humans suffering from C-J infections where the vector of infection is infected meat are said to have CJD variant (Collinge, 2005).

Huntington’s disease is an inherited degenerative disease linked to chromosome 4 with disturbances of cognition, emotion, and movement. The disorder is usually diagnosed in the late 30’s to early 40’s but may begin as early as age 4 or as late as age
Onset is often noted by changes in behavior and personality (e.g., depression, irritability, anxiety). Some individuals present with abnormalities of movements that resemble increased fidgeting that later progress into generalized choreoathetosis. Deficits in memory retrieval, executive functioning, and judgment are common. Disorganized speech and psychotic features also may be present. Late in the disease “boxcar ventricles” may be seen on structural brain imagery (APA, 2000), suggesting pronounced brain atrophy and corresponding ventricular enlargement.

Parkinson’s disease leads to another form of dementia. This degenerative condition is the result of a slow progressive neurological process characterized by tremor, rigidity, bradykinesia, and postural instability (Bondi, Salmon, & Kaszniak, 1996). Dementia associated with Parkinson’s disease is noted in 20-60% cases and is characterized by cognitive and motoric slowing, executive dysfunction, and impairment in memory retrieval (APA, 2000). Onset is more common in the advanced stages of the disease process (APA, 2000), but can been seen as early as age 40, with a peak onset in the sixth decade (Bondi, et al., 1996). The source of dementia is controversial, considered by some to be due to effects of subcortical degeneration (Bondi, et al., 1996). These symptoms are often exacerbated by depression (APA, 2000).

The Lewy Body subtype of dementia was identified in the 1980’s and has been briefly discussed in the DSM-IV TR (APA, 2000). Researchers suggest that this is possibly the second leading cause of dementia and may account for 11% of all cases of dementia (Ford et al., 2003). Lewy bodies (round deposits occurring within damaged nerve cells) have been found in postmortem studies of both patients suffering from Alzheimer’s and Parkinson’s disease. Studies indicate that between 10-20% of elderly
demented patients present with Lewy bodies (Burns et al., 1990; Dickson, et al., 1991; Hansen, et al., 1990; Koska, 1990; Perry et al., 1990). It is thought that dementia of the Lewy body type has been misdiagnosed as Alzheimer’s disease or Parkinson’s disease in the past. The primary characteristic of the disease is the development of dementia that mirrors features of both Alzheimer’s and Parkinson’s disease. Unique clinical features include impaired attention, disproportionate impairment of problem solving, visuospatial difficulties, and frontal lobe dysfunction. Recent literature suggests depression and REM sleep disturbance may be additional features supportive of a diagnosis of dementia of the Lewy body type (PubMed, 1996; 1999). Symptoms of this disorder seem to fluctuate across hours, and over weeks and months. Variability in functioning often will lead caregivers to question whether the individual is deliberately influencing symptoms. Overall, there have been limited studies regarding progression of cognitive impairment related to Lewy Body dementia; however, the preponderance of these studies suggest that individuals with Lewy Body dementia decline more rapidly than individuals with Alzheimer’s (Ballard, O’Brien, & Morris, 2001). The presence of apolipoprotein E4 allele is associated with greater progression of cognitive deficits because they have chronic brain disease and AD. One study found a mean annual decline of 17.5 points for individuals with apolipoprotein E4 allele as compared with a mean decline of 8.3 on the Mini Mental State Exam for individuals who were negative for apolipoprotein E4 (Ballard et al., 2001). Frequently, there is the presence of hallucinations and Parkinsonism without a resting tremor. Parkinsonian features include: flexed posture, shuffling gait, reduced answering, tendency to fall, paucity of spontaneous movement, and tremor. However, it should be noted that tremor is the least common feature for
those who first present with dementia. Myoclonus (twitching or spasm of a muscle or group of muscles) is also common and is usually mild, spontaneous and multifocal. If myoclonus is prominent early in the disease there is concern of C-J disease (PubMed, 1996; 1999). Individuals with extrapyramidal symptoms and visual hallucination may decline more rapidly (Ballard, et al., 2001).

The treatment of Lewy Body dementia is a lose-lose situation. Pharmacological treatment of the movement disorder worsens cognitive decline and the presentation of psychotic symptoms, while treatment of the psychotic symptoms may result in physical immobilization (McKeith, et al., 1992; 1997). Ultimately, there is no therapy that can yet stop or substantially suppress the process of neurodegeneration of this form of dementia. Studies by McKeith et al., (1992) (c.f. Ince, Perry, & Morris, 1998) indicated that half of all patients with this form of dementia who are exposed to neuroleptics experienced severe adverse drug reactions which included deterioration in cognitive functioning, Parkinsonism, drowsiness, and features of neuroleptic malignant syndrome. These patients have a three-fold increase in mortality compared with those not exposed to the drug. McKeith et al., (1997) (c.f. Ince, et al., 1998) noted negative responses might occur with atypical neuroleptic drugs. Various researchers noted that neuroleptic use with Lewy Body dementia as compared with Alzheimer’s patients resulted in severe reactions within two weeks of neuroleptic administration or dose change. Neuroleptic use has also been associated with a reduction in survival for Lewy Body patients (PubMed, 1996; 1999).

In summary, dementias associated with primary dysfunction of the cerebral cortex (e.g., AD and Pick’s) often produce aphasias, amnesias, apraxia, agnosia, and impaired judgment and insight (Harper, 1993). Cummings and Benson (1983) note cognitive
processes slow gradually, forgetfulness appears, and concurrent affective disorders are not uncommon. In the early stages, personality style and behavioral patterns are relatively well-preserved in cortical dementias. Disinhibition and increases in behavioral impulsivity also are recognized features later in the disease process (Harper, 1993). In subcortical dementias, which include Vascular, movement disorders (i.e., Huntington’s Chorea and Parkinson’s disease), psychiatric disorders, hydrocephalus, metabolic, and toxic based dementias, individuals generally remain free of motor system involvement until late in the disease. Metabolic and toxic dementias share features of acute confusional states (such as fluctuating arousal, slowness, hallucinations), and may last for many weeks or months. The intellectual impairments associated with toxic and metabolic conditions resemble those observed in subcortical brain structures (i.e., slowing in responsiveness, cognitive declines, and movement disorders). Rigidity, tremors, and myoclonus are not uncommon in toxic and metabolic dementias (Cummings & Benson, 1983).

**Behavior Change**

Behavior changes are common among individuals with dementia. These changes are thought to be the result of the combined effects of brain damage and the external environment (Mace et al., 1993). These behavior changes are not limited to decline in adaptive behaviors, but also to changes in personality and the development of problem behaviors. Commonly identified problem behaviors include: wandering, irritability, disruptive nocturnal behaviors, aggression, non-compliance, inappropriate sexual behaviors, and repetitious/preservative behaviors (Keene et al., 2001; Knopman, et al., 1998; Walsh et al., 1990). In addition to these issues, loss of coordination and motor
Skills often result in a diminished ability to perform activities of daily living. Researchers suggest that the development of behavior problems, the loss of ambulation, and caregiver distress often result in institutionalization for the individual presenting with dementia (Cacabelos, 2002; Keene et al., 2001; Mace et al., 1993). In a prospective study by Keene et al., (2001), 75% of the 91 subjects were institutionalized during the course of 11 years. Ninety percent of these individuals presented with significant behavior problems.
INTELLECTUAL DISABILITIES AND DEMENTIA: OVERVIEW

Janicki, Heller, Seltzer, and Hogg (1996) define dementia among individuals with ID as a “progressive and invariant decline in adaptive, cognitive, social, and physical functioning”. This definition is different from standards set forth in the DSM-IV TR as deficits are tied to adaptive behaviors as well as cognitive functioning and does not specifically identify aphasia, apraxia, agnosia, or disturbance of executive functioning (APA, 2000). Janicki et al., (1996) further delineated dementia among the ID population into three primary stages of decline (early, mid, and late). In the first stage, cognitive impairment includes memory decline, temporal disorientation, reduced motor output, attentional decline, apathy, and reduced social output. The second stage is characterized by decrements in instrumental skill functions, declines in self-care routines, emergence of extra-pyramidal motor signs, and development of seizures. In the final stage, there is progressive motor involvement, loss of ambulatory capacity, and incontinence (Slomaka & Berkey, 1997). It should be noted, however, that the identification of aphasia, agnosia, or disturbed executive function is not insurmountably challenging when the neuro-diagnostician is confronted with persons who present with declines in adaptive, cognitive, social and physical functioning, and as such, the apparent definitional dispute between Janicki & DSM-IV may be more of words than substance.

Prevalence

Prevalence rates of dementia among the ID population have shown variability across studies and have been a source of debate (Harper, 1993; Haveman, Maaskant, & Sturman, 1989; Hewitt & Fenner 1986; Janicki & Dalton, 2000; Reid & Aungle, 1974) (See Table 2). Some researchers contend that the ID population is subject to higher
prevalence rates of dementia than the general population (Hewitt & Fenner 1986; Reid & Aungle, 1974; Slomaka & Berkey, 1997). In comparison to the general population with rates ranging from 1-15% after age 65, the ID population is shown to have prevalence rates ranging from 6-15% (Cacabelos, 2002; Cooper, 1997; Janicki & Dalton, 2000), which suggests there is not a notable difference in prevalence rates across the groups.

