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Intellectual Disability in Children; a Systematic Review

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ABSTRACT

Aims Intellectual disability is a condition characterised by the inability of a person to undertake normal psychological activities. The purpose of this study was to systematically review the intellectual disability in children and discuss the implications of different environmental and genetic factors, which describe particular categories of intellectual disable cases.

Information & Methods This systematic review was performed in 2014 by searching the existing literature in PubMed database in the scope of "intellectual disability in children". 38 articles written from 1987 to 2014 were selected and surveyed for review.

Findings The prevalence of ID in the general population is estimated to be approximately 1%. ID disorder is multi-causal, encompassing all factors that interfere with brain development and functioning. Causes usually are classified according to the time of the insult, as prenatal, perinatal, and postnatal or acquired. Some causes, such as environmental toxins or endocrine disorders, may act at multiple times. Others, such as genetic disorders, have different manifestations during postnatal development. The outcome for ID is variable and depends upon the aetiology, associated conditions, and environmental and social factors. The goals of management of ID are to strengthen areas of reduced function, minimize extensive deterioration in mental cognitive and adaptability, and lastly, to promote optimum or normal functioning of the individuals in their community.

Conclusion Prominent features of ID include significant failures in both intellectual functioning and adaptive behaviour, which comprises daily social and practical life skills, commencing earlier in life.

Keywords Intellectual Disability; Disabled Children; Review, Systematic

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Introduction

Intellectual disability (ID) is an encephalopathy with multiple aetiologies, ranging from disability by physical literal limitations to inadequacy in adapting to strengths of life skills, which disables persons to operate properly. The older version of this condition was termed mental retardation, but due to the varying paradigm of manifesting the manner in different ages, the term ID was preferred to be an umbrella of all ailments associated with mental growth. In young children (from infants to the under-fives), "developmental delay" refers to the same condition. This group influences special interests because at this developmental stage brain cells usually become mature and cognitive defects start manifesting, therefore the validity of the results of IQ tests done on this age-group is nullified, not only regionally but also globally [1-3].

As advised by paediatrics, the developmental surveillance should be performed at every crucial stage of child development. At this point, the examination should target screening at periodic visits by regular intervals of 9-, 18- and 30 months [2]. The above program allows close monitoring and evaluation of growth parameters, which facilitates quick early identification of either developmental delay or ID signs in children. The importance of mental developmental delay as a public health phenomenon, just like all health events, is that they contribute to the statistic of prevalence, which determines the level of need and the manner in which the operation of the extensive support services rolls out. As proposed by health practitioners, the remedy to these conditions is dependent upon prompt comprehensive diagnosis and proper intervention. This is because of the nature of the brain cells failure to regenerate, and for effective outcome, the approach should integrate access to and consumption of health care, and relevant educational resources [3].

Intellectual literacy is a functional capacity of the brain, signifying normal mental developmental growth, which commences at childhood and described by huge abilities in intelligence and adaptive skills. Whereas ID and other forms of mental retardations, emanate from childhood experiences and expressed in various forms, including, but not limited to, below-average intelligence levels, and abject deficits in life skills that are necessary for human being's productive functioning. Persons with intellectual disabilities have the opportunity to understand and learn important life skills, though at a slower pace than those with normal mental development [4].

As pointed out earlier, there are various levels or forms of ID, evident as limitations in the following areas. The first area is that of "Intellectual functioning" commonly referred to as Intelligence Quotient (IQ), which is determined by an IQ test. The IQ of a person with optimal mental development is approximately 100. Therefore, a person is "mentally retarded" if his/her intelligence quotient falls below 70-75%, which determines a person's ability to learn and comprehend events, make reasonable judgements, make rational decisions, and effectively solve problems [5].

The purpose of this study was to systematically review the intellectual disability in children and discuss the implications of different environmental and genetic factors, which describe particular categories of intellectual disable cases.

Information & Methods

This systematic review was performed in 2014 by searching the existing literature in PubMed database in the scope of "intellectual disability in children".

Using "disability in children" as keyword, more than 100 articles were recognized under the scope, though not all of them were relevant to the topic. By more evaluations and narrowing the criteria of selection, finally, 38 articles written from 1987 to 2014 were selected and surveyed for review.

Findings

Features and characteristics of intellectual disability

The severity of cognitive impairment is characterized by the extent of deviation of the IQ below 100, the estimated mean for the population. The lower limit than normal characterized by either the IQ below 70 or failure in adaptive behaviours [3-5]. Gradations of severity include IQs in the following ranges:

Mild: from 50-55 to approximately 70;

Moderate: from 35-40 to 50-55;

Severe: from 20-25 to 35-40;
Profound: from <20 to 25; and
Unspecified: not readily testable but presumed low (<70).

