Intellectual Disability

34

Barbara Tylenda, Rowland P. Barrett,
and Henry T. Sachs, III

Introduction

Change in Terminology

Intellectual disability has been recognized perhaps longer than anything else we currently study in psychiatry and psychology (Berkson, 2004; Tylenda, Hooper, & Barrett, 1987). However, the term intellectual disability (ID) is relatively new. It replaces the previous and long-used term mental retardation (MR).

In 2007, the American Association on Mental Retardation (AAMR) successfully voted to rename its organization the American Association on Intellectual and Developmental Disabilities (AAIDD). At that time, the organization also put forth the formal diagnostic name change from MR to ID (Schalock et al., 2007).

According to the AAIDD, the diagnostic term ID is preferred over the term MR because it reflects a changed construct of disability (AAMR, 2002; Buntinx, 2006; World Health Organization, 2001). Specifically, the construct changes from disability as a problem residing within a person to understanding disability as residing at least partly in the gap between a person's capacities and the demands of typical contexts or environments (AAIDD, 2012). Further, the construct changes from a static, unchanging condition to a condition that can change over time (Harris, 2006).

The AAIDD was clear to stipulate that "… anyone eligible in the past for a diagnosis of MR is now considered eligible for a diagnosis of ID. The term ID covers the same population of individuals previously diagnosed with MR in number, kind, level, type and duration, and the need for services and supports; and every individual who is or was eligible for a diagnosis of mental retardation is eligible for a diagnosis of ID" (AAIDD, 2010, p. xvi).

Overview

ID is a categorization for a heterogeneous group of individuals with deficits in cognitive and adaptive behavior functioning manifest prior to their 18th birthday. Those presenting with ID display individual patterns of strengths and weaknesses across academic, language, social, emotional, and physical skill performances. The developmental course for individuals with ID is not as inflexible as once thought (AAIDD, 2012). Multiple, unique developmental challenges face these children and their caregivers alike. How these challenges are resolved contributes significantly to the eventual developmental outcomes of children with ID.

ID is not a medical disorder, although it may be coded in a medical classification of diseases. Further, despite its inclusion in all the editions of

B. Tylenda, Ph.D., A.B.P.P. (✉) • R.P. Barrett, Ph.D.
H.T. Sachs, III, M.D.
Alpert Medical School of Brown University,
Providence, RI 02912, USA
e-mail: BTylenda@Lifespan.org

M. Lewis and K.D. Rudolph (eds.), *Handbook of Developmental Psychopathology*,
DOI 10.1007/978-1-4614-9608-3_34,

the APA's *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) (American Psychiatric Association, 1952, 1968, 1980, 1987, 1994, 2000), ID is not a mental disorder. Rather, it is a deviation in development that can increase the risk of mental disorder. In general, the definition and severity of ID refers to a level of behavioral performance without reference to etiology.

Until recently, ID was wrongly viewed as a protective or exclusionary factor in mental disorders. "Diagnostic overshadowing" (Reiss, Levitan, & Szyszko, 1982) attributed most behavioral and emotional abnormalities in those with ID to their cognitive limitations or underlying medical condition. However, it is now clear that individuals with ID have three to four times the general population risk for the full range of psychiatric disorders (cf. Matson & Barrett, 1993). This is not surprising when one considers the factors contributing to developmental psychopathology: biology, learned behavior, psychodynamic issues, social factors, family systems functioning, and cognitive abilities. ID and its various etiologies are likely to impact on most or all of these components. In this regard, individuals with ID and psychiatric disorder represent a unique population requiring the integration of numerous treatment modalities.

Diagnosis of ID

Diagnostic Criteria

While there are several different diagnostic criteria for ID (e.g., WHO, 2008), the authoritative definition of ID is that of the AAIDD (Harris, 2006). The 2010 definition in 11th edition of the *Manual* is essentially the same definition as that from its 2002 *Manual* with the minor edit that substitutes the term ID for MR: "ID is characterized by significant limitations both in intellectual functioning and in adaptive behavior as expressed in conceptual, social, and practical adaptive skills. This disability originates before age 18" (p. 1).

The 2010 *Manual* also provides the listing of five *assumptions* that are now *an explicit part* of the definition because they clarify the context from which the definition arises and indicate how the definition must be applied (AAIDD, 2010). The five AAIDD definition *assumptions* are as follows: "(*1*) Limitations in present functioning must be considered within the context of community environments typical of the individual's age peers and culture; (*2*) Valid assessment considers cultural and linguistic diversity as well as differences in communication, sensory, motor, and behavioral factors; (*3*) Within an individual, limitations often coexist with strengths; (*4*) An important purpose of describing limitations is to develop a profile of needed supports; and (*5*) With appropriate supports over a sustained period, the life functioning of the person with ID generally will improve" (p. 1).

Since 2002, the AAIDD definition no longer includes a classification system based on degrees of severity of ID. This came about from the AAIDD's position of incorporating measured intelligence only in the initial diagnosis. The AAIDD definition emphasizes identification of an individual's specific areas of "ability" rather than "disability" and then classifies the intensity of needed support services in various cognitive and adaptive behavior domains. The intent has been to drive recognition of the relative strengths of individuals with ID and create awareness among service providers, researchers, and policymakers that the population is vastly heterogeneous, even within sets of individuals possessing a common IQ.

Review of APA's *Diagnostic and Statistical Manual of Mental Disorders*, *Fifth Edition* (*DSM-5*) (American Psychiatric Association, 2013), reveals a significant shift of definitional criteria for ID by APA. The APA definition of ID is now quite consistent with the AAIDD definition. The APA *DSM-5* definition for ID (referred to as "Intellectual Disability (Intellectual Developmental Disorder")) is as follows:

> "Intellectual Disability (Intellectual Developmental Disorder) is a disorder with onset during the developmental period that includes both intellectual and adaptive functioning deficits in conceptual, social, and practical domains. The following three criteria must be met:

A. Deficits in intellectual functions, such as reasoning, problem solving, planning, abstract thinking, judgment, academic learning, and learning from experience, confirmed by both clinical assessment and individualized, standardized intelligence testing.

AND

B. Deficits in adaptive functioning that result in failure to meet developmental and sociocultural standards for personal independence and social responsibility. Without ongoing support, the adaptive deficits limit functioning in one or more activities of daily life, such as communication, social participation, and independent living, across multiple environments, such as home, school, work, and community.

AND

C. Onset of intellectual and adaptive deficits during the developmental period." (p. 33)

In doing so, *DSM-5* (American Psychiatric Association, 2013) has not only eliminated the terminology MR to be consistent with AAIDD practices and international opinion, it also has added rigor to wording regarding cultural sensitivity and adaptive behavior functioning.

Clinical Profile of ID Severity Level

While the AAIDD and APA have eliminated ID IQ subtypes from definitional criteria for diagnostic purposes, general clinical profiles are associated with respective identified ranges of cognitive functioning. These profiles of severity levels have been the way that educators, psychologists, adult service providers, and researchers have identified the ID population and are considered to have continued qualitative value (McMillan, Gresham, & Siperstein, 1993, 1995).

Individuals with IQ scores falling in the range of 2 to 3 SD below the mean comprise approximately 85 % of all persons diagnosed with ID (APA, 2000). Comparable limitations with regard to adaptive functioning also are present.

Individuals in this range of functioning often are not identified as having ID until they enter school (Grossman, 1983; Tylenda, Beckett, & Barrett, 2007). Parents of these children, however, frequently report that their child displayed delays in acquisition of developmental speech and motor milestones. Children with cognitive functioning in this range may have a history of referral for speech and occupational therapy services in early childhood. Once identified as having ID, these children become eligible for special education services to aid acquisition of academic, vocational, and life skills. Individuals with cognitive functioning in this range typically develop social and communication skills by age 4 and achieve academic skills approximating the sixth-grade level by their late teenage years. For many children in this range of functioning, cognitive deficits will be misinterpreted as decreased motivation, an oppositional response style, or attention deficits, and appropriate special education interventions will not be sought.

