

# THE “TEETH” IN DISEASE?

## READERS SUMMARY:

1. HOW SCIENCE PRESENTS A CURVEBALL YOU DID NOT SEE COMING?
2. HOW YOUR MOUTH MIGHT BE THE KEY TO DIABETES, OBESITY AND INFLAMMATION?
3. IS THERE A HYPOTHALAMIC PAROTID AXIS?
4. IS THE PAROTID GLAND THE MOUTH’S PANCREAS?

Most of you may not know that before I was a neurosurgeon I was a dentist and oral and maxillofacial surgeon. Many people know that Weston A. Price was a dentist too, who was ostracized from organized dentistry for many political reasons. Much of his accomplishments have been largely under appreciated ,because he was painted as an “renegade” by the American Dental Association and by organized dentistry. Most dental students never learn a thing about him or his work as a result. The ancestral community knows about Weston A. Price largely, because of his foundation and people like Sally Fallon and the newer generation of young bucks, like, Chris Masterjohn. When I went to dental school at UCONN, my classmates and I never learned a thing about Weston A Price. We never were told about his work or his books. In the last 25 years, my education has evolved from dentistry to oral surgery, then to neurosurgery, and now to evolutionary molecular biology and it hopefully it will end in Optimal longevity.

My background is different, and kind of gives me a unique view

point into medicine, surgery, and aging. Unlike many other ancestral practitioners, I look at health and disease a bit differently. If you have read my QUILT, you know that levee one is the top of the heap. The cellular terroir determines all things in biologic terms for the future's of our cells. Lately, there has been a lot of "discussion" in the blogosphere about macronutrient theories of obesity and the role of carbohydrates, and most of you know that my views on the situation are different than some of these "food guru" luminaries. Instead of drawing a line in the sand on macronutrients, I have chosen to look at the problem through the optic of how our brain accounts for electrons from our food, and how it responds to those signals via the hormonal signals from our hypothalamus in our brain. This is a job of quantum biology. The main hormone that determines how we partition electrons is leptin, as I have laid in my Quilt document, and many of my blogs. Today, we are going to talk about an area that has never been tapped in our community—How might diabetes and obesity begin when we view the problem through an "old" scientific finding that has been largely ignored since it was discovered in 1968.

I'd like to introduce you to John Leonora, Ph.D in endocrinology. He was a medical/dental researcher who like, Weston A. Price, was largely ignored by most of organized dentistry and medicine. He toiled at Loma Linda University and he recently died in February of 2006. I first heard his name in 1986, when I was a first year dental student at UCONN during a class on the origin of dental caries. Back then, I heard a presentation on a new theory of dental caries based upon a lecture that Dr. Leonora gave in 1983. I became reacquainted with some more of his work from some of my current patients who are 7<sup>th</sup> Day Adventists and one of my former dental school classmates, Dr. John Sorrentino recently. My 7<sup>th</sup> Day Adventist friends turned me back on to

his work in 2006 and I largely ignored this information again after I read it. I think I never fully grasped the implications of it, honestly. I compiled the articles he wrote, and in 2006, put them in a file cabinet under the tab called the "oral hypothalamus". Then in October 2011, my old buddy Dr. Sorrentino, from UCONN, sent me an article in a general dentistry magazine to read. As soon as I saw the name, John Leonora, lightening struck my own memories. It was time to synthesize what I remembered from those articles in 2006 and the 1986 talk, and what I now know and write something cogent for you to consider.

Dr. Leonora had some new theories about how dental caries (tooth decay) were caused. He rejected the dogma I was taught in dental school that the acid from bacteria caused demineralization of enamel and dentin in teeth to cause dental decay. He took it much further. He postulated that that dental caries was caused not by bacteria, but caused by inflammation that was due to hypothalamic interruption of hormones that controlled avascular structures in teeth and other endocrine organs. **He believed that the parotid gland in our mouth acted just like our pancreas does in our gut.** He believed that the parotid gland indirectly controlled the metabolism of avascular structures like dentin, enamel, and the islets of Langerhans that control insulin release in the pancreas. This axis essentially helped the brain sense the initial carbohydrate breakdown in the mouth, and relayed that data to our parotid gland and pancreas to formulate the brain/gut axis response hormonally to this energy substrate. This is why salivation and insulin secretion can often occur before a sugary meal is even eaten. In fact, he was the first person hinting that insulin and salivary flow maybe linked to sun exposure. Leptin receptors are also now known to exist in the mouth, gingiva, and on taste receptors.