Adaptive behaviours are skills necessary for routine normal functioning while engaging in life events, for example, to enhance one's ability to communicate effectively and sustain fruitful interpersonal relations through guided interactions and being self-conscious at all times regarding security and time management. Adaptive skills are the skills of daily living, which facilitates living, working, interactions, learning or even during communal out-door games. The above requires competence in one's ability to communicate, socialize and have good interpersonal skills, and exercise self-care as required. Conversely, adaptive behaviours improves on home-living, the manner in which societal resources are put to use, inculcate sense of self-direction, and instil foundational academic skills (which necessitate reading, writing, and basic mathematics), work, health and safety. Adaptive functioning is considered impaired when there is a deficit in at least two of these areas compared to children of the same age and culture [6].

Diagnosis of intellectual disability

In order to assess children's adaptive behaviours, specialists have to keenly observe, record and analyse several mental development parameters, e.g., life skills. Thereafter, comparative evaluation with the results from other children's performance of the same age and on the same study parameters must be done.

The observed study parameters should capture as much as possible adaptive features in children. This should include all forms of adaptation with respect to their immediate surrounding conditions and the environment influencing their lives, e.g., child's ability to feed and dress up properly, the capability of a child to communicate effectively to people and understand them in return, the child's level of interaction with his/her immediate family, friends and significantly, other children of his/her age group. To do so, some questionnaires are used:

Ages and Stages Questionnaire (ASQ): ASQ provides age-specific questions about 4-60 month old children that are completed by parents that can be used to screen development of children [2]. These are used in clinical and research settings and applied in some screening and intervention programs [7].

Bayley Infant Neuro-developmental Screener (BINS): BINS can be used to screen development in 3-25 month old children. It uses 10 to 13 directly extracted items per three- to six-month age range to screen neurologic processes (reflexes and tone), neuro-developmental skills (fine motor, language), and cognitive processes [8].

Brigance Screens-II: It can be used to screen development in new-borns to 90 month old children and consists of 9 separate forms, one for each 12-month age range, each of which takes 10 to 15 minutes to administer. It uses parents' report (in the new-borns to 24 months age range), direct observation, and elicitation to screen speech-language, motor skills, and general knowledge at younger ages and reading and math at older ages [9].

Denver Developmental Screening Test-II (DDST-II): DDST-II is a directly administered tool that is designed to screen expressive and receptive language, gross motor, fine motor and personal-social skills in new-borns to 6-year-old children [6]. It has limited sensitivity and specificity to detect language delay, mild ID, learning disabilities, and functional developmental delay and is used to track developmental skills during longitudinal paediatric follow-up and to detect children who require further evaluation of delays.

Infant-Toddler Checklist for Language and Communications (ITCLC): ITCLC can be used to screen language and communication development in 6-24 month old children. Early language delay may be the first sign of atypical development in ID [10].

Parents' Evaluation of Developmental Status (PEDS): PEDS can be used as a brief developmental screen in new-borns to 8-year-old children. In addition to function as a screening test, it provides longitudinal surveillance and helps to determine when referrals are necessary and when patient education, in-office counselling, watchful waiting, or additional screening is needed [11].

When a screening test suggests developmental delay, further evaluation is needed, e.g., complete history and physical examination, performing comprehensive

developmental assessment, complete vision and auditory screening, reviewing the new-born screening results and performing a chromosomal microarray.

Signs and symptoms of intellectual disability in children

Signs and symptoms of ID, in children, differ depending on the child's genetic disposition, social life and the living conditions. Though the actual cause of ID is not known, speculative conclusion are linked to the above-mentioned factors with some levels of doubt. The signs and symptoms associated with the emergence of ID usually become evident at the early days of children growth and development. In most instances, the symptoms may remain latent or clinically invisible and thereafter detected clinically, at full blown, when the child has reached the age of school. Thus, manifestation of this condition is determined by the severity or level of disability [3-5].

Evident features, which are commonly associated with mental retardation or ID, ranges from late or delayed rolling over, sitting up, crawling or walking, failure to talk on time or developing the sense of speech, and slow pace of mastering routine activities. In other incidences, the same signs could be coupled by immense difficulties in comprehending things or events, e.g., failure to harmonise or connect actions with possible consequences or translating behavioural problems, which include unstable, bad temper and difficulty with problem-solving or logical thinking [12].

