SCIENTIFIC ARCHIVES OF DENTAL SCIENCES

Volume 2 Issue 6 June 2019

Mini Review

Oral Stomodeum and the Gastrointestinal System Obesity a Second Brain Dilemma

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Received: May 31, 2019; **Published:** June 03, 2019

Abstract

The Gastrointestinal Tract with its nerve cells, receptors and hormones functions like a 'second brain'. The incretins that function like hormones increase insulin secretion in response to the presence of glucose. The Cephalic phase is a nerve pathway of insulin secretion independent of glucose. The body anticipates food as soon as it goes into the mouth /stomodeum long before nutrients enter the stomach. All foods cause weight gain carbohydrates, proteins and fats. The dietary fats tend to have the weakest effect. Thus, all foods cause obesity or fat deposition in the connective tissues when excessive foods are administered. Indeed maternal obesity causes foetal obesity It has been found that this results in early eruption of the dentition due to excessive collagen cross linkage in the extracellular mesoderm commensurate with the PhD research findings of Thomas NR 1966 which was inhibited by oral administering aminoacetonitrile lathyrogens and is supported by recent research findings by cell biologists recently reviewed by Jenkins and Thomas in the J California Dental Association in August 2018'.

Keywords: Oral Stomodeum; Gastrointestinal System; Obesity; Brain Dilemma

Introduction

Cahill and Marks [1] formulated a Hypothesis that a clock exists in the formative stages of the enamel organ of each tooth that times progressive development of dental lamina, amelogenesis, dental follicle, gubernaculum, preocclusal and occlusal tooth eruption. Thus, the assessment of the relationship between body mass indexes (BMI) is a reliable index of overweight and obesity in children and adolescents. Obesity in children can lead to skeletal problems in the head and neck region. Alteration in the order of tooth eruption denotes a disorder in normal development of the teeth that in the obese condition leads to precocious development of tooth eruption [2].

Maternal obesity (MO) has harmful effects on both fetal development and offspring health. It has been shown that MO enhances collagen cross-linkage in fetal tissues. Mature collagen cross-linkage was increased as much as 36.3 +/- 9.9% ($p < 0.05$) compared to control offspring (22.3%) in maternal obesity while tissue inhibitor of metalloproteinases (TIMP) which remodels collagen was higher in obese offspring muscle. Lysyl oxidase (TGF-Beta) and tumor necrosis factor (TNF alpha) are key enzymes regulating collagen biosynthesis, collagen cross linking and inflammatory breakdown of collagen was higher in subjects given obesogenic diet (74.0 +/- 32.3%, 32.3%, $p < 0.05$ [3].

Hence the aberrant occlusal and oropharyngeal apparat necessitate emergent craniomandibular orthopedics, neuromuscular reconstruction, correction of forward head posture, myofascial dysfunctions including infant death syndrome, obstructive sleep apnea, gastroesophageal reflux syndrome and orthodontic collapse. It is recognized that the pre-functional 'clock' is evidence of a second brain that tests the clinician's skill and training in physiology extending from fetal physiology to the new born infant's physiology of breast feeding/suckling, myoglossal obesity effects on the forming occlusion and the neurophysiology of deglutition and mastication which does acknowledge the importance of swallowing with apnea.

The enamel organ leading from amelogenesis of the dental crown is followed by the development of the dental follicle, alveolar bone, root formation and periodontal ligament (PDL) defined as a tractional theory of tooth support and eruption by Thomas 1966 [4] and later Berkovitz and Thomas 1969 [5] although Berkovitz, Migdalski and Solomon 1972 [6] recused themselves from the tractional theory offering only that eruption is a multifaceted process. This is most certainly true since the graph accompanying their paper comparing eruption of lysyl oxidase inhibitor treated experimental animals compared with controls shows that eruption from the second to the eighth day is 20% decreased in mean eruption rate. Subsequently there appears no difference between control and experimental rates which appeared to be due to loss of tooth support with root bending (dilaceration) indicating that collagen is critical for both eruption and tooth support. Indeed the latter authors acknowledged that the teeth were easily extracted from their socket by the fingers. It is generally acknowledged that crosslinking of collagen stiffens the collagen providing for both tooth support and eruption due to the added effect of lysyl oxidase on the cell defined as tensegrity together with resultant differentiation of the periodontal stem cells into contractile myofibroblasts [7].

Studies of the effect of obesity show that early tooth eruption occurs suggesting a much wider concept of timing of events than a clock. Many medicines given orally are incompletely absorbed or deactivated by the liver before reaching the blood stream. Intravenous delivery tends to be much more effective. In the case of blood glucose level oral administration is identical to that given by the intravenous route. But despite the same level of blood sugar insulin response differs greatly. Remarkably the insulin response to oral glucose is much more powerful. Intensive research revealed that the stomach produces hormones called incretins that increase insulin secretion by gut hormones that are secreted from entero endocrine cells into the blood within minutes after eating. The incretin effect is insulin effect and may account for 50 - 70 per cent of the excessive insulin secretion after oral glucose intake. Rather than simply being a mechanism for food absorption and excretion. The stomodeum, gastrointestinal tract, with its nerve cells, receptors and hormones functions like a 'second brain'. The two human incretin hormones described so far are glucagon-like peptide 1(GLP) and glucose-dependent insulin tropic polypeptide (GIP). However, studies reported in 1966 show that in the case of oral administration of the amino acid leucine also causes insulin secretion [8]. Thus, oral administration of fats, proteins and carbohydrates all release and thus increase insulin levels. Even non-nutritive sweeteners which have no calories at all can stimulate the insulin response [9].