Table 2
Comparison of Prevalence Rates Across Diagnostic Groups and Age

<table>
<thead>
<tr>
<th>Age</th>
<th>General Population</th>
<th>ID Population</th>
<th>Down’s Population</th>
<th>Reference</th>
</tr>
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<tbody>
<tr>
<td>45</td>
<td>---</td>
<td>7%</td>
<td>---</td>
<td>Reid &amp; Aungle, 1974</td>
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<tr>
<td>&gt;50</td>
<td>&lt; 1%</td>
<td>---</td>
<td>36%</td>
<td>Fisher &amp; Ketti, 2005</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>50%</td>
<td>Thompson, 2001</td>
</tr>
<tr>
<td>60-69</td>
<td>2%</td>
<td>---</td>
<td>54%-56%</td>
<td>Prasher, 1995</td>
</tr>
<tr>
<td>&gt;65</td>
<td>1-15%</td>
<td>13%</td>
<td>---</td>
<td>Cacabelos, 2002</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Harper, 1993</td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td>Reid &amp; Aungle, 1974</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Janicki &amp; Dalton, 2000</td>
</tr>
<tr>
<td>65-74</td>
<td>6%</td>
<td>15%</td>
<td>67%</td>
<td>Cooper, 1997</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Fisher &amp; Ketti, 2005</td>
</tr>
<tr>
<td>75-84</td>
<td>---</td>
<td>23%</td>
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<td>Cooper, 1997</td>
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<tr>
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<td>---</td>
<td>Insel &amp; Badger, 2002</td>
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<tr>
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<td>---</td>
<td>18%</td>
<td>15-51%</td>
<td>Hewitt &amp; Fenner, 1986</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Lott et al., 2002</td>
</tr>
<tr>
<td></td>
<td></td>
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<td></td>
<td>75%</td>
</tr>
<tr>
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<td>Watchman, 2003</td>
</tr>
<tr>
<td>Tabular</td>
<td></td>
<td>13%-23%</td>
<td>22%</td>
<td>50%-56%</td>
</tr>
</tbody>
</table>

These findings corroborate assertions made by Zigman et al., (2004), which suggest that the incidence and prevalence rates among the developmentally disabled are consistent with the general population. However, prevalence studies across individuals 75 years of age and older suggest a possible divergence from these findings. In comparison, prevalence studies for the general population over the age of 75 yield rates of 16-64%
(APA, 2000; Cacabelos, 2002; Insel & Badger, 2002; Klag, 1999), while the ID population yielded rates of 23-70% with increasing prevalence rates noted a decade in advance of the general population (Cooper, 1997). While features affecting prevalence rates may be attributed to the methodology of each individual study, it is clear that continued research in this area is needed. The increases in prevalence rates across successive decades also suggest a need to examine the course of decline among the two populations as there may be a critical time of onset that has yet to be established among the ID population.

A separate consideration when investigating dementia among the ID population is the presence of Down’s syndrome. There has been heightened interest in dementia among individuals with Down’s syndrome for the last thirty years (Lott 1992; 1986; 1982). The majority of prevalence studies suggest that individuals with Down’s syndrome present with a heightened risk for the development of dementia (APA, 2000; Fisher & Ketti, 2005; Lott, Osann, Doran, & Nelson, 2002; Prasher, 1995), with some suggesting that all individuals with Down’s will present with features of dementia if they survive into the upper ranges of old age (Lott & Head, 2001; Watchman, 2003). Onset of dementia is noted as early as the third decade for this group (Evenhuis, 1990; Lai & Williams, 1989) with prevalence rates ranging from 36-56% (Fisher & Ketti, 2005; Prasher, 1995; Thompson, 2001) after the age of 50, in comparison to rates of 1-15% for the general population, and the general ID population after the age of 60 (Cacabelos, 2002; Cooper, 1997; Harper, 1993). Lifetime prevalence rates for the Down’s subgroup, is reported as high as 75% (Watchman, 2003). Postmortem studies have yielded parallels in brain pathology identifying AD dementia in the group (Lott & Head, 2001; Thase, 1982;
Wisniewski, 1990; Zigman et al., 2004), yielding increased interest in the contributing factors to chromosomal 21 deficiencies to AD (APA, 2000; Lott & Head, 2001). Additionally the rapid progression of AD in this group has resulted in this group becoming a model for research for AD among the general population (Lott, 1992; 1986; 1982; Lott & Head, 2001; Slomaka, & Berkey, 1997).

When researching dementia among the general ID population, one must consider the existing parallels between the ID population and the general population, as well as the potentially confounding features presented by individuals with Down’s syndrome. Specifically, advances in health care have shown an overall increase in mean life expectancy for all groups (Bush & Beaill, 2004). It is estimated that with aging baby boomers and improvements in health care that the overall number of individuals with dementia will continue to increase across time. This concern holds true for those with intellectual disabilities. Today, younger adults with ID are expected to live as long as individuals in the general population (life expectancy 76.9 years) (Horwitz, Kerker, Owens, & Zigler, 2005). While life expectancy for the Down’s population is not equivalent to the general population, there have been notable advances as illustrated by Holland (2003). This report suggests that for the Down’s subgroup life expectancy has extended from 10 years of age in the 1930’s to over 60 years of age at present (Holland, 2003). Although Down’s syndrome is the most common single cause of ID in America (Fisher & Ketti, 2005; Hook, 1982), it remains a subset of ID and represents a fraction of the ID population that presents with needs secondary to cognitive decline (Zigman, Schupf, Haveman, & Silverman, 1997). Due to the heightened risk factors for dementia
among the Down’s population, dementia studies among the ID population should control for the presence of Down’s syndrome so results are not skewed or confounded.

**Risk Factors**

Research suggests that etiology, level of mental retardation, as well as moderating psychosocial influences predict mortality risk in the elderly (Slomka & Berkey, 1997). Individuals with developmental disabilities who are at risk for developing dementia include those over the age of 50 years and individuals with Down’s syndrome who are over the age of 40 (Janicki, Heller, Seltzer, & Hogg, 1996). The most salient risk factor for dementia among persons with ID is the presence of Down’s syndrome (Bush & Beaill, 2004; Prasher, Chowdury, Rowe, & Bain, 1997). Dementia related to the presence of Down’s syndrome will be reviewed separately in the following section, as this group is only a subset of the individuals being considered in this research.

The ID population is similar to the general population, with increasing age being established as a more salient predictor for dementia (Aylward, Burt, Thorpe, Lai, & Dalton, 1995; Schultz, et al, 2004). The presence of apolipoprotein E-4 and usually high numbers of Lewy Body inclusions are two other risk factors. Other factors include family history, head injury (Bondi, Salmon, & Kasznaik, 1996), gender, oxidative damage, and premorbid levels of mental retardation (Bush & Beaill, 2004). Eveinhuis (1996) further suggests prevention and intervention of psychiatric symptoms may further influence risks of dementia.

Prevalence estimates of psychiatric disorders among people with ID range from 20-40% (Pary, 2002). Additionally, more than two thirds of individuals in this
population are over the age of 55 and receive psychotropic medications (Pary, 2003), providing a large target group at risk for dementia.

For individuals with static etiologies of mental retardation (e.g., post traumatic development, infection, anoxia), their risk factors are reported to be similar to those of the general population. Whereas, in cases with progressive syndromes (e.g., congenital conditions, errors in metabolism, neurodegenerative processes), the syndromes themselves become predictive factors of progressive central nervous system decline resulting in dementia (Slomka & Berkey, 1997).

**Dementia and Down’s Syndrome**

Risk of AD is increased in the presence of Down’s syndrome. Postmortem studies reveal early signs of similar brain pathology among individuals with Down’s syndrome by the age of 40 (Thase, 1982; Zigman et al., 2004). Although clinical presentation may not be evident until later in life (Carr, 2003), noted brain atrophy is demonstrated in CT and MRI studies (APA, 2000). Findings include the presence of wider cortical sulci, and larger cerebral ventricles (APA, 2000). Research by Lott and Head (2001) indicates the presence of “Trisomy 21 leads to dose-dependent increase in the production of the amyloid precursor protein and subsequently the production of the amyloidogenic fragments leading to early and predominant senile plaque formation”. These proteins are noted in the third decade (Hof et al., 1995; Hyman et al., 1995) and increase exponentially after age 35 (Wisniewski, 1990). By the sixth decade, additional neuropathological features of AD including neurofibrillary tangles and neural cell loss are evident (Mann, 1993). Meta analysis by Deb et al., (2000) also concluded that there
was significantly higher frequency of E-4 alle of the apo-lipoprotein gene among the Down’s group studied.

Individuals with Down’s syndrome are also at risk for reversible forms of dementia secondary to the presence of hypothyroidism. Thyroid abnormalities contribute to the presence of reversible dementias among the general population and are more common in those with ID. Rates of hypothyroidism are noted at 49% among those residing in institutions (Van Buggenhout et al., 1999). Additional research suggests poor screening of thyroidism among the mentally retarded, reporting 50% of individuals Down’s syndrome never had a thyroid test (c.f. Fisher & Ketti, 2005).

**Rate of Decline**

Janicki and Jacobson (1986) empirically determined that decline in the ID population typically began within the mid-fifties and further suggested that individuals functioning in the community were at less risk and were able to maintain skills for longer periods of time than were individuals in an institutional setting. Among individuals with Down’s syndrome, decline has been noted as early as the third decade (Holland, 1999). Pathological aging consists of emotional and behavioral disturbances, decreases in motivation, appearance of depressive symptoms, decreases in daily living skills, disturbances in gait, increases in motor disturbances (potentially accompanied with primitive repetitive behaviors), and onset of seizures prior to the age of sixty (Lott, et al., 2002). The progression of decline has been identified across three stages. Stage one includes initial deterioration of cognitive abilities. In the second stage of decline, there is expected loss of over-learned behaviors and decline in social and adaptive skills. The final stage of decline is associated with a more accelerated rate of neuro-degeneration and
neurofibrillary tangle formulation (Silverman, Schupf, Zigman, et al., 2004; Slomka & Berkey, 1997). Much like Alzheimer’s disease, for individuals with Down’s syndrome, neuropathological aging is associated with loss of acetylcholine, neuronal loss, senile plagues, neurofibrillary tangle formulation, and reduced cerebral metabolism. The key differences between Alzheimer’s in the general population and dementia among individuals with Down’s syndrome are the rates of disease progression and the average age of onset (Slomka & Berkey, 1997). Mortality is expected between 4.9 years and 5.2 years following initial diagnosis (Eveinhuis, 1990).

