Special Issue

Advances in Treatments for