Beyond the school-age years, individuals with cognitive functioning in this range develop sufficient social and vocational abilities to live and work independently without coming to the attention of the professional service community. Some may need assistance when facing unusual personal, economic, or social stressors. Some also will require ongoing vocational, social, and self-care support.

Individuals with IQ scores falling in the range of more than 3 SD below the mean make up the remaining 15 % of all individuals diagnosed with ID (APA, 2000). Comparable limitations with regard to adaptive functioning also are present.

Individuals whose eventual IQ scores fall between 3 and 4 SD below the mean usually are first identified during infancy or early childhood secondary to displaying delays in attaining developmental milestones (Tylenda, Beckett, & Barrett, 2007). Individuals functioning in this range usually develop communication skills in early childhood. With the support of special education services, these individuals may acquire academic skills similar to a second- to fourth-grade student, usually by the period of late adolescence. They usually will be able to interpret social cues but may have difficulty organizing a timely and appropriate response to

social interactions. Even as adults, these individuals will likely require increased support, supervision, and/or assistance in most areas of vocational and daily living in comparison to their mildly affected counterparts. These individuals make up approximately 10 % of the ID population (Harris, 2006).

Individuals whose eventual IQ scores fall 4 to 5 SD below the mean commonly are identified as needing supports/services in infancy as they manifest delays in acquiring motor and language skills (Tylenda et al., 2007). Physical abnormalities are not unusual, and they often have concurrent medical problems. These individuals develop very limited language. While they acquire some basic self-help skills, they cannot function independently and usually require significant daily support and professional supervision throughout their entire lives. This group constitutes 3 to 4 % of those diagnosed with ID (Harris, 2006).

Individuals whose eventual IQ scores fall more than 5 SD below the mean are typically identified as needing supports/services at birth or soon thereafter (Tylenda et al., 2007). Early identification is usually secondary to their apparent physical abnormalities and/or compromise. Such may hinder or preclude the ability to ambulate or speak. Neurological impairments are most common in this group. Skills for simple tasks, such as basic communication, may be learned with frequent repetition and dedicated individual attention. Others will have to take on responsibility for all basic care and activities of daily living for individuals in this range of functioning. This level of care will be lifelong. This group makes up 1 % to 2 % of persons with ID (Harris, 2006).

Epidemiology

The statistical component of the definition of ID would presume a population prevalence of approximately 3 % (2 SD below the mean in a normal distribution of intelligence). An early study by Heber (1961) put the prevalence of ID in the United States at 3 %, a very significant number. A very well-conducted population study of the Isle of Wight by Rutter, Tizard, and Whitmore (1970) confirmed these findings. However, more recent studies (cf. Murphy, Boyle, Schendel, Decoufle, & Yeargin-Allsopp, 1998) have repeatedly demonstrated a prevalence rate equal to or less than 1 % (Baird & Sadovnick, 1985) with males being 50 % more likely to have ID. Males also are at greater risk of genetic abnormality (McLaren & Bryson, 1987) and are more likely to come to professional attention for psychiatric (i.e., aggressive, disruptive behaviors) disorders. Approximately 2.5 million individuals in the United States have ID (Centers for Disease Control, 1996; Committee on Disability Determination for Mental Retardation, 2002), which is identified as the largest categorical disability among children. The prevalence rate of ID in children between the ages of 6 and 17 is 11.4/1,000.

The reasons for the apparent overestimation of ID prevalence are probably multifactorial. The mortality rate for those with moderate-profound ID is elevated (McLaren & Bryson, 1987). Genetic counseling and prenatal testing have decreased the likelihood of children with chromosomal abnormalities. Abortion service for high-risk pregnancies also is a factor. Improved obstetrical techniques have lowered the incidence of brain damage at birth (Harris, 2006). Newborn screening (e.g., PKU), hormone replacement, vaccination, and immunotherapy have nearly eliminated some causes of ID (Alexander, 1998). Finally, the reduction in poverty and improvements in early childhood nutrition and education (e.g., lead exposure) have decreased the rate of mild ID, the classification most likely impacted by environmental variables associated with poverty (Thompson & Hupp, 1992).

Undiagnosed ID also plays a role in suppressing prevalence rates. As individuals become adults and function independently in society, many no longer meet the adaptive impairment criteria of ID.

Etiology

Overall, the potential etiologies for ID are as diverse as they are numerous (Accardo & Capute,

1998). Such reflect the interaction of genetics and environmental factors and can occur prenatally, perinatally, or postnatally during the developmental period (AAMR, 2002). Chromosomal abnormalities, deficits of metabolism, intrauterine infections, and toxic exposure or brain developmental errors are among the prenatal causes of ID (Bale, 2002; Burd, Cotsonas-Hassler, Martsolf, & Kerbeshian, 2003; Jones, Lopez, & Wilson, 2003; Mochida & Walsh, 2004; Rhead & Irons, 2004; Rovet, 2002). Normally developed fetuses may experience perinatal insult. Toxemia, obstetrical trauma, intracranial hemorrhage, hydrocephalus, seizures, and infections may create permanent deficits (Vannucci, 1990). Throughout the child's development, head injuries, infections, seizure disorders, genetic disorders, toxic exposures, and/or environmental deprivation may contribute to a presentation consistent with ID (Matson & Barrett, 1993).

The roles of deprivation and cultural and familial factors have been cited as dominant etiologically for individuals where the degree of ID is less severe. Specifically, low socioeconomic status, maternal education, positive family histories of ID, consanguinity, child abuse, and child neglect have been identified as risk factors for individuals with less severe ID (Zigler, 1967).

However, advances in biomedical technology have enabled identification of a neurobiological etiology for an increasing number of ID syndromes (Harris, 2006). A cause can be confirmed for approximately 80 % of individuals presenting with ID in the more severe forms. Neurobiological factors are also being found to play a significant etiological role in milder ID—where a variety of biomedical abnormalities may be responsible for 30 % to 45 % of cases of milder ID (Lamont and Dennis, 1988). These results have led to a reexamination of the causative role of psychosocial disadvantage and polygenic inheritance in those presenting with ID in the milder form of severity level. Current evidence does not support the existence of the etiological distinction between "organic ID" and "cultural/familiar ID" as newer knowledge about brain functioning and behavior becomes available (Rutter, Simonoff, & Plomin, 1996).

Disabilities Frequently Associated with ID

In comparison to the general population, individuals with ID are more likely to have significant disabilities (Frazier, Barrett, Feinstein, & Walters, 1997). The frequency of associated physical disabilities increases in proportion to the level of cognitive and adaptive delay. Blindness and hearing impairment occur at 20 to 30 times the rate of the general population, respectively (Baroff, 1986), in those with the most severe degree of ID. Cerebral palsy, scoliosis, kyphosis, and other impairments of motor functioning are much more frequent. Even constipation, enuresis, and encopresis have significant impacts on social and life skill development in those with ID (Matson, Anderson, & Bamburg, 2000).

Comorbid psychopathology also is present in the ID population. Rutter et al. (1970) in their study of the Isle of Wight showed that mental illness occurred about five times as often among individuals with ID compared to the non-ID population. Menolascino (1970) published a compendium on psychiatric approaches to ID and coined the term "dual diagnosis" to designate people who have both ID and mental illness simultaneously. A more complete discussion of comorbid psychopathology among individuals with ID can be found later in this chapter.

