Salivary sIgA as a predictor of caries risk in stunting children

by Udijanto Tedjosasongko

Submission date: 02-Jun-2022 10:41AM (UTC+0800)
Submission ID: 1848831913
File name: WJARR-2022-0391.pdf (782.49K)
Word count: 3145
Character count: 17626

World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(Review Article)

Check for updates

Salivary sIgA as a predictor of caries risk in stunting children

Udijanto Tedjosasongko *, Aisyah Marwah and Ardianti Maartrina Dewi

Department of Pediatric Dentistry, Faculty of Dental Medicine, Universitas Airlangga, Surabaya, Indonesia.

World Journal of Advanced Research and Reviews, 2022, 14(02), 052–056

Publication history: Received on 29 March 2022; revised on 02 May 2022; accepted on 04 May 2022

Article DOI: https://doi.org/10.30574/wjarr.2022.14.2.0391

Abstract

Background: Stunting has a detrimental influence on the health and development of children and has a number of negative consequences, including an increase in mortality as a result of a loss in bodily immunity, which makes youngsters more susceptible to illness. Additionally, there are impairments in cognitive capacities, as well as learning and productivity in adulthood. Stunting prevalence is relatively high in Indonesia, at 30.8%, compared to the global prevalence of 22.2%. Stunting children tend to have protein energy malnutrition (PEM) condition, which can lead in salivary gland atrophy, decrease sIgA levels, and disrupting the buffering system, cleansing, and antibacterial agents. It is able to increase the risk of dental caries.

The purpose of this review is to determine whether salivary sIgA levels can be used to predict the risk of developing caries in stunted children.

Keywords: Stunting; sIgA; *Streptococcus mutans;* Caries; Children

1. Introduction

Stunting in children is a result of dietary inadequacies during the first thousand days of life, which cause irreversible disruptions in the physical development of the child, causing a reduction in cognitive and motoric abilities, as well as a reduction in activity performance[1]. Children who are stunting between the ages of 0 and 2 years will continue to be short between the ages of 4-6 years and are at risk of being short 27 times until reaching puberty. Preventing this requires early dietary intervention[2]. Interventions at a critical stage of life, around the age of 1000 days, are critically needed through WASH (Water, Sanitation, and Hygiene) improvements, education, and nutrition interventions. WASH improvement and education work together to prevent the incidence of Environmental Enteric Dysfunction (EED), a condition that impairs nutritional intake in stunting children[3]. Malnutrition during the vulnerable tooth's developing stages, particularly in early childhood, can result in enamel hypoplasia, causing teeth more susceptible to demineralization and dental caries[4].

Underweight and delayed growth are associated with dental caries and predict delayed permanent tooth eruption in children from Cambodia, Indonesia, and Laos[5]. Caries affects 90.5 percent of preschool children aged 4-5 years in urban areas and 95.9 percent of preschool children in rural areas in Indonesia. Caries affects 90 percent of school-aged children globally and the majority of adults. Preschool children are particularly vulnerable to dental and oral problems, as they often maintain personal behaviors or routines that are detrimental to dental health[6]. *Streptococcus mutans* (*S. mutans*) is the potential cause for dental caries. *S. mutans* and *Streptococcus sobrinus* (*S. Sobrinus*) are the most prevalent bacteria in human tooth plaque and are believed to be the primary cause of dental caries[7]. *S. mutans* attaches to its host through its own receptor, the salivary pellicle, which acts as a mediator for oral bacteria adhering to tooth surfaces and restorations[8].

* Corresponding author: Udijanto Tedjosasongko
Department of Pediatric Dentistry, Faculty of Dental Medicine, Universitas Airlangga, Surabaya – Indonesia.

