ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 14, Issue, 09, pp.22320-22326, September, 2022
DOI: https://doi.org/10.24941/ijcr.43964 .09.2022

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

REVIEW ARTICLE

A SYSTEMATIC REVIEW ON INCIDENCES OF MENTAL RETARDATION INTERLINKAGES WITH HOLISTIC DEVELOPMENT AMONG CHILDREN

Shreeya, R.[1], Manjula Sheshadri, M.S.[2] and Sushma B. V.[1]

[1]Department of Nutrition & Dietetics, School of Life Sciences, JSSAHER, Mysore
[2]Department of food science & Nutrition, Maharani Science college for women Mysuru

ARTICLE INFO

Article History:
Received 20th June, 2022
Received in revised form
27th July, 2022
Accepted 09th August, 2022
Published online 30th September, 2022

Key words:

Climate, Comfort,
Energy Efficiency,
Environment.

Corresponding Author:
Shreeya, R.

ABSTRACT

Early childhood is a basic stage for the child's later life. During this period the brain development and the foundations for cognitive development and social-emotional skills, is laid. Nutrition plays a key role in optimal brain function. When a child is adequately fed, the basis for smooth brain function is created. Nutritional deficiencies found to affect the child's cognitive skills and behavior. While rapid brain development occurs during the early years, this period could particularly sensitive to deficiencies in the diet. Recent data has shown that diet seems to be one of the most important growth and maturation factors in the brain both in infancy and childhood. In particular, fatty acids play a central role in brain tissue, iron deficiency causes disturbances in attention, memory and behavior, and is closely linked to lower cognitive indices. Long-term studies have shown that children with iron deficiency have lower school performance. In addition, zinc, as well as iodine and vitamins D and B12 deficiencies are likely to adversely affect brain function and memory. An inadequate diet usually causes multiple deficiencies of micronutrients with short-term and long-term effects on the functioning of the child's brain.

Citation: ***Shreeya, R., Manjula Sheshadri, M.S. and Sushma B. V.*** **2022.** "A systematic review on incidences of mental retardation interlinkages with holistic development among children*". International Journal of Current Research*, 14, (09), 22320-22326.

INTRODUCTION

Mental retardation or MR: currently termed as intellectual disability [ID]; describes a constellation of symptoms that includes severe deficits or limitations in an individual's developmental skills in several areas or domains of function: cognitive, language, motor, auditory, psychosocial, moral judgment, and specific integrative adaptive (i.e., activities of daily living) (33). This article reviews concepts of ID in children and adolescents useful for the primary care clinician who cares for these individuals (17). The majority of youth with ID can live independent or semi-independent lives as adults if they have received the appropriate personalized support over a sustained period of their lives, especially during the formative years (21). Mental retardation (MR) is one of the major clinical and socially relevant conditions that affect 3% of the total pediatric population (4). The deleterious effect of early malnutrition on later intellectual development in children in developing countries has been clearly documented (54). It is also known that severely mentally disabled children are at a high risk for developing malnutrition which may partly explain the growth retardation generally encountered in such children and associated complications in later life (6). The extent of adaptive psychological impairment is key to defining ID and its severity (1). The term ID replaces the older term of "mental retardation" (20). Intellectual disability (ID) is a neurodevelopment disorder that begins in childhood and is characterized by limitations in both intelligence and adaptive skills, affecting at least one of three adaptive domains (conceptual, social, and practical), with varying severity (34). Often Intellectual disability is associated with multiple etiologies (27). It is characterized by deficits in intellectual and adaptive functioning of varying severity presenting before 18 years of age (2). ID encompasses a broad spectrum of functioning, disability, needs, and strengths (45). It is an important public health issue because of its prevalence and the need for support services. ID affects approximately 1 percent of the population (42). Its management requires early diagnosis and intervention, including access to health care and appropriate supports (61). Nutritional status often has a significant impact on overall health and quality of life in children with neurodevelopment disabilities; both under and over nutrition generally lead to increased health care use and reduced participation in educational and social activities (62). Malnutrition is frequently associated with impairment of linear growth, reduced peripheral circulation and wound healing, increased spasticity and irritability (9). The overall prevalence of malnutrition in neurologically impaired (NI) children is difficult to estimate, due to the heterogeneity of neurological disorders (24). The majority of scientific literature on nutrition in NI children has focused on the population with cerebral palsy (CP) in which malnutrition has been observed in 46%–90% of cases (9). Etiology of malnutrition in NI children is multifactorial including both nutritional and no nutritional factors (30). Among the nutritional factors, the main is represented by inadequate dietary intake as a consequence of gastrointestinal disorders including oral motor dysfunction, gastro esophageal reflux and constipation (49). Among non-nutritional factors, the type and severity of underlying

