Wnt signaling regulates pulp volume and dentin thickness.

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Public Summary:
The cells that form mineralized tissues in the body are all responsive to a Wnt stimulus. In this paper we demonstrated the consequences of inadequate Wnt signaling. We showed that mice carrying a mutation in a protein called Wntless exhibit severe dental defects in addition to their brittle bones. These data demonstrate the the importance of Wnt signaling in the homeostasis of mineralized tissues of the craniofacial complex.

Scientific Abstract:
Odontoblasts, cementoblasts, ameloblasts and osteoblasts all form mineralized tissues in the craniofacial complex, and all these cell types exhibit active Wnt signaling during postnatal life. We set out to understand the functions of this Wnt signaling, by evaluating the phenotypes of mice in which the essential Wnt chaperone protein, Wingless was eliminated. The deletion of Wls was restricted to cells expressing Osteocalcin, which in addition to osteoblasts includes odontoblasts, cementoblasts, and ameloblasts. Dentin, cementum, enamel, and bone all formed in OCN-Cre;Wlsfl/fl mice but their homeostasis was dramatically affected. The most notable feature was a significant increase in dentin volume and density. We attribute this gain in dentin volume to a Wnt-mediated mis-regulation of Runx2. Normally, Wnt signaling stimulates Runx2, which in turn inhibits DSP; this inhibition must be relieved for odontoblasts to differentiate. In OCN-Cre;Wlsfl/fl mice, Wnt pathway activation is reduced and Runx2 levels decline. The Runx2-mediated repression of DSP is relieved and odontoblast differentiation is accordingly enhanced. This study demonstrates the importance of Wnt signaling in the homeostasis of mineralized tissues of the craniofacial complex.

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