Finally, difficult behaviors expressed by individuals with ID also are not uncommon. Unfortunately, these difficult behaviors often grab unwanted attention by the public or can create feelings of apprehension and/or danger for family members, professionals, or direct care staff. For individuals with ID who are nonverbal, behavioral disruption may indicate a very mild annoyance to a serious medical or mental condition (Lowe et al., 2007). For example, some of the causative sources for difficult behavior could include internal triggers (e.g., pain, seizure, sensory, fear, psychosis), external triggers (e.g., threats, environmental cues, lack of safety), trauma (e.g., physical, sexual, posttraumatic,

stress), limited range of expression (e.g., through illness, disability, or habit, different emotions are expressed in few visible expressive behaviors), and differentiation from mental illness diagnoses (e.g., overlap of conditions and behaviors which could include behavioral syndromes, learned behaviors, mannerisms, or manifestations of other disease processes such as change in arousal secondary to toxic effects of medication). Each of these categories requires assessment for potential causes, and they are not mutually exclusive of another category. The task for the evaluator is to assess the likely reasons for the disruptive behavior and to address individual and environmental contributions to the situation in which it arose.

Differentiating Children with ID from Children with Other Handicaps

In general, diagnostic issues with individuals who may be presenting with an ID may not be as straightforward as one would think. It is possible that an individual is presenting with an ID, is manifesting dual diagnosis, or is presenting with another disability altogether. Thus, psychiatrists, psychologists, and other evaluators who will be specializing in the diagnosis of ID would be well served to understand the unique concerns that present when undertaking such an evaluation.

It is possible that young children, particularly those of preschool age, who are referred for a first time developmental/cognitive evaluation for clarification of a diagnosis of ID are actually manifesting either (1) a diagnosis other than ID or (2) one or more additional diagnoses concomitant with the diagnosis of ID. Consequently, an evaluator who will be specializing in the evaluation and diagnosis of ID should be well versed in the presenting features and profiles for a range of other disorders and problems frequently diagnosed in the preschool and school-age population. Table 34.1 lists a range of these disorders and problems.

Table 34.1 List of disorders and problems to consider when a child is referred for a diagnostic evaluation to "rule in/rule out" intellectual disability

When conducting an assessment with a child for a possible diagnosis of intellectual disability (ID), the evaluator needs to consider the following range of disorders or problems as possible alternative or additional diagnoses/conditions:
1. Developmental delay
2. The "umbrella of neurological impairment" which includes:
(a) Autism spectrum disorder[a]
(b) Childhood disintegrative disorder
(c) Neurological inefficiency/nonverbal learning disability
(d) Attention deficit/hyperactivity disorder
3. Language/communication disorder
4. Hearing impairment
5. Visual impairment
6. Cerebral palsy
7. Rett's syndrome
8. Motor coordination disorder
9. Regulatory disorder
10. Reactive attachment disorder
11. Elective mutism
12. Psychosocial deprivation
13. Other psychiatric condition
14. Some form of a behavioral disorder
15. Dyadic problem between caretaker and child
16. Challenging temperament and/or inconsistency of temperament between caretaker and child

[a]Childhood disorders subsumed under "autism spectrum disorder" in *DSM-5* include (1) pervasive developmental disorder, not otherwise specified; (2) high-functioning autism; and (3) Asperger's disorder

Components of a Comprehensive Evaluation

The determination of ID is rarely a simple matter. While assessment of an individual's cognitive and adaptive behavior functioning during the developmental period is the core criteria for diagnosis of ID, a comprehensive evaluation for ID typically goes beyond this. A comprehensive evaluation includes consideration of genetic and nongenetic etiologies and assessment of cognitive and adaptive behavior functioning. Associated medical conditions (e.g., cerebral

palsy or a seizure disorder) and mental, emotional, and behavioral problems should also be closely reviewed as they may influence cognitive functioning. A comprehensive evaluation may involve several assessment visits over time and, for a younger child, may occur within the context of a multidisciplinary treatment team.

Determining the etiology of ID can be critical. If a genetic disorder is suspected, genetic testing may be used to confirm a diagnosis. Confirmation of a genetic disorder also may lead to uncovering other associated medical and behavioral features requiring attention. Information on the etiology of ID may impact trajectory as some developmental disorders can be arrested or sometimes prevented through early detection and treatment. Knowledge of etiology can inform for the future of the individual with ID or other family members as they consider their own family planning. Finally, etiological clarification can identify an appropriate support group and, in certain cases, facilitate funding for special services.

A full medical history and a thorough physical examination are first steps to deciphering the etiology of ID. Following these events, laboratory and diagnostic studies are chosen based on the clinical findings. Formal evaluation of cognitive and adaptive behavior functioning is also completed along with evaluation of comorbid emotional and/or behavioral problems. A comprehensive treatment plan is created based on all evaluation findings which may include medical, psycho-educational, genetic, and/or behavioral counseling and family support services.

Not all evaluations will result in a clear etiology or a diagnosed condition. The cause for ID can be identified in 40 % to 60 % of those undergoing an evaluation (Curry et al., 1997). Diagnostic accuracy is gradually increasing with improved neuroimaging techniques and cytogenetic techniques (Harris, 2006).

Harris (2006) has provided a comprehensive review of guidelines for a medical and genetic evaluation of an individual with ID. Tylenda et al. (2007) have provided an extensive examination of various standardized verbal and nonverbal intelligence tests useful in assessing for ID in individuals across the age range (and who may present with various forms of concomitant challenge). They review in detail the history, conceptual bases, method of test construction, psychometric properties, testing procedures, scoring protocols, and examiner qualifications, as well as indications and contraindications for the use of each test. They also walk professionals through the intricacies of accurate intelligence testing and reporting of results for children and adolescents with ID. A companion review for the assessment of adaptive behavior functioning can be found in Dixon (2007). Aman has (1991a, 1991b) reviewed instruments for assessing emotional and behavioral disorders in individuals at all levels of ID.

Developmental Challenges for Individuals with ID

Development is a complex process of growth and change through which children acquire a variety of skills and abilities that allow them to understand and function in their world. The normal trajectory of development enables a child to progress from complete dependency on others to near- or complete independency for his/her needs and well-being. Although there is great variability in development, there are earlier and later limits to what is considered "normal/typical" development. Statistically, children with ID are those who develop at a rate significantly below average—the lowest 3 % on the normal, bell-shaped curve distribution—indicating why ID is called a "developmental disability."

These children make progress at a rate that is significantly slower than is expected of children their age. However, just as the development of children who do not have ID varies, so does the development of children with ID. Further, for any child with ID, the development for different areas (i.e., cognitive abilities, language and speech skills, gross motor skills, fine motor skills, social-emotional skills, and play skills) may proceed at different rates, on different timelines, and in different orders. As a result, the timetable for achievement of developmental milestones for a child with ID can be difficult to predict as well as

eventual developmental trajectories. However, over time, such trajectories become more predictable based on the child's prior rates and breadth of performance. Variables key to the eventual developmental progression for any child with ID will include the following: (1) the child's inborn biological and neurological capacity (which set the general limits for the rate and eventual endpoints of development), (2) the ongoing environmental factors to which the child is exposed (e.g., type and amount of stimulation), (3) any associated disabilities or medical problems of the child, and (4) the child's support network's ability to assist him/her in addressing these challenges.

Disruption of Mastery of Developmental Skills Specific to Developmental Periods

The mastery of developmental "skills" specifically associated to each developmental period can present unique challenges for a child or adolescent with ID. A disruption in the mastery of specific developmental skills also can present unique challenges for the child or adolescent's caregivers and family, in how they initiate as well as respond to the child.

Infancy

Infancy is characterized by the development of attachment, self-regulation, and environmental awareness and exploration (Lieberman & Pawl, 1988). ID and associated disorders may disrupt mastery in each of these areas. Many developmental processes in infancy focus on strengthening attachment. Eye contact, a social smile, and cooing and other vocalizations often are delayed or nonexistent in children with ID. For infants with significant neurological or physical disabilities, uncertainty about their survival or prognosis, long-term postnatal hospitalization, or prolonged intrusive medical interventions also inhibit normal attachment.

Families often experience anger, denial, sorrow, and a prolonged grieving process (Lewis & MacLean, 1982) in response to having a child with ID. This also may interfere with the attachment process. In children with more subtle delays, the inability to achieve milestones at expected intervals may lead to misgivings about parental skills and increasing frustration. Autism spectrum disorders (American Psychiatric Association, 2013), often associated with ID, create further obstacles to attachment.