Saliva is a biological fluid made up of 99 percent water. Saliva contains a variety of components, both organic (peptides and proteins) and inorganic (electrolytes and water), that contribute to the prevention of dental caries. Saliva contains immunoglobulins IgA, IgG, and IgM. IgA constitutes over 60% of total immunoglobulins in saliva[9]. sIgA concentrations in saliva, tears, and nasal secretions are frequently decreased in malnourished children[10]. Children with dietary inadequacies develop salivary gland atrophy, resulting in decreased saliva output and a decrease in the buffering, cleaning, and antimicrobial functions of saliva[11]. Nutritional deficits, such as protein energy deficiency, can impact tooth development by pathways similar to those affecting physical development. Salivary flow is lower in malnourished children, both stimulated and unstimulated. This is consistent with prior reports based on animal and human research[11][12]. Viruses, poisons, and bacteria that cling to the mucosal epithelium and tooth surfaces are neutralized by salivary secretory immunoglobulin A (sIgA). sIgA can impede the demineralization process in teeth by preventing *S. mutans* from adhering to the tooth surface and forming glucan. Low SIgA levels in the oral cavity have been linked to a significant caries risk in several studies[13]. Caries prevalence was substantially higher in stunting children than in non-stunting children. Delgado-Angulo, Hobdell, and Bernabé (2013) found that stunting children had a higher rate of caries than non-stunting children after three years of follow-up[14].

sIgA is a risk factor for the severity of dental caries, however it is uncertain if stunting in children has an effect on it. The purpose of this review is to identify whether SIgA can be utilized as a predictor of caries risk in stunting children.

2. Literature Reviews

2.1. **Stunting**

Stunting is caused by a variety of factors, including family and environmental factors, complementary foods, breastfeeding, infection, and social factors. These factors can result in EED (Environmental Enteric Dysfunction) conditions and macronutrient and micronutrient deficiencies, resulting in a decrease in dietary protein, calories, vitamins, and calcium intake, resulting in Protein Energy Malnutrition in children (PEM)[15]. EED arises as a result of intestinal inflammation, which results in impaired nutritional absorption. Numerous studies have established a link between intestinal inflammation and stunting[16].

Previously scientific studies showed that a deficiency of vitamin D was correlated with stunting. Vitamin D is critical for bone health, as well as bone mineralization and maintenance. Vitamin D deficiency over time disrupts the transcriptional regulation of skeletal homeostasis and linear growth, which can lead into stunting. Vitamin D is biologically active in the 1,25(OH)2D form, which is regulated by the parathyroid glands. Vitamin D's active form is primarily bound to the albumin protein superfamily and is referred to as Vitamin D Binding Protein in the blood (VDBP). 1,25(OH)2D reaches the target organ via the Vitamin D Receptor (VDR), which is responsible for increasing calcium and phosphorus absorption by the intestine, bone resorption, and calcium and phosphorus excretion by the kidneys, as well as for bone health maintenance in conjunction with parathyroid hormone (PTH) and calcitonin (1,25(OH)2D). After binding to the VDR, the active vitamin D metabolite enters the nucleus of the cell and activates gene expression. Vitamin D deficiency decreases the level of 1.25(OH)2, impairing the function of the VDR receptors in the intestines, and serum calcium deficiency impairs the osteoclast and osteoclast processes, impairing bone growth. Chronic deficiency of vitamin D disrupts the transcriptional regulation of skeletal homeostasis and linear growth, which can result in stunting[17]. Vitamin D's biologically active form is 1,25(OH)2D, which is regulated by the parathyroid glands. Vitamin D's active form is primarily attached to the protein superfamily albumin by a molecule called Vitamin D Binding Protein (VDBP). 1,25(OH)2D reaches the target organ via the Vitamin D Receptor (VDR), which is responsible for increasing calcium and phosphorus absorption by the intestine, bone resorption, and calcium and phosphorus excretion by the kidneys, as well as for maintaining bone health in conjunction with parathyroid hormone (PTH) and calcitonin (1,25(OH)2D). After adhesion to the VDR, the active vitamin D metabolite enters the cell nucleus and activates gene expression. Vitamin D deficiency decreases the level of 1.25(OH)2, impairing the action of the VDR receptors in the intestine, and serum calcium deficiency impairs the osteoclast and osteoclast processes, resulting in decreased bone development[18].

