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Elements deficiency that causes brain diseases

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ABSTRACT

The trace elements deficiency is the most common micronutrient deficiency in the world. Trace metals such as iron, zinc, cobalt, copper, potassium, lithium, iodine, manganese, molybdenum etc., are necessary for the growth and function of the brain. The transport of trace metals into the brain is strictly regulated by the brain barrier system. The homeostasis of trace metals in the brain is important for brain function and also for the prevention of brain diseases. Women of reproductive age, young children and age person are particularly vulnerable to the element deficiency diseases.

Keywords: Trace elements, brain barrier system, homeostasis.

INTRODUCTION

Trace elements are chemical elements needed in minute amounts for normal physiology. Some of the physiologically relevant trace elements include iodine, copper, iron, manganese, zinc, selenium, cobalt and molybdenum. Of these, some are metals, and in particular, transition metals[1]. Trace metals usually serve the function of metalloproteins in neurons and glial cells, while a portion of trace metals exists in the presynaptic vesicles and may be released with neurotransmitters into the synaptic cleft. Zinc and manganese influence the concentration of neurotransmitters in the synaptic cleft, probably via the action against neurotransmitter receptors and

transporters and ion channels. Zinc may be an inhibitory neuromodulator of glutamate release in the hippocampus, while neuromodulation by manganese might mean functional and toxic aspects in the synapse. Dietary zinc deficiency affects zinc homeostasis in the brain, followed by an enhanced susceptibility to the excitotoxicity of glutamate in the hippocampus. Transferrin may be involved in the physiological transport of iron and manganese into the brain and their utilization there. It is reported that the brain transferrin concentration is decreased in neurodegenerative diseases such as Alzheimer's disease and Parkinson's

disease and that brain iron metabolism is also altered[2].

ELEMENTS IN BRAIN

The some of the important elements present in brain are as follows;

- IRON
- ZINC
- COPPER
- POTTASIIUM
- LITHIUM
- COBALT
- RUBIDIUM
- SELENIUM
- CHROMIUM
- MANGANESE
- CESIUM
- ANTIMONY
- CALICUM
- SCANDIUM

IRON

The increase in the iron in brain can accelerate dementia and brain deterioration. The decrease in the iron may causes energy problem. The iron is primary component in blood, without it causes anemia and fatigue[3].

RISK FACTORS

- Alzheimer's disease
- Parkinson disease

DIETARY REQUIRMENTS

- Women -18mg
- Men - 8mg

IRON RICH FOODS

- Nuts
- Green leafy vegetables
- Lentils
- Beef
- Sea foods

ZINC

The highest amount of zinc is present in the brain. The zinc constitutes structure and function of brain. It leads to cognitive development and helps to bring back to health, boost the immune system, and in the brain it controls the neuro impulses. High dose of zinc are

known to help in reduce epilepsy. Zinc also helps in smooth neuron connection and make recall easier[4].

ZINC DEFICIENCY IN CHILD

- Deflects in brain growth
- Immune system
- Motor development
- Change in behaviour

RISK FACTORS

- Seizure
- Depression
- Wilson diseases
- Malformation of brain

RECOMMENDED SOURCE

- Women-8mg
- Men-11mg

DIETARY SOURCE

- Beef
- Spinach
- Wheat germ
- Chocolate
- Pumpkin seed
- Nuts

RUBIDIUM

The rubidium content of brain differs significantly between defined functional regions, and rubidium can apparently enhance the turnover of brain norepinephrine (noradrenaline) and cause electroencephalogram activation in monkeys and rats[4].

RISK FACTOR

- Alzheimer's disease
- Cerebral infarction
- Stroke

RECOMMENDED SOURCE

- Men - 10.3 mg/kg-day
- Women – 5.6 mg/kg-day

DIETARY SOURCE

- Spinach
- Collards
- Broccoli

CALCIUM

It's a crucial element in how the cells in your brain communicate. As an electrical signal speeds down the axon, it opens pores that let calcium ions rush into the cell. The high calcium concentrations let the neuron know that it's time to release its neuro-transmitters, the

chemical messages neurons use to communicate with each other[5].