While there are numerous studies detailing the ID population (more specifically the Down’s population) as an at risk group for early onset dementia, not all individuals develop dementia (Das & Mishra, 1995; Devenny & Krinsky-McHale, 1998; Holland, 1998; Zigman et al., 1987). Moss, Hogg, and Horne (1992) noted age related decline is not an inevitable consequence of aging. Among 122 adults with an intellectual disability over the age of 50 years, there were no significant differences in daily living skills across cohorts of 50 to 74 years of age and 75 years and older. In contrast to this study, Brown, Greet, Aylward, and Hunt (1990) note there are significant declines in social and adaptive skills with increasing age. Various researchers suggest that skill deficits occur in healthy adults as a function of aging (Huppert, 1994; Petersen et al., 1991; Wingfield & Stine, 1989).

As with the general population there are also concerns for early identification of cognitive decline, as there have been positive treatment effects with medications (Lott & Head, 2001). Devenny, Zimmerli, Kittler, and Krinsky-McHale (2002) suggested the possibility of identifying mild cognitive impairment based on cued recall memory
assessments among individuals functioning within the mild and moderate ranges of ID. Janicki and Jacobson (1986) sampled a population of 10,532 individuals and noted earlier general decline was associated with motor skill decline for individuals functioning within the severe and profound levels of mental retardation. This is especially relevant as differential diagnosis of dementia is difficult to determine when intellectual functioning is estimated to be less than an IQ of 25 (Harper, 1993).

Various studies among the Down’s population suggest prior to clinical symptoms of dementia there is no decline in adaptive skills (Prasher & Chung, 1996; Prasher, Chung, & Haque, 1988; 1998). Considering research suggesting consistent presence of features of AD in postmortem studies for individuals with Down’s (Lott 1992; 1986; 1982), it is surprising that there is an absence of notable cognitive decline throughout the aging process with this sub-group. Studies among the general population suggest that features of AD may develop 20 years prior to diagnosis (Slomaka & Berkey, 1997). The above findings illustrate a need for longitudinal assessment with sensitive measures to establish differential rates of cognitive decline across dementia and non-dementia groups with ID. As the presence of dementia and level of functioning predict decline in adaptive skills (Prasher, Chung, & Haque, 1998) opportunities for early intervention need to be established.

From the existing literature on rates of decline, Janicki and MacEachron (1984) reported decline in daily living skills and mobility were reflective of aging and most notable among individuals with severe ID and among the age cohort of 73-99. Schupf et al., (1989) support the finding of decline secondary to aging and further indicated decline was three to four times greater among individuals with Down’s syndrome. Collacott
(1992) reported that deterioration occurred across all domains of the AAMR ABS among individuals with Down’s syndrome between the ages of 50 and 59 years.

Table 3
Review of Sample Sizes Across Age Groups for Cross Sectional and Longitudinal Designs Among the ID Population

<table>
<thead>
<tr>
<th>Participants</th>
<th>Years</th>
<th>Ages</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cross Sectional</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>34</td>
<td></td>
<td>34-70</td>
<td>Miniszek, 1983</td>
</tr>
<tr>
<td>40</td>
<td></td>
<td>40+</td>
<td>Hauber, et al., 1985</td>
</tr>
<tr>
<td>23</td>
<td></td>
<td>50-61</td>
<td>Hewitt, et al., 1985</td>
</tr>
<tr>
<td>63</td>
<td></td>
<td>20-85</td>
<td>Linter, 1986</td>
</tr>
<tr>
<td>70</td>
<td></td>
<td>65+</td>
<td>Barcikowska et al, 1989</td>
</tr>
<tr>
<td>122</td>
<td></td>
<td>50+</td>
<td>Moss, et al., 1992</td>
</tr>
<tr>
<td>Longitudinal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>198</td>
<td></td>
<td>20-69</td>
<td>Schupf et al., 1989</td>
</tr>
<tr>
<td>109</td>
<td>4</td>
<td></td>
<td>Rasmussen et, al.</td>
</tr>
<tr>
<td>34</td>
<td>3</td>
<td>22-56</td>
<td>Burt et al. 1995</td>
</tr>
<tr>
<td>128</td>
<td>3</td>
<td>16-72</td>
<td>Prasher et al., 1998</td>
</tr>
<tr>
<td>23</td>
<td></td>
<td>50+</td>
<td>Hewitt, et al, 1985</td>
</tr>
<tr>
<td>57</td>
<td>5</td>
<td>17-71</td>
<td>Prasher et al., 1998</td>
</tr>
<tr>
<td>17</td>
<td></td>
<td>40+</td>
<td>Eveinhuis, 1990</td>
</tr>
<tr>
<td>70</td>
<td>7</td>
<td></td>
<td>Kittler, et al., 2004</td>
</tr>
<tr>
<td>48</td>
<td>4</td>
<td></td>
<td>Prasher et al., 2004</td>
</tr>
<tr>
<td>14</td>
<td>10</td>
<td></td>
<td>Ikeda &amp; Arai, 2002</td>
</tr>
<tr>
<td>66</td>
<td>4</td>
<td></td>
<td>Devenney et al, 2000</td>
</tr>
<tr>
<td>67</td>
<td>4.5</td>
<td></td>
<td>Roedden, et al, 1997</td>
</tr>
<tr>
<td>46</td>
<td>5</td>
<td>27-55</td>
<td>Devenney et al., 1992</td>
</tr>
<tr>
<td>22</td>
<td></td>
<td>27-64</td>
<td>Johanson et al., 1991</td>
</tr>
</tbody>
</table>

Note table partial adaptation from Prasher, 1998 pgs. 166-168.

Recent research on rates of decline among the ID population has made general comparisons across individuals with and without Down’s syndrome, as well as between individuals above and below the age of 50. However, there has been a paucity of longitudinal studies reviewing sequential decline across individual years.

One such study by Rasmussen and Sobsey (1994) reviewed retrospective data of adaptive skills with a cross sectional and longitudinal design. Findings of this study suggested there were no significant differences in adaptive skills. Limitations of this study cited by
Prasher (1999) were that few of the individuals in the study were over 50 years of age, thus creating a population too young to establish notable decline. Overall, there is a need for clinically sensitive assessment to establish sequential rates of decline for dementing and non-dementing individuals who present with ID.

While there have been a limited number of studies assessing age-related decline of adaptive behavior (Prasher, 1996), researchers have found particular utility in longitudinal designs (Dalton et al., 1999; Devenny et al., 1996; Haxby & Schapiro, 1992; Oliver et al., 1998). In previous studies of dementia among individuals with ID, sample sizes ranged from 22 to 198 (Holland, 1999). Studies with geriatric ages have ranged from samples of 17 to 63 for individuals 40 years old and above (Holland, 1999; Prasher, 1999). Longitudinal studies have ranged from 3 to 10 years (Prasher, 1999). Utility has been found in studies with smaller sample sizes as they allow for more detailed assessments with more “hands-on” data collection (Prasher, 1999).

**Clinical Assessment**

Dementia screenings may be facilitative in identifying factors of decline that may be chronic versus reversible such as depression, thyroidism, etc. (Janicki, et al., 1996). Identifying dementia among individuals with ID is difficult, as cognitive impairments exist prior to age-related decline (Schultz et al., 2004). Dementing disorders have to be assessed in light of pre-existing impairment (Burt & Aylward, 2000). Decline in functioning for individuals with low cognitive functioning is identified through combined direct and indirect assessments (Aylward et al., 1995; Burt & Aylward, 2000), as well as by noting changes from baseline in adaptive, emotional functioning and/or the onset of physical changes (Harper, 1993; Varnhagen et al., 1987). The presence of floor effects
and difficulty in testing the limits impedes interpretation of single assessment screening tests (McDaniel, Foster, Compton, & Courtney, 1998). Combination assessments are noted as more useful among both the general population and the developmentally disabled populations (Mackinnon & Mulligany, 1998; Strydom & Hassotis, 2003). Recommendations are that assessments be administered longitudinally with a baseline prior to age 40 and follow up every 1-5 years once dementia is suspected (Bush & Beail, 2004). As there is no one assessment instrument or battery (Silverman et al., 2004; Watchman, 2003) and there is difficulty distinguishing between cognitive declines associated with normal aging versus more rapid or large declines tied to dementia (Crayton, Oliver, Holland, Bradbury, & Hall, 1998), it is important to have a clear standard of decline when considering diagnosis.

Diagnosis of dementia should only be made when substantial change has been noted over time or can be inferred with some confidence (Silverman et al., 2004). Issues to be considered when making a diagnosis include changes from baseline functioning for the individual rather than cutoffs typical for general population, perception of change should be based on premorbid functioning and demands noted in everyday life, and changes over time should exceed what is expected for typical aging adults who have mental retardation (Aylward, Burt, Thorpe, Lai, & Dalton, 1997). Traditional intelligence tests typically underestimate decline because they are not sensitive to individuals with reduced abilities (Watchman, 2003).