In stunting, PEM occurs an imbalance between protein intake and energy alongside a decrease in the body's ability to function optimally. PEM decreases salivary flow, buffer system, saliva composition, secretion rate, calcium, protein secretion, and bacterial agglutination defense components[12]. PEM impairs the ability of immunity to perform its roles in stunted children, resulting in three things: disruption to the T helper population, a significant decrease in particular memory and suppressor cells, and a reduction in IgA levels. Furthermore, there is a reduction in plasma mucosal cells that make IgA and mucosal epithelial atrophy, which results in a reduction in sIgA[19].

3. Salivary Secretory Immunoglobulin A (sIgA)

The oral defense mechanism begins with mucin and other antibacterial substances being secreted by the salivary glands to preserve the mucosa and tooth surface. Salivary glands serve as mucosal effector sites for the differentiation of B cells into polymeric IgA-secreting plasma cells. Salivary IgA is secreted in the form of sIgA[20].

Secretory immunoglobulin A (SIgA) is a type of immunoglobulin found in all saliva and is regarded a critical component of the mouth cavity's particular defensive mechanism[21]. sIgA is present in saliva and other exocrine secretions, including those from the respiratory, urinary, and digestive tracts. sIgA is the primary defense in the mucosa; it functions by limiting pathogen invasion and neutralizing and eliminating pathogenic antigens from penetrating the host. sIgA is a polymeric molecule formed up of approximately two IgA monomers, a J chain, and a secretory component. Saliva, tears, and breast milk all include these components. Each IgA monomer is composed of four polypeptides, two of which are heavy chains and two of which are light chains[22]. Antigen exposure, level of stress or emotional state, diet, salivary flow, salivary stimulation, age, activity intensity, hormonal and genetic background have an effect on the production and concentration of salivary sIgA[23].

sIgA antibodies inhibit bacterial binding, colonization, metabolism, and penetration across mucosal surfaces, as well as neutralize viruses, enzymes, and toxins. Increased sIgA secretion has a beneficial effect on the mucosa by reducing infection. sIgA is a unique primary defensive mechanism found in saliva and has a role in maintaining oral homeostasis. sIgA has the ability to inhibit bacterial cell adhesion to the teeth and oral mucosa. The amount of antigen induces sigA levels in two ways. First, the antigen stimulates lymphoid cell proliferation and differentiation in the local lymphoid tissue. Additionally, it involves the migration of antigen-sensitized IgA precursor B cells from the gut-associated lymphoid tissue (GALT) to the salivary glands[24]. As the primary particular defense, sIgA aids in the protection of the oral cavity against caries-causing bacteria (*S. mutans).* sIgA was able to inhibit *S. mutans* colonization by decreasing bacteria' initial adherence to the tooth surface and neutralizing extracellular enzymes. *S. mutans* colonization is reduced by sIgA because it inhibits the activity of GTF, preventing the production of glucans. As a result, bacterial adhesion does not occur during the plaque formation mechanism[23]. sIgA inhibits attachment facilitated by Glucan Binding Protein (GBP) and the accumulation of *S. mutans* in acidic or critical environments where the biofilm becomes pathogenic by releasing lactic acid and causing tooth demineralization[20]. Numerous studies have reported that low sIgA levels in the oral cavity result in an increased risk of caries, whereas high sIgA levels result in a decreased risk of caries[13].

Salivary sIgA levels have been widely researched in both healthy and pathological individuals. sIgA levels in healthy persons without systemic or immunologic illness typically range between 4 and 30 mg/dL. This level is affected by a variety of factors, including malnutrition, obesity, infection, stress, smoking, salivary flow rate, hormonal factors, emotional state, and physical activity. Decreased sIgA levels are correlated with an increased risk of caries and candidiasis in the elderly.[25]

