Id2 acts downstream of BMP signaling in chondrogenesis

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Objectives:

This study aimed to conduct a case-control investigation in maxilofacial morphogenesis, using a sample of Id2 homozygous knockout (KO) and wild-type (WT) mice. A special interest was to establish a relationship among Id2, BMP signals and chondrogenesis.

Materials and Methods:

Appropriate ethics approval has been received. Crania collected from mice at the age of 0 day, 2 weeks and 12 weeks were assessed with a Euclidean Distance Matrix Analysis method. Cultured synchondroses of the murine cranial base were inspected with a histological approach and examined with a reverse transcription polymerase chain reaction technique.

Results:

A shorter nasofrontal profile was seen in Id2 KO 12-week-olds but not 0-day-olds. The WT-KO gap in the skull length instead of the width increased with age. KO 2-week-olds showed a narrower hypertrophic zone and an inhibited proliferative zone in the presphenoid and the spheno-occipital synchondroses. Id2 expression in WT synchondroses was identified. Distribution of Type X collagen other than osteopontin was downregulated in Id2 KO samples. The WT murine cranial base showed a wider hypertrophic zone, a higher degree of ectopic hypertrophy and a larger number of proliferative chondrocytes in the presence of exogenous BMP2, BMP4 and BMP7, respectively. Such acquired growth was not detected in KO subjects. A 5-fold upregulation of Smad7 transcripts and a decrease in phosphorylation of Smad1-Smad5-and-Smad8-positive cells were identified in Id2 KO cartilage.

Conclusions:

Postnatal chondrogenesis was related to Id2 that acted downstream to enhance BMP signals by inhibiting Smad7 expression.