Prevalence factors of intellectual disability

The incidence of ID varies significantly among studies due to differences in study design, diagnostic approach, severity of the condition, and demographic characteristics such as age and population composition. In addition, 85% of the known cases, who have brain developmental deficits, report a mild level of ID [6]. Mental retardation affects nearly 1% of the entire population [7]. Based on IQ alone, the estimated prevalence of ID increases to approximately 3% [6]. However, prevalence varies with age and gender and is highest in school age and male individuals [13, 14]. The prevalence of mild ID is more variable than severe ID, varying with environmental factors of maternal education, educational access, or opportunities and access to healthcare [15]. ID and mild ID in particular, are more prevalent in developing countries or areas with lower socioeconomic status [15].

Children with severe mental retardation are prone to other opportunistic health problems. The consequences of these problems are seizures, poor vision and associated problems, or hearing difficulty [8]. The causes of ID are numerous, distinct but limited to disorders, which are known to have direct influence on brain development and functioning. Identifying a cause enables suitable counselling, focused interventions, treatments, surveillance, and anticipation of possible medical or behavioural complications, with a more specific prognosis. The most common causes of ID are genetic conditions problems during pregnancy, problems in childbirth and illness or injury [11]. Genetic and biological factors are associated in many cases of ID. Among the known prenatal causes of ID, the majority are genetic abnormalities. Down syndrome or Trisomy 21 is the commonest known cause of ID, affecting 1 in 700–1000 live births. Down syndrome (DS) differs from other congenital syndromes in that routine antenatal testing is recommended in all pregnant women over 35 years old. Up to 50% of infants with DS have congenital heart defects, which are mostly mild or surgically correctable [15]. Of 100 identified physical features of DS, facial features are most easily recognizable. These include a flat face with a large protruding tongue, upward slanting eyes, small ears and mouth. Individuals have poor muscle tone and short height and are horizontal to obesity and hypothyroidism. Eye conditions such as strabismus and refractive errors are common and dry genetic causative conditions are increasingly being diagnosed by technological advances in genetic testing, particularly chromosomal microarray testing. Chromosomal microarray testing is currently the most valuable tool to identify genetic causes of ID [12].

Causes, prevalence risks and examples of intellectual disability in some regions

The distribution of causes of ID varies among different populations. 0.03% (178 children) of the Norwegian population births between

1980 and 1985 have reported to have severe (IQ<50) or mild (IQ=50-70) ID [8]; The period distribution of severe ID were 70, 4 and 5% in prenatal, perinatal and postnatal stages, respectively and 18% of severe cases were undetermined and for mild ID these levels were 51, 5 and 1% for prenatal, perinatal and postnatal stages, respectively and 11% were undetermined.

The prevalence of ID varies with age and sex, and is highest in school age and male populations [14]. However, the sex difference reduces with more severe ID. The prevalence of mild ID is more changeable across populations than severe ID is and varying with ecological factors of maternal education, educational access or access to healthcare [15]. Important risk factors for ID are maternal age (older age is related with greater risk) and maternal education [16].

The leading cause of prenatal disorders was central nervous system malformations. CNS anomalies were identified in 4% of Californian children with ID [17]. An identifiable malformation syndrome of unknown origin accounts for less than 10% of ID cases [18]. Other important prenatal causes of ID are congenital infection and potent environmental toxins, e.g., alcohol, lead and mercury. Internationally, prenatal exposure to alcohol is an important and potentially preventable cause of ID. Radiation exposure, especially between 9 and 15 weeks gestation, is associated with ID. Congenital hypothyroidism that is unrecognized may cause cognitive delay [15].

Perinatal abnormalities that may lead to ID are intracranial haemorrhage, preterm birth, hypoxia, infection, and trauma. Some of these causes may have prenatal origins. Postnatal and acquired causes of ID may be easier to identify, as they usually occur in an individual who was previously normal; e.g., accidental or non-accidental trauma, CNS haemorrhage, hypoxia, environmental toxins, psychosocial deprivation, malnutrition, intracranial infection, CNS malignancy, or acquired hypothyroidism [13].