As treatments are tied to diagnosis, diagnostic accuracy is essential (Jarvik, 1989). A diagnostic evaluation geared toward the assessment of dementia should include a detailed medical history (e.g., history medication use, past and present illnesses, previous
medical treatments), and checking for family history of dementia. The goal of the assessment is to determine whether there has been a progressive deterioration of skills (Janicki, et al, 1996). Identification of progressive decline is often accomplished by the review of longitudinal data from behavioral and clinical assessments of cognitive and adaptive skills (Harper, 1993; Slomka & Berkey, 1997). Such information assists in ruling out other causes of decline (e.g. psychiatric fluctuations) (Janicki, et al, 1995). A formal assessment should include information from multiple informants and behavioral observations across multiple settings. Additional referrals should be made when appropriate to rule-out differential diagnoses (Janicki, et al, 1996). Longitudinal assessments aid in documenting and extrapolating the course of illness, which further facilitates determining the appropriate dementia subtype (Gurland & Cross, 1987).

Assessment information suggests that in the early stage of dementia the most obvious failings are in the area of immediate memory. In individuals functioning below the moderate range of intellectual functioning, this may be difficult to assess as they often possess limited verbal skills and their responses may be variable across assessment (Harper, 1993). Early signs may be expressed as a failure to carry out normal daily routines or previously completed simple tasks. Towards the end stage of dementia, memory loss is frequently global and is followed by general incontinence, and behavioral deterioration. As the deterioration continues, seizures may develop and death is typically the result of infection or cardiovascular disease. Within the Down’s population, isolated psychotic episodes may indicate the beginning of an organic form of dementia (Harper, 1993).
Behavioral Problems

Behavioral problems among individuals with ID, is relatively common and occurs between 4-22% percent across the population (Harris, 1996; Schroeder, Rojahn, Oldenquist, 1989). Often, these problems are related to communication deficits. Given these considerations, it is not surprising that behavioral excesses occur in the presence of dementia for individuals in the general population as receptive and expressive skills often decline (Keene et al., 2001; Knopman, et al., 1998; Walsh et al., 1990). The onset of behavioral problems are increasing incidence of behavioral problems among an aging ID population can be considered a hallmark of dementia and or seen in the early and middle stages of AD (Devenny et al., 2002). Fisher and Ketti (2005) have indicated utility in treating cognitive decline among the ID population, but did not find relative improvement in behavioral symptoms. Although behavioral issues may require separate treatment, development of these problems may be indicative of the presence of dementia.
PURPOSE/HYPOTHESES

Nearly all research investigating relationships between developmental disabilities and dementia is relatively recent. Numerous studies examine the parallels between Down’s syndrome and Alzheimer’s disease (Hyman, West, Rebeck, Lai, & Mann, 1995; Lott, & Head, 2001; Watchman, 2003; Weigiel, Wisniewski, Dziewiatkowski, Popovitch, Tarnawski, 1996), as well as the methodology for diagnosing dementia among individuals with ID (Burt & Aylward, 1998; Janicki, Heller, Seltzer, & Hogg, 1996). Despite this research, relatively little is known regarding genetic susceptibilities, environmental variables, and improving the quality of life for those afflicted. Furthermore, the course of the disease has yet to be studied among individuals with ID.

Individuals functioning within the severe and profound ranges of ID represent the smallest percentage of individuals with ID across functioning levels and they are among the most difficult to assess. Barriers in assessment among individuals with severe and profound ID include deficits in communication and cognitive abilities. The absence of personal verbal reports makes it difficult to assess mental state and cognitive deficits often resulting in failure to achieve a basal level on many traditional tests of intellectual and neuropsychological functioning (Aylward, Burt, Thorpe, Lai, & Dalton, 1995; Burt & Aylward, 1998). Establishing patterns of change across assessments standardized with the ID population, and measuring social skills, adaptive behaviors and negative behaviors can aid in diagnosis as well as define course of decline. While course changes in adaptive measures are anticipated in the diagnosis of dementia among individuals with ID, they may lack sensitivity in identifying early onset decline. One measure to assist with this is the Matson Evaluation of Social Skills for the sEverely Retarded (MESSIER) (Matson,
1995). The MESSIER evaluates a wider range of social skills (Matson et al., 1998) compared to the VABS, and also includes a survey of negative behaviors that may assist in the early identification of diagnosis of dementia, given that increases of problem behaviors are often coupled with diagnosis of dementia (Keene et al., 2001; Knopman, et al., 1998; Walsh et al., 1990).

Assessment efforts to address the barriers resulting from communication deficits include the use of both direct and indirect assessment. Direct assessments include responses provided by the individual being assessed, performance of skills, and behavioral observations. When individuals lack the ability to provide verbal responses, mental status assessments often include observations of affect, review of mealtime data to assess appetite, and review of sleep charts to assess disturbance in sleep. However, these forms of data are not readily available and clinicians have to rely on indirect assessments. Indirect assessments include structured and unstructured interviews with the family members or caregivers who are most familiar with the individual being assessed.

Although specific gold standard assessments have yet to be established for this population with a dual diagnosis of ID and dementia, some guidelines for assessments have been offered. The International Association for the Scientific Study of Intellectual Disability (IASSID) and the American Association on Mental Retardation (AAMR)(1998) recommend baseline assessments for individuals including both direct and indirect assessment procedures by the age of 25 (Burt & Aylward, 1999). Assessments that are administered routinely over time allow for longitudinal comparisons (Prasher, 1999). While some cognitive decline is anticipated with typical aging (Devenny et al., 2002; Huppert, 1994; Petersen et al., 1991; Wingfield & Stine, 1989), there are
benchmarks in which rates of cognitive decline become atypical or pathological. As standards of assessment have recently been established, it becomes clear that considerations for rate of decline have yet to be established for this group with dual diagnosis.

By utilizing diagnostic information, caregivers are provided the tools and information to address issues of cognitive decline and provide supporting services. For example, individuals with decline secondary to medical conditions (Thase, 1982) or environmental situations (Geyde, 1998) are more apt to receive the requisite medical and environmental supports to arrest the cognitive decline. Knowledge of differential rates of decline may someday allow clinicians to more accurately diagnose sub-classifications of dementia among individuals with ID. For example, clinicians will be able to differentiate between the declines associated with C-J (APA, 2000) Lewy body dementia (McKeith et al., 1992), stepwise decline typical of Vascular dementia, and the progressive decline characterized by Alzheimer’s dementia (APA, 2000). This information is vital to ensure appropriate medical interventions and to enable caregivers to plan for future services.

In an initial effort to garner this information, the current study employed the standards set by the IASSID-AAMR (1998) workgroup. Baseline measures were established by reviewing assessments administered two years prior to the existing diagnosis of dementia for the treatment group and their individually matched controls. A mixed multivariate analysis of variance (MANOVA) design was utilized to study within group longitudinal effects and differences between diagnostic groups. The mixed MANOVA design allowed examination of differing rates of decline between groups independently diagnosed with dementia and matched controls. Differences across
adaptive behaviors as measured by the Vineland Adaptive Behavior Scales (Sparrow et al., 1984) and the Positive scales of the MESSIER, and problem behaviors as measured by the Negative scales of the MESSIER were assessed to establish if they aided in determining treatment needs for the dually diagnosed group. Additionally, by ascertaining the strong relationship between the VABS and the MESSIER positive scales across diagnostic groups there was converging support/reliability in the diagnosis of dementia. Given that diagnosis of dementia is difficult in the ID population, one would expect increased difficulty in assessing dementia in individuals who experience decreased cognitive abilities as a result of the initial diagnosis of mental retardation. By establishing variability in the presentation of negative behaviors with a standardized measure across individuals diagnosed with dementia, there is a novel component added to the diagnosis of dementia that might assist in confirming the diagnosis as well as provide some indication of future treatment needs. Preparation for the presence of problem behaviors is routinely seen in the general population and this would reflect a parallel for service delivery based on the existing model of services for dementia as a broad class.

Therefore, the current investigation compared rates of decline within a typically aging group of individuals with developmental disabilities and a group of individuals with developmental disabilities and dementia. Establishing differential rates of decline is a primary step in delineating differences between groups. Additionally, the variance of negative behaviors across groups was assessed as a secondary means of demonstrating assessments differences more likely in the presence of dementia. Established group differences and differential rates of decline across these populations may provide
The following hypotheses were offered for study:

Hypothesis 1: The skill changes during pre-diagnosis baseline measures across the VABS will not be significantly different for individuals now diagnosed with dementia as compared to non-demented matched controls. Testing of this hypothesis establishes that groups maintain sufficient similarities prior to the onset of dementia, that differences noted post diagnosis are more likely based on cognitive decline secondary to dementia rather than occurring by chance. This also works to meet guidelines of the AAMR-IASSID that recommend baseline assessments are used to make comparisons to more confidently make a diagnosis of dementia.

Hypothesis 2: Percentage change of adaptive behaviors from baseline to post diagnosis of dementia will be significantly different for the dementia group, while showing no significant differences from baseline to post diagnosis for the non-demented matched controls.

![Diagram](image)

**Figure 1**
Adaptive Skills as Measured with the Vineland Adaptive Behavior Scales
This hypothesis served to establish there are notable differences across diagnostic groups providing statistical support/reliability of the exiting diagnosis of dementia and highlighting the content relevance of decrease in adaptive skills for the diagnostic group. It was proposed if the findings yielded a trend of decline in the Non-Dementia group that was not statistically significant this may provide support for diagnostic accuracy as well as highlight the percentage of decline anticipated in normal healthy aging among persons with ID.

