Laron syndrome is caused by mutations or deletions of the Growth Hormone Receptor (GHR) gene leading to deficient IGF-1. This results in dwarfism associated with both tooth and bone malformations. GH receptors and IGF-I are expressed at all stages of tooth development and growth factors influenced by these products are reduced in the absence of GH receptors and IGF-I. A mouse model for Laron syndrome was developed by knocking-out the GHR gene.

Objectives: Although Laron syndrome is often characterized by the presence of tooth crowding, delayed eruption, and defective teeth, the effect of the syndrome on tooth morphology and enamel structure has not been examined. The purpose of this study was to evaluate the effect of GH insensitivity on tooth development. Methods: GHR knock out mice and Laron Syndrome human teeth were used for this study. Mouse molars were measured in multiple planes using digital morphometry. Primary and permanent teeth of an individual with Laron Syndrome were sectioned and the enamel thickness and structure evaluated using light microscopy. Results: The homozygous GHR knock-out mice had significantly (ANOVA/Fishers Multiple Mean p<0.05) smaller molar dimensions (mesial/distal widths) compared with the wild type mice. The Laron human teeth showed similar to increased enamel thickness compared with controls. The human Laron enamel structure was prismatic and in some regions showed more pronounced Striae of Retzius compared with normal teeth. The lack of GH/IGF-I activity in humans did not result in a diminished enamel thickness or alter the basic prismatic enamel architecture. Supported in part by Grover Hunt Fellowship UNC

Seq #93 - Amelogenesis & Enamel Proteins
3:45 PM-5:00 PM, Thursday, 13 March 2003 Henry B. Gonzalez Convention Center Exhibit Hall C

Back to the Mineralized Tissue Program
Back to the 32nd Annual Meeting and Exhibition of the AADR (March 12-15, 2003)