4. Caries

S. mutans has several components that contribute to dental caries progression, including adherence to the enamel surface, the ability to produce acid, the ability to accumulate glycogen reserves, and the ability to synthesize extracellular polysaccharides present on the tooth surface[26]. Various laboratory researches have revealed that *S. mutans* can alter the local environment by developing a high Extracellular Polysaccharide (EPS) and low pH environment, hence encouraging the development of other acidogenic and aciduric species[27]. *S. mutans* virulence factors are divided into two categories: glucosyltransferase enzymes (GTF) and protein antigens (AgI/AgII). The GTF enzyme catalyses sucrose to glucans, which aid in sucrose's adhesion to the tooth surface. In the oral cavity, AgI/AgII interacts with salivary glycoprotein agglutinins. Extracellular polysaccharides increase bacteria's adhesion to the tooth surface and contribute to the biofilm's formation. Polysaccharide matrix structure has a significant effect on plaque virulence through affecting the biofilm's physical and biochemical properties[26]. *S. mutans* possesses the ability to rapidly transport and convert a variety of carbohydrates into organic acids (acidogenicity), as well as the capacity to grow in adverse environmental conditions, most notably low pH. (aciduric environment). If bacteria on the tooth surface are not eliminated, cariogenic plaque forms, which involves the accumulation and interaction of microbial cells, salivary proteins, and food particles, leading to greater plaque retention and concurrent acid formation[20]. Bacteria utilize the acid produced during metabolism to produce energy, causing damage to the tooth structure. Plaque on the enamel surface will absorb acid, resulting in a lower pH in the oral cavity. If the pH remains below normal for an extended period of time, it will result in tooth demineralization and enamel degradation, leading to caries[28].

5. Conclusion

sIgA in saliva has potency in predicting caries risk in stunting children.

Compliance with ethical standards

Acknowledgments

The authors thank the reviewers for their insightful suggestions.

Disclosure of conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this document.

References

[1] Kemenkes RI. Buletin Stunting. Kementeri Kesehat RI. 2018; 301(5): 1163–78.

[2] Aryastami NK. Government Studies and Overcoming Indonesia's Suffocating Nutritional Issues. Bul Penelit Kesehat. 2017; 45(4): 233–40.

[3] Budge S, Parker AH, Hutchings PT, Garbutt C. Environmental enteric dysfunction and child stunting. Nutr Rev. 2019; 77(4): 240–53.

[4] Achmad H, Ramadany S, Sukmana BI, Hanan N, Hartami E, Ramadhany YF, et al. A Review of Stunting Growth in Children : Relationship to the Incidence of Dental Caries and its Handling in Children. 2020; 11(6): 230–5.

[5] Dimaisip-Nabuab J, Duijster D, Benzian H, Heinrich-Weltzien R, Homsavath A, Monse B, et al. Nutritional status, dental caries and tooth eruption in children: A longitudinal study in Cambodia, Indonesia and Lao PDR 11 Medical and Health Sciences 1117 Public Health and Health Services 11 Medical and Health Sciences 1105 Dentistry. BMC Pediatr. 2018; 18(1): 1–11.

[6] Hidayati S, Utami NK, Amperawati M. Def-t index for kindergarten children in Banjarbaru South Kalimantan. Jurnal Skala Kesehatan Volume 5 No. 2. 2014; 5(2): 1–7.

[7] Singla D, Sharma A, Sachdev V, Chopra R. Distribution of Streptococcus mutans and Streptococcus sobrinus in dental plaque of Indian pre-school children using PCR and SB-20M agar medium. J Clin Diagnostic Res. 2016; 10(11): ZC60–3.

[8] Anggraeni A, Yuliati A, Nirwana I. Streptococcus mutans colony attachment to the visible light composite resin surface. Dental Journal Majalah Kedokteran Gigi. 2005; 38(1): 8.

[9] Sood LI, Al-Ezzy MYH, Diajil AR. Correlation between Streptococci Mutans and Salivary IgA in Relation to Some Oral Parameters in Saliva of Children. J Baghdad Coll Dent. 2014; 26(1): 71–9.

[10] Rytter MJH, Kolte L, Briend A, Friis H, Christensen VB. The immune system in children with malnutrition - A systematic review. PLoS One. 2014; 9(8).

[11] Ahmad MS, Bhayat A, Zafar MS, Al-Samadani KH. The impact of hyposalivation on quality of life (QOL) and oral health in the aging population of Al madinah Al munawarrah. Int J Environ Res Public Health. 2017; 14(4): 8–10.