Delays in motor coordination and exploration of the environment often create a greater dependence on caregivers that is a harbinger of future interactive patterns. This may be enhanced by comorbid medical disorders, such as seizures, that enhance parental vigilance. Conversely, social withdrawal and isolation are frequent presentations.

Early Childhood

Many children's ID and associated delays are identified during this period. Parental response, both emotionally and in terms of expectations, impacts on this period of personal mastery. Maintaining unrealistic expectations of trying to "prove the experts wrong" leads to increasing frustration and tension in the parent–child relationship. Conversely, removing or minimizing expectations may inhibit the development of many key skills. This may create an environment of overprotection or chronic parental apathy that squelches individual initiative.

Language development is usually delayed in persons with ID. Mild delays are often overlooked or misinterpreted. Early intervention, which can be very helpful, is often unintentionally delayed. Deficits in language and communication development are some of the best predictors of behavioral difficulties in children with developmental disabilities (Carr & Durand, 1985). Frustration at not being able to communicate needs or desires may lead to disruptive, aggressive, or self-injurious behavior. Social failures are often the result of an inability to follow the flow of communication and basic interpersonal cues. Isolation or increased reliance on selected caregivers may be inadvertently reinforced. In this regard, it is important to recognize that children with specific language disorders may develop effective alternative communication systems to express their needs (Bondy & Frost, 1994).

Self-care skills are frequently delayed. Associated fine and gross motor delays may prevent children from successfully dressing, going to the toilet, or feeding themselves. Children with less severe delays may express the desire to perform these tasks without the requisite skills. This may lead to increasing conflict with caregivers. For those with a more severe form of ID, there is often a lifelong inability to contribute effectively to activities of daily living. Opportunities for child care may be greatly reduced by the child's lack of self-care skills. Unfortunately, it is just those parents who must continue providing their children with intensive assistance who would benefit most from more readily available child care.

Spontaneous, meaningful play may be delayed or missing. Children with a more severe form of ID may engage in seemingly undirected or self-stimulatory behavior instead of appropriate play. Children with lesser delays may only develop some symbolic play as they are about to enter school. Isolated or parallel play may predominate, especially when communicative skills are significantly impaired.

As with all children, many factors during this formative period contribute to personality development. The challenges of skill mastery, communication, emotional and physiological self-regulation, and how caregivers address these issues have significant implications. For children with milder severity ID, self-esteem, trust, and perceived competence form the basis of interpersonal relationships and a sense of self in the world. For those with more severe delays, the caregiver's ability to assist the child effectively in regulating responses to internal and environmental stimuli helps create a lifelong style of behavior.

Childhood

For many children with ID, beginning school is the first exposure to a large number of children without disabilities. It may be the first time descriptors such as "intellectually disabled," "slow learner," or other pejorative terms are encountered. This may be particularly challenging to children with mild ID. While increased academic mainstreaming has elevated the awareness of many typical children regarding disabilities, children with ID often still are perceived as different and are the target of peer taunting and rejection. As important, they perceive themselves as different. This becomes particularly challenging in the later elementary grades as peers become less tolerant of anyone seen as different. It is also a time when children with mild to moderate severity ID become increasingly aware of their limitations. Social withdrawal, isolation, and depression often manifest during this period. Some children display externalizing or acting-out behavior as an increasing desperation to be socially accepted coincides with peers increased willingness to use them as foils.

Participation in extracurricular and community activities is a hallmark of this age. Athletics may be inaccessible for some with significant associated physical handicaps. The nationwide Special Olympics initiative and greater understanding and support from many school districts have increased the participation rate of children with ID and other developmental delays in athletics. Group activities such as scouting have created subgroups that are more geared toward children with special needs but may isolate them from the mainstream, increasing their awareness of perceived differences. Dance and martial arts classes are often very well received by parents and children alike.

Most children with ID need support in the classroom in terms of either special resource support or placement in a self-contained special education classroom. As peers tackle more demanding language and abstract concepts, children with mild ID increasingly struggle to keep up. Academic failures are common. For children with more severe delays, the goals of education often change from preparation for higher education to life skills and vocational activities, further differentiating them from peers. The rigid demands of an academic schedule may be very different from the previous flexibility of home. Children with ID will have greater difficulty adapting to this change. This difficulty often will be expressed behaviorally as they are unable to convey via communicative

skills the ensuing frustration and confusion. The subtleties of communication and behavioral routines, well learned by families, may be lost on teachers caring for numerous children with varying special needs.

Adolescence

Adolescence is a challenging period for every teen as well as for those living and working with them. Physical changes, striving for greater independence, and social acceptance are even more difficult for teens presenting with cognitive and adaptive skill deficits.

Increasing sensitivity of the erogenous zones may lead to inappropriate touching in children unable to master social rules. Some females with more severe levels of ID may be unable to understand the physical sensation of menstruation. Physical discomfort may lead to increased irritability, self-injury, and aggression (Kaminer, Feinstein, Barrett, Tylenda, & Hole, 1988). Personal hygiene also may be a problem.

The commonly held prejudice that those with ID are likely perpetrators of sexual assaults belies the reality that they more likely may be victims of sexual mistreatment or abuse (Ammerman, Hersen, van Hasselt, Lubetsky, & Sieck, 1994). Adolescence is a particularly risky period. Many children at this age are living in institutional settings, such as residential group homes, which may further increase the risk of abuse.

Deficits in social skills are particularly debilitating during this period (Borden, Walters, & Barrett, 1995). Complex social interactions, rapidly changing trends, and group cohesiveness are the norm. It is very challenging for adolescents with mild ID to keep up with their developmentally intact age-mates. Friendships with non-delayed peers, which may have flourished for years, are strained as these peers begin dating, working, and expressing their own independence. Children with ID may have the same dreams and expectations as their peers. Status symbols such as driving or a "cool" job may be out of reach. Medical or neurological conditions may contribute to physical abnormalities at an age when personal appearance has heightened significance. Depression and withdrawal are common as social failures accumulate. Suicidal ideation is not unusual (Walters, Barrett, Knapp, & Borden, 1995). Relationships with adults are often more rewarding. Somatization or creative storytelling may increase as a means of soliciting professional help or to otherwise fill the void of loneliness.

Academic challenges often change during this period. Vocational skill development predominates. Those with more severe levels of ID will frequently be taught repetitive "prevocational" tasks that are often minimally rewarding. For adolescents with milder delays, attending vocational classes may be stigmatizing and a source of shame (Lewis, 1998). Self-worth may diminish rapidly as teens with ID come to blame themselves for having a developmental disability.

Families face different stressors. Parents are aging and may feel increasingly overwhelmed by the demands of a teenager with ID. The normal developmental trajectory of increasing child independence may be disrupted, forcing families to face issues that have previously been avoided. For example, the opportunity for increased freedom for parents as their children leave the nest may prove illusory. In addition, issues of long-term care may arise when parents are no longer able to support more seriously delayed children. This is frequently a period when professional agencies become involved in the child's life to plan vocational, social, and residential opportunities. However, many families find it stressful to relinquish some or all of the care of their children to others. Brothers and sisters may be overprotective or resentful of a sibling with special needs and find that the changes of adolescence have a greater impact on family functioning.

Adulthood

As individuals with mild forms of ID reach adulthood, some might no longer be considered intellectually disabled. Relieved of the imposed structure of academics, some individuals with ID find appropriate jobs and housing arrangements that allow them to live independently. Unfortunately, stressors around child rearing, occupational or financial matters, and social relationships may lead to minor or major regression that requires

additional family or professional support. Others with milder severity levels of ID receive support around occupational, residential, and social issues, as needed. Self-motivation and time management may be a challenge when rewards (i.e., a paycheck) are not immediate. In less supported worksites, relationships with coworkers can be challenging. Being criticized or taken advantage of is common.