The cognitive effects of coexisting exposure to multiple environmental toxins are not known. Congenital hypothyroidism frequency is about 1:2000 to 1:4000 of new-borns. This condition is a mild form of ID, which is preventable. Global statistics collected from various screening centers on congenital hypothyroidism depicts great variation of incidence. These variations have significance in that it indicates different geographic locations and ethnic groups. A 20-year period study of French screening program has shown that the incidence of permanent congenital hyperthyroidism is 1:4000 and the figure is rising [15]. The 11-year period study in Greece from 1990 to 2000 has shown the incidence of congenital hypothyroidism as 1:1800. Another case study has shown the effect of TSH level in 3-6 day old infants as a cause of hypothyroidism [19]. About 85% of permanent cases of congenital hypothyroidism are sporadic and 15% are hereditary (most caused by one of the inborn errors of thyroid hormone synthesis). Screening of new-borns has become a recommendable routine in the United States of America, Canada, and Europe. The program was also successful in Israel, Japan, and recently in New Zealand. This screening program is being rolled out in parts of Eastern Europe, countries in South America, Central Asia, and among African countries. In the USA, approximately 4million infants undergo congenital hyperthyroidism screening annually. This facilitates the early detection of congenital hyperthyroidism condition in children [10-12].

Phenylketonuria (PKU) is a disorder occurring in aromatic amino acid called phenylalanine. The above condition emanates from a deficit in phenylalanine hydroxylase, which is a direct cause of ID. The frequency of occurrence of PKU ranges from one among 13,500 to 19,000 births [20]. The mechanism by which the elevated concentration of phenylalanine causes ID is unknown. Excessive phenylalanine is thought to interfere with brain growth, myelination, and neurotransmitter synthesis. New-born infants are asymptomatic before the initiation of feeds containing phenylalanine (breast milk or standard infant formula). If undetected by metabolic screening, PKU may not cause symptoms until early infancy. In untreated patients, the hallmark of the disease is ID; epilepsy occurs frequently [18].

Some environmental toxins and other heavy metals, e.g. lead and mercury, cause devastative and irreparable damages on the development of brain and the entire nervous system. The government of USA has combated

the prevalence of heavy metal toxicity from early 1970's. Screening programs launched in schools during enrolment, advanced public advocacy, aided this and awareness, and lastly a ban on usage of gasoline, lead and paint products. Although the trend of the incidence and degree of severity caused by lead poisoning is declining, approximately, 450,000 infants in USA were beyond 5mcg/dl, which is a recommended reference value in 2012. Children younger than 6 years old (and particularly those between 12 and 36 months) were more susceptible to toxic effects of lead than adults were. This is because they have an incomplete blood-brain barrier that permits the entry of lead into the developing nervous system and because they have a greater prevalence of iron deficiency, which is associated with lead poisoning and may permit increased absorption of lead from the gastrointestinal tract [21]. In addition, they are at greater risk of exposure to lead dust because of crawling and hand-to-mouth behaviour. The prevalence of elevated lead levels is highest among inner-city children who live in deteriorating housing that was built before 1970s. Lead poisoning is more common among urban than rural, low-income than middle-income, and children who live in older housing [21].

The prevalence of lead poisoning is increased in refugee children who have arrived recently in the United States. Children typically are exposed to environmental lead through ingestion or inhalation. Common sources include chips of paint or lead dust from lead-painted surfaces, food or beverages that purchased, stored, or served in lead-soldered cans and most significantly, automobile emissions from the lead-using industry [22]. The clinical manifestations of lead poisoning vary depend on the lead exposure and the age of the exposed individuals [22]. The primary symptoms of acute lead poisoning, as pointed out in many research findings, is episodic and nonspecific, especially in children. Lead poisoning causes developmental delay of brain neurones, or loss of milestones, particularly in language, to encephalopathy.

Alcohol consumption among expectant mothers is thought to be one of the prerequisites of ID. In addition, though preventable, alcohol is the leading cause of ID. Recently, scientific research implicates smoking to be among the risk factors for ID. Other adverse consequences include malnutrition, and the ailing pregnant mother, leading to conditions such as toxoplasmosis, cytomegalovirus, rubella and syphilis. Prematurity and low birth weight predicts congenital hyperthyroidism disorders and a possible case of ID. Problems associated with difficulties during birth processes magnify this condition in cases that original cause was genetic or congenital. These problems include temporary oxygen deprivation, dropping maternal pressure or sustained birth injuries may be crucial ingredients to cause intellectual disabilities [19].

Another group of a possible cause to congenital hyperthyroidism and mental retardation is untreated childhood diseases, which damages the brain. In most pronounced cases, children who grow up in poverty are vulnerable to malnutrition and exposure to toxic health hazards in their surroundings. The above factors increase the risks of children developing mental retarding. However, the cause of ID often is unknown. A study from California identified risk factors for idiopathic ID according to the level of severity [22]. Increased risk for severe ID was seen in males, low-birth-weight infants, and children of Hispanic, black, or Asian, compared to white, mothers. The risk for severe ID increases with maternal age and decreasing maternal education.