neurological disability, influencing ambulatory and cognitive status, and antiepileptic use are crucial factors involved in determination of nutritional status (53). Malnutrition as revealed by anthropometric variables is highly prevalent among children with MR; and the prevalence of malnutrition increases with age, deterioration of mental functioning and cerebral palsy (14). Evidences on micronutrients consumption among on-obese MR children with Down's syndrome reported to be 80% less than Recommended Dietary Allowances (RDA) and had low energy intake; though their body composition did not differ in comparison to the control group (31). Further study showed the significant difference with regard to the energy intake among male and female individuals with MR. However, there was no marked effect on energy intake with degree of the mental handicap or body mass index (16). Children and adolescents (2-18 years) with Down's syndrome, developmental and chromosomal disorder exhibited less satisfactory level of self-feeding skills (8).To greater extent preschool children with mental illness as well as their siblings reported lack of chewing skills. Markedly food habits reported to have a significant influence on nutritional status both in biochemical and anthropometric parameters in mentally retarded children (13). Evidently biochemical parameters were also influenced by socio-economic status (23). Even though low IQs are inevitably linked with cognitive deficits, there is considerable variability among individuals with and without psychiatric disorders (15). Children with ID generally have worse phonological functioning, sensory motor skills and working memory than youth with higher IQs (23). Heterogeneous cognitive deficits are also commonplace in adolescents with ID, including verbal memory, working memory, and executive functioning impairments (26). Deficits in verbal memory, comprehension, and executive functioning are similarly common in adults with ID (29).

OBJECTIVES

In accordance the aim of the proposed review investigation addresses MR in children and adolescents, in light of neuroscience, diagnostic criteria, Nutritional strategies with respect to treatment and healing

REVIEW OF LITERATURE

The human brain is a self-adjusting organ, the result of co-operative hereditary and environmental influences. Research has shown that the structure of the fine texture of the brain the formation of neural pathways or "neural networks''- is guided by environmental stimuli, natural and social (Katsiou-Zafrana, 2018; Bhatnagar &Taneja, 2001). Cognitive development is affected by many factors, including nutrition. A big bulk of bibliographical references indicates the correlation between healthy nutrition and optimal brain function. Nutrients provide structural elements which play a critical role in cell multiplication, DNA composition and the neurotransmitter and hormone metabolism, as they are vital components of enzyme systems in the brain (Nyaradi, Li, Hickling, Foster, Oddy, 2013; Fenech, 2013; De Souza, Fernandes, do Carmo, 2011). During the first 3 years of a child's life, nutrition plays an important role in the child's health and physical growth. This is a critical period for brain formation, which will serve as the foundation for the development of cognitive, motor and socio-emotional skills throughout life.

Literature in connection to the proposed research is highlighted under following:

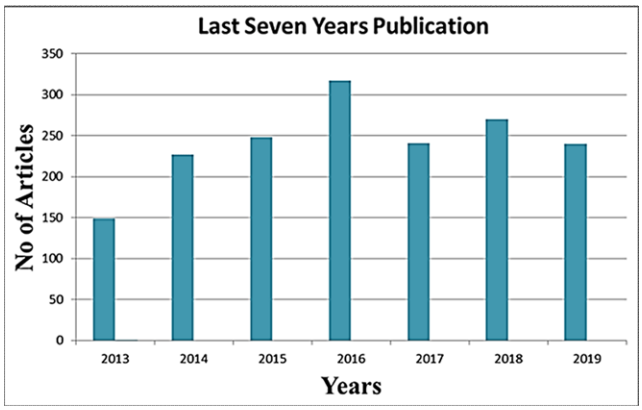
▪ Neuromechanisms in mental retardation ▪ Epidemiology of mental retardation ▪ Mental retardation: Definition and Classification ▪ Prevalence and Incidence of MR/ Intellectual Disabilities: Current Trends and Issues ▪ Etiological factors responsible for the occurrence of mental retardation ▪ Influence of nutrient on cognitive development in early Development years

NEUROMECHANISMS IN MENTAL RETARDATION: The human genome mapping project and gene knockout studies with laboratory animals allow determining specific intracellular changes in each gene mutation and the correlation of a deficient molecule with the resulting cognitive deficit, thus establishing the cellular bases of cognition (58). Neurons, which conduct nerve impulses, have two types of extensions: several short dendrites, which are branched and receive signals from other neurons, and a single long axon, which sends out the signals (37). Dendritic spines are tiny protrusions found in postsynaptic regions of excitatory synapses; serving as a bridge between axons and dendrites, they mediate the synaptic plasticity that determines learning, memory and cognition (35). In other words, synapse remodeling and changes in dendritic spine shape and density underlie many brain functions such as learning and memory (56). Furthermore, various proteins encoded by genes whose mutations produce X linked MR activate the signaling paths that regulate the morphology of dendritic spines, release of neurotransmitters, growth of axons and actin cytoskeleton (44). The current theory is that MR originates from a defect in the structure and function of neuronal synapses (59). MR has been associated with changes in dendrites and dendritic spines for several decades (5). Recently, studies on pyramidal neurons in the cerebral cortex and hippocampus of patients with Down's syndrome, Rett's syndrome and fragile X syndrome have confirmed changes in dendritic spine shape and density (44). Plasticity refers to the capacity of the brain to be molded by experience, learn, recollect, reorganize itself and recover after injury (55).Plasticity develops from the interaction of excitatory and inhibitory synapses, especially excitatory ones, mediated by glutamate (37).Learning and memory are related to short-term changes in the strength or efficiency of synaptic neurotransmission, and long-term changes in the structure and number of synapses (55). Gene transcription is required for the activity of long-term memories and construction of mature neuronal circuits in the developing brain (41).Thus, plasticity involves the stimulation of receptors on the cell surface by neurotransmitters, activation of intracellular signaling cascades, gene transcription, and synthesis of new proteins that modify the physical shape and number of synapses (51).