In 1986, as a first year as an ignorant dental student, I had

no idea how brilliant this man was. Moreover, I was unaware of how incredibly ignorant the rest of the dentistry and medicine was to his findings that began in the late sixties. Today, I now know about the tremendous accomplishments of Weston A Price, so it comes as no surprise that another dental researcher findings are finding the light of day today. Dr. Leonora found that rats fed a high cariogenic diet (high sucrose diet) ***had the flow of dentinal fluids completely reversed in their teeth, than we expect in normal non-diseased teeth.*** This reversal of dentinal fluid caused the teeth to become susceptible to decay because it fostered an inflammatory terroir in the tooth. In 1978, he wrote about the systemic role of trace elements in glucose metabolism (magnesium and zinc). Remember, from the Gnolls blog post we spoke about how the loss of intracellular magnesium was the first biochemical step in developing diabetes, but not the first quantum effect that causes it. *Dr. Leonora found the exact same thing in the hypothalamic parotid axis too.* I don't think this is a coincidence either. Prior to this in 1975, he wrote about the effects of carbohydrates on the hypothalamic parotid gland endocrine axis and how it mimicked the endocrine and exocrine pancreatic physiology. He followed this line of research up with working out how the parotid gland also had dual endocrine and exocrine function like the pancreas. He showed in the earlier 1980's that dental caries was largely influenced by hormonal fluctuations and not from bacterial acid production to cause disease but from hormonal changes due to a foods carbohydrate load. This hypothesis was largely rejected at the time. Even today, most dentists and physicians are ignorant of this man's work. I knew about it because I listened to his 1983 talk on this at UCONN medical library and my professor pretty much shot it down after I asked him about it. He was quick to point out citing the "accepted research at the time in the literature." I totally forgot about it until recently.

Mainline scientific theories may have long lifespans, but are inevitably overturned as accumulating evidence renders them obsolete and brings alternative theories to the fore. This happened in physics in 1905 with the advent of the theory of relativity, and it happened 150 years ago in biology, when many diseases did not spontaneously generate but whose etiologies became explainable when germs and viruses became known. It has now happened again today in molecular biology with ROS and antioxidants effects on mitochondria as well. Dr. Leonora's work was pretty shocking for the early 1980's scientific community, and I failed to realize its significance until recently as well. My knowledge back then was fairly limited. Today, I am a bit more well rounded and I think his work merits some further attention.

When such a major scientific change in paradigm happens, there is usually a period of significant and often bitter controversy between scientists seeking to hold on to the theories of the older paradigm, and those espousing the newer one. The recent disputes on obesity from Taubes, Guyenet, and Harris come to mind in the blogging community. Often, this is followed gradually by changes in basic thinking patterns and a subsequent long period of fertile discovery. That fertile ground takes many shapes. Today's post is planting some more of those seeds for you to think about.

It appears that obesity and diabetes development "might begin" in the oral cavity. In this way our mouth maybe an ideal redox sensor for most diseases. We know that carbohydrates are first broken down by salivary gland amylase. Amylase breaks down long-chain carbohydrates. Most human amylases tend to be alpha amylases. Both the parotid gland and pancreas share this exact physiology. It appears the endocrine function of the parotid gland's acinar cells respond

to a releasing hormone of hypothalamus. This ability is only found in the human parotid gland. ***If the human parotid gland is removed, the hypothalamic parotid axis ceases to work on dentinal flow in teeth.*** Oral inflammation occurs and sets the stage for disease propagation. It appears that this axis is coupled to lingual leptin receptors, and possibly to parotid gherlin levels. This releasing hormone signal from the brain releases a parotid specific hormone that directly effects the flow of fluid in dentin and helps forms teeth and to calcify and re-calcify decayed teeth (with Vitamin K2) to keep them in optimal health. When the process is broken down, dentinal fluid reverses towards the apex of the tooth, and we absorb things from the oral cavity we should not and it causes an inflammatory response. This is precisely how inflammation enters the tooth and jaw to cause inflammation. It also helps explain why diseased teeth show apical abscesses as the inflammation become chronic. It also appears to be the mode of action in how we absorb heavy metals from amalgams and metal dental framework. It further sheds light on why bad periodontal and dental health correlates well with cardiac risk and elevated cardiac CRP and diabetes. It appears the oral cavity is the initial sensory sensor for foods for the hypothalamus as well.