[12] Psoter WJ, Spielman AL, Gebrian B, St. Jean R, Katz R V. Effect of childhood malnutrition on salivary flow and pH. Arch Oral Biol. 2008; 53(3): 231–7.

[13] Soesilawati P, Notopuro H, Yuliati Y, Ariani MD, Firdauzy MAB. The role of salivary sIgA as protection for dental caries activity in Indonesian children. Clin Cosmet Investig Dent. 2019; 11: 291–5.

[14] Aviva NN, Pangemanan DHC, Anindita PS. Overview of Primary Dental Caries in Stunting Children in Indonesia. e-GiGi [Internet]. 2020; 8(30): 73–8.

[15] Putri R, Nuzuliana R, Kurniawati HF. Management of stunting to improved children nutritional status and cognitive. 1st International Respati Health Conference (IRHC). 2019; 490–500.

[16] Goddard FGB, Knee J, Sumner T, Nalá R, Clasen T, Brown J. Child salivary siga and its relationship to enteric infections and eed biomarkers in Maputo, Mozambique. International Journal of Environmental Research and Public Health. 2020; 17(9).

[17] Yu EA, Huey SL, Peña-Rosas JP, Mehta S. The effects of oral vitamin D supplementation on linear growth and non-communicable diseases among infants and children younger than five years of age. Cochrane Database of Systematic Reviews. 2017; (11).

[18] Antonucci R, Locci C, Clemente MG, Chicconi E, Antonucci L. Vitamin D deficiency in childhood: Old lessons and current challenges. J Pediatr Endocrinol Metab. 2018; 31(3): 247–60.

[19] Bourke CD, Berkley JA, Prendergast AJ. Immune Dysfunction as a Cause and Consequence of Malnutrition. Trends Immunol [Internet]. 2016; 37(6): 386–98.

[20] Jenkinson HF, Lamont RJ HG. Oral Microbiology and Immunology. 2nd ed. Lamont, R. J.; Hajishengallis, G. N.; Jenkinson HF, editors. Washington DC: ASM Press, 2013. P-209-17.

[21] Chawda JG, Chaduvula N, Patel HR, Jain SS, Lala AK. Salivary SIgA and dental caries activity. Indian Pediatr. 2011; 48(9): 719–21.

[22] Pratiwi TD. Differences in SIga Saliva Levels in Patients with Gingivitis between Normal Beta Major Thalassemia Children (thesis). Jakarta. Fakultas Kedokteran Gigi Universitas Indonesia. 2014.

[23] Rosdiana R, Rizal MF. The relation between salivary sIgA level and caries incidence in Down syndrome children. Dent J (Majalah Kedokt Gigi). 1 Jun 2012; 45(2): 79.

[24] Ruth MR, Field CJ. The immune modifying effects of amino acids on gut-associated lymphoid tissue. J Anim Sci Biotechnol. 2013; 4(1): 1.

[25] Gornowicz A, Tokajuk G, Bielawska A, Maciorkowska E, Jabłoński R, Wójcicka A, et al. The assessment of sIgA, histatin-5, and lactoperoxidase levels in saliva of adolescents with dental caries. Med Sci Monit. 2014; 20: 1095–100.

[26] Sandi IM, Bachtiar H, Hidayati. JURNAL Volume 2 Nomor 2 , Desember 2015 Daftar Isi. 2015; 2(2): 90.

[27] Lemos JA, Palmer SR, Zeng L, Wen ZT, Kajfasz JK, Freires IA, et al. The Biology of Streptococcus mutans . Gram-Positive Pathog. 2019; 7(1): 435–48.

[28] Listrianah. Dental caries index in terms of common diseases and salivary secretions in snacks at Public Elementary School 30 Palembang 2017. Jurnal Kesehatan Palembang. 2017; 12(2): 136–48.