Pathophysiology: On the basis of our current knowledge, MR resulting from constitutive or somatic mosaic deregulation of genetic information and programs might occur through (i) chromosomal rearrangements that result mainly into deleterious gene dosage effect, (ii) deregulation of the imprinting of specific genes or genome regions and (iii) dysfunction of single genes (monogenic causes of MR), which are individually required for development of cognitive functions (56). Resulting from these monogenic causes is either the only clinical manifestation of the disorders or a symptom of a clinical syndrome with or without detectable brain abnormalities (21). The section below should be regarded as an attempt to provide an updated view (but not an exhaustive list) of the genetic causes of MR, according to their involvement in the emerging molecular pathways and cellular processes thought to contribute to the physiopathological mechanisms underlying cognitive impairment (26).

EPIDEMIOLOGY OF MENTAL RETARDATION: Epidemiological characteristics of MR in California between 1987 and 1994 reported 11,114 children with unclassified MR excluding children diagnosed with cerebral palsy, autism, chromosome aberrations, infections, endocrine or metabolic disorders, traumas or intoxications, brain malformations, and central nervous system diseases or neoplasms (19).Findings reveals that the birth weight < 2.500 g was the strongest predictive factor for MR, and observed other risk factors associated with MR, such as low educational level, advanced maternal age at time of delivery, and multiple births (3). The risk of MR is higher in children with congenital structural defects (7).A study compared the presence of a congenital structural defect at the age of one year with the diagnosis of MR at 7 to 9 years (10).The results showed that congenital structural defects, either involving or not the central nervous system, increased the risk of MR by 27 times (62). Children with Down's syndrome and those with sex-linked chromosome disorders were at greater risk for MR, however, the presence of spina bifida had a relative prevalence of 91.2, compared to children without any congenital disorder; skin disorders showed a prevalence of 70.9; and musculoskeletal disorders had a prevalence of 47.1 (29). Significantly congenital defects evidenced to be the reason for the increased risk of developmental disabilities — MR, cerebral

palsy, hearing impairment and visual loss— in a group of 9,142 children born between 1981 and 1991 (20).Prevalence ratios of MR for each congenital defect in comparison with children without congenital defect, and found the following values: chromosome disorders: 62.5; central nervous system disorders: 30.2; fetal alcohol syndrome: 29,1; TORCH infections: 24.3; ophthalmic disorders: 7.2 (25). The association of MR with multiple disorders suggests that some cases are not caused directly by concurrent congenital defects, but instead, that they could be brought on by other factors found during embryonic development, which would be common causes of congenital defect and MR (11).11



Source Il yas M, Mir A, Efthymiou S and Houlden H. The genetics of intellectual disability: advancing technology and gene editing [version 1]. *F1000Research* 2020, **9**(F1000 Faculty Rev)

Mental retardation: Definition and Classification: Mental retardation is a condition diagnosed before age 18 years that includes below average general intellectual function, and a lack of the skills necessary for daily living (22). Mental retardation is characterized by sub average intellectual functioning, existing concurrently with limitations in conceptual, social, and practical adaptive skills (22). Mental retardation is a clinically and socially important condition (54). Under nutrition may influence brain development by directly affecting brain processes or indirectly by affecting children's experiences and behavior (57). Disabled children are known to be at high risk for developing malnutrition, which may partly explain the growth retardation often encountered in such children (61). The most common problems associated with malnutrition in disabled children, are inadequate nutrient intake either due to feeding problems or poor feeding knowledge among care providers. Mental retardation affects 3% of the total pediatric population (32). According to the Disabilities in Turkey study reports, the ratio of the disabled population and the normal population to be 12.29% and 15.5% respectively (59).Evidently research addresses. Anthropometric parameters and dietary intake to be the indicators in assessing nutritional status of mentally disabled children (61).

Mental retardation refers to a general lower IQ in which an individual is unable to perform present functioning (9). Markedly mental retardation is one of the psychological developmental disability characterized by sub average intelligence and irregular impairments in adapting one of the daily life skills (18). Often this condition is usually prevalent at developing period i.e before 18 years, which is clubbed with numerous biological, environmental and socio-cultural causes (40). Significant characteristic features include sub-average intellectual functioning which exists concurrently with related limitations (41). Noticeably the clinical characteristic features of mental retardation is observed at standard deviation scores less than 2 exhibiting with limited adaptive function in communication, self care, daily living skills in home or community or social skills (45). The fundamental criterion for defining mental retardation depends on the clinical characteristic feature, etiologic feature and ethnicity (29). Various policies and practices have been designed as an assessment strategies and treatment modalities with respect to mental retardation (39).

Defining of mental retardation based on ICD-9-CM codes:

- Mild mental retardation
- Moderate mental retardation
- Severe mental retardation
- Profound mental retardation
- Mental retardation, unspecified
- Global days

According to AAMR (America Association of Mental Retardation); mental retardation refers to enumerate imitating functions happening in present (39). Significantly average intellectual function notably proven to exist concurrently with certain related limitations in two or more of the applicable following adaptations in skill areas: communication, taking care of one self, safety and health, academicals function, leisure or free hours and work (21).