Bilder *et al.* have indicated that prenatal and perinatal factors, attributed to ID, are advanced maternal age, still births, and foetal distress [23]. The incidence of membranes' premature rupture and or repeated caesarean sections, together with low birth weight alongside assisted ventilation, usually leads to developmental delay. In other instances, low Apgar scores, and congenic infection are possible causative agent to ID [18]. Mann *et al.* have shown that the risk of children to be born with ID escalated in mothers who showed morbid obesity or pre-pregnancy obesity [24]. While similar risk factors were identified for mild ID with unknown cause, increased risk was identified in multiple births and children born second or later. However, compared to children born from white mothers, the risk of having a child with mild ID was greater for black mothers, less for Asian mothers, and similar for Hispanic

mothers [24]. Maïano has shown that in children and youth with ID, the overall prevalence level of overweight ranged from 11 to 24.5% and 7 to 36% for obesity [25]. Children and youth with disability are believed to be at greater risk of obesity due to lower levels of physical activity, inappropriate behaviours related with their disability, medicine and related chronic health conditions.

Epidemiology and trauma of intellectual disability

The prevalence of ID is about 1% in general [7, 8-12]. ID is mild in 85% of influenced persons. The prevalence of ID differs with sex and age, and is highest in school age and males [29]. Approximately, about 30% more males are diagnosed with ID as compared with females [30]. Prevalence of mild ID changes by environmental factors of educational access, maternal education, and access to healthcare [15]. ID and mild ID are more common in developing countries with lower socioeconomic status [15].

The effect of trauma is increased for children with developmental disabilities due to cognitive and processing delays that interfere with considerate of what is happening in violent situations. Tuberous sclerosis complex (TSC) is a genetic disorder and 45% of persons with TSC have ID. High rates of violent behaviour and self-injury [30, 31] have been described in individuals with TSC. Numerous physical and behavioural features related with TSC signify that persons with TSC may be a high-risk group for these behaviours. Persons with severe or deep ID are considerably more possible to employ in self-injury and aggression than individuals with mild or moderate ID [32]. Eden *et al.* have described that individuals with TSC are at a high risk of appealing in self-injury and aggression and are more expected to show these behaviours if they have low mood, high levels of activity and autism spectrum disorder [33].

Intervention strategies and prevention of intellectual disability

Early life prevention is one of the most effective ways to improve physical and psychological health and reduce social and economic burdens for families, communities and health care services. The general objectives of curbing ID are mainly focused on ensuring that individuals grow up to be independent, with improved functionality at the same time sustain the needed family supported and enhances optimal societal functioning. Children with ID require on-going health surveillance similar to normal children. Developmental, academic, and psychosocial progress should be monitored. Slower developmental progress should be expected in children with more severe cognitive-adaptive disability [26].

Most individuals with ID require a broad range of interventions that should be applied early to improve short-term and long-term outcomes. If an underlying cause is identified, it should be promptly treated. Some of interventions are speech and language therapy, occupational therapy, physical therapy, including mobility support, family counselling and support, behavioural intervention and educational assistance. Behaviour interventions are usually needed to improve community skills and behavioural functioning in children with ID. Behaviour interventions are usually needed to improve social skills and behavioural functioning in children with ID. These should be individualized and applied every time to give confidence appropriate thinking, expression, adaptive function, behaviour, and environmental manipulation. Interventions should be suitable to the child's level of functioning (particularly language functioning) rather than chronological age [34]. Behavioural goals and psychotherapy interventions for the child and family should concentrate on understanding, the positive use of strengths, building social skills and problem solving, self-esteem, communication strategies, and independence. Interventions for the family are counselling, training, home visitation and social services. The outcome of ID is variable and depends upon the aetiology, associated conditions (biomedical disorders may be associated with a range of functions and/or a reduced lifespan and mental health disorders may damage communication abilities and social functioning and affect the child's profile of intellectual strengths and weaknesses [35]), and environmental (appropriate condition of services, such as education and treatment [36]) and social

factors (hopes, attitudes, socioeconomic characteristics and social opportunities).