RISK FACTOR

- Alzheimer disease
- Bipolar Disorder
- Schizophrenia

DIETARY SOURCE

- Yogurt
- Almonds
- Beans and Lentils
- Whey Protein
- Leafy greens

CHROMIUM

Chromium is important in the breakdown of fats and carbohydrates. It stimulates fatty acid and cholesterol synthesis. They are important for brain function and other body processes. Chromium also aids in insulin action and glucose breakdown. The predominant form of chromium that is found in the body is trivalent chromium (Cr^{3+}). Cr^{3+} , which can be sourced from many foods, is believed to be involved in normal insulin function. Insulin is key to maintaining the living state and storing carbohydrates, lipids, and proteins within the human body[5].

RISK FACTOR

- Lung Cancer
- Asthma
- Dermatitis

RECOMMENDED SOURCE

- Men - 35 mcg/day
- Female - 25 mcg/day

DIETARY SOURCE

- Wheat Germ
- Broccoli
- Chicken
- Eggs
- Liver

COPPER

Copper is the trace element present in all tissues and is required for cellular respiration, peptide-amidation, neuro transmitter biosynthesis, pigment formation and connective tissue strength. It plays an role in central system development[5,6].

RISK FACTOR

- Wilson's disease
- Myelo-neuropathy

- Neurological dysfunction

RECOMMENDED SOURCE

- Women-900mcg/day
- Men -900mcg/day

DIETARY SOURCE

- Beef liver
- Sunflower seeds
- Lentils
- Almond
- Asparagus
- Mushrooms

POTASSIUM

Potassium act as an electrolyte it promotes muscle activity it supports the brain memory function. Because potassium plays a role in the basic function of brain neurons, supplementation may be recommended as put of therapeutic approach to treating dementia potassium boosts memory and learning in healthy individuals

LOW POTASSIUM CAUSES

Memory loss and confusion are common side effects, decrease memory function. A low potassium level has many causes but usually results from vomiting, diarrhea, adrenal gland disorders, or use of diuretics[6].

RECOMENDED SOURCE

- Infants-3,000mg(day)
- Adults-4,700mg/day

RISK FACTOR

- Memory loss
- Confusion

DIETARY SOURCE

- White beans
- Yogurt
- Potato
- Sweet potato

LITHIUM

Lithium is an essential nutrient in the human body that prevent mental disorder such as Alzheimer's disease and dementia. It help in control depression and mania

RECOMMENDED SOURCE

- Adult-1-3mg

DIETARY SOURCE

- Mollusks
- Fish
- Egg
- Meat

- Scallops
- Oysters

COBALT

The cobalt influences brain development and function. The body may use cobalt in place of zinc. Cobalt also helps in regulate and stimulate the production of some enzyme such as thyroxin, thyroid hormone. It helps in the formation of haemoglobin. It also benefits in same cases of fatigue, digestive disorder and neuromuscular problem[7].

DAILY REQUIREMENT

- Adult – 5-8mg/day

DIETARY SOURCES

- Calms
- Fish
- Leafy green vegetables
- Liver
- Milk
- Nuts
- Oysters
- Red meat

MANGANESE

The manganese is one of the essential trace elements which are required for normal body growth and development. It also plays a role in cellular homeostasis[7]. The deficiency in the Mn May leads to a brain defects, malformation of bones, infertility, enhances the chances of seizures and muscle weakness. Mn act as a cofactor for certain enzymes, which is required for the glial cells and neuronal function, which also involves in synthesis of some neurotransmitter and metabolism. The human highest level of Mn was found in the globus pallidus. The increase in the Mn level may alter the effect of dopamine, glutamate and γ -aminobutyric acid in brain. The major organ affected by increased level of manganese is brain and the lungs. It also leads to an impairment of haemostasis of iron, mitochondrial dysfunction, excitotoxicity, induction in the aggregation of protein and oxidative stress[8]. The accumulation of Mn in the brain may leads to an basal ganglia dysfunction, which may causes the neurological disorders. The Mn may get exposure to the body via dietary intake, inhalation and dermal absorption.

