

Importance of Zinc in Human Diet

Shraddha Soni¹, Ashutosh Chaturvedi¹, Jai Prakash Singh²

¹Research Scholar, ²Associate Professor, Department of Kayachikitsa, Faculty of Ayurveda, Institute of Medical Sciences, Banaras Hindu University, Varanasi

Corresponding Author: Shraddha Soni

ABSTRACT

It is well recognized that zinc is an essential trace element, influencing growth and affecting the development and integrity of the immune system and many more physiological functions in human body. Social and economic conditions can adversely affect dietary choices and eating patterns and it cause many kinds of health problems because of low nutritional level. It is clear that this trace element has a broad impact on key immunity mediators, such as enzymes, thymic peptides and cytokines, explaining the paramount importance of zinc's status on the regulation of lymphoid cell activation, proliferation and apoptosis. The total amount of Zn in the human adult has been estimated to be approximately 2 g. More than 80% of this is found in bone, muscle, hair and skin, and a large number of enzymes require Zn for maximum catalytic activity, e.g., alcohol dehydrogenate, RNA nucleotidyl transferase and alkaline phosphates. So, it plays a major role in human body to continue a healthy active life. In this study here we are going to discuss how zinc affect health and how it works for immunity, nervous system, taste acuity (mainly in elderly), for healthier bone and wound healing.

Key Word: Zinc, Human diet.

INTRODUCTION

Zinc is a trace element for human and other animal, a component of more than 300 enzymes. Deficiency of zinc is very widespread throughout the world may even be as prevalent as iron deficiency anemia. It is typically the second most abundant transition metal which appears in all enzyme classes. In small amount zinc presents in all tissues and also required for the synthesis of insulin by the pancreas. Human body contains 2-4g of zinc, but plasma zinc only occurs in co-concentration of 12-16 umol/L (96 ug/100ml). Apart from people with genetic or clinical disorders, certain apparently-healthy members of the population may be susceptible to Zn deficiency. These include infants, growing children, and pregnant and lactating women, whose requirements are especially high in order to support growth. There are some

groups that are at high risk of zinc deficiency such as elderly people, vegetarians and patients with renal insufficiency.

Zinc Deficiency in Human

The consequence of zinc deficiency are several. They impact on human health severally, growth retardation, male hypogonadism and neurosensory changes (abnormal behavior and change in taste in acuity) and delayed wound healing, abnormal immune system, impaired cognitive function and decrease in night vision are some major effect of human zinc deficiency which are reversible with zinc full diet and supplementation.^[1] Moderate level of zinc deficiency occur due to dietary factors, malabsorption syndrome, alcoholic liver disease, chronic renal disease, chronically debilitated condition pernicious anemia, thalassemia and myocardial

infarction. [2] One from the severe zinc deficiency is acrodermatitis enteropathica a rare genetic disease in which zinc absorption is significantly impaired. [3]

Zinc is essential for the maintenance of protein structure and nucleic acids. The maintenance and replication of genetic material (DNA and RNA) and the use of genetic information to generate specific protein are dependent on zinc. [4] it also involved in zinc transport and in maintaining the stability of lipids within the cell membrane. Zinc dependent enzymes are involved in the synthesis of long-chain fatty acids and various prostaglandins. [5]

Psychological Function

Zinc deficiency appears to be a factor in a number of psychological processes due to its important role in the functioning of the central nervous system. [6] Studies have showed deficiency of Zinc may affect cognitive development by alterations in attention, activity, neuropsychological behavior and motor development. Research has shown that certain micronutrients, including Zn, are significantly depleted in depressed patients. [7] And Zn depletion has also been implicated in mood disorders. [8] Zn can act as a neuromodulator or neurotransmitter. [9] Studies in animals show that zinc deficiency during the time of rapid brain growth, or during the juvenile and adolescent period affects cognitive development by decreasing activity, increasing emotional behavior, impairing memory and the capacity to learn. [10] Zinc has an important role in the formation of key enzymes involved in uptake or release of neurotransmitters. All neural tissue contains zinc, but the hippocampus, an area important for memory and other integrative functions, is especially rich in zinc, zinc is concentrated in the hippocampal mossy fiber pathway. [11] Serum Zn concentrations have been associated with impaired cognitive function in older individuals. [12] In addition, zinc is involved in the metabolism of thyroid hormones, receptor function and transport of other hormones that could influence the

central nervous system. [13] Evidence from the available literature suggests that both deficiency and excess of Zn may have profound positive and negative consequences, respectively, on human behavior.