Classification of Mental Retardation: The terms mental retardation/developmental retardation have been replaced with Intellectual disability in the recent classification DSM 5 (44). It is diagnosed before 18 years of age(1). Intellectual disability is a condition of arrested or incomplete development of mind of a child and is specifically characterized by sub average intellectual functioning existing concurrently with limitations in conceptual, social, practical adaptive skills (22). Intellectual disability involves impairment of general mental abilities that impact adaptive functioning in 3 domains. The conceptual domain includes skill in language, reading, writing, math, reasoning, knowledge and memory. The social domain refers to empathy, social judgement, inter personal communication skills, the ability to make and maintain friendships, and similar capacities. The practical domain centre's on self-management in areas such as personal care, job responsibilities, money management, recreation, and organizing school and work tasks (45).

In DSM-5 Intellectual disability is considered to be approximately two standard deviations or more below the normal population which equals to an IQ score of 70 or below (43). The prevalence of intellectual disability in India is 2-3% of general population (1).1 Disabled children are known to be at high risk for developing malnutrition which may partly explain the growth retardation often encountered in such children (5). Non-nutritional factors perhaps could influence growth, but nutritional factors such as insufficient calorie intake, excessive nutrient losses and abnormal energy metabolism also contribute to growth failure (49). The deleterious effect of early malnutrition on later intellectual development in children in developing countries has been clearly documented (32). Under nutrition often associated with a decrease in cerebral function (Stoch *et al*, GranthamMcGregor *et al*. 1991) with possible exacerbation of existing neurological impairments (25). Chronic under nutrition could be associated with increased irritability, and decreased motivation and energy available for non-essential activities such as play and rehabilitation (Stallings *et al*) (25). Significant developmental progress has been shown to accompany improved nutritional status (Sanders *et al*). 30-80% of children with developmental disability have feeding difficulty (32). They are at high risk of oral and pharyngeal dysphagia due to or motor dysfunction (32). Furthermore because of communication difficulties many of them are unable to indicate when they are hungry and unable to request food or drink (49).

A TheClinical characteristic feature to be considered in detecting and defining MR includes:

- Communication
- Self-care
- living styles
- Social skills
- Community use
- Self-direction
- knowledge on Health and safety

- Functionality at academics
- Leisure
- productivity at Work

Classification of Mental retardation according to Indian policies

- Mild mental retardation-IQ from 50-55 to 70
- Moderate mental retardation-IQ from 35-40 to 50-55
- Severe mental retardation-IQ from 20-25 to 35-40
- Profound mental retardation-IQ below 20-25

METHODOLOGY

The proposed systematic review was focused on specified two elements: the prevalence/ incidence with population of interest (Intellectualdisability) and Nutritional effects on quality of children health (7). Study aims at analyzing scientific review and original article findings from PubMed and MEDLINE. With respect to the objectives the research criteria include scientific articles evidences those in English and full-text from epidemiological, cross-sectional, experimental and cohort studies.

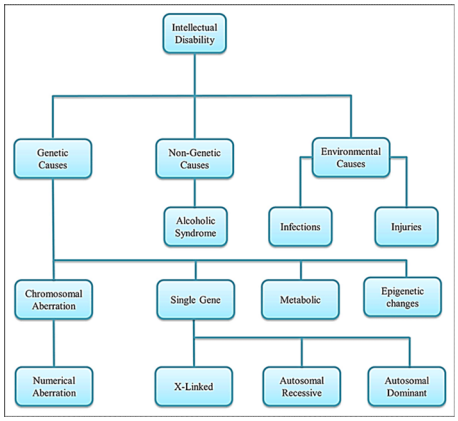
However, studies which did not address the case definition and adequate explanation with respect to prevalence or incidence were excluded (4). Data documentation elicits the compilation of evidence on the prevalence of intellectual disabilities e.g., mild, moderate, or severe cases from specific young populations (e.g, individuals living in correctional facilities, visiting pediatric hospitals, or attending special schools) (4).Research reports focusing on individuals born prematurely, with chromosomal abnormalities, with autism were excluded. Further the findings on prevalence of MR correlated with nutritional factors were emphasized (56).