This is important in the connection with obesity because insulin response to macronutrients in non-diabetic and none insulin dependent diabetes mellitus (NIDDM) decreases the incretin effect. The cephalic phase is another pathway of insulin secretion independent of glucose. The body anticipates food as soon as it goes into the mouth and long before nutrients enter the stomach. All foods can thus cause weight gain. Thus, restricting carbohydrates may not always be as beneficial as believed. Dietary fat tends to have the weakest effect of all foodstuffs. In summary drugs that raise insulin levels cause weight gain progressing to obesity. A recent study showed that 75% of the weight change response is predicted by insulin levels [10] and are dependent upon the same enzymes that increase collagen cross-linkage TGF-alpha (weight gain) and decreased collagen cross-linkage TFN beta (weight loss).

Conclusion

The profibrotic cytokine TGFβ promotes the expression of genes involved in collagen maturation and cross-linking. At down-stream TGF-β signaling was more active in obesity compared to controls because TGF-β signaling regulates collagen synthesis and fibrosis as well as increasing eruption

Intermolecular crosslinking of collagen provides stability and tension to collagen fibrils. Crosslinking is initiated by oxidative deamination of selected telopeptides and helical collagen lysine residues, a critical step catalysed by lysyl oxidase. Inflammation induces lysyl oxidase expression.

Obesity is now considered the most important health problem. The relationships between obesity and specific oral diseases and disorders: periodontal disease, dental caries and early eruption are biologically plausible. Data supporting relationship between obesity and periodontal disease are the strongest. Inflammatory response reduces insulin sensitivity and elevated blood glucose levels leading to greater accumulation of advanced glyoxylate end stages (AGES) suggest that obesity exacerbates periodontal disease.

Chronic non-communicable diseases have overtaken communicable diseases as leading health problems in most parts of the world.

Periodontitis, a chronic inflammatory disease initiated by dental plaque biofilm is perpetuated by a deregulated immune response.

More recently a major public health problem has occurred as overweight and obesity prevalence which has trebled since the 1980s, especially in developed countries Overweight and obesity are defined as abnormal or excessive fat accumulation that represents a risk to general health. Furthermore, insulin resistance dyslipidaemia and a state of low-grade inflammation is evident in obesity. In addition, numerous other comorbidities are currently being investigated including diabetes, coronary artery disease, stroke, respiratory disorders, hypertension, cancers, osteoarthritis, liver and gall bladder disease.

Dentists have an important role in preventing and detecting oral and systemic diseases because of their diagnostic and screening abilities and the frequency of patient visits. These skills and practice paradigms should be considered in solving the obesity epidemic. Periodontal disease and diabetes are a reason for dentists to intervene in the rise of obesity. Dentists can play a role in raising awareness of overweight status and obesity risk behaviours in children.

Bibliography

1. Cahill DR, Marks SM. Chronology and histology of exfoliation and eruption of mandibular premolars in dogs. J Morphol. 1982;171(2):213-218.

2. Zangouei-Booshehri M, Ezoddini-Ardakani F, Agha Aghili H, Sharifi H. Assessment of the relationship between body mass index (BMI) and dental age. Health. 2011;3(5):253-257.

3. Huang Y, Zhao Jun-Xing, Yan Xu, Zhu MJ, Long NM, McCormick RJ, Ford SP, Nathanielsz PW, Min Du. Maternal Obesity Enhances Collagen Accumulation and Crosslinking in Skeletal Muscle of Ovine Offspring. PLoS One 2012;7(2):e31691.

4. Thomas NR. The Process and Mechanism of Tooth Eruption. PhD thesis University of Bristol, UK, 1966.

5. Berkovitz BKB, Thomas NR. Unimpeded eruption in the root-resected lower incisor of the rat with a preliminary note on root resection. Archs oral Biol. 1969;14(7):771-780.

6. Berkovitz BKB, Migdalski A, Solomon M. The effect of the lathyritic agent aminoacetonitrile on the unimpeded eruption rate in normal and root-resected rat lower incisors. Archs oral Biol. 1972;17(12):1755-1763.

7. Thomas NR, Jenkins DN. Inhibition of Collagen Crosslinking produces Significant Retardation of Impeded and unimpeded eruption rates. Journal of the California Dental Association. 2018;46(8):497-504.

8. Floyd JC. Insulin response to protein ingestion. J Clin Invest. 1966;45(9):1479-1486.

9. Fung J. The Obesity Code. pub Greystone 2016.

10. Kong LC, Wuillemin PH, Bastard JP, Sokolovska N, Gougis S, Fellahi S, Darakhshan F, Bonefont-Rousselot D, Bittar R, Dore J, Zucker JD, Clement K, Rizkalla S. Insulin resistance and inflammation predict kinetic body weight changes in response to dietary weight loss and maintenance in overweight and obese subjects by using a Bayesian network approach. Am J Clin Nutr. 2013;98(6):1385-1394.

Volume 2 Issue 6 June 2019