# Abstract

Ectodysplasin A (EDA) is a transmembrane protein of the TNF family, which plays an important role in the development of ectodermal derivates, such as teeth, hair or glands. Mutation in E*da* gene causes the Hypohidrotic ectodermal dysplasia (HED) in humans. Sonic hedgehog (*Shh*) as a downstream of *Eda* signalling pathway is an important signalling molecule involved in the initiation of tooth development.

In frame of the present study, we aimed to evaluate the involvement of *Eda* gene during the development of mice teeth and its relation to *Shh* signalling, since *Shh* is an important marker of the normal tooth development. We focused on *Shh* expression in *Eda* mutant mice teeth with spontaneous mutation in *Eda* gene, since these mice represent a natural model for X-linked HED. First, we compared the *Shh* expression pattern during the tooth development in *Eda* mutant mice with CD1 control using the dissociation of dental epithelia and fluorescent microscopy. Consequently, we focused on the cell line expressing *Shh* observation using Cre-loxP system. We also visualized SHH expression in *Eda* mutants using imunohistochemistry and qantified SHH protein in the embryonic jaws using western blot.

According to our results, the development of teeth in *Eda* mutants seems to be approximately one day delayed compared to controls. We can summarize that *Eda* gene with its protein seems to influence the region of successive *Shh* expressions formation in the sense of a reduction of its size and duration of the presence but we did not observe any deficiency of SHH protein in *Eda* mutant mice.

Keywords: *Eda* gene, Hypohidrotic ectodermal dysplasia, tooth development, patology of teeth, mouse