FINDINGS

PREVALENCE AND INCIDENCE OF MR/ INTELLECTUAL DISABILITIES: CURRENT TRENDS AND ISSUES: Intellectual disabilities are lifelong conditions that manifest during the developmental years and are characterized by below-average general intellectual function and limitations in adaptive functioning (31). In the 1970s, when diagnostic criteria focused on standardized intelligence quotient tests (IQ) cut-offs, it was estimated that 3 % of annual births could be expected to acquire such disabilities at some point in their lives (incidence) (38). This proportion corresponded to a cut-off of approximately 70 on a Gaussian curve for the distribution of intelligence scores (55). Using this cut-off, the proportion of a given population affected (prevalence) to be 3 %.The recognition that IQ alone is not sufficient to diagnose intellectual disabilities, that intellectual disabilities manifest at different ages and under different conditions, and that there are identified causes of the disabilities has led to a reconsideration of both incidence and prevalence (7). As incidence reflects risk in a population, recent focus has been on incidence of intellectual disabilities from specific known causes, such as genetic abnormalities, prenatal exposure to alcohol or infection, trauma during birth, early childhood infections, exposure to heavy metals, and severe malnutrition (10). Trend analyses, including those obtained through systematic reviews of the literature, contribute to the identification of changes in risk factors (12). In 2011, Maulik *et al*. published a systematic review and meta-analysis that evaluated studies of the prevalence of intellectual disabilities published between 1980 and 2009 (15). Prevalence estimates based on psychological assessments were found to be 1% in low- and middle income countries, in children/adolescents populations (20). Evidences on psychiatric disorders in adults with ID suggest that the overall incidence of psychopathology is between 14% and 41% (22). While investigations of severe psychiatric disorders in children and adolescents with ID are scarce, estimates on the rate of comorbid disorders as between 30% and 50% (45). In fact, epidemiological studies indicate that the overall prevalence rates of psychiatric disorders in youth with ID are higher than those in the general population with similarly high incidence rates for children and adolescents with IQs equal to or lower than 50 (19). Meta-analyses demonstrate that individuals with ID are at least three times more likely to experience psychosis than those with higher cognitive functioning. However, the prevalence rates of Schizophrenia Spectrum Disorder (SC), Bipolar Disorder (BD), Posttraumatic Stress Disorder (PTSD) and Disruptive Mood Dysregulation Disorder (DMDD) in children and adolescents with ID all seem uncertain. Relatively major Depressive Disorder (MDD) is noted to be prevalent in youth with ID (5). Certainly diagnosing depression among individuals with a limited ability to describe subjective, internal mental states found to be difficult (8).

ETIOLOGICAL FACTORS RESPONSIBLE FOR THE OCCURANCE OF MENTAL RETARDATION: The discovery of phenylketonuria in 1934 quickly led to the conclusion that a low-phenylalanine diet could prevent MR associated with the disease, 38 and this model of diagnostic definition, which clarified the physiopathology of the disease and indicated possible treatment, encouraged further studies about MR (19). In fact, there are many reasons for establishing the etiology of MR: the family wants to elucidate the problem, and the definition of its cause helps determine the risk of recurrence, request appropriate lab exams, implement proper treatment whenever available, predict the prognosis, and refer patients and family to support groups (26). Nowadays, a careful clinical evaluation can identify the etiology of MR in up to 50 to 70% of cases, a percentage value that is far better than that observed in older case series (34). For instance, in 715 cases investigated from 1985 to 1987, the etiology of MR could be identified in only 22% of children (42). The most prevalent causes, in decreasing order of frequency, were the following: prenatal asphyxia, Down's syndrome, neonatal or postnatal CNS infection and fetal Alcohol syndrome (21). In a more recent study including 99 children younger than five years with global developmental delay, 44 (44%) had a definite diagnosis (26). Seventy seven percent of the cases with known etiology included only four diagnoses— cerebral dysgenesis, hypoxicischemic encephalopathy, intrauterine exposure to toxins, and chromosome aberrations (49). Inborn errors of metabolism were not included in the diagnoses because they had already been identified by universal neonatal screening (60). Using logistic regression, the authors detected clinical characteristics associated with a greater probability of determining the etiology of MR: prenatal exposure to toxins, microcephaly, focal motor symptoms, and absence of autistic behavior (4). A diagnostic survey carried out in southern Brazil included 202 individuals with MR from the Association of Parents and Friends of the Mentally Handicapped (APAE) (8). The authors conducted a careful clinical examination and laboratory investigation to define the diagnosis in 132 patients (65.3%) (41). Down's syndrome was detected in 32.2% of cases, followed by Mendelian inheritance disorders in 12.4%, acquired conditions including infections in 10.4%, and CNS malformations in 4.0%. The high percentage of Down's syndrome probably shows a selection bias (61).

Multiple factors are involved in intellectual disability including genetic inheritance and environmental conditions



NUTRITIONAL INTERVENTIONS ON MENTAL DISORDERS

Nutrient	RDA(3-8 yrs)	Impact of Supplementation in Young Children
Protein	3 yrs – 15.7g/d 4-6 yrs -20.3g/d 8yrs- 29.6g/d	Improvement in cognition [22]
Polyunsaturated fats	25g/d (total visible fat)	Fish oil supplementation in diets of school children have showed improvement in cognition. [14]
Iron	3 yrs – 9mg/d 4-6 yrs -13mg/d 8yrs- 16mg/d	Improvement in cognition and school performance [23]
Zinc	3 yrs – 5mg/d	Improved neuro-psychological functions [24]
	4-6 yrs -7mg/d 8yrs- 8mg/d	
Iodine	90mcg/d 25	Supplementation of moderately iodine deficient school age children improves cognitive function[6]
Vitamin B12	3-6 yrs – 80- 100mcg/d 7-8yrs – 120- 140mcg/d	Supplement in children improves neurodevelopment & cognitive function[27]
Vitamin D	15mcg/d 26	Supplementation in children with attention deficit hyperactivity disorder was found to improve cognitive function [28]
Vitamin A	3 yrs – 400mcg/d 4-6 yrs -600mcg/d 8yrs- 600mcg/d	Insufficient evidence in 3-8 yrs children
Thiamine (Vitamin B1)	3 yrs – 0.5mg/d 4-6 yrs -0.7mg/d 8yrs- 0.8mg/d	Better intelligence scores Better memory Quicker reaction times [6]
Pyridoxine (Vitamin B6)	3 yrs – 0.8mg/d 4-6 yrs -1.1mg/d 8yrs- 1.4 mg/d	Insufficient evidence in 3-8 yrs children[6]
Folic acid	3-6 yrs- 80-100mcg/d 7-8 yrs- 120- 140mcg/d	Supplement in children improves neurodevelopment & cognitive function