This parotid signaling system is how the brain readies the "avascular parts" of the gut for the onslaught of carbohydrates that will eventually hit the duodenum. The Islets do not have a direct blood supply. They have a portal blood supply much like that of the pituitary gland. It readies the avascular Islets of Langerhans of the pancreas to further deal with a carbohydrate load. It seems to somehow effect the islets portal blood supply and its gene regulation even before the carbohydrates hit the duodenum to signal the pancreas to action. There is research ongoing to see if the parotid gland somehow is involved in the biphasic release of insulin. It

also appears that Dr. Leonora discoveries should have illuminated some light on the significance of the hypothalamic parotid gland endocrine axis as it relates not only to dental care, but also to endocrine pancreatic function and the possible development of diabetes and obesity. Sadly this curious finding remains largely unknown by even the most learned neurobiologists and clinicians. I hope this changes soon, because it might just uncover more information to help us help people restore their health.

#### **CITES:**

1.

<http://www.sciencedirect.com/science/article/pii/S1568163703000667>

2. Biological Significance of Glutamate Signaling during Digestion of Food through the Gut-Brain Axis, Akihiko Kitamura, Tomokazu Tsurugizawa, Kunio Torii, Nutritional Physiology Fundamental Research Group, Frontier Research Laboratories, Institute for Innovation, Ajinomoto Co., Inc., Kawasaki, Japan Digestion 2011;83 (Suppl. 1):37-43 (DOI: 10.1159/000323407)

3.

<http://www.nature.com/oby/journal/v8/n3/full/oby200024a.html>

4.

<http://jme.endocrinology-journals.org/content/34/2/353.full>  
(salivary leptin)

5.

<http://onlinelibrary.wiley.com/doi/10.1111/j.1601-0825.2011.01820.x/abstract> (leptin's effects on dentin stem cells)

6. <http://www.springerlink.com/content/x482348327311k50/>  
(leptin and oral cancer)
7.  
<http://www.ispub.com/ostia/index.php?xmlFilePath=journals/ijds/vol6n1/leptin.xml> (leptin receptors on gingiva)
8. Groschl M, Rauh M, Wagner R, Neuhuber W, Metzla M, Tamguney G, et al. Leptin in human saliva. *J Clin Endocrinol Metab.*2000; 86: 5234-9.
9. Ken Southward, DDS, FAGD, "The systemic theory of dental caries." Featured in *General Dentistry*, September/October 2011. Pg. 367-373.  
(<http://www.agd.org/publications/articles/?ArtID=9892>)
10. Chaussain-Miller C, Fioretti F, Goldberg M, Menashi S. The role of matrix metalloproteinases (MMPs) in human caries. *J Dent Res* 2006;85(1): 22-32.
11. Schatz A, Martin JJ, Schatz V. The chelation and proteolysis-chelation theories of dental caries: Their origin, evolution and philosophy. *NY State Dent J* 1972;38(5):285-295.
12. Kato MT, Leite AL, Hannas AR, Buzalaf MA. Gels containing MMP inhibitors prevent dental erosion in situ. *J Dent Res* 2010;89(5):468-72. Epub March 3, 2010.
13. Requejo R, Chouchani ET, Hurd TR, Menger KE, Hampton MB, Murphy MP. Measuring mitochondrial protein thiol redox state. *Methods Enzymol* 2010;474:123-147. Epub June 20, 2010.
14. Leonora J, Tieche JM, Steinman RR. Further evidence for a hypothalamus-parotid gland endocrine axis in the rat. *Arch Oral Biol* 1993;38(10): 911-916.
15. Leonora J, Tjaderhane L, Tieche JM. Effect of dietary carbamyl phosphate on dentine apposition in rat molars. *Arch Oral Biol* 2002;47(2): 147-153.



16. Leloup C, Turrel-Cuzin C, Magnan C, Karaca M, Castel J, Carneiro L, Colombani AL, Ktorza A, Casteilla L, Penicaud L. Mitochondrial reactive oxygen species are obligatory signals for glucose-induced insulin secretion. *Diabetes* 2009;58(3):673-681. Epub December 10, 2008.

17. Stowe DF, Camara AKS. Mitochondrial reactive oxygen species production in excitable cells: Modulators of mitochondrial and cell function. *Antioxid Redox Signal* 2009;11(6):1373-1414.

18. Potula R, Hawkins BJ, Cenna JM, Fan S, Dykstra H, Ramirez SH, Morse B, Brodie MR, Persidsky Y. Methamphetamine causes mitochondrial oxidative damage in human T lymphocytes leading to functional impairment. *J Immunol* 2010;185(5):2867-2876. Epub July 28, 2010.