Managing home finances, food shopping, laundry, and other life skills may be daunting tasks for individuals with milder severity levels of ID (Antonello, 1996). Those individuals are more at risk for con artists and "scams." Some engage in inappropriate or illegal activities without understanding the full import of their actions. Many have legal guardians. Sometimes, individuals and guardians disagree on issues, which may lead to tension and frustration. All of these factors may contribute to greater dependence on others than the individual desires.

Those with ID may have physical handicaps, limited mobility, or limited access to transportation. This enhances difficulties in keeping appointments and job expectations and creates further isolation. Individuals with more severe levels of ID in adulthood often work in a very structured and supportive environment. Behavioral issues such as aggression, self-injury, compulsions, and opposition are common and limit productivity. Ongoing assistance around self-care skills requires close supervision by family or agencies.

Family Stress

Having a child with ID can change family life in many ways, impacting on the family's time, finances, and physical and emotional energy. Krantz (1993) extensively reviewed the many facets of family life that can be affected while raising a child with ID.

Family stress is often significantly increased while parents care for a child with ID. These children will often have coexisting conditions requiring frequent medical involvement (Knoll, 1992). The paroxysmal nature of seizures, loss of functioning in numerous associated degenerative genetic disorders, and monitoring of multiple medications and their potential side effects contribute to heightened parental vigilance and a perception of greater fragility in the child.

The normal maturation of the family is inhibited or may even regress as the child fails to meet developmental milestones. Constant advocacy for special education and support services diverts energy from other family responsibilities. Parental career progress may be impacted. Siblings often feel ignored or overburdened and may exhibit problematic behaviors (Lobato, 1990; Stoneman & Berman, 1993). The family's sense of loss and guilt in not having the anticipated "perfect child" cannot be overlooked (Lewis, 1998). "Managing" extended family dynamics also can be extremely challenging when, for example, grandparents consistently respond that there is nothing wrong with their grandchild and that the child's parents are "reading too much into things" when developmental progression begins to fall behind or goes awry.

Parents may have to master many skills unique to their situation (Baker, 1989). More intensive behavioral management techniques or medical care interventions may be needed. Up to 80 % of children with developmental delays have significant sleep difficulties (Quine, 1986). This may lead to pervasive family sleep deprivation that further increases household stress and limited adaptability. There is also an increased incidence of ID and illiteracy in family members of probands with ID. This may impact on a family's coping ability in the face of the unique demands associated with raising a child with ID (Knoll, 1992).

Finally, children and adolescents with ID have three to four times the general population risk for the full range of psychiatric disorders (cf. Matson & Barrett, 1993). This can lead to a family becoming totally overwhelmed (Dykens, 2000), particularly if periods of inpatient psychiatric hospitalization are required. Sometimes, hospitalization of the child requires the temporary domestic relocation of a family member near to where the child is hospitalized, given the limited number of inpatient facilities that treat

children and adolescents with ID and comorbid psychopathology. Understanding of the various psychotropic medications used for children and adolescents with ID and a coexisting psychiatric condition may be unfamiliar territory for the family. Equally unfamiliar may be the necessity of treating the child's psychiatric condition via multiple medication trials, various behavioral intervention strategies, and ongoing data collection. Such also requires the close co-collaboration and development of trust with the hospital clinical team which can become a long-standing relationship as the child may move between inpatient, day treatment, outpatient, and/or residential care.

Comorbid Psychopathology for Individuals with ID

The incidence of psychopathology in those with ID is elevated above the rate observed in non-ID individuals (Dekker & Koot, 2003; Reiss, 1990). One study (Einfield & Tonge, 1996) found that 20 % of children with an IQ less than 70 had severe emotional or behavioral disorders, and only 1 in 10 of these children and adolescents with major psychiatric disorders had received specialized psychiatric care. Individual and family stress, neurological impairment, sensory deficits, and limited adaptive skills increase the risk of developing psychopathology.

Controversy persists as to the efficacy of *DSM* diagnoses in those with greater severity of ID. It is not clear if abnormal behaviors seen in this population meet the specific criteria for psychiatric disorders. However, in those with milder severity ID, presentations meeting *DSM* diagnostic criteria are readily apparent (Dekker & Koot; 2003; Harden & Sahl, 1997; Szymanski, 1994).

Abnormal behaviors are often the presenting complaint of dually diagnosed children and adolescents. More often than not, a functional analysis of behaviors, including review of antecedent issues and behavioral consequences, is a necessary first step to better understand the etiology of a given behavior. More recently, professionals in the field have been able to supplement these behavioral analyses with the development of many new diagnostic scales specific to the ID population across a variety of psychiatric disorders. Matson (2007) has extensively reviewed this body of assessment materials with thoughtful commentary regarding diagnostic efficacy with regard to this population for the following areas: self-injurious behavior, aggressive behavior, feeding disorders, pain, and depression, anxiety, and related disorders.

Self-Injurious Behavior

Self-injurious behavior (SIB) is common in persons with ID, occurring in 16 % of the population (cf. Barrett, 2008). Prevalence rates vary in accordance with the level of severity of ID. Self-injury is rare (1 %) in children with mild ID, but more common in children with moderate ID (9 %), severe ID (16 %), and profound ID (27 %). Self-injurious responses may range from skin picking and head-banging to severe self-mutilation. There is an inverse correlation between the amount and severity of SIB and expressive language and cognitive development (Schroeder, Schroeder, Smith, & Dalldorf, 1978). Self-injury may be a final common pathway for several psychiatric and behavioral phenomena (Schroeder, Oster-Granite, & Thompson, 2002). Identifying specific etiologies will presumably dictate appropriate treatment interventions.

Inadvertently reinforced self-injury may occur in children with limited communication skills or sensory deficits (Barrett, 2008). These children appear to learn rapidly that engaging in self-mutilating behaviors commands instant and close attention from caregivers. Often, these behavioral patterns arise from periods of physical discomfort such as headaches, earaches, dental pain, menstrual periods, constipation, or eczema. The child's inability to describe discomfort leads to physical expression of pain and frustration.

For some children, self-injury appears to be internally reinforced by the release of endogenous opiates (Barrett, Feinstein, & Hole, 1989). Repeated self-injury apparently induces endorphin release and a temporarily favorable sensory consequence. In these situations, medications that

inhibit the effectiveness of endogenous opiates effectively extinguish the drive to self-injury (Sandman et al., 1998).

Some SIBs suggest an underlying affective disorder or obsessive–compulsive disorder (Matson, 1986). Cyclic presentations, co-occurring vegetative symptoms, and associated affective changes may indicate a mood disorder. In milder severity ID, a child or adolescent will often be able to describe depressed mood, anhedonia, or even manic symptoms. The apparent ego-dystonic nature of self-injury and the desire for imposed physical restraint to prevent the acts in some are consistent with a compulsive drive. This may be difficult to differentiate from stereotypical behaviors often seen in individuals with severe developmental delays. Interestingly, medications that address mood disorders and obsessive–compulsive behaviors have diminished SIB in some individuals (Cook, Rowlett, Jaselskis, & Leventhal, 1992; Kastrom, Finesmith, & Walsh, 1993; King, 2000). Mental disorders such as Lesch–Nyhan syndrome, and chromosomal abnormalities such as Cornelia de Lange syndrome (Opitz, 1985), have phenotypic presentations that include severe self-injury.

Abnormal Movements

Individuals with ID and associated neurological impairments are at increased risk for movement disorders. Motor tics and Tourette's syndrome are common in this population. Differentiating motor tics from stereotypies and vocal tics from echolalia is challenging and impacts treatment recommendations. Vocal and complex motor tics often are misdiagnosed as oppositional, aggressive, and disruptive behaviors.

