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Tracing the effects of Epigenetic factors on midface growth, upper airway collapse and intermittent Hypoxia

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onsidering the causes of the sharp rise of primary human pathologies during the last half of the 19th century, rationalization showed that while alcohol and tobacco were major factors, it is sucrose (cane and beet sugar) that poses the greatest problem. The pathway starts with exposure to these three epigenetic factors in early embryonic growth, before the facial skeleton is determined. Such exposure induces HOX genes into midface growth, normally the sole prerogative of Neural Crest cells. Such induction leads to a truncation of midface growth and resulting in a smaller nasomaxilla and oro-pharynx. This opens two pathways, the first being impaired breathing where intermittent falls and rise of cerebral oxygen induces recruitment of Hypoxia- Induced-Factors [HIF's]. These peptides 'turn- on' the systemic inflammatory chemical factory producing the chemical products that generate systemic inflammation that sponsors and maintains most of human pathology. The second pathway that that of distalising of the mandible and compressing the

soft tissues of the vascular bed of the TMJ which are highly populated with type 4 nociceptors and is thereby arguably converted into the most 'most-painful' joint in the body. This is now a well evidenced claim and the combination of systemic inflammation and of a cortical barrage of sub-clinical barrage of nociceptive signaling subtends most disorders associated with this pattern, principally those ranking high in morbidity.

Speaker Biography

David Zimmerman has graduated as a general dentist long ago. An interest in orthodontics became one in craniofacial growth, TMD and sleep. The broad spectrum of maladies with a common thread of OSA/SDB demanded rationalizing. The presentation is a short form of these links and of their clinical significance. Dr. Zimmerman is currently involved in examining the relationship between adaptive posture, altered weight distribution and its involving lumbar flexion and damage in attaining weight equilibrium. Understanding adverse lumbar loading is key to reducing both lateral segment damage and reducing the incidence of spinal surgeries.

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