Most causes of ID cannot be prevented. When possible, strategies should be implemented to reduce the risk of ID, to detect treatable conditions, to minimize the extent of potential disability, and to improve the functioning of affected individuals. Primary prevention is aimed at preventing conditions that may cause ID; avoidance of prenatal exposure to alcohol or other toxins, use of prenatal multivitamins, appropriate prenatal care, new-born screening programs for metabolic disease, routine childhood immunizations, use of car seats and restraints, prevention of motor vehicle accidents, violence, and other trauma. Secondary prevention is directed at treating an underlying condition to reduce potential cognitive-adaptive dysfunction. Tertiary interventions are designed at maximizing function and quality of life by attempting to reduce functional disability [21].

Early identification of learning disabilities is crucial to provide interventions to optimize learning and prevent secondary emotional problems. Because early recognition may affect ultimate outcome, paediatric healthcare providers should have a low threshold for considering learning disabilities in children who are at risk and those who have problems at school. Any child who is failing at school should be presumed to have a learning disability until proven otherwise. Children with learning disabilities frequently are referred to healthcare providers when the parent expresses concerns about the child's learning failure. Such concerns should always be taken seriously. Parental concerns are a sensitive indicator of developmental and learning problems. The paediatric healthcare provider can help the family by informing them of their right to evaluation/testing through the child's school district and by providing access to resources that help families of children with disabilities [7].

Learning problems are usually first identified by teachers, who may initiate referral for a special education evaluation. Paediatric clinicians are not likely to identify learning disabilities unless the child is already known to have learning problems and/or the child's parents express concerns about the child's failure to learn. However, because early recognition may affect ultimate outcome, paediatric healthcare providers should be quick to consider learning disabilities in children who are at the risk and children who have problems at school. The diagnosis of learning disabilities is made primarily by history. Psychometric measures help to confirm the presence of learning disabilities and identify targets for intervention. An appropriate assessment for learning disabilities includes information from standardized psychometric measures, a review of the student's educational history, and a description of classroom observations [32].

Intellectually disabled children are vulnerable to increased risks leading to accidental injuries, inflicted pain, and neglect of parental role on their children. Consequently, behaviour, and physical cognitive features of a child, together with the immediate environments forms a pillar, which determines a child's disability and increases a child's risks of mental retarding. Children with behaviours disorders such as increased activity are prone to engage in hazardous behaviours that may result in accidental injury. The prevalence of injury in children and adolescents with intellectual disabilities is 1.5-2 times more than children and adolescents without intellectual disabilities [35]. The types of injuries that occur with increased prevalence among children and adolescents with intellectual disabilities include burns, poisonings, foreign body injuries, fractures, dislocations, and internal injuries [36]. Cognitive skills that are necessary to prevent injury and that may be impaired in children with intellectual disabilities include the accuracy to comprehend and follow safety rules to the later as required to estimate physical abilities (e.g., strength and balance), attend to and understand the risks of a given situation, and understand the cause of injury after it occurs. Factors that may contribute to the increased risk of injury include behavioural problems, cognitive impairment, medical conditions, environments not well adapted to the needs of disabled children, psychosocial stressors associated with caring for a child with disability, and the increased susceptibility of a child with disability. Prevention of unintentional injury in children with disabilities encompasses the injury prevention strategies that are recommended for all children [20, 36-38].

It has become apparent, from more than two decades ago, that treatment programs for the intellectually disabled should contain provisions for the training of appropriate social skills. The final goal of such programs has shifted to facilitate the development of effective interpersonal functioning so that these individuals can move into community-based living situations. Because the efficiency of any treatment program relies greatly on the sufficiency of the based assessment, the correct assessment of social skills excesses and deficits is of serious importance.

Conclusion

Prominent features of ID include significant failures in both intellectual functioning (that is, ability to reason, comprehend ideas, and solve problems) and adaptive behaviour, which comprises daily social and practical life skills, commencing earlier in life (below 18 years old). The prevalence of ID in the general population is estimated to be approximately 1%. ID disorder is multi-causal, encompassing all factors that interfere with brain development and functioning. Causes usually are classified according to the time of the insult, as prenatal, perinatal, and postnatal or acquired. Some causes, such as environmental toxins or endocrine disorders, may act at multiple times. Others, such as genetic disorders, have different manifestations during postnatal development. The outcome for ID is variable and depends upon the aetiology, associated conditions, and environmental and social factors. Most causes of ID cannot be prevented. The goals of management of ID are to strengthen areas of reduced function, minimize extensive deterioration in mental cognitive and adaptability, and lastly, to promote optimum or normal functioning of the individuals in their community. Therefore, interventions should commence immediately and be progressive. The approach should be joint and multidisciplinary.

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