Salivary sIgA as a predictor of caries risk in stunting children

ORIGINALITY REPORT

19%	12%	14%	0%
SIMILARITY INDEX	INTERNET SOURCES	PUBLICATIONS	STUDENT PAPERS

PRIMARY SOURCES

1	Www.mdpi.com Internet Source	2%
2	pure.uva.nl Internet Source	2%
3	J.A. Lemos, S.R. Palmer, L. Zeng, Z.T. Wen, J.K. Kajfasz, I.A. Freires, J. Abranches, L.J. Brady. " The Biology of ", Microbiology Spectrum, 2019 Publication	1%
4	jfmpc.com Internet Source	1%
5	Neculina Anyinkeng, Godlove A. Neba, Afui M. Mih, Aaron S. Tening. "Phytoremediation Potential of Some Macrophytes from a Car Wash Stream in Buea, South Western Cameroon", Journal of Environmental Protection, 2020 Publication	1%
6	Www.dovepress.com Internet Source	1%
7	mafiadoc.com	

	Internet Source	1 %
8	repositorio-aberto.up.pt Internet Source	1 %
9	topics.sciencedirect.com Internet Source	1 %
10	repository.uksw.edu Internet Source	1 %
11	mmbr.asm.org Internet Source	1 %
12	content.iospress.com Internet Source	1 %
13	www.walshmedicalmedia.com Internet Source	1 %
14	Novia N. Aviva, Damajanty H. C. Pangemanan, Pritartha S. Anindita. "Gambaran Karies Gigi Sulung pada Anak Stunting di Indonesia", e-GiGi, 2020 Publication	1 %
15	Brogan Ashley, Claire Simner, Antigoni Manousopoulou, Carl Jenkinson et al. "Placental uptake and metabolism of 25(OH)Vitamin D determines its activity within the fetoplacental unit", Cold Spring Harbor Laboratory, 2021 Publication	1 %

16	Springer Series on Biofilms, 2014. Publication	<1 %
17	Arass Jalal Noori, Fadil Abdullah Kareem. "The effect of magnesium oxide nanoparticles on the antibacterial and antibiofilm properties of glass-ionomer cement", Heliyon, 2019 Publication	<1 %
18	Ying Yang, Yuhong Li, Yuhong Lin, Minquan Du, Ping Zhang, Mingwen Fan. "Comparison of immunological and microbiological characteristics in children and the elderly with or without dental caries", European Journal of Oral Sciences, 2015 Publication	<1 %
19	www.spandidos-publications.com Internet Source	<1 %
20	Silvia Savastio, Erica Pozzi, Francesco Tagliaferri, Roberta Degrandi, Roberta Cinquatti, Ivana Rabbone, Gianni Bona. "Vitamin D and Cardiovascular Risk: Which Implications in Children?", International Journal of Molecular Sciences, 2020 Publication	<1 %
21	"7. Results and Discussion", Pharmacology and Toxicology, 9/2000 Publication	<1 %

22	Rytter, Maren Johanne Heilskov, Lilian Kolte, André Briend, Henrik Friis, and Vibeke Brix Christensen. "The Immune System in Children with Malnutrition—A Systematic Review", PLoS ONE, 2014. Publication	<1 %
23	cdr.lib.unc.edu Internet Source	<1 %
24	Advances in Salivary Diagnostics, 2015. Publication	<1 %
25	Francesca Sassi, Cristina Tamone, Patrizia D'Amelio. "Vitamin D: Nutrient, Hormone, and Immunomodulator", Nutrients, 2018 Publication	<1 %
26	Ciro Dantas Soares, Roman Carlos, Juan Pablo Díaz Molina, Thayná Melo de Lima Morais, Oslei Paes de Almeida. "Laryngeal Epstein–Barr Virus-Associated Smooth Muscle Tumor in an Undernourished Child", Head and Neck Pathology, 2018 Publication	<1 %
27	karyailmiah.unisba.ac.id Internet Source	<1 %

Exclude quotes	Off	Exclude matches	Off

Exclude bibliography On

Salivary sIgA as a predictor of caries risk in stunting children

GRADEMARK REPORT

FINAL GRADE

/0

GENERAL COMMENTS

Instructor

PAGE 1

PAGE 2

PAGE 3

PAGE 4

PAGE 5