Hypothesis 3: Results of the VABS and MESSIER Positive subscale raw scores will show a high correlation across scales for both subgroups of dementia and non-dementia, while each subgroup will maintain differences reflective of presentation/absence of dementia.

Figure 2
Convergence of Findings for the Matson Evaluation of Social Skills Among the Severely Retarded and the Vineland Adaptive Behavior Scales

This hypothesis was tested to establish similar trends across global adaptive behaviors and social skills. By establishing both measures follow the same trend as
expected across diagnostic groups, convergent validity in the diagnosis of dementia is supported.

Hypothesis 4: Results of the VABS and MESSIER Negative subscale raw scores will show a divergence of scores and variance across the subgroups of dementia and non-dementia. Specifically, the dementia subgroup will show a broader range of variance of negative behaviors while the non-dementia subgroup will show a narrow and stable presentation of negative behaviors. This trend will be established by plotting upper and lower quartiles of negative scores on the MESSIER for each group.

![Variability of Response Based on Diagnosis](image)

**Figure 3**

Variability of Negative Scores on the Matson Evaluation of Social Skills Among the Severely Retarded Based on the Presence and Absence of Dementia.

Visual analysis was reviewed to establish if there was an interaction effect of adaptive skills and negative skills demonstrating a decline in adaptive behaviors and an increase of negative behaviors for individuals presenting with dementia, while there would be no expected cross over effect noted for individuals in the control group. These findings were expected as there is evidence of the development of problem behaviors during the course of dementia among both general and ID populations. However, in end-
stage dementia, the loss of motor skills was expected to create the possibility of decline in negative behaviors, allowing for the possibility of more variability in negative behaviors for the dementia group than the non-dementia group.

By establishing a wider variation of negative behaviors in the dementia group, discriminate validity was expected. In the presence of dementia, it is common that negative behaviors are evinced with the decline of skills thought to be related to cognitive decline as well as decline in social and communication skills, thus supporting features of the hypothesis anticipating increasing rates across the dementia group. Lower ranges were anticipated as not all individuals present with negative behaviors and in the later stages of decline more vegetative features and diminished motor skills were anticipated, thus resulting in diminished abilities to present with negative behaviors. Stable rates were anticipated in the control group as it was anticipated there would be little change in adaptive behaviors yielding no expectation in diminished skills. Differences in variability
across the groups were expected to yield an additional feature to validate the diagnosis of dementia.

An interaction effect was anticipated between positive and negative behaviors in the dementia group as with the decline in adaptive behaviors increases in negative behaviors were anticipated. As there are little to no changes anticipated in adaptive behaviors for the control group it was expected that the relationship between positive and negative behaviors were to remain linear.
METHOD

Experimental Design

A 2 (groups) x 3 (measures) x 4 (repeated intervals) Mixed Multivariate Analysis of Variance (MANOVA) design was used to analyze differences both between and within subjects. All data was retrospective with exception of the dementia rule out screening administered to the control group. The independent blocking variable was the diagnosis of dementia, yielding two groups one with a diagnosis of dementia and one without (control group). Dependent measures included the total raw score of VABS, the total raw score of the Positive scales of the MESSIER, and the total raw score of the Negative scales of the MESSIER. The repeated measures element of the design included independent measures for the 3 dependent measures across five years. Two years of data before the diagnosis of dementia was considered baseline data. The additional three years were examined as changes following the diagnosis of dementia. Each assessment battery was administered within a one year time frame every year over the five years reviewed. Ratio of decline for each individual was established by subtracting total raw scores for each dependent measure from the previous year and dividing by the total of the measure from the first year. For example, an individual that had a total raw score of 100 on the VABS in the first year and a total raw score of 80 in the second year, the ratio was computed as (100-80)/100 yielding a 20% rate of decline between year 1 and 2. If year 3 resulted in a total raw score of 60 on the VABS the corresponding ratio of decline was reflected as (80-60)/100 yielding another decline of 20% for the year and a cumulative decline of 40% from baseline. A ratio of decline was established so that each individual served as their own control as differences across individuals may be variable dependent
on baseline levels of functioning. By creating a ratio of decline, individual decline was compared across subjects yielding a 1%, 2%, etc. decline as opposed to an absolute raw score. An absolute raw score decline of 5 points may be significant for an individual with a total raw score of 30, whereas a decline of 5 points may not be as significant or equivalent for an individual with a total raw score of 100, the first reflecting a 17% decline and the later reflecting a 5% decline. With 5 points of measurement for each individual there were a total of 4 ratios available for evaluation, reflecting the last component of the design.

| Year 1-Year 2 | Percentage of Decline (Annualized Percentage of Decline) |
| Year 2-Year 3 | Percentage of Decline (Annualized Percentage of Decline) |
| Year 3-Year 4 | Percentage of Decline (Annualized Percentage of Decline) |
| Year 4-Year 5 | Percentage of Decline (Annualized Percentage of Decline) |

Figure 5
Experimental Design

The mixed model design allowed for multiple comparisons to be made between the groups (i.e. dementia versus non-dementia groups, pre versus post diagnostic time frames), as well as within groups (i.e. significant difference across years within groups) (Hays, 1994; Meyers, & Well, 1991).
Participants

Participants were selected from a population of 635 residents of an Intermediate Care Facility for Mental Retardation in central Louisiana. An a priori statistical power analysis was conducted using G*Power, a statistical software package (Faul & Erdfelder, 1992). A large effect size of .80 was used as differences between the dementia group and the control groups were expected to be vast, with predominant differences expected with decline of over-learned behaviors and motor skills typically seen in the mid and late stages of decline (Harper, 1993; Janicki & Jacobson, 1986). For a large effect size of .80, power of .80, and an alpha level of .05, results indicated the need for a total sample size of 42. This sample size was consistent with previous standards set forth in the research literature studying effects of adaptive skill decline among individuals with ID and dementia (See Table 3). The sample was obtained from archival data maintained at the facility mentioned above. Only individuals classified as functioning within the Moderate to Profound ranges of mental retardation based on criteria established in the DSM-IV TR (APA, 2000) and Association of Mental Deficiency criteria were eligible for inclusion in this study (Grossman, 1983).

Institutional consent for use of data was obtained as a part of a broader study for the norming of the MESSIER. Oversight approvals have been made by the Louisiana State University Internal Review Board, the Department of Health and Hospitals of Louisiana Review Board, and the Human Rights Committee for Pinecrest Developmental Center. Instruments reviewed in this study were scales routinely used in annual assessments. All subject information was coded into a larger database to ensure anonymity and confidentiality.
The study group consisted of twenty-two individuals independently diagnosed with dementia based on standards detailed in the AAMR-LASSID Practice Guidelines for Care Management of Alzheimer’s Disease Among Adults with Mental Retardation (Janicki, Heller, Seltzer, & Hogg, 1996). Two subjects were eliminated as there was insufficient archival data available for review. Among the study group, individuals ranged in age from 32-82 at point of diagnosis. The average age at time of diagnosis was 58 years. The years under review ranged from 1996-2005. Of the individuals in the study group, six individuals died during the time period under review. The average time of death post diagnosis of dementia was 2 years. These deaths resulted in missing data for five of the individuals. Two individuals were missing the last two years of data and three individuals were missing the last year of data.

Table 4
Demographic Information Related to Age of Onset of Dementia.

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Mean Age</th>
<th>Age Ranges</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dementia</td>
<td>20</td>
<td>58</td>
<td>32-82</td>
</tr>
<tr>
<td>Down’s Syndrome</td>
<td>06</td>
<td>48</td>
<td>39-78</td>
</tr>
<tr>
<td>Males</td>
<td>11</td>
<td>58</td>
<td>32-78</td>
</tr>
<tr>
<td>Females</td>
<td>09</td>
<td>58</td>
<td>51-66</td>
</tr>
<tr>
<td>Severe ID</td>
<td>06</td>
<td>58</td>
<td>32-78</td>
</tr>
<tr>
<td>Profound ID</td>
<td>14</td>
<td>58</td>
<td>39-82</td>
</tr>
</tbody>
</table>

For individuals not already diagnosed with dementia an Early Signs of Dementia Checklist (Visser & Kuilman, 1990) was administered to rule-out the possibility of onset of symptoms of decline characteristic of dementia. Twenty-three individuals were screened as possible controls for the dementia group. Three individuals received a score of 5 or higher and were excluded from consideration as a control subject and were referred for dementia screening. Visser et al., (1997) suggested that responses of 5 or higher with the ESDC are indicative of potential cognitive decline. This rule-out out
measures were completed in an effort to prevent the potential confound of unidentified early onset dementia in the control group. Individuals in the control group were matched based on age, race, gender, and level of ID. Level of intellectual disability was based on independent determinations registered in subject records. Clinical standards at this ICF/MR were consistent with diagnostic criteria detailed in the DSM-IV-TR requiring both intellectual and adaptive assessments in determining level of cognitive functioning. In cases where Down’s syndrome was identified, individuals were matched for this genetic condition, as it was a relevant variable when considering dementia. Individuals included in the control group ranged in age from 31-81. An average age post baseline was established to match time of diagnosis for the Dementia group. Average age for the control group was 58 years of age. The years under review for the data considered ranged from 1999-2005. There were 3 variables of missing data for this group resulting in one variable for three separate subjects (2 Vineland measures and 1 MESSIER measure). These missing points of time measures were excluded from analysis.

**Measures**

The current standard for assessing dementia is comparing skills assessments across time; therefore, primary assessment information was archival and consisted of social and adaptive skills. The Vineland Adaptive Behavior Scales and the Matson Evaluation of Social Skills for the Severely Retarded was used in measuring skills across time. The Early Signs of Dementia Checklist was used to rule out the presence of cognitive decline among individuals in the control group.