Many adolescents and young adults with ID have been on neuroleptics for extended periods, targeting aggression or disruptive behavior. Comorbid neurological abnormalities increase the risk of tardive dyskinesia, involuntary muscle movements often centering on the oral musculature. The use of neuroleptics also may increase a sense of restlessness, known as akathisia. This may present as hyperactivity, irritability, or dysphoria and, if not properly diagnosed, could lead to a cascade of inappropriate psychopharmacological and behavioral interventions (Wilson, Lott, & Tsai, 1998).

Aggressive/Destructive Behaviors

Aggressive behaviors appear in approximately 25 % of the community sample of children and adolescents with developmental disabilities (Emerson, 2003). These behaviors contribute significantly to the social isolation and institutional placement of this population (Hill & Bruininks, 1981). Frustration stemming from communication deficits is a common source of aggressive and destructive behavior. Adjustment difficulties created by environmental changes may be expressed aggressively. Disruption in living arrangements, support staff, or routine that is not understood or explained can lead to significant outbursts. If these outbursts lead to the reinstatement of the status quo, such behavioral patterns are strongly reinforced.

Mood disorders, both major depression (Dosen, 1984) and mania (Sovner, 1989), may present as disruptive behavior. This is especially challenging to diagnose in nonverbal children. Associated changes in sleep, appetite, and energy patterns are frequently seen. There is also often an ebb and flow to the presenting symptoms.

Previous traumatic events including abuse and neglect may create a long-standing pattern of disruptive behaviors in an attempt at self-protection. Withdrawal and social isolation often may be seen in these situations. The interictal, or between-seizure, phase in some seizure disorders may increase aggressive tendencies. Comorbid schizophrenia or substance abuse may lead to increased confusion and poor impulse control, exacerbating aggressive tendencies.

Attention and Motivational Deficits

Attention difficulties and motivational difficulties are found as common presentations for numerous disorders, including attention deficit/hyperactivity

disorder (ADHD), depression, mania, anxiety, and hyperthyroidism. Differentiation of these disorders in cognitively limited and often medically complicated populations is particularly challenging but important. There is evidence that treatments helpful to otherwise intact children with ADHD are useful for ID children with a diagnosis of ADHD, but their rate of beneficial response appears to be well under that found for those children in the normal IQ range, and their response to treatment shows great variability (Aman, Buican, & Arnold, 2003). The symptom presentation in these individuals may represent underlying neurocognitive deficits in attention and arousal, or other psychiatric disorders, and not ADHD per se. Medications for seizures or other medical conditions as well as sensory deficits also may diminish attention (cf. Aman & Singh, 1988; Reiss & Aman, 1998).

Anxiety, Mood Disorders, and Related Disorders

Rates of significant anxiety symptoms appear to be much higher in children with ID, approximately 25 % (Benson, 1985), than the general population estimate of 2–5 % (Clum & Pickett, 1984). The typical predominance of females is not seen. There is an increased prevalence for anxiety disorders in specific neurogenetic syndromes (Dykens, 2003). It remains unclear if an individual's level of cognitive delay contributes to the severity and frequency of anxiety disorders.

Mood disorders (e.g., major depression, bipolar disorder, and dysthymia) occur commonly in individuals with ID. Approximately 2 % to 10 % of individuals with ID manifest major affective disorders, and approximately 25 % suffer from dysthymia (Cooper, Melville, & Einfeld, 2003). Cain et al. (2003) reported that bipolar disorder can be distinguished from behavioral and psychiatric diagnoses in adults with ID. Specifically, those with clinical symptoms of bipolar disorder had significantly more mood-related and non-mood-related symptoms and greater functional impairment than those with major depression.

The inability of many with developmental delays to describe internal states accurately is particularly problematic around anxiety disorders and mood disorders. Careful observation is often the most helpful diagnostic tool. One must differentiate general anxiety, avoidance, panic, posttraumatic stress, and obsessive–compulsive behaviors from several other diagnoses. This list should include mania, ADHD, stereotypical movements, akathisia, hyperthyroidism, and seizures.

Limited adaptability, sensory deficits, concrete thought processes, and increased family stress also may contribute to an increased rate of anxiety symptoms in this population. If identified, these symptoms are frequently responsive to typical interventions (cf. Barlow, 1993; Reiss & Aman, 1998; Werry & Aman, 1993).

Adjustment Disorders

Those with ID often thrive and rely on consistency in routine. Even minor changes in the environment can have a disproportionately significant impact on behavior and mood. Careful analysis of recent changes in living situation, education, or caretaker interaction is necessary when sudden changes in behavior are seen. The *DSM* criterion of resolution of symptoms in 6 months is not always relevant in this population. It is not unusual for children with ID to be excluded from full or partial explanations for the sudden disappearance of a relative from death or moving. Entering the school situation where one is teased or ignored can lead to withdrawal or provocative behaviors across settings. Entering or leaving institutions may create changes in mood or behavior as long-standing routines are disrupted. Understanding underlying issues is essential for efficacious treatment.

Conclusion

The societal perception of individuals with ID and, consequently, their social acceptance has changed dramatically over the past 40 years. The

results of parent advocacy, deinstitutionalization, academic and vocational mainstreaming, increased respect for individual rights, increased interdisciplinary collegiality between various professional groups, and the de-emphasis of chemical restraint have caused an enormous shift in paradigms for treatment and support. While challenges remain for individuals with ID and their families, advances in understanding and treating children with ID, while still evolving, have resulted in marked improvements in the quality of life.

References

Accardo, P. J., & Capute, A. J. (1998). Mental retardation. *Mental Retardation and Developmental Disabilities Research Reviews, 4*, 2–5.

Alexander, D. (1998). Prevention of mental retardation: Four decades of research. *Mental Retardation and Developmental Disabilities Research Reviews, 4*, 50–58.

Aman, M. G. (1991a). *Assessing psychopathology and behavioral problems in persons with mental retardation: A review of available instruments.* Rockville, MD: S. Department of Health and Human Services (DHHS) (DHHS Publication No. [ADM] 91-1712.).

Aman, M. G. (1991b). Review and evaluation of instruments for assessing emotional and behavioral disorders. *Australia and New Zealand Journal of Developmental Disabilities, 17*, 127–145.

Aman, M. G., Buican, B., & Arnold, L. E. (2003). Methylphenidate treatment in children with borderline IQ and mental retardation: Analysis of three aggregated studies. *Journal of Child and Adolescent Psychopharmacology, 13*, 29–40.

Aman, M. G., & Singh, N. N. (Eds.). (1988). *Psychopharmacology of the developmental disabilities*. New York: Springer.

American Association on Intellectual and Developmental Disabilities (AAIDD). (2010). *Intellectual disability: Definition, classification, and systems of support* (11th ed.). Washington, DC: Author.

American Association on Intellectual and Developmental Disabilities (AAIDD). (2012). *User's guide—To accompany the 11th edition of intellectual disability: Definition, classification, and systems of support*. Washington, DC: Author.

American Association on Mental Retardation (AAMR). (2002). *Mental retardation: Definition, classification, and systems of support* (10th ed.). Washington, DC: Author.

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders* (1st ed.). Washington, DC: Author.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: Author.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., revised). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

Ammerman, R. T., Hersen, M., van Hasselt, V., Lubetsky, M. J., & Sieck, W. R. (1994). Maltreatment in psychiatrically hospitalized children and adolescents with developmental disabilities: Prevalence and correlates. *Journal of the American Academy of Child and Adolescent Psychiatry, 33*, 567–576.

Antonello, S. J. (1996). *Social skills development. Practical strategies for adolescents and adults with developmental disabilities*. Boston, MA: Allyn & Bacon.

Baird, P. A., & Sadovnick, A. D. (1985). Mental retardation in over half a million consecutive live births: An epidemiologic study. *American Journal of Mental Deficiency, 89*, 323.

Baker, B. L. (1989). *Parent training and developmental disabilities*. Washington, DC: American Association on Mental Retardation.

Bale, J. F. (2002). Congenital infections. *Neurology Clinics, 20*, 1039–1060.