INFLUENCE OF NUTRIENT ON COGNITIVE DEVELOPMENT IN EARLY YEARS

Nutrients often found to be the vital component for the development and function of the human system. During rapid growth, there is a great need for provision of nutrients, necessary for the development of the brain (57). Nutrient inadequacy, however, can endanger the structural development of the brain, as it is likely to cause serious and permanent damage (3). This is because nutrients can influence the anatomy of the neurons by reducing their multiplication or diversity (Nurliyana, MohdShariff, MohdTaib, Gan, Tan, 2016; Benton, 2008) (7). Recent findings have established that the point of time, the duration and the severity of nutrient inadequacies have a different impact on brain development and subsequent cognitive and emotional procedures (Black, 2018) (16). Energy is nothing but the calorie content of the food; it is derived from the carbohydrates, protein, fat and alcohol found in the food and beverages (29). Even vitamins and minerals are essential to the body which provides no energy (31). The brain of the human is very active metabolically and uses for about 20 to 30% of energy intake when the person is in rest (47).

Individuals who do not consume much of calories from food to meet the requirements of energy will surely experience the change in the mental function (52). Skipping breakfast for no reason is bonded with the lower fluency and ability of solving problems, especially for the ones who are slightly malnourished (33). The person who is hungry has no ability of energy to get motivated (5). Chronic hunger and energy deprivation affects mood and responses (8). The body usually responses for the energy deprivation by closing or slowing down non-essential functions, altering the activity of levels, hormonal levels, nutrient and oxygen transport, the ability of the body to fight infections , and other many functions in the body that is direct or indirect affects the brain (17). Individual with consent of low energy intake often feel apathetic, sad or hopeless (37). Fetus which are developing and young infants are particular to the susceptible of brain damage due to malnutrition. The more the energy deprivation the more the damage, the timing in these two stages of the development (44).

NUTRITIONAL INTERVENTIONS ON MENTAL DISORDERS

Nutritional interventions on dietary improvements thereby reduce the risk or may even lock the progression of certain psychiatric disorders (52). Clinical studies are backbone for the use of certain nutrients, which drives away a range of neuron chemical activity benefits for treating mental disorders in the form of medical supplements (52). Certain evidence from clinical research drives the use of natural medicines on psychiatric disorders (56). Typically, most of the mental disorders are treated with medicines or prescribed drugs adverse side effects (55). Western diet has a correlation with mental disorders (1). individual who suffer from mental retardation found to exhibit nutrient deficiencies such as; omega 3 fatty acids, N-acetylcysteine [NAC], S- adenosyl methionine [SAM], zinc, magnesium, vitamin D and B vitamins [including folic acid] (47). Natural compounds such as micro biotic derived from laboratory synthesis or fermented foods, amino acids plant-based antioxidants found to influence the brain health. Certain natural compounds having brain chemical- modulating effects, noted to play an important role in treating mental disorders. Evidently nutritional therapies with specific bioactives comprising neuroprotective actions has shown a greater impact in treating mental retardation (47,49).

SUMMARY AND CONCLUSION

The elasticity of the brain is the means nature uses to protect the brain from external influences, allowing for its adaptation to environmental effects. Yet, it is all dependent on the time, the duration and the severity of the influence. Optimum nutrition, with adequate quantities of essential micronutrients, proteins and calories, provided in the appropriate time period, can ensure proper development of the brain and its functioning. Significant congenital malformations, such as hydrocephaly, mental retardation and behavior problems often could be corrected with specific nutritional therapies. Notably nutrition deficiencies are regarded as a public health issue of the utmost importance. Long term nutritional issues could raise burden of disease resulting to various morbidities. Often this could impair childhood development milestones. Importantly isolated micronutrient components, including n-3 fatty acids, zinc, iron and iodine have shown a significant effect on cognitive development. Markedly, parents, as they act as models for their children, need to be sensitized towards healthy dietary practices and ensuring nutritional surveillance in childhood development is imperative.

REFERNCES

1. https://www.webmd.com/parenting/baby/intellectual-disability-mental-retardation
2. https://www.pediatrics.emory.edu/centers/pehsu/health/mental.html
3. https://www.healthline.com/health/mental-retardation
4. https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/mentalretardation
5. https://en.m.wikipedia.org/wiki/Intellectual_disability
6. https://www.medicinenet.com/script/main/art.asp?articlekey=20174
7. https://pedsinreview.aappublications.org/content/27/6/204
8. https://pedsinreview.aappublications.org/content/27/6/204
9. https://emedicine.medscape.com/article/289117-overview
10. https://www.aafp.org/afp/2000/0215/p1059.html
11. Jacka FN, *et al*. Western diet is associated with a smaller hippocampus: a longitudinal investigation. (2015). BMC Med. 13:215.