The Vineland Adaptive Behavior Scales (VABS) (Sparrow, Balla, & Cicchetti, 1984) served as both a measure to diagnose mental retardation and as a more sensitive
measure for comparison of declining adaptive skills across time. The VABS is a well-validated measure of adaptive behavior across four domains (socialization, communication, daily living skills, and motor skills). Construct validity has been established through principle component analysis. Subscales were identified and yielded loadings of .56 to .79. Factor analyses confirm organization of sub-domains into respective domains. Convergent and discriminate validity have been established with the VABS showing stronger correlations with adaptive measures and weaker correlations with intellectual assessments (Sparrow et al., 1984). Questions are subdivided across the four domains, and are listed in the order consistent with developmental achievement. Scoring of items are rated on a 3 point Likert scale (2=yes usually, 1=sometime partially, 0=no never), as well as correcting for lack of opportunity or knowledge of specific ability (N=no opportunity, DK=don’t know). The VABS has been extensively used in the field of developmental disability delineating acquisition of adaptive skills across time. Reliability coefficients reported for internal consistency, inter-rater reliability, and test-retest concordance averaging in the .80’s and .90’s (Sparrow et al, 1984).

The MESSIER (Matson, 1995) was used to assess positive and negative social skills. The scale consists of 85 items generated from existing social skills measures, adaptive skills measures, and nominated items by experts. Items are grouped into six clinically derived subscales: positive verbal, positive non-verbal, positive general, negative verbal, negative non-verbal, and negative general. Items are rated on a four point Likert scale (0=never, 1=rarely, 2=sometimes, and 3=often). Internal consistency, interrater-reliability, and test-retest reliability for the scale have been noted as .93, .93, and .97, respectively (Matson, LeBlanc, & Weinheimer, 1999; LeBlanc, Matson, Cherry,
& Bamburg, 1999). Convergent validity has been established with the Socialization domain of the VABS (Matson, Carlisle, & Bamburg, 1998). The MESSIER evaluates social skills and deficits as well as the maladaptive behavior excesses (Matson, 1995).

The Early Signs of Dementia Checklist (Visser & Kuilman, 1990) is an indirect measure of cognitive decline that is particularly effective in testing the limits despite floor effects noted in other assessments (Cosgrave et al., 1998). The ESDC is a 37-item measure that assesses nine separate categories of decline: general, personality changes, decrease in performance, deterioration of language skills, deterioration of gait, disorientation, incontinence, epilepsy, and loss of school acquired skills. Internal consistency and inter-rater reliability have been established at .82. The nine categories are considered symptom areas predictive of dementia (Ballard et al., 2001). When combined with social skill inventories, the ESDC is effective in reliably detecting Alzheimer type dementia at an early stage (Visser et al., 1997).

**Procedures**

Standard annual assessments utilizing the VABS and the MESSIER from a five year period were reviewed archivally to establish rates of decline of adaptive skills and rates of negative skills. Time frames were established based on year of diagnosis for the dementia group. Two years of data prior to the diagnosis of dementia were reviewed and three years post diagnosis of dementia was reviewed. These time frames were matched for the controls, by matching the control’s age at the time of diagnosis for the dementia match. Two years prior to this age and three years post this age were reviewed for each control subject. All assessments were given every 10 to 12 months yielding a single point of measurement across each year.
Assessments of all measures were completed by interviewers possessing a master’s degree in psychology and were supervised by a licensed clinical psychologist. Interviewers had been trained in assessment procedures and had worked in the above listed setting under supervision for at least one month prior to conducting interviews. Informants consisted of direct care service providers who had worked with the participants for a minimum of one year. Doctoral level psychology practicum students and interns who received training from a supervising licensed psychologist administered dementia assessments requiring participation by subjects.

Data was retrieved by accessing general databases for the entire population of the developmental center and dementia screening databases. All individuals with a diagnosis of dementia were selected from the dementia screening database which details time of diagnosis with a written report. These diagnoses were completed by pre-doctoral and postdoctoral trainees under the supervision of a licensed neuropsychologist. The general database has been maintained by psychological services of the facility. In situations in which data was missing or was unclear, the physical assessment was retrieved from medical records. For individuals in the control group, a screening was administered utilizing the ESDC to rule out the presence of dementia.
RESULTS

A 2 (groups) X 3 (measures) X 4 (repeated measures) Mixed Multivariate Analysis of Variance (MANOVA) was completed to assess rate of decline within groups with the Vineland Adaptive Behavior Scales (Sparrow, Balla, & Cicchetti, 1984) and corresponding changes in positive and negative behaviors as measured by the Matson Evaluation of Social Skills for the Severely Retarded (Matson, 1995). Hypotheses one and two were assessed by completing analysis of variance. Specifically, Hypothesis 1 predicted the percentage of change from baseline measures across the VABS was not significantly different for individuals now diagnosed with dementia as compared to non-demented matched controls (F (1, 40) = 1.086, p = .304). In Figure 1, Hypothesis 1 is reflected as the data point prior to a diagnosis of dementia (listed as pre-diagnosis). Hypothesis 1 was assessed with 2 X 3 X 1 MANOVA was used to compare the first ratio of change for all three measures between year 1 and year 2 across the dementia and non-dementia groups. Non-significant differences across baseline measures were also noted for the MESSIER Positive scales (F (1, 40) = .298, p=.589) and the MESSIER Negative scales (F (1, 36) = .192, p = .664). This suggests that groups were not significantly different prior to the diagnosis of dementia for the experimental group. Homogeneity of variance analysis was also completed to establish that the group variances were not significantly different prior to period of diagnoses. Results of these analysis supported homogeneity of variance across groups for all three dependent measures (VABS (F (1, 38) = 2.098, p = .156); MESSIER Positive (F (1, 38) = .383, p = .540); MESSIER Negative (F (1, 34) = .696, p = .410). These results support Hypothesis 1 and the
likelihood that changes after the diagnosis of dementia are related to the symptoms of dementia.

*Purple and yellow reflect independent variables compared. Green reflects dependent variables.

Figure 6
Hypothesis 1 Reflected Across the Experimental Design

Hypothesis 2 suggested percentage change across adaptive behaviors from baseline (Year 1 to Year 2) to post diagnosis of dementia (Year 2 to Year 3) would be significantly different for the dementia group, while showing no significant differences from baseline to post diagnosis for the non-demented matched controls. This was assessed by completing a 2 X 1 X 4 MANOVA. Results of the assessment yielded no significant differences across VABS measures across time (F (1, 32) = 1.134, p = .299) and no significant interaction differences between VABS scores and time of assessment (F (1, 32) = .321, p = .590). Between group differences were also non-significant (F (1,
30) = 3.156, p = .086). Therefore, Hypothesis 2 was rejected; suggesting rate of decline was not significantly different from baseline to post diagnosis. Post hoc comparisons were not completed as there were no significant findings for the main effect of adaptive skills across groups. While statistical significance was not evident across groups, means of percentage change between each measurement epoch were plotted to examine trends for both groups. From the graph below the means of Annualized Percentage Decline show positive scores for the dementia group reflecting decline. As the differences were assessed by using the following formula for Annualized Percentage Decline: (Year 1-Year 2)/Year 1 (see Method section) any positive scores reflect decline in skills and negative scores reflect growth (e.g. (100-110)/100 = -.1). From the graph below decline is noted across all years for the dementia group and growth is noted for all years for the control group. Also noteworthy, is that group differences are evident at baseline while statistical significance difference was not established.

Figure 7
Percentage Change in Adaptive Skills as Measured with the Vineland Adaptive Behavior Scales
Figure 8
Hypothesis 2 Reflected Across the Experimental Design

Figure 9
Percentage Change in Positive Social Skills as Measured with the MESSIER

Additionally, there were significant differences in rate of decline across the MESSIER Positive scale across time (F (3, 96) = 3.887, p = .011, β = .811). There were no clear interaction effects within group differences (F (3, 96) = 1.122, p = .344), or
between group differences ($F(1, 32) = 1.402, p = .245$). Pairwise comparisons yielded significant differences across rate of decline between years 1 and 2, 2 and 3, 2 and 4, all noted with probability of .01.

Figure 10
Hypothesis 3 Reflected Across the Experimental Design

Hypothesis 3 was assessed utilizing Pearson product-moment correlations between scales within groups. Results of the VABS and MESSIER Positive subscale raw scores showed linear relationships and an overall high correlation between scales $r = .738, p = .01$. Across groups significant correlations and linear relationships were maintained between the VABS and the MESSIER Positive Scales (Dementia $r = .701, p = .01$; Control $r = .856, p = .01$). The table below shows that significant correlations were maintained at each point of measure for each diagnostic group. As a result of these findings, Hypothesis 3 was supported.
Table 5
Table of Correlations of the Matson Evaluation of Social Skills Among the Severely Retarded Positive Subscales and the Vineland Adaptive Behavior Scales.

<table>
<thead>
<tr>
<th>Group</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Year 4</th>
<th>Year 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td>.652*</td>
<td>.677*</td>
<td>.778*</td>
<td>.823*</td>
<td>.830*</td>
</tr>
<tr>
<td>Control</td>
<td>.840*</td>
<td>.700*</td>
<td>.826*</td>
<td>.895*</td>
<td>.893*</td>
</tr>
<tr>
<td>Dementia</td>
<td>.611*</td>
<td>.709*</td>
<td>.779*</td>
<td>.780*</td>
<td>.791*</td>
</tr>
</tbody>
</table>

* denotes Significance = .01

Group means across each year of measurement were plotted and establish a pattern of decline for individuals with dementia across both scales. Plotting of means also illustrates the relationship between the MESSIER and the VABS in that stable patterns were noted with the control group and decline was noted across both instruments for the dementia group.

