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CHANGES IN ZINC CONCENTRATION IN ORAL ENVIRONMENT AS A RISK FACTOR OF PERIODONTAL DISEASE

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Risqa Rina Darwita : Changes In Zinc Concentration In Oral Environment As A Risk Factor Of Periodontal Disease. 2000; 9(2) : 20 - 22

Abstract

The Zinc (Zn) concentration was determined from salivary gland of Wistar King A (WKA) rats. The salivary glands are divided in to sub-lingual, sub-mandible and parotis. The Zn concentration was determined within 15-60 minutes of restraint stress. Concentration of Zn in sub-mandible, parotis and sub-Lingual glands was significantly higher ($p<0.001$) than in the control group. Further more at. Zinc concentration in sub-mandible gland at 30 min restraint stress was increase ($p<0,001$), and decrease significantly at 49 min by restraint stress. By the way, Zn concentration in parotis gland was increase significantly ($p<0.0001$) after 60 min restraint stress. The results suggest that Zn is linked to oral saliva under physiological stimuli, and that Zn accumulates in the salivary gland during salivary enzyme activity.

Introduction

The prevalence of periodontal disease in Indonesia has almost reached 100%[1]. Understanding the biological factors that influence the pathogenesis of periodontal disease could help to prevent the problem. The tooth surface consists of mainly of proteins and minerals. The periodontal disease is indicated by a close relationship with mineralized and non mineralized deposits on external surface of teeth[2-3].

The essential mineralized dental plaque can cover the external surface of teeth is called dental calculus. In the prevailing view, until 1960, dental calculus is the major causative factor of periodontal disease. Its pathogenicity is attributed to its rough outer surface, which mechanically irritates the adjacent tissues. The irritates tissues with the initial damage to the gingival margin in periodontal disease is due to immunological and enzymatic effects of the microorganism in the dental plaque[2]. There may also be loosely held covering of material alba, shed bacteria, desquamated epithelial cells, and blood cells derived from the crevicular area. Poor oral hygiene, lack of adequate masticatory function, and tooth malpositition can contribute to an increased rate and extent of dental calculus deposition. The tightly adherent calcified deposit, classified as supragingival calculus, is formed on the clinical crowns of the teeth above the free gingival margin. Another form of calcified deposit from sub gingival plaque mineralization is called subgingival calculus, which is not formed from direct extension of the supragingival calculus. It is located on the root surface below the free gingival margin and extends into the periodontal pocket. The subgingival and supragingival plaque are primarily responsible for inflammatory changes in the gingival and supporting tissues, which generate the gingival crevicular fluids[2-4].

The dental calculus is a biological apatite as well as enamel and dentin of the teeth. A review study[5] on dental calculus composition has revealed that the dental calculus lacks homogeneity regarding its density, distribution in the mouth, and crystal structure[5]. The gross distribution of dental calculus varies depending whether it is subgingival, supragingival or tooth-to-tooth location. These variations may influence the elements incorporated into calculus formation because of various oral biochemical environments. For example, gingival exudates will influence

sub gingival calculus, and its proximity to salivary gland ducts will produce different biochemical environments. Another study has elucidated that the presence of essential trace elements had a close relation with the formation of dental calculus synthesized during precipi-tation. Also the presence or absence of some elements, such as alkaline earth, may affect the appatite's nuclei crystallization or bacterial proliferation and their activities on the surface of dental calculus.

Zn is a common trace element in dental calculus which have a strong relationship with periodontal disease process[5]. Zinc plays an important role as an essential trace element in many biological functions[6]. The four-fold higher content of Zn in sub gingival calculus has been related to the fact that Zn level in serum is higher than in saliva[3]. Saito has reported that the changes of Zinc concentration in whole blood have a strong relation with diabetes mellitus disease. The correlation between diabetes mellitus and the severity of periodontal disease are well known[7]. Diabetes mellitus has been one of the major systemic predis-posing factors that affect a periodontal tissue. Recent study elucidated that periodontal disease had relationship with stress and delayed wound healing of connective tissue and bone. In addi-tion, acute necrotizing ulcerative gingi-vitis (ANUG) has strong correlation with psychologic stress. Various stresses such as starvation, infection, psycholo-gical and inflammatory stresses induce changes of essential trace elements in human and animal[3].

