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Isolation and characterization of dental epithelial cells derived from amelogenesis imperfecta rat.

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Abstract

OBJECTIVE: Disruption of the third zinc finger domain of specificity protein 6 (SP6) presents an enamel-specific defect in a rat model of amelogenesis imperfecta (AMI rats). To understand the molecular basis of amelogenesis imperfecta caused by the Sp6 mutation, we established and characterized AMI-derived rat dental epithelial (ARE) cells.

MATERIALS AND METHODS: ARE cell clones were isolated from the mandibular incisors of AMI rats, and amelogenesis-related gene expression was analyzed by reverse transcription polymerase chain reaction (RT-PCR). Localization of wild-type SP6 (SP6WT) and mutant-type SP6 (SP6AMI) was analyzed by immunocytochemistry. SP6 transcriptional activity was monitored by rho-associated protein kinase 1 (Rock1) promoter activity with its specific binding to the promoter region in dental (G5 and ARE) and non-dental (COS-7) epithelial cells.

RESULTS: Isolated ARE cells were varied in morphology and gene expression. Both SP6WT and SP6AMI were mainly detected in nuclei. The promoter analysis revealed that SP6WT and SP6AMI enhanced Rock1 promoter activity in G5 cells but that enhancement by SP6AMI was weaker, whereas no enhancement was observed in the ARE and COS-7 cells, even though SP6WT and SP6AMI bound to the promoter in all instances.

CONCLUSION: ARE cell clones can provide a useful in vitro model to study the mechanism of SP6-mediated amelogenesis imperfecta.

KEYWORDS: Sp6 mutation; amelogenesis imperfecta; in vitro disease model; tooth phenotype

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