



Pathogenetic Mechanism and Therapy Strategies of Achondroplasia

Guest Editor:

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Message from the Guest Editor

Achondroplasia (ACH), the most common form of short-limbed skeletal dysplasia, is caused by activating mutations in the fibroblast growth factor receptor 3 (FGFR3) gene. Patients with ACH suffer from various neurological, otolaryngologic and orthopaedic problems for a lifetime. Multidisciplinary medical approaches from early childhood are required to maintain a long-term patient's quality of life. Several researches to develop therapeutic drugs have focused on target inhibition of the activated FGFR3 signaling in growth plate chondrocytes. Some of the drugs including BMN111, infgratinib, and TransCon CNP, are currently on-going clinical trials.

In this special issue, we would like to review current therapeutic approaches for diverse disease-specific problems, including short stature, spinal canal stenosis, sleeping apnea, foramen magnum stenosis, and upper-airway obstruction. We will also discuss exciting advances of novel pharmacological development for future clinical applications and describe molecular mechanisms of these candidate drugs on the therapeutic potential.





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Message from the Editor-in-Chief

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