Barlow, D. (Ed.). (1993). *Clinical handbook of psychological disorders* (2nd ed.). New York: Guilford Press.

Baroff, G. S. (1986). *Mental retardation: Nature, causes, and management* (2nd ed.). Washington, DC: Hemisphere.

Barrett, R. P. (2008). Atypical behavior: Self-injury and pica. In M. L. Wolraich, D. D. Drotar, P. H. Dworkin, & E. C. Perrin (Eds.), *Developmental-behavioral pediatrics: Evidence and practice* (pp. 871–885). Philadelphia, PA: Mosby Elsevier.

Barrett, R. P., Feinstein, C., & Hole, W. (1989). Effects of naloxone and naltrexone on self-injury in autism: A double blind, placebo controlled analysis. *American Journal on Mental Retardation, 93*, 644–651.

Benson, B. A. (1985). Behavioral disorders in mental retardation: Association with age, sex, and level of functioning in an outpatient clinic sample. *Applied Research in Mental Retardation, 6*, 79–85.

Berkson, G. (2004). Intellectual and physical disabilities in prehistory and early civilization. *Mental Retardation, 42*, 195–208.

Bondy, A. S., & Frost, L. A. (1994). The picture exchange communication system. *Focus on Autistic Behavior, 9*, 1–19.

Borden, M. C., Walters, A. S., & Barrett, R. P. (1995). Mental retardation and developmental disabilities. In V. van Hasselt & M. Herson (Eds.), *Handbook of adolescent psychopathology: A guide to diagnosis and treatment* (pp. 497–524). New York: Lexington Books.

Buntinx, W. H. E. (2006). The relationship between WHO-ICF and the AAMR-2002 system. In H. Switzky & S. Greenspan (Eds.), *What is mental retardation? Ideas for an evolving disability in the 21st century* (pp. 303–323). Washington, DC: American Association on Mental Retardation.

Burd, L., Cotsonas-Hassler, T. M., Martsolf, J. T., & Kerbeshian, J. (2003). Recognition and management of fetal alcohol syndrome. *Neurotoxicology and Teratology, 25*, 681–688.

Cain, N. N., Davidson, P. W., Burhan, A. M., Andolsek, M. E., Baxter, J. T., Sullivan, L., et al. (2003). Identifying bipolar disorders in individuals with intellectual disability. *Journal of Intellectual Disability Research, 47*, 31–38.

Carr, E. G., & Durand, V. M. (1985). Reducing behavior problems through functional communication. *Journal of Applied Behavior Analysis, 18*, 111–126.

Centers for Disease Control. (1996). State-specific rates of mental retardation—United States. 1993. *Morbidity Mortality Weekly Reports, 45*, 61–65.

Clum, G. A., & Pickett, L. (1984). Panic disorder and generalized anxiety disorders. In P. B. Sutkon & H. Adams (Eds.), *Comprehensive handbook of psychopathology* (pp. 123–146). New York: Plenum Press.

Committee on Disability Determination for Mental Retardation. (2002). Introduction. In D. J. Reschly, T. G. Myers, & C. R. Hartel (Eds.), *Mental retardation: Determining eligibility for social security benefits* (pp. 15–37). Washington, DC: National Academy Press.

Cook, E. H., Rowlett, R., Jaselskis, C., & Leventhal, B. L. (1992). Fluoxetine treatment of children and adults with autistic disorder and mental retardation. *Journal of the American Academy of Child and Adolescent Psychiatry, 31*, 739–745.

Cooper, S. A., Melville, C. A., & Einfeld, S. L. (2003). Psychiatric diagnosis, intellectual disabilities, and diagnostic criteria for psychiatric disorders for use with adults with learning disabilities/mental retardation (DC-LD). *Journal of Intellectual Disability Research, 47*(suppl. 1), 3–15.

Curry, C. J., Stevenson, R. E., Aughton, D., Byrnes, J., Carey, J. C., Cassidy, S., et al. (1997). Evaluation of mental retardation: Recommendations of a consensus conference: American College of Medical Genetics. *American Journal of Medical Genetics, 72*, 468–477.

Dekker, M. C., & Koot, H. M. (2003). DSM-IV disorders in children with borderline to moderate intellectual disability. II: Child and family predictors. *Journal of the American Academy of Child and Adolescent Psychiatry, 42*, 923–931.

Dixon, D. R. (2007). Adaptive behavior scale. In J. L. Matson (Vol. Ed.), *International review of research in mental retardation: Vol. 34. Handbook of assessment in persons with intellectual disability* (pp. 99–140). San Diego, CA: Elsevier.

Dosen, A. (1984). Depressing conditions in mentally handicapped children. *Acta Paedopsychiatrica, 50*, 29–40.

Dykens, E. M. (2000). Psychopathology in children with intellectual disability. *Journal of Child Psychology and Psychiatry, 41*, 407–417.

Dykens, E. M. (2003). Anxiety, fears, and phobias in persons with Williams syndrome. *Developmental Neuropsychology, 23*, 291–316.

Einfield, S. L., & Tonge, B. J. (1996). Population prevalence of psychopathology in children and adolescents with intellectual disability: II. Epidemiological findings. *Journal of Intellectual Disabilities Research, 40*, 99–109.

Emerson, E. (2003). Prevalence of psychiatric disorders in children and adolescents with and without intellectual disability. *Journal of Intellectual Disability Research, 47*, 51–58.

Frazier, J., Barrett, R. P., Feinstein, C. B., & Walters, A. S. (1997). Moderate to profound mental retardation. In J. Noshpitz (Ed.), *Handbook of child and adolescent psychiatry* (Vol. 4, pp. 397–408). New York: Wiley.

Grossman, H. J. (1983). *Classification in mental retardation*. Washington, DC: American Association on Mental Deficiency.

Harden, A., & Sahl, R. (1997). Psychopathology in children and adolescents with developmental disorders. *Research in Developmental Disabilities, 18*, 369–382.

Harris, J. C. (2006). *Intellectual disability: Understanding its development, causes, classification, evaluation, and treatment*. New York: Oxford University Press.

Heber, R. (1961). *Epidemiology of mental retardation*. Springfield, IL: Charles C. Thomas.

Hill, B. K., & Bruininks, R. H. (1981). *Physical and behavioral characteristics and maladaptive behavior of mentally retarded individuals in residential facilities*. Minneapolis: University of Minnesota, Department of Psychoeducational Studies.

Jones, J., Lopez, A., & Wilson, M. (2003). Congenital toxoplasmosis. *American Family Physicians, 67*, 2131–2138.

Kaminer, Y., Feinstein, C., Barrett, R. P., Tylenda, B., & Hole, W. (1988). Menstrually related mood disorders in developmentally disabled adolescents: Review and current status. *Child Psychiatry and Human Development, 18*, 239–249.

Kastrom, T., Finesmith, R., & Walsh, K. (1993). Long-term administration of valproic acid in the treatment of affective symptoms in people with mental retardation. *Journal of Clinical Psychopharmacology, 15*, 448–451.

King, B. H. (2000). Pharmacological treatment of mood disturbance, aggression, and self-injury in persons with developmental disabilities. *Journal of Autism & Developmental Disabilities, 30*, 439–445.

Knoll, J. (1992). Being a family: The experience of raising a child with a disability or chronic illness. In V. J. Bradley, J. Knoll, & J. M. Agosta (Eds.), *Emerging*

issues in family support (pp. 9–56). Washington, DC: American Association on Mental Retardation.

Krantz, J. Z. (1993). Family and community life. In R. Smith (Ed.), *Children with mental retardation* (pp. 259–301). Bethesda, MD: Woodbine House.

Lamont, M. A., & Dennis, N. R. (1988). Aetiology of mild mental retardation. *Archives of Diseases of Children, 63*, 1032–1038.

Lewis, M. (1998). Shame and stigma. In P. Gilbert & B. Andrews (Eds.), *Shame: Interpersonal behavior, psychopathology, and culture* (pp. 126–140). New York: Oxford University Press.