Bone Metabolisms

Zn is essential for growth. It has been reported to exert an effect upon enzymes and enzymatic function, [14] protein synthesis, [15] carbohydrate metabolism, [16] and bone formation. [17] Bone growth retardation is a common finding in Zn deficiency both experimentally induced deficiencies in growing animals [18] in children as a result of dietary insufficiency, [19] and in elderly. Zn and bone metabolism diseases osteoporosis is a average age-related condition, [20] and is a major cause of morbidity and mortality in the elderly of both sexes. Bone is lost at a rate of 0-2-0-5 %/year in both men and women after the age of 40-45 years. Subclinical Zn deficiency, due to a reduced dietary intake and/or impaired intestinal absorption of Zn may be a contributory factor for age-related osteoporosis. [21] The causes of age-related changes in bone mass are multifactorial and include genetic predisposition, nutritional factors, endocrine changes, habitual exercise levels and body weight. Bone loss is accelerated to 2-5 % year immediately before and for up to 10 years post-menopause. [22] Zn deficiency results in a reduction in femur Zn concentration, [23] a reduction in cancellous bone mass and a deterioration of trabecular bone architecture. [24]

Taste Disorder

Taste, one of the major senses in humans, it is the sensory system devoted primarily to a quality check of food to be ingested. Although aided by smell and visual inspection, the final recognition and selection relies on chemoreceptive events in the mouth. [25] There are different- different type of taste disorder --- Dysgeusia defined as (distorted sense of taste), which is often used as a general description for any taste abnormality, more specific terms include

ageusia (complete loss of taste sensation); hypogeusia (impairments of the sense of taste or decrease in taste perception, leading to an increase in the taste threshold); hypergeusia (increased sensitivity for taste stimuli); parageusia (bad taste in the mouth); phantogeusia that is a gustatory hallucination (perception of a taste in the absence of a stimulus); gustatory agnosia (loss of the ability to identify a given taste stimulus, although still able to recognize between different stimuli), while normogeusia (is the name given to normal taste. [26])

Taste disorders are basically classified into two types, on the basis of the presence or absence of taste: ageusia and dysgeusia. Ageusia is the complete loss of the ability to taste, which is caused by the redundant gustatory innervation of the tongue. Dysgeusia is the impairment of taste sensation and is the most common type of taste disorder, occurring in about 34% of all patients with taste disorders. [27] Dysgeusia is classified into two types: hypogeusia and hypergeusia. On the basis of the state of impairment, taste disorders can also be classified into three categories. The first category involves external damage to the gustatory papillae and taste buds, and is caused by dry mouth; saliva plays an important role in taste perception. [28] As taste stimulants require salivary secretion to get to the taste buds, so patients with decreased or absent saliva exhibit decreased taste acuity (xerostomia, hyposalivation), tongue coating, atrophic glossitis, iatrogenic causes (e.g., dental treatment or exposure to radiation), burns, exposure to toxic substances and other external sources of damage. The second category involves internal damage to the gustatory papillae and taste buds, [29] and is caused by zinc deficiency, aging, excessive medication intake, vitamin deficiency, systemic disease (e.g., bulimia, anorexia, hypothyroidisms, Cushing's syndrome, diabetes mellitus, [30] liver disease, kidney disease and others, [31] infections of the upper respiratory tract, and exanthema dysgeusia. [32] The third category

involves disturbance of the taste sensation neural pathway as a result of peripheral or central nerve damage, [33] such as taste bud degeneration occurring after chord tympani nerve injury or head trauma. [34] However, it is possible for taste cells to regenerate, with a half-life of approximately 10 days. Taste distortions in human beings have been attributed to various physiological and environmental factors including aging and disease conditions. In fact, age-related decline in taste acuity may be both a cause and an effect of zinc depletion, and it may be associated with an increased requirement of zinc. Moreover, this age-related decline in taste acuity will likely be observing globally. [35] And the results of the 1994 USA survey also indicated that 40% of patients with taste and smell disorders were more than 65 years old. [36] Taste is one of the most important factors in food preference, selection, and consumption, the decreased appetite in the elderly, probably due to disease conditions, may lead to dietary restrictions that could negatively impact nutritional and health status. Some studies have revealed that carbonic anhydrase (CA) IV, a zinc metalloenzyme, is found in salivary glands and has been localized to taste buds in rats. [37] Zinc an important substance for healthy taste bud, taste buds are known to contain various zinc containing enzymes so if there is a zinc deficiency it can cause taste disorder.