RISK FACTORS

- Parkinson's disease(PD)
- Damage brain dopaminergic system

RECOMMENDED SOURCE

Mn level in brain tissue average approximate of 1-2mcg/gm

DIETARY SOURCE

- Grains
- Nuts
- Seeds
- Tea
- Legumes
- Pineapple
- Beans

SELENIUM

The selenium is an essential trace element, which distributed widely in the human body, mainly present in the brain. The selenium has an importance function in brain in maintaining motor performance, memory, coordination and cognition. It act against the free radicals thus considered as an protective agent, which also enhance the enzyme activity[9]. The selenium leaves from the body within 24 hours after enters into the body. The selenium side effects may include nausea, vomiting, hair loss, mild rash, tremors, flushing, feel tired and lack of energy. The changes in the concentration level of selenium in the brain and the blood may leads to Alzheimer's disease[10].

DIETARY REQUIREMENTS

- Adult -200 microgram, above which is considered as an over dose.

DIETARY SOURCES

- Brazil nuts
- Ham
- Fish
- Pork
- Turkey
- Beef
- Chicken

CONCLUSION

It shows that the zinc and iron deficiency may concerned with most diseases and these elements has been performed for over 50 years, but there are still no satisfactory theories that can be integrated with trace element deficiency. The above review states, that the body elements should be in the normal level to maintain the body homeostasis. The increase and decreased level of various elements in the brain may leads to a various neurological disorders.

REFERENCES

1. Taylor A. Detection and monitoring of disorders of essential trace elements. *Ann Clin Biochem.* 1996 Nov;33(6):486-510. doi: 10.1177/000456329603300603, PMID 8937580.
2. Prohaska JR. Functions of trace elements in brain metabolism. *Physiol Rev.* 1987 Jul;67(3):858-901. doi: 10.1152/physrev.1987.67.3.858, PMID 3299411.
3. Schroeder HA, Frost DV, Balassa JJ. Essential trace metals in man: selenium. *J Chronic Dis.* 1970 Oct 1;23(4):227-43. doi: 10.1016/0021-9681(70)90003-2, PMID 4926392.
4. Abboud S, Haile DJ. A novel mammalian iron-regulated protein involved in intracellular iron metabolism. *J Biol Chem.* 2000 Jun 30;275(26):19906-12. doi: 10.1074/jbc.M000713200, PMID 10747949.
5. Adlard PA, Cherny RA, Finkelstein DI, Gautier E, Robb E, Cortes M et al. Rapid restoration of cognition in Alzheimer's transgenic mice with 8-hydroxyquinoline analogs is associated with decreased interstitial A β . *Neuron.* 2008 Jul 10;59(1):43-55. doi: 10.1016/j.neuron.2008.06.018, PMID 18614028.
6. Altamura S, Muckenthaler MU. Iron toxicity in diseases of aging: Alzheimer's disease, Parkinson's disease and atherosclerosis. *J Alzheimers Dis.* 2009 Jan 1;16(4):879-95. doi: 10.3233/JAD-2009-1010, PMID 19387120.
7. Bell RD, Zlokovic BV. Neurovascular mechanisms and blood-brain barrier disorder in Alzheimer's disease. *Acta neuropathol.* 2009 Jul 1;118(1):103-13. doi: 10.1007/s00401-009-0522-3, PMID 19319544.
8. Bishop GM, Robinson SR, Liu Q, Perry G, Atwood CS, Smith MA. Iron: a pathological mediator of Alzheimer disease? *Dev Neurosci.* 2002;24(2-3):184-7. doi: 10.1159/000065696, PMID 12401957.
9. Gallagher JJ, Finnegan ME, Grehan B, Dobson J, Collingwood JF, Lynch MA. Modest amyloid deposition is associated with iron dysregulation, microglial activation, and oxidative stress. *J Alzheimers Dis.* 2012 Jan 1;28(1):147-61. doi: 10.3233/JAD-2011-110614, PMID 21971404.
10. Gebril OH, Simpson JE, Kirby J, Brayne C, Ince PG. Brain iron dysregulation and the risk of ageing white matter lesions. *NeuroMolecular Med.* 2011 Dec 1;13(4):289-99. doi: 10.1007/s12017-011-8161-y, PMID 21979376.