Convergence of Findings for the Matson Evaluation of Social Skills Among the Severely Retarded and the Vineland Adaptive Behavior Scales

Correlations across scales were used to test Hypothesis 4. It was hypothesized that results of the VABS and MESSIER Negative subscale raw scores would show a divergence of scores for the dementia group. Overall there was a negative correlation between the VABS and the MESSIER Negative scores $r = -.047$ across total responses. Negative correlations were maintained across groups (Control $r = -.18$, Dementia $r = -$
The table below demonstrates that no significant correlations between scales were established at any point in time for either group. These findings partially support Hypothesis 4 as divergences of scores across the MESSIER and VABS was maintained. These findings also yield additional discriminate validity for the MESSIER, as the Negative subscales do not correlate with adaptive scales.

Table 6
Table of Correlations of the Matson Evaluation of Social Skills Among the Severely Retarded Negative Subscales and the Vineland Adaptive Behavior Scales.

<table>
<thead>
<tr>
<th>Group</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Year 4</th>
<th>Year 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td>-.081</td>
<td>-.307</td>
<td>.035</td>
<td>-.034</td>
<td>.261</td>
</tr>
<tr>
<td>Control</td>
<td>-.384</td>
<td>-.404</td>
<td>-.136</td>
<td>.085</td>
<td>.323</td>
</tr>
<tr>
<td>Dementia</td>
<td>.074</td>
<td>-.270</td>
<td>.153</td>
<td>-.070</td>
<td>.200</td>
</tr>
</tbody>
</table>

Variances of negative behaviors as measured by the MESSIER Negative subscales are listed below for each group for each successive year. While the variances

Figure 12
Hypothesis 4 Reflected Across the Experimental Design
are notably different across some epochs, assessment of homogeneity of variance for
each measurement period revealed that there were no significant differences in variance
across populations (Epoch 1 (F(1,29) = .369, p = .548), Epoch 2 (F(1,29) = .881, p =
.356), Epoch 3 (F (1,29) = .451, p = .507), Epoch 4 (F (1,29) = .663, p = .422). These
findings do not support Hypothesis 4, as significant differences across variances were
expected. Variances for each year of measurement are listed for the MESSIER Negative
scales to establish variance rates for the assessment, as opposed to variance rates for each
epoch, which would denote variance for rate of decline. Variances across groups for the
MESSIER Negative scales are listed below. Larger variances were noted for the control
group, rather than the dementia group.

Table 7
Variance of Negative Behaviors as Measured by the MESSIER Negative Scales Across
Groups for each Year of Measurement.

<table>
<thead>
<tr>
<th>Group</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dementia</td>
<td>277.88</td>
<td>250.59</td>
<td>78.22</td>
<td>249.95</td>
<td>228.61</td>
</tr>
<tr>
<td>Control</td>
<td>159.01</td>
<td>1371.22</td>
<td>251.22</td>
<td>152.52</td>
<td>209.67</td>
</tr>
</tbody>
</table>

Group means were plotted for upper and lower quartiles across each assessment
period and support statistical findings yielding a lack of variance across groups (Figure
13) and a divergence of scores across the VABS and the MESSIER Negative scores
(Figure 14).

These findings suggest that while divergence of across scales was maintained,
there was little variability in negative behaviors across diagnostic groups. This resulted in
only partial support of Hypothesis 4.
Figure 13
Upper and Lower Mean Scores Across Negative Behaviors as Measured by the Matson Evaluation of Social Skills Among the Severely Retarded for Individuals with and without Diagnoses of Dementia

Figure 14
Divergence of Findings for the Matson Evaluation of Social Skills Among the Severely Retarded and the Vineland Adaptive Behavior Scales.
DISCUSSION

By using standardized measures of adaptive and social skills across time, clinicians are able to track changes in behaviors. These changes may be related to adaptive skills and reflect learning, as well as establishing declines in skills. The MESSIER has specific utility in assessing social skills strengths and deficits, adding the dimension of negative behaviors. Examining the profile as strengths are related to deficits, clinicians often look for improvement in skills secondary to learning objectives to result in the decrease in negative behaviors. However, when assessing cognitive decline, one can look for an opposing inverse relationship (i.e., the loss of social skills and subsequent increases in negative social behaviors). This study sought to identify both rates of cognitive decline as measured by adaptive and social skills measures, in addition to identifying features of social skills that assist in confirming a diagnosis of dementia. The goal was to establish a benchmark of decline so that clinicians may be equipped with information to further detail subtypes of dementia, which could possibly assist in providing more specific interventions.

In an effort to demonstrate changes across time were a result of pathological aging, the dementia group was matched with control subjects on the basis of age, gender, level of functioning, and the presence of Down’s syndrome. The demographics for each group are therefore largely identical. Age ranges across groups were 31 years of age to 82 years of age. The average age of onset of dementia was noted at 58 years of age, with a range of 32 - 82 years of age for the general dementia group. These findings support results by Janicki and Jacobson (1986), which purport age of onset in the mid-fifties for individuals with intellectual disability.
Among the 6 individuals with Down’s syndrome, the average age of onset for the dementia group was 48 years of age. This represents a 10-year difference of age of onset compared to individuals without Down’s syndrome. These findings do not support literature by Wisniewski (1990) and Holland (1999) suggesting decline begins as early as the third decade, but does support literature by Janicki et al. (1996), Thase (1982), and Zigman, et al. (2004) which note decline in the fourth decade for individuals with Down’s syndrome. Furthermore, these findings support the notion that onset of dementia is earlier among individuals with Down’s syndrome than for individuals without Down’s syndrome (Deb et al., 2000; Lott & Head, 2001).

Overall, there were a total of 40 subjects, 22 males and 18 females. There was no difference in age of onset of dementia across gender when examining average age; however, when looking at age ranges across groups there was a notable difference. Among the participants in the dementia group, onset ranged from age 32-78 for males and 51-66 for females, yielding almost a 20-year differential in age of onset across gender. While these findings do not support shorter life expectancies across gender, they do support the presence of gender differences among individuals with dementia (Aguerro-Torres, et al., 1998; Keene, et al., 2001).

As the age range for the study group began at age 32, there is support for Cummings and Cole (2002), who report early onset dementia as early as 30 years of age. This further validates concerns that dementia is under-diagnosed in younger people, and men more specifically (Newens et al., 1993). Mild cognitive impairment is often the earliest sign of dementia (Galvin, et al., 2005; Ralph et al., 2003) and can be seen 10, 15, and 30 years prior to a diagnosis of dementia (Klag, 1999). Therefore, recommendations
for baseline measures prior to age 40 (Bush & Beail, 2004) are warranted. Early identification is essential as it facilitates treatment planning, creates opportunities for use of disease modifying drugs, and allows for long range planning (Berg, 2003; Lott & Head, 2001; Ralph et al., 2003).

When considering level of functioning, there were 12 individuals functioning within the severe range of intellectual disabilities and 28 individuals functioning within the profound range of intellectual functioning. Average age of dementia onset was 58 years of age. There was not a notable difference in age of onset across groups.

Hypothesis 1 established that groups were not significantly different and variance across groups was not significantly different for all three measures (VABS, MESSIER Positive, MESSIER Negative) during the baseline levels. These results lend support to further interpretation of hypotheses 2, 3 and 4, suggesting that the changes across groups are the result of the presence of features of dementia.

Testing Hypothesis 2 demonstrated both cross sectional and longitudinal analysis comparing measures at baseline to post-diagnostic periods for dementia across adaptive behavior skills. The results of the assessment yielded no significant differences between the dementia and control group after the diagnosis of dementia was made. There was also no significant difference within subjects across times. Therefore, post hoc comparisons were not warranted.

While significant differences were not evident, group means for each year of assessment were reviewed and provide a rationale for why group differences were not statistically significant. The first notable consideration is that while baseline measures were not significantly different, the control group presented with lower means at baseline.
Mean responses at baseline differed by 56 and 42 points. Secondly, the declining means across the dementia group began to approach the scores of the control group, making statistical significance improbable.

Table 8
Table of Means of the Vineland Adaptive Behavior Scales for each Year of Measurement Across each Group.

<table>
<thead>
<tr>
<th>Group</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Year 4</th>
<th>Year 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>102</td>
<td>102</td>
<td>102</td>
<td>102</td>
<td>110</td>
</tr>
<tr>
<td>Dementia</td>
<td>158</td>
<td>144</td>
<td>108</td>
<td>98</td>
<td>92</td>
</tr>
</tbody>
</table>

One consideration for future studies would be to match subjects based on adaptive skill measures, which would be a more sensitive and accurate match than level of functioning. The current presentation (Figure 7) does support a pattern increase of adaptive measures for the control group, and a pattern of decline for the dementia group consistent with what was anticipated.

Further review of the data revealed that, the dementia group showed a sharp decline from baseline measures after the diagnosis of dementia. At the time in which dementia was considered, there was an average drop in responses by 36-points with the VABS and 18-points with the MESSIER Positive scales for the dementia group. In contrast, the matched control group presented with a 6-point increase with the VABS and a 1-point decrease with the MESSIER Positive scales. Decline was persistent and progressive across time for the dementia group across adaptive behaviors. The decline between the first and second baseline responses may be indicative of mild cognitive decline that are below the threshold of a diagnosis of dementia. Given that statistical significance was not established and baseline measures were visibly different, rate of decline can not be confidently determined for each group and visual analyses should be
cautiously interpreted. It is possible that reanalysis of these data, using non-parametric procedures in the manner Advokat, Martino, Hall and Gouvier (in press) propose might reflect the statistical significance implied by the visual inspection.