Immunity

The innate immunity as the first line of defense represents a natural protection against infections. Zinc is known to play a vital role in normal immune system. The functions of the innate immunity are disturbed by altered zinc levels because it is essential for high proliferating cell in human body. It is well known that zinc affects multiple aspects of the immune system from the barrier of the skin to gene regulation to lymphocytes. Zinc is crucial for normal development and function of cell mediating-, specific immunity such as B & t cells and nonspecific immunity (active) such as neutrophils and natural killer cells. [38] A

vivo and vitro effect of zinc on immune cell mainly depends on the zinc concentration. By zinc deficiency all functions of Monocytes and activation function of T lymphocytes are impaired and B lymphocytes development and antibody production are reduced, [39] particularly immunoglobulin G is compromised, where as in natural killer cell's cytotoxicity is decrease, [40] and in neutrophils granulocytes, phagocytosis is reduced. [41] B and T cells of the specific immune system have a great variety of specific receptors (antibodies and T-cell receptors) and can produce memory cells that respond quickly and powerfully to antigens to which they have been primed. B cells were shown to be less dependent on zinc for proliferation than T cells; [42] Compare to T lymphocytes, B lymphocytes and their precursors (especially pre-B and immature B cells) are started to more reduced in absolute number during zinc deficiency than T lymphocytes. [43] According to all aspect the effect of zinc on these key immunological mediators is rooted in the myriad roles.

CONCLUSION

Enough is now known about the clinical and public health importance of zinc deficiency to establish beyond doubt the outstanding practical relevance of this trace element in human nutrition. Zinc protects against UV radiation, enhances wound healing, contributes to immune and neuropsychiatric function, and decreases the relative risk of cancer and cardiovascular disease. A zinc rich diet and supplementation could be an interesting strategy aimed at improving physiological and cognitive functions. The roles of this metal in so many aspects of metabolism, including those that are central to cellular growth and differentiation, are very compatible with the wide variety of problems that have been linked with zinc deficiency. All this leaves no reason to doubt the exceptional importance of zinc in human nutrition and public health but Zinc administration must be adjusted to the

patient's actual requirements, because high dosages show negative effects on the immune system. By a proper diet so many problem caused by zinc deficiency can be decrease. Furthermore, zinc might be used as a very promising metal and in a large scale as a theuroptic trace element in sever kind of disease like cancer.

REFERENCES

1. Prasad AS. Impact of the discovery of human zinc deficiency on health. *Journal of the American College of Nutrition*. 2009 Jun 1; 28(3):257-65.
2. Cakmak I, Kalaycı M, Ekiz H, Braun HJ, Kılınc Y, Yılmaz A. Zinc deficiency as a practical problem in plant and human nutrition in Turkey: a NATO-science for stability project. *Field Crops Research*. 1999 Jan 1; 60(1):175-88.
3. Wang K, Zhou B, Kuo YM, Zemansky J, Gitschier J. A novel member of a zinc transporter family is effective in acrodermatitis enteropathica. *The American Journal of Human Genetics*. 2002 Jul 31; 71(1):66-73.
4. Scrutton MC, Wu CW, Goldthwaite DA. The presence and possible role of zinc in RNA polymerase obtained from *Escherichia coli*. *Proceedings of the National Academy of Sciences*. 1971 Oct 1; 68(10):2497-501.
5. Arnold LE, DiSilvestro RA. Zinc in attention-deficit/hyperactivity disorder. *Journal of Child & Adolescent Psychopharmacology*. 2005 Sep 1; 15(4):619-27.
6. Frederickson CJ, Suh SW, Silva D, Frederickson CJ, Thompson RB. Importance of zinc in the central nervous system: the zinc-containing neuron. *The Journal of nutrition*. 2000 May 1; 130(5):1471S-83S.
7. Nowak G, Szewczyk B, Pilc A. Zinc and depression. An update. *Pharmacol Rep*. 2005 Nov 1; 57(6):713-8.
8. Kaplan BJ, Simpson JS, Ferre RC, Gorman CP, McMullen DM, Crawford SG. Effective mood stabilization with a chelated mineral supplement: an open-label trial in bipolar disorder. *Journal of Clinical Psychiatry*. 2001 Dec 12; 62(12):936-44.
9. Kay AR, Tóth K. Is zinc a neuromodulator? *Science signaling*. 2008 May 13; 1(19):re3.