Lewis, M. H., & MacLean, W. E., Jr. (1982). Issues in treating emotional disorders of the mentally retarded. In J. L. Matson & R. P. Barrett (Eds.), *Psychopathology in the mentally retarded* (pp. 1–36). New York: Grune & Stratton.

Lieberman, A. F., & Pawl, J. H. (1988). Clinical applications of attachment theory. In M. Greenberg (Ed.), *Attachment beyond infancy* (pp. 327–351). Chicago: University of Chicago Press.

Lobato, D. J. (1990). *Brothers, sisters, and special needs: Information and activities for helping young siblings of children with chronic illnesses and developmental disabilities*. Baltimore: Paul H. Brooks.

Lowe, K., Allen, D., Jones, E., Brophy, S., Moore, K., & Jones, W. (2007). Challenging behaviors: Prevalence and topographies. *Journal of Intellectual Disability Research, 51*, 625–636.

Matson, J. L. (1986). Self-injury and its relationship to diagnostic schemes in psychopathology. *Applied Research in Mental Retardation, 7*, 223–227.

Matson, J. L. (Vol. Ed.). (2007). *International review of research in mental retardation: Vol. 34. Handbook of assessment in persons with intellectual disability.* San Diego, CA: Elsevier.

Matson, J. L., Anderson, S. J., & Bamburg, J. W. (2000). The relationship of social skills to psychopathology for individuals with mild and moderate mental retardation. *The British Journal of Developmental Disabilities, 46*, 15–22.

Matson, J. L., & Barrett, R. P. (Eds.). (1993). *Psychopathology in the mentally retarded* (2nd ed.). Boston, MA: Allyn & Bacon.

McLaren, J., & Bryson, S. E. (1987). Review of recent epidemiological studies of mental retardation: Prevalence associated disorders and etiology. *American Journal of Mental Retardation, 92*(5), 243–254.

McMillan, D. L., Gresham, F. M., & Siperstein, G. N. (1993). Conceptual and psychological concerns about the 1992 AAMR definition of mental retardation. *American Journal of Mental Retardation, 98*, 325–335.

McMillan, D. L., Gresham, F. M., & Siperstein, G. N. (1995). Heightened concerns over the 1992 AAMR definition: Advocacy versus precision. *American Journal on Mental Retardation, 100*, 87–97.

Menolascino, F. J. (Ed.). (1970). *Psychiatric approaches to mental retardation*. New York, NY: Basic Books.

Mochida, G. H., & Walsh, C. A. (2004). Genetic basis of developmental malformations of the cerebral cortex. *Archives of Neurology, 61*, 637–640.

Murphy, C. C., Boyle, C., Schendel, D., Decoufle, P., & Yeargin-Allsopp, M. (1998). Epidemiology of mental retardation in children. *Mental Retardation and Developmental Disabilities Research Reviews, 4*, 6–13.

Opitz, J. M. (1985). The Brachmann-de Lange syndrome. *American Journal of Medical Genetics, 22*, 761–767.

Quine, L. (1986). Behavior problems in severely mentally handicapped children. *Psychological Medicine, 16*, 761–767.

Reiss, S. (1990). Prevalence of dual diagnosis in community-based day programs in the Chicago metropolitan area. *American Journal of Mental Retardation, 94*, 578–585.

Reiss, S., & Aman, M. G. (Eds.). (1998). *Psychotropic medications and developmental disabilities*. Columbus: Ohio State University Press.

Reiss, S., Levitan, G. W., & Szyszko, J. (1982). Emotional disturbance and mental retardation: Diagnostic overshadowing. *American Journal of Mental Deficiency, 86*, 567–574.

Rhead, M. J., & Irons, M. (2004). The call from the newborn screening laboratory. *Pediatric Clinics of North America, 51*, 803–818.

Rovet, J. F. (2002). Congenital hypothyroidism: An analysis of persisting deficits and associated factors. *Neuropsychology, Development and Cognition, Section C, Child Neuropsychology, 8*, 150–162.

Rutter, M., Simonoff, E., & Plomin, R. (1996). Genetic influences on mild mental retardation: Concepts, findings, and research implications. *Journal of Biosocial Science, 28*, 509–526.

Rutter, M., Tizard, J., & Whitmore, K. (1970). *Education, health, and behavior*. New York: Wiley.

Sandman, C. A., Thompson, T. T., Barrett, R. P., Verhoeven, W. M., McCubbin, J. A., Schroeder, S. R., et al. (1998). Opiate blockers. In S. Reiss & M. G. Aman (Eds.), *Psychotropic medications and developmental disabilities* (pp. 291–302). Columbus: Ohio State University Press.

Schalock, R. L., Luckasson, R. A., Shogren, K. A., Borthwick-Duffy, S., Bradley, V., Buntinx, W. H. E., et al. (2007). The renaming of mental retardation: Understanding the change to the term intellectual disability. *Intellectual and Developmental Disabilities, 45*, 116–124.

Schroeder, S. R., Oster-Granite, M. L., & Thompson, T. (2002). *Self-injurious behavior: Gene-brain-behavior relationships*. Washington, DC: American Psychological Association.

Schroeder, S. R., Schroeder, C. S., Smith, B., & Dalldorf, J. (1978). Prevalence of self- injurious behaviors in a large state facility for the retarded: A three year follow-up study. *Journal of Autism and Childhood Schizophrenia, 8*, 261–269.

Sovner, R. (1989). The use of valproate in the treatment of mentally retarded persons with typical and atypical bipolar disorders. *Journal of Clinical Psychiatry, 50*, 40–43.

Stoneman, Z., & Berman, P. W. (Eds.). (1993). *The effects of mental retardation, disability, and illnesses on sibling relationships: Research issues and challenges*. Baltimore: Paul H. Brookes.

Szymanski, L. S. (1994). Mental retardation and mental health: Concepts, etiology, and incidence. In N. Bouras (Ed.), *Mental health in mental retardation: Recent advances and practices* (pp. 19–33). Cambridge: Cambridge University Press.

Thompson, T., & Hupp, S. C. (Eds.). (1992). *Saving children at risk: Poverty and disabilities*. London: Sage.

Tylenda, B., Beckett, J., & Barrett, R. P. (2007). Assessing mental retardation using standardized intelligence tests. In J. L. Matson (Vol. Ed.), *International review of research in mental retardation: Vol. 34. Handbook of assessment in persons with intellectual disability* (pp. 27–97). San Diego, CA: Elsevier.

Tylenda, B., Hooper, S. R., & Barrett, R. P. (1987). Developmental learning disorders. In C. L. Frame & J. L. Matson (Eds.), *Handbook of assessment in child psychopathology: Applied issues in differential diagnosis and treatment evaluation* (pp. 187–217). New York: Plenum Press.

Vannucci, R. C. (1990). Experimental biology of cerebral hypoxia-ischemia: Relation to perinatal brain damage. *Pediatric Research, 27*, 317–326.

Walters, A. S., Barrett, R. P., Knapp, L., & Borden, M. C. (1995). Suicidal behavior in children and adolescents with mental retardation. *Research in Developmental Disabilities, 16*, 85–96.

Werry, J. S., & Aman, M. G. (Eds.). (1993). *Practitioner's guide to psychoactive drugs for children and adolescents*. New York: Plenum Press.

Wilson, J. G., Lott, R. S., & Tsai, L. (1998). Side effects: Recognition and management. In S. Reiss & M. G. Aman (Eds.), *Psychotropic medications and developmental disabilities* (pp. 95–114). Columbus: Ohio State University Press.

World Health Organization. (2001). *International classification of functioning, disability, and health (ICF)*. Geneva: Author.

World Health Organization. (2008). *Revision of the international classification of diseases (ICD).* Available at http://www.who.int/classifications/icd/ICDRevision/en/index.html

Zigler, E. (1967). Familial mental retardation: A continuing dilemma. *Science, 155*, 292–298.