Following the main problems mentioned above, the hypothesis is that the presence of zinc in dental plaque, serum, and saliva have some influence in dental calculus formation, which induces periodontal disease.

There have not been any studies on the changes of zinc as causative factors in periodontal disease. This study will investigate the effect of stress on the changes of zinc level in salivary gland of WKA rat.

Research Methodology

The experiments have be con-ducted on male Wistar-King-A rats weighing 280-340 gram. The animals were allowed a free access to rat chow and tap water and were maintaned on a 12 hour light-dark cycle.

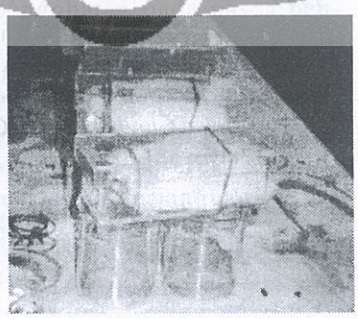
Experimental procedure

After preparing the acrylic restrainer The rats were put into the acrylic re-strainer in 15, 30, 45, 60, 75, 90, 105 and 120 minutes. Further the rats anesthe-tized by ether, put the blood from the heart to glass reaction. Put serum, after blood centrifuging in 20000 gravitate. After that, cut all salivary glands including to sub lingual gland, sub mandible gland, and parotids to each dishes.

Determination of zinc concentration in salivary gland after stressed by acrylic restainer.

Samples were prepare of sub lingual, sub mandible, parotids of WKA rats. X-Ray Fluorescent was used to determinate zinc concentration. The differences valye of Zn concentra-tion was analyze by student s T-test, and P values less than 0.05 was considered to be significant.

Figure 1 : The rats in acrylic restrainer in from 15-20 minutes



Result

Effects of restraint stress on zinc, in sub lingual, sub mandible and pa-rotids gland are indicated in Figure 1, 2, and 3.

Figure 1.
Dynamic of zinc in sub mandible Gland of WKA Rats after Restrain Stress

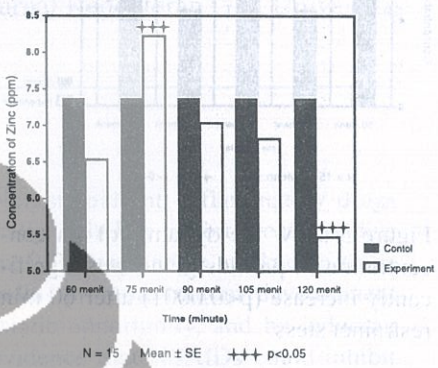


Figure 1 indicated that Zn concentration in sub-mandible gland at 75 min restrain stress was increase in significant ($p<0.0001$), and decreases significantly ($p<0.0001$) at 120 min by restraint stress.

Figure 2.
Dynamic of Zinc in Sub lingual Gland of WKA Rats after Restrain Stress

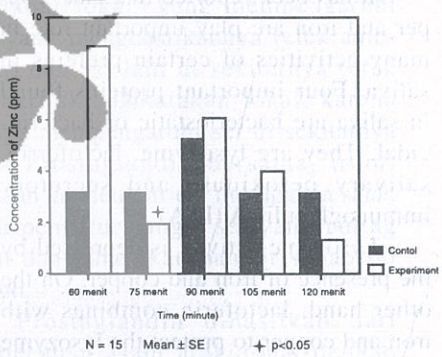


Figure 2 indicated that the dynamic of Zn concentration in sub-lingual gland was decrease significantly at 75 min by re-straint stess.

Figure 3.
Dynamic of Zinc in Parotis Gland of WKA Rats after Restrain Stress

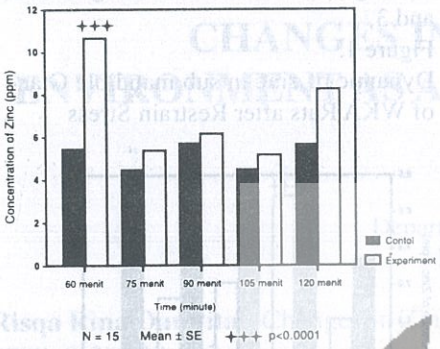


Figure 3 show the dynamic of Zn concentration in paroids gland was significantly increase ($p<0.0001$) after 60 min restrainet stess.