10. Takeda A. Movement of zinc and its functional significance in the brain. *Brain research reviews*. 2000 Dec 31; 34(3):137-48.
11. Hesse GW. Chronic zinc deficiency alters neuronal function of hippocampal mossy fibers. *Science*. 1979 Sep 7; 205(4410):1005-7.
12. Ortega RM, Requejo AM, Andrés P, López-Sobaler AM, Quintas ME, Redondo MR, Navia B, Rivas T. Dietary intake and cognitive function in a group of elderly people. *The American journal of clinical nutrition*. 1997 Oct 1; 66(4):803-9.
13. Frederickson CJ, Jae-Young K, Bush AI. The neurobiology of zinc in health and disease. *Nature reviews. Neuroscience*. 2005 Jun 1; 6(6):449..
14. Matthews RG, Goulding CW. Enzyme-catalyzed methyl transfers to thiols: the role of zinc. *Current opinion in chemical biology*. 1997 Oct 1; 1(3):332-9.
15. Richards MP, Cousins RJ. Mammalian zinc homeostasis: requirement for RNA and metallothionein synthesis. *Biochemical and biophysical research communications*. 1975 Jun 16;64(4):1215-23.
16. Brand IA, Kleineke J. Intracellular zinc movement and its effect on the carbohydrate metabolism of isolated rat hepatocytes. *Journal of Biological Chemistry*. 1996 Jan 26; 271(4):1941-9.
17. Yamaguchi M. Role of zinc in bone formation and bone resorption. *The Journal of Trace Elements in Experimental Medicine*. 1998 Jan 1;11(2-3):119-35
18. Yamaguchi M, Ozaki K. Effect of the new zinc compound beta-alanyl-L-histidinato zinc on bone metabolism in elderly rats. *Pharmacology*. 1990; 41(6):345-9.
19. Hambidge KM, Hambidge C, Jacobs M, Baum JD. Low levels of zinc in hair, anorexia, poor growth, and hypogeusia in children. *Pediatric research*. 1972 Dec 1;6(12):868-74.
20. . Yamaguchi M. Role of nutritional zinc in the prevention of osteoporosis. *Molecular and cellular biochemistry*. 2010 May 1; 338(1-2):241-54.
21. New SA, Robins SP, Campbell MK, Martin JC, Garton MJ, Bolton-Smith C, Grubb DA, Lee SJ, Reid DM. Dietary influences on bone mass and bone metabolism: further evidence of a positive link between fruit and vegetable consumption and bone health?. *The American journal of clinical nutrition*. 2000 Jan 1; 71(1):142-51.
22. Heaney RP. Calcium bone health and osteoporosis. In *Bone and Mineral Research*. 1986 vol. 4, pp. 255-301.
23. Lowe NM, Bremner I, Jackson MJ. Plasma Zn Kinetics in the rat. *British journal of nutrition*. 1991 May 1; 65(03):445-55.
24. Ryz NR, Weiler HA, Taylor CG. Zinc deficiency reduces bone mineral density in the spine of young adult rats: a pilot study. *Annals of Nutrition and Metabolism*. 2009 Jun 9;54(3):218-26.
25. Dessirier JM, Simons CT, Carstens MI, O'Mahony M, Carstens E. Psychophysical and neurobiological evidence that the oral sensation elicited by carbonated water is of chemogenic origin. *Chemical Senses*. 2000 Jun 1;25(3):277-84.s
26. Goto T, Shirakawa H, Furukawa Y, Komai M. Decreased expression of carbonic anhydrase isozyme II, rather than of isozyme VI, in submandibular glands in long-term zinc-deficient rats. *British Journal of Nutrition*. 2008 Feb 1; 99(02):248-53.
27. Landis BN, Lacroix JS. Postoperative/posttraumatic gustatory dysfunction. In *Taste and Smell 2006* (Vol. 63, pp. 242-254). Karger Publishers.
28. Pedersen AM, Bardow A, Jensen SB, Nauntofte B. Saliva and gastrointestinal functions of taste, mastication, swallowing and digestion. *Oral diseases*. 2002 May 1; 8(3):117-29.
29. Vijay kumarAmbaldhage,Jaishankar Hombhalli Puttabuddi, Purnachandrarao Naik Nunsavath, Yehoshuva R Tummuru. Taste disorders: A Review. *Journal of Indian Academy of Oral Medicine & Radiology* 2014(Vol.26,pp.69-76).
30. Ship JA. Diabetes and oral health: an overview. *The Journal of the American Dental Association*. 2003 Oct 31;134:4S-10S.
31. Akar H, Akar GC, Carrero JJ, Stenvinkel P, Lindholm B. Systemic consequences of poor oral health in chronic kidney disease patients. *Clinical Journal of the American Society of Nephrology*. 2011 Jan 1;6(1):218-26.
32. Yagi T, Asakawa A, Ueda H, Ikeda S, Miyawaki S, Inui A. The role of zinc in the treatment of taste disorders. *Recent patents on food, nutrition & agriculture*. 2013 Apr 1;5(1):44-51.

