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The Correlation between Zinc and Cardiovascular Ailments

Glory Coppe*

Department of Cardiovascular medicine, Florida State University, United States

Introduction

The aging process is a significant contributor to the development of cardiovascular diseases due to the buildup of waste materials, such as damaged mitochondria, and a decline in stress response mechanisms. This leads to an increase in oxidative stress over time. According to the free radical theory of aging, elevated levels of reactive oxygen species (ROS) cause DNA damage in the mitochondria and genome, which leads to sustained oxidative stress and contributes to tissue dysfunction and aging. Within the cardiovascular system, ROS are produced by NADPH oxidases as a part of normal function. However, excessive ROS production by these enzymes, along with reduced expression of antioxidant enzymes, is linked to vascular dysfunction and disease [1]. Zinc is a potential mediator of vascular aging and disease as altered zinc homeostasis can accelerate cellular senescence in vascular smooth muscle cells (VSMCs) through a ROS-dependent mechanism. Zinc deficiency has also been linked to increased risk of cardiovascular diseases such as atherosclerosis, and in vitro experiments suggest that intracellular zinc not buffered by zinc regulatory mechanisms creates a vicious cycle that promotes oxidative damage and senescence in VSMCs. Therefore, understanding the role of intracellular zinc levels and distribution in response to zinc deficiency in the cardiovascular system is crucial for the development of potential interventions for vascular aging and disease. Zinc has been linked to the development of cardiovascular diseases, such as atherosclerosis, although the exact mechanisms are not fully understood. Zinc deficiency, which is prevalent worldwide, has been associated with an increased risk of cardiovascular diseases. Studies have shown that low dietary zinc intake promotes atherosclerosis and increases inflammation in mice. As mammalian organisms age, cellular senescence becomes a prominent characteristic, which is a permanent cessation of cell division. This process has been associated with the progression of age-related illnesses, such as atherosclerosis. Recent studies have shown that senescence plays a causative role in the development of these diseases. This is supported by in vivo studies that demonstrate delayed onset of age-related illnesses upon selective removal of senescent cells. Despite losing their ability to replicate, senescent cells retain a secretory and pro-inflammatory/oxidative phenotype that is believed to contribute to age-related organ dysfunction [2-7]. The role of nutritional status in disease development is well-established, with overconsumption of high-calorie foods being a known risk factor for diseases like metabolic syndrome, atherosclerosis, and diabetes. However, the impact of micronutrient deficiencies on cardiovascular disease progression is less understood. Zinc deficiency, which affects 31% of the global population according to the World Health Organization, has been linked to an increased risk of cardiovascular diseases such as atherosclerosis, although the mechanisms are not well defined. Studies in mice have shown that low dietary zinc intake is associated with increased inflammation and promotes atherosclerosis. Zinc deficiency in rats during pregnancy has also been correlated with increased susceptibility to cardiovascular diseases in the offspring. Additionally, zinc has been found to be required for the protective anti-inflammatory function of PPAR [8-13]. Nutritional status is an important factor in the development of various diseases. High calorie intake is a known risk factor for metabolic syndrome, atherosclerosis, and diabetes. However, the impact of micronutrient deficiencies, such as zinc, on the development of cardiovascular disease is less well-known. Zinc deficiency, which affects 31% of the global population according to the World Health Organization, has been linked to increased risk of cardiovascular diseases such as atherosclerosis, although the exact mechanisms are not yet understood. Previous research has suggested a possible link between zinc and senescence, which is a process of permanent cell cycle arrest that contributes to aging and age-related diseases. In vitro experiments have shown that altered zinc homeostasis accelerates cellular senescence by a reactive oxygen species (ROS)-dependent mechanism in vascular smooth muscle cells (VSMCs). Zinc deficiency may induce changes in the expression of zinc regulators such as zinc transporters, which could lead to a redistribution of intracellular zinc, potentially causing a rise in cytosolic zinc. Increased intracellular zinc in vitro and zinc deficiency in vivo lead to oxidative stress that may mediate vascular disease [14,15]. Zinc is also known to be a mediator of oxidative damage and neuronal cell death in neurological diseases like Alzheimer's disease [16,17]. Overall, these findings suggest that zinc deficiency may contribute to the development of cardiovascular disease through its impact on senescence and oxidative stress.

Conclusion

To gain a comprehensive understanding of how zinc affects vascular aging and disease, it is necessary to investigate intracellular zinc levels and distribution, as well as the expression of zinc transporters in response to zinc deficiency in the cardiovascular system. It is important to note that zinc deficiency is not only associated with lower zinc intake but also with chronic diseases. Therefore, further development of this emerging area of research is crucial.

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Conflict of Interest

Author declares no conflict of interest.

References

1. Lassegue B, San Martin A, Griendling KK (2012) Biochemistry, physiology, and pathophysiology of NADPH oxidases in the cardiovascular system. Circ Res 110:1364-1390.

2. Hayflick L, Moorhead PS (1961) The serial cultivation of human diploid cell strains. Exp Cell Res 25:585-621.

***Corresponding author:** Glory Coppe, Department of Cardiovascular medicine, Florida State University, United States, E-mail: glorycop@yahoo.com

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3. Fuster JJ, Andres V (2006) Telomere biology and cardiovascular disease. Circ Res 99:1167-1180.

4. Erusalimsky JD, Kurz DJ (2005) Cellular senescence in vivo: its relevance in ageing and cardiovascular disease. Exp Gerontol 40:634-642.

5. Minamino T, Miyauchi H, Yoshida T, Ishida Y, Yoshida H, et al. (2002) Endothelial cell senescence in human atherosclerosis: role of telomere in endothelial dysfunction. Circulation 105:1541-1544.

6. Baker DJ, Wijshake T, Tchkonia T, LeBrasseur NK, Childs BG, et al. (2011) Clearance of p16Ink4a-positive senescent cells delays ageing associated disorders. Nature 479:232-236.

7. Coppe JP, Desprez PY, Krtolica A, Campisi J (2010) The senescence-associated secretory phenotype: the dark side of tumor suppression. Annu Rev Pathol 5:99-118.

8. Black RE, Allen LH, Bhutta ZA, Caulfield LE, De Onis M, et al. (2008) Maternal and child undernutrition: global and regional exposures and health consequences. Lancet 371:243-260.

9. Little PJ, Bhattacharya R, Moreyra AE, Korichneva IL (2010) Zinc and cardiovascular disease. Nutrition 26:1050-1057.

10. Beattie JH, Gordon MJ, Duthie SJ, McNeil CJ, Horgan GW, et al. (2012) Suboptimal dietary zinc intake promotes vascular inflammation and atherogenesis in a mouse model of atherosclerosis. Mol Nutr Food Res 56:1097-1105.

11. Shen H, Oesterling E, Stromberg A, Toborek M, MacDonald R, et al. (2008) Zinc deficiency induces vascular pro-inflammatory parameters associated with NF-kappaB and PPAR signalling. J Am Coll Nutr 27:577-587.

12. Tomat AL, Inserra F, Veiras L, Vallone MC, Balaszczuk AM, et al. (2008) Moderate zinc restriction during fetal and postnatal growth of rats: effects on adult arterial blood pressure and kidney. Am J Physiol Regul Integr Comp Physiol 295:543-549.

13. Tomat A, Elesgaray R, Zago V, Fasoli H, Fellet A, et al. (2010) Exposure to zinc deficiency in fetal and postnatal life determines nitric oxide system activity and arterial blood pressure levels in adult rats. Br J Nutr 104:382-389.

14. Patrushev N, Seidel-Rogol B, Salazar G (2012) Angiotensin II requires zinc and down regulation of the zinc transporters ZnT3 and ZnT10 to induce senescence of vascular smooth muscle cells. PLoS One 7:33211.

15. McCord MC, Aizenman E (2014) The role of intracellular zinc release in aging, oxidative stress, and Alzheimer's disease. Front Aging Neurosci 6:77.

16. Granzotto A, Sensi SL (2015) Intracellular zinc is a critical intermediate in the excitotoxic cascade. Neurobiol Dis 81:25-27.

17. Chowanadisai W, Kelleher SL, Lonnerdal B (2005) Zinc deficiency is associated with increased brain zinc import and LIV-1 expression and decreased ZnT-1 expression in neonatal rats. J Nutr 135:1002-1007.