12. Jacka, FN, *et al*. A randomised controlled trial of dietary improvement for adults with major depression (the 'SMILES' trial). (2017). BMC Medicine. 15(1): 23.
13. Parletta, N, *et al*. A Mediterranean-style dietary intervention supplemented with fish oil improves diet quality and mental health in people with depression: A randomized controlled trial (HELFIMED)." (2017). Nutritional Neuroscience. Dec 7;1-1
14. Opie RS, *et al*. Assessing Healthy Diet Affordability in a Cohort with Major Depressive Disorder. (2015). Journal of Public Health and Epidemiology. 7(5):159-69.
15. Opie RS, *et al*. A modified Mediterranean dietary intervention for adults with major depression: Dietary protocol and feasibility data from the SMILES trial. (2017). Nutritional Neuroscience. Apr 19:1-
16. Grosso G, *et al*. Dietary n-3 PUFA, fish consumption and depression: A systematic review and meta-analysis of observational studies. (2016). Journal of Affective Disorders. 205:269-81.
17. Mocking RJ, *et al*. Meta-analysis and meta-regression of omega-3 polyunsaturated fatty acid supplementation for major depressive disorder. (2016). Translational Psychiatry. 6:e756.
18. Tolmunen T, *et al*. Dietary folate and the risk of depression in Finnish middle-aged men. A prospective follow-up study. (2004). Psychotherapy and Psychosomatics. 73:3349.
19. Ng TP, Feng L, Niti M, Kua EH, Yap KB. Folate, vitamin B12, homocysteine, and depressive symptoms in a population sample of older Chinese adults. (2009). Journal of the American Geriatrics Society. 57:871-876.
20. Anglin R, Samaan Z, *et al*. Vitamin D deficiency and depression in adults: systematic review and meta-analysis. (2016). British Journal of Psychiatry. 202:100-107.
21. Ainsworth, Patricia, and Pamela Baker. Understanding Mental Retardation. Jackson, MS: University Press of Mississippi, 2004.
22. Libal, Autumn. My Name Is Not Slow: Youth with Mental Retardation. Broomall, PA: Mason Crest Publishers, 2004.
23. Wehmeyer, Michael L., *et al*. Teaching Students with Mental Retardation: Providing Access to the General Curriculum. Baltimore: Brookes Publishing Co., 2001.
24.Cinamon, Rachel Gali, and Limor Gifsh. "Conceptions of Work among Adolescents and Young Adults with Mental Retardation." Career Development Quarterly 52 (March 2004): 212–24.
25. Howard, Barbara J. "Mental Retardation Challenges." Pediatric News 38 (September 2004): 20–1.
26. Kerker, Bonnie D., *et al*. "Mental Health Disorders among Individuals with Mental Retardation: Challenges to Accurate Prevalence Estimates." Public Health Reports 119 (August 2004): 409–17.
27. Ly, Tran M., and Robert M. Hodapp. "Maternal Attribution of Child Noncompliance in Children with Mental Retardation: Down Syndrome versus Other Causes." Journal of Developmental & Behavioral Pediatrics 23 (October 2002): 322–29.
28. Sampaio RC, Truwit CL. Myelination in the developing human brain. In: Nelson CA, Luciana M, eds. Handbook of Developmental Cognitive Neuroscience. Cambridge, MA: MIT Press; 2001:35–44.
29. Oppenheim RW. Cell death during development of the nervous system. Annu Rev Neurosci. 1991;14:453–501.
30. Fugelstad A, Rao R, Georgieff MK. The role of nutrition in cognitive development. In: Nelson CA and Luciana M, ed. Handbook of Developmental Cognitive Neuroscience, 2nd ed. Cambridge, MA: MIT Press; 2008:623–641.
31. Tolsa CB, Zimine S, Warfield SK, *et al*. Early alteration of structural and functional brain development in premature infants born with intrauterine growth restriction. Pediatr Res. 2004;56:132–138.
32. Winick M, Rosso P. The effect of severe early malnutrition on cellular growth of human brain. Pediatr Res. 1969;3:181–184.
33. Cordero ME, D'Acuna E, Benveniste S, *et al*. Dendritic development in neocortex of infants with early postnatal life undernutrition. Pediatr Neurol. 1993;9:457– 464.
34. Cordero ME, Valenzuela CY, Rodriguez A, *et al*. Dendritic morphology and orientation of pyramidal cells of the neocortex in two groups of early postnatal undernourished-rehabilitated rats. Brain Res Dev Brain Res. 2003;142:37–45.
35.. Jones DG, Dyson SE. The influence of protein restriction, rehabilitation and changing nutritional status on synaptic development: a quantitative study in rat brain. Brain Res 1981;208:97–111.