33. Heckmann JG, Lang CJ. Neurological causes of taste disorders. In *Taste and Smell 2006* (Vol. 63, pp. 255-264). Karger Publishers.
34. Frank ME, Bieber SL, Smith DV. The organization of taste sensibilities in hamster chorda tympani nerve fibers. *The Journal of General Physiology*. 1988 Jun 1; 91(6):861-96.
35. Murphy C. Nutrition and chemosensory perception in the elderly. *Critical Reviews in Food Science & Nutrition*. 1993 Jan 1; 33(1):3-15.
36. Seiden AM. *Taste and smell disorders*. New York: Thieme; 1997.
37. Brown D, Garcia-Segura LM, Orci L. Carbonic anhydrase is associated with taste buds in rat tongue. *Brain Res* 1984; 324(2):346-8
38. Ravaglia G, Forti P, Maioli F, Bastagli L, Facchini A, Mariani E, Savarino L, Sassi S, Cucinotta D, Lenaz G. Effect of micronutrient status on natural killer cell immune function in healthy free-living subjects aged ≥ 90 y. *The American journal of clinical nutrition*. 2000 Feb 1; 71(2):590-8.
39. Ostan R, Alberti S, Bucci L, Salvioli S, Pasi S, Cevenini E, Capri M, Di Iorio A, Ginaldi L, De Martinis M, Franceschi C. Effect of zinc ions on apoptosis in PBMCs from healthy aged subjects. *Biogerontology*. 2006 Oct 1; 7(5-6):437-47.
40. Honscheid A, Rink L, Haase H. T-lymphocytes: a target for stimulatory and inhibitory effects of zinc ions. *Endocrine, Metabolic & Immune Disorders-Drug Targets (Formerly Current Drug Targets-Immune, Endocrine & Metabolic Disorders)*. 2009 Jun 1;9(2):132-44.
41. Ch.Bimola Devi, ThNandkishore,Sangeeta N,Gomti Basar, N.Omita Devi, Sundgdsirenla Jamir, M.Amuba Singh. Zinc in Human health. *IOSR Journal of Dental and Medical Sciences*.2014 July (Vol 13 PP18-23)
42. Zazonico, R., Fernandes, G. & Good, R. A. (1981) The differential sensitivity of T cell and B cell mitogenesis to in vitro Zn deficiency. *Cell. Immunol.* 60: 203–211.
43. Rink L, Kirchner H. Zinc-altered immune function and cytokine production. *The Journal of nutrition*. 2000 May 1; 130(5):1407S-11S.

How to cite this article: Soni S, Chaturvedi A, Singh JP. Importance of zinc in human diet. *Int J Health Sci Res*. 2017; 7(8):462-467.