An additional consideration is that the VABS was not a sensitive enough measure to discriminate dementia across time. While the VABS is a gold standard assessment of adaptive skills (AAMR, 2002; Sparrow, et. al, 1984), the MESSIER was developed as a more specific measure of social skills. Overall, the MESSIER presents with a larger range of items with greater specificity in demonstrating social skills among individuals with Severe and Profound ID (Bielecki & Swender, 2004; Matson, et. al., 1998). The MESSIER was supported as a more sensitive measure of social skills as the comparisons of the MESSIER positive skills across the dementia and non-dementia group were significantly different. While this was not a hypothesis in this study, it is a novel finding that warrants further investigation, with the potential to improve our technology for assessing performance changes associated with dementia.

The visual analysis illustrated in Figure 9 shows that the two groups were similar at baseline. Rates of decline post-diagnosis of dementia were significant among the experimental group. Interestingly, while there was an initial decline post diagnosis, there was also evidence of skill acquisition in the presence of dementia. This creates difficulty in establishing a rate of decline, but it does support the notion that skills can be learned despite presentation of symptoms of dementia. This finding also supports the idea that individuals with dementia require and benefit from continued active treatment (Dick, Shankle, Beth, Dick-Muehlke, Cotman, & Kean, 1996). Both sets of findings provide support for the use of adaptive measures as tools for diagnosis of dementia (Aylward et
al., 1997; Schultz et al., 2004; Strydom & Hassiotis, 2003). Furthermore, the goal of dementia assessments are to determine progressive deterioration of skills (Janicki et al., 1996), the current study adds support to the use of the MESSIER as additional assessment to be used in the diagnosis and monitoring of dementia and interventions.

Correlations between the MESSIER Positive subscales and the VABS were significant at each point in time across five measurements, thus supporting Hypothesis 3. These findings add to the existing convergent validity of the MESSIER. While convergent validity had previously been established for the MESSIER with the VABS (Matson et al., 1998), that study was strictly cross sectional from a single point of measure (i.e., time). The current study establishes longitudinal stability of the correlation across both time and cognitive decline secondary to dementia (stability across time and sensitivity across diagnostic group of dementia).

Additionally, the pattern of decline for the dementia group is noted across both scales, providing support to the existing diagnosis of dementia as well as giving a secondary benchmark of decline across social skills. Information from the MESSIER Positive scales can be used to develop interventions to minimize social skills decline. Further review of Figure 9 shows that despite decline in social skills after a diagnosis of dementia, there was improvement in skills in year 5. This supports the notion of skill acquisition in the face of decline, and it creates promise for enhanced quality of life for individuals faced with a diagnosis of dementia. These results create an opportunity for changes in standards of care from a medical support/custodial care model to an active treatment model (i.e., skill acquisition and maintenance) for those dually diagnosed.
Hypothesis 4 suggested that a negative correlation exists between MESSIER Negative subscales and the VABS for the individuals in the dementia group. Specifically, as adaptive skills decline, negative behaviors will increase. This hypothesis was only partially supported. There were non-significant correlations between the MESSIER Negative scales and the VABS across each measurement period. This finding contributes to the existing discriminant validity of the MESSIER as one does not expect negative behaviors to correlate with adaptive skills. However, measures of homogeneity of variance did not yield significant differences in population variances post diagnosis of dementia. Therefore, the assessment of negative behaviors is not supported as a diagnostic variable for dementia at this time, nor was the expectation of increasing variance across post diagnosis epochs supported.

Visual analysis yielded an opposing finding to what was hypothesized with the control group showing more variability in negative behaviors than the dementia group. Sharp declines in negative behavior among the control group may be related to effective behavioral intervention and learning opportunities. Given individuals with dementia are losing cognitive abilities yet maintaining some ability to learn skills, their response to behavioral interventions creates an additional area of study. The stability in variance for the dementia group may loosely be interpreted as a struggle between treatment gains and the loss of cognitive skills.

The goal of this study was to extend the research base by establishing rate of decline and adding novel elements to aid in diagnosing dementia among individuals who present with severe and profound ID. The lack of significant findings leads to further examination of limitations of the existing study. One primary limitation noted is that of
effect size. While the current sample size is consistent with treatment studies in the literature for individuals with ID and older age, review of effect size of previous studies suggests that the use of a large effect size was not appropriate in determining a sample size for this study. A review of effect size for previous studies was completed after data analysis and can be seen in Table 9 below. For studies in which data was available to compute effect size, findings support the use of a small effect size resulting in a need for a significantly larger study group. For a future study replicating the existing methodology a total sample size of 620 individuals would be needed.

Table 9
Review of Sample Sizes and Effect Size Across Age Groups for Cross Sectional and Longitudinal Designs Among the ID Population

<table>
<thead>
<tr>
<th>Participants</th>
<th>Years</th>
<th>Ages</th>
<th>Effect Size</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cross Sectional</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>34</td>
<td>34-70</td>
<td>.49</td>
<td>Miniszek, 1983</td>
<td></td>
</tr>
<tr>
<td>40</td>
<td>40+</td>
<td></td>
<td>Hauber, et al., 1985</td>
<td></td>
</tr>
<tr>
<td>23</td>
<td>50-61</td>
<td></td>
<td>Hewitt, et al., 1985</td>
<td></td>
</tr>
<tr>
<td>63</td>
<td>20-85</td>
<td></td>
<td>Linter, 1986</td>
<td></td>
</tr>
<tr>
<td>70</td>
<td>65+</td>
<td>.23</td>
<td>Barcikowska et al, 1989</td>
<td></td>
</tr>
<tr>
<td>122</td>
<td>50+</td>
<td></td>
<td>Moss, et al., 1992</td>
<td></td>
</tr>
<tr>
<td>Longitudinal</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>198</td>
<td>20-69</td>
<td>.25</td>
<td>Schupf et al., 1989</td>
<td></td>
</tr>
<tr>
<td>109</td>
<td>4</td>
<td></td>
<td>Rasmussen et al., 1994</td>
<td></td>
</tr>
<tr>
<td>34</td>
<td>3</td>
<td>22-56</td>
<td>.99</td>
<td>Burt et al. 1995</td>
</tr>
<tr>
<td>128</td>
<td>3</td>
<td>16-72</td>
<td>.19</td>
<td>Prasher et al., 1998</td>
</tr>
<tr>
<td>23</td>
<td>50+</td>
<td></td>
<td>Hewitt, et al, 1985</td>
<td></td>
</tr>
<tr>
<td>57</td>
<td>5</td>
<td>17-71</td>
<td>.19</td>
<td>Prasher et al., 1998</td>
</tr>
<tr>
<td>17</td>
<td>40+</td>
<td></td>
<td>Eveinhuis, 1990</td>
<td></td>
</tr>
<tr>
<td>70</td>
<td>7</td>
<td>.27</td>
<td>Kittler, et al., 2004</td>
<td></td>
</tr>
<tr>
<td>48</td>
<td>4</td>
<td></td>
<td>Prasher et al., 2004</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>10</td>
<td></td>
<td>Ikeda &amp; Arai, 2002</td>
<td></td>
</tr>
<tr>
<td>66</td>
<td>4</td>
<td>.35</td>
<td>Devenney et al, 2000</td>
<td></td>
</tr>
<tr>
<td>67</td>
<td>4.5</td>
<td></td>
<td>Roedern, et al, 1997</td>
<td></td>
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<tr>
<td>46</td>
<td>5</td>
<td>27-55</td>
<td>.54</td>
<td>Devenney et al., 1992</td>
</tr>
<tr>
<td>22</td>
<td>27-64</td>
<td></td>
<td>Johanson et al., 1991</td>
<td></td>
</tr>
</tbody>
</table>

Note table partial adaptation from Prasher, 1998 pgs. 166-168.
Upon attempting to establish a number of hypotheses it is also clear that the initial study of rate of decline could be strengthened matching subjects on baseline measures of adaptive behaviors as measured by the VABS. While groups were measured as equal at baseline, examination of means makes it clear that a more stringent matching of groups would have strengthened this research. Given that trends of means support persistent decline among the individuals with dementia on the measure of adaptive behaviors, research in this area should be continued to establish a standard measure of decline among typical aging and decline secondary to dementia. Further research identifying features of MCI among the ID population should also be considered as there are promising studies supporting the efficacy of medications that slow the progression of dementia among the ID population (Lott, et al., 2002). Likewise, as the diagnosis of dementia was supported across time with the measures of the VABS and the MESSIER Positive scales, there was evidence of learning in the presence of symptoms of dementia. Future studies should focus on active treatment gains as they may improve the quality of life for individuals dually diagnosed.
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VITA

Julia D. Lott was born in Alexandria, Louisiana. She received her bachelor of science at Louisiana College in Pineville, Louisiana, in the field of psychology in 1993. Her master’s of science in clinical psychology was earned at Northwestern State University in Natchitoches, Louisiana, in 1996. At this time, she began working with individuals with special needs at Pinecrest Developmental Center in Pineville, Louisiana. Her combined interest in the field of psychology and serving individuals with intellectual disabilities resulted in an application to Louisiana State University in Baton Rouge, Louisiana, in 1999. While in training at Louisiana State University, she completed an internship with Kennedy Krieger Institute at Johns Hopkins Medical School in Baltimore, Maryland. The completion of this document has resulted in the fulfillment and completion of her Doctor of Philosophy in clinical psychology. She is currently serving as the Clinical Director at Northwest Developmental Center in Bossier City, Louisiana.