Identification of genes involved in fluoride resistance in oral streptococci

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（口腔レンサ球菌のフッ化物耐性に関与する遺伝子の同定）

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論 文 内 容 の 要 旨

Recently, it has been reported that *eriC* and *crcB* are involved in bacterial fluoride resistance. However, the fluoride-resistance mechanism in oral streptococci remains unclear. BLAST studies showed that two types of *eriC*s (*eriC1* and *eriC2*) and two types of *crcB*s (*crcB1* and *crcB2*) are present across 18 oral streptococci, which were selected based on the following criteria: identification in Ó10% of 166 orally healthy subjects and Ó0.01% of the mean relative abundance. They were divided into three groups based on the distribution of these four genes: group I, only *eriC1*; group II, *eriC1* and *eriC2*; and group III, *eriC2*, *crcB1*, and *crcB2*. Group I consisted of *Streptococcus mutans*, in which one of the two *eriC1*s predominantly affected fluoride resistance. Group II consisted of eight species, in which *eriC1* was involved in fluoride resistance but *eriC2* was not in *Streptococcus anginosus* as a representative species. Group III consisted of nine species, in which both *crcB1* and *crcB2* were crucial for fluoride resistance, but *eriC2* was not, in *Streptococcus sanguinis* as a representative species. Based on these results, either EriC1 or CrcB play a role in fluoride resistance in oral streptococci. Complementation between *S. mutans* EriC1 and *S. sanguinus* CrcB1/B2 was confirmed in both *S. mutans* and *S. sanguinis*. However, neither transfer of *S. sanguinis* CrcB1/B2 into wild-type *S. mutans* nor *S. mutans* EriC1 into wild-type *S. sanguinis* increased the fluoride resistance of the wild-type strain. It is possible that EriC1 and CrcB1/B2 are responsible for fluoride resistance in oral streptococci by sharing specific pathways.