3049 Growth Hormone Receptor Defects Effect on Tooth Morphology and Structure

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Laron syndrome, an autosomal recessive disorder, is caused by mutations in the Growth Hormone Receptor (GHR) gene leading to deficient IGF-1. In humans, this results in dwarfism associated with bone and tooth malformations (hypodontia of the teeth in 30% of patients, tooth crowding, delayed eruption, and defective teeth). Homozygous (−/−) and heterozygous (+/−) Laron mice were produced and identified by Southern analysis. In both mice and humans GHR and IGF-1 are expressed at all stages of tooth development and growth factors influenced by these products are reduced in the absence of these factors. Objectives: Our preliminary mouse model data showed homozygous GHR knock-out mice (−/−) had statistically smaller (ANOVA/Fishers Multiple Mean p<0.05) molar dimensions (mesial/distal widths) compared with the heterozygous and wild-type mice. The purpose of this study was to evaluate the effect of GH insensitivity on enamel structure and morphology in humans. Methods: Primary and permanent teeth of individuals with treated (IGF-1 replacement therapy) and untreated Laron syndrome were compared to control teeth. Mesial-distal and facial-lingual widths were recorded. The teeth were then sectioned and the enamel thickness and structure evaluated using light microscopy. Results: The mesial-distal and facial-lingual measurements for the Laron teeth were smaller than the controls. However, the teeth from the treated and untreated Laron syndrome subjects had significantly increased enamel thickness compared with controls (P <0.05). The enamel structure of the teeth from the Laron syndrome individuals was prismatic, but had more pronounced Striae of Retzius and more darkened areas of enamel than the control teeth. Conclusions: Congenital IGF-1 deficiency leads to a decreased tooth size in the GHR knock-out mice and Laron patients. However, the lack of GH/IGF-I activity in humans resulted in increased enamel thickness and more pronounced Striae of Retzius and apparent dead space. Supported in part by Dental Foundation Research Fellowship UNC

Seq #330 - Basic Science Category
10:15 AM-11:30 AM, Saturday, 13 March 2004 Hawaii Convention Center Exhibit Hall 1-2

Back to the Caulk/Dentsply Competition Program
Back to the IADR/AADR/CADR 82nd General Session (March 10-13, 2004)