.
36. Wiggins RC, Fuller G, Enna SJ. Undernutrition and the development of brain neurotransmitter systems. Life Sciences. 1984;35:2085–2094.
37. Hulshoff HE, Hoek HW, Susser E, *et al*. Prenatal exposure to famine and brain morphology in schizophrenia. Am J Psychiatry. 2000;157:1170–1172.
38. Antonow-Schlorke I, Schwab M, Cox LA, *et al*. Vulnerability of the fetal primate brain to moderate reduction in maternal global nutrient availability. Proc Natl Acad Sci U S A. 2011;108:3011–3016.
39. Coti Bertrand P, O'Kusky JR, Innis SM. Maternal dietary (n-3) fatty acid deficiency alters neurogenesis in the embryonic rat brain. J Nutr. 2006;136:1570– 1575.
40. Uauy R, Dangour AD. Nutrition in brain development and aging: role of essential fatty acids. Nutr Rev. 2006;64(Suppl):S24–S33. discussion S72–91.
41. Miller SL, Klurfeld DM, Loftus B, *et al*. Effect of essential fatty acid deficiency on myelin proteins. Lipids. 1984;19:478–480.
42. McKenna MC, Campagnoni AT. Effect of pre- and postnatal essential fatty acid deficiency on brain development and myelination. J Nutr. 1979;109:1195– 1204.
43. Rao R, Tkac I, Schmidt AT, *et al*. Fetal and neonatal iron deficiency causes volume loss and alters the neurochemical profile of the adult rat hippocampus. Nutr Neurosci. 2011;14:59–65.
44. Jorgenson LA, Wobken JD, Georgieff MK. Perinatal iron deficiency alters apical dendritic growth in hippocampal CA1 pyramidal neurons. Dev Neurosci. 2003;25:412–420.
45. Jorgenson LA, Sun M, O'Connor M, *et al*. Fetal iron deficiency disrupts the maturation of synaptic function and efficacy in area CA1 of the developing rat hippocampus. Hippocampus. 2005;15:1094–1102.
46. Beard JL, Connor JR. Iron status and neural functioning. Annu Rev Nutr. 2003;23:41–58.
47. Beard J. Recent evidence from human and animal studies regarding iron status and infant development. J Nutr. 2007;137(Suppl):524S–530S.
48. Kwik-Uribe CL, Gietzen D, German JB, *et al*. Chronic marginal iron intakes during early development in mice result in persistent changes in dopamine metabolism and myelin composition. J Nutr. 2000;130:2821–2830.
49. de Escobar GM, Obregon MJ, del Rey FE. Iodine deficiency and brain development in the first half of pregnancy. Public Health Nutr. 2007;10: 1554–1570.
50. Chen ZP, Chen XX, Dong L, *et al*. The iodine deficient rat. In: Medeiros-Neto G, Maciel RMB, Halpern A, eds. Iodine Deficiency Diseases and Congenital Hypothyroidism. Sao Paolo: Ache Press; 1986:46–51.
51. Dussault JH, Ruel J. Thyroid hormones and brain development. Annu Rev Physiol. 1987;49:321– 334.
52. Jia-Liu L, Zhong-Jie S, Yu-Bin T, *et al*. Morphologic study on cerebral cortex development in therapeutically aborted fetuses in an endemic goiter region in Guizhou. Chin Med J (Engl). 1984;97:67–72.
53. Sandstead HH. W.O. Atwater memorial lecture. Zinc: essentiality for brain development and function. Nutr Rev. 1985;43:129–137. Nutrition Reviews® Vol. 72(4):267–284 281 Downloaded from https://academic.oup.com/nutritionreviews/article-abstract/72/4/ 2 67/1859597 by guest on 20 May 2020

54. Walsh CT, Sandstead HH, Prasad AS, *et al*. Zinc: health effects and research priorities for the 1990s. Environ Health Perspect. 1994;2:5–46.
55. McNall AD, Etherton TD, Fosmire GJ. The impaired growth induced by zinc deficiency in rats is associated with decreased expression of the hepatic insulinlike growth factor I and growth hormone receptor genes. J Nutr. 1995;125:874– 879.
56. Blusztajn JK, Cermak JM, Holler T, *et al*. Imprinting of hippocampal metabolism of choline by its availability during gestation: implications for cholinergic neurotransmission. J Physiol Paris. 1998;92:199–203.
57. Molloy AM, Kirke PN, Troendle JF, *et al*. Maternal vitamin B12 status and risk of neural tube defects in a population with high neural tube defect prevalence and no folic acid fortification. Pediatrics. 2009;123:917–923.
58. Chang SJ, Kirksey A, Moore DM. Effects of vitamin B6 deficiency on morphological changes in dendritic trees in Purkinje cells in developing cerebellum in rats. J Nutr. 1981;111:848–857.
59. Groziak SM, Kirksey A. Effects of maternal restriction of vitamin B6 on neocortex development in rats: neuron differentiation and synaptogenesis. J Nutr. 1990;120:485–492.
60. Guilarte T. Vitamin B6 and cognitive development: recent research findings from human and animal studies. Nutr Rev. 1993;51:193–198.
61. Guilarte TR, Wagner HN, Frost JJ. Effects of perinatal vitamin B6 deficiency on dopaminergic neurochemistry. J Neurochem. 1987;48:432–439.
62. Moore DM, Kirksey A, Das GD. Effects of vitamin B6 deficiency on the developing central nervous system of the rat. Myelination. J Nutr. 1978;108:1260–1265.
63. Kolb B, Whishaw IQ. Brain plasticity and behavior. Annu Rev Psychol. 1998;49:43–64.

****