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Genetic Disorders of the TGFβ Signaling Family

Guest Editor:

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Deadline for manuscript submissions:

closed (25 October 2020)

Message from the Guest Editor

Dear Colleagues,

The TGF β superfamily of signaling molecules, in humans, includes over 30 ligands that are clustered into several subfamilies, all that signal through one or more of the five type Il receptors, seven type I receptors, five receptor-associated Smad signal transducers, Smad1, Smad2, Smad3, Smad5 and Smad8, and a single Smad4 nucleocytoplasmic shuttling co-Smad, Smad4. These signaling pathways play critical roles in embryonic development and are frequently perturbed in common disease processes such as cancer, cardiovascular disease and immunity, and drugs that target pathway components have been developed for therapeutic purposes. Gain- or loss-of-function mutations in pathway components cause various human genetic disorders that manifest as abnormal patterns of skeletalmuscular growth and dysmorphology, as well as cardiovascular and premalignant syndromes. This Special Issue will focus on human genetic disorders caused by mutations in components of the $TGF\beta$ signaling superfamily, the novel molecular mechanistic insights gained from study of these genetic disorders, and therapeutic approaches developed for their treatment.













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Message from the Editor-in-Chief

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