Discussion

The present investigation showed that zinc, concentrations in specific region of salivary gland affected by restraint stress and that differenees in these concentrations vary with the duration of restraint stress. The increase zinc concentration caused by relatively short-term restraint stress.[6,7]

It has been reported that zinc, copper and iron are play important role in many activities of certain proteins in saliva. Four important proteins found in saliva are bacteriostatic or bactericidal. They are lysozyme, lactoferrin, salivary peroxidase and secretory immunoglobulin A (Ig A). [8,9]

Lysozyme activity is depressed by the presence of iron and copper. On the other hand, lactoferin combines with iron and copper to protect the lysozyme action, while at the same time depriving bacteria of some of their essential simply of those two metals. These activities indicate that there was disturbance in the activity of lysozyme and lactoferin. On the other hand, the levels of zinc, in sub mandible gland saliva was decreased. This it is inhibit this activity of lysozyme and lactoferin. The decreasing of iron, copper and zinc will inhibits the activity of lactoferin and lysozyme. Depriving of lysozyme activity will elevate the capability of bacteria to use glucose. [3,7,9]

By the way, the level of concentration of zinc in parotids was increase within 60 min, this condition indicates that Zn-binding protein is stimulates the parotids gland saliva to secrete carbonic anhydrase in parotids gland. Whereas the function of carbonic anhydrase is relatied to control the taste and smell function.[9,10] While the increaseing of copper and iron concentration level in paritids gland will activated the lactoferin and lysozyme activity.[9]

The results suggest that the changes in the trace element of zinc, sopper and iron in the saliva from salivary gland of sub lingual, sub mandible, and parotids gland may be closely related to the processes of enzyme activations induced by mild physiology stimuli and that the metabolism of these metals in differently regulated according to the functional role of each element in the salivary gland system.

References

1. Ministry of Health. *Oral Health Profile in Indonesia*. Depkes RI 1999.
2. DeMa ZJ. Zhang JZ. Changes in serum zinc level of periodontitis with kidney deficiency. *Chung-Kuo Chung Hsi i Chieh Ho Tsa Chih*. 1993 13(10): 581, 606-7.
3. Polenik P. Zinc in etiology of periodontal disease. *Med Hypoth*. 1993 40(3): 182-5.
4. MEJ Curzon, *Trace Elements and Dental Disease*. John Wright London 1982: 199-236.
5. Speirs RL. Beeley JA. Food and oral health: 2. Periodontium and oral mucosa *Dent Update*. 1992; 19(4): 161-2, 164-7
6. Irwin DM. Calculus Update: Prevalence, Pathogenicity and Prevention. *JADA* 1995: 573-580.
7. Saito T. *Changes in Zinc and Copper levels in rat brain regions and theirsynap tosomal fractions induced by restrain stress*. In: Fungtional Roles of Trace Elements in Neuritransmissions of the central nervous system. Hokkaido University, Japan 1995: 9-20.
8. Kuraner T. Beksac MS. Kayakirilmaz K. Caglayan F. Inderoglu LS. Ozgunes H. Serum and parotid saliva testosterone, calcium, magnesium, and zinc levels in males, with and without periodontitis. *Biol Trace Elem Res*. 1991; 31(1): 43-9.
9. Henki RI, Martin BM, Agarwal RP. Decreased parotid saliva gustin/carbonic anhydrease VI secretion: an enzyme disorder manifested by gustatory and olfactory dysfunction. *Am J Med Scie* 1999; 318(6): 380-91.
10. Norman O H, Richard P S. *Oral biology defenses and the demineralization and remineralization of teets*. In: Prim Prev Dent.3rd ed. Appleton & Lange. 1991: 261.
11. Etzel KR, Hempel JD, Koepsel RR. Identtification of zinc in rat parotid saliva. *Arch Oral Biol*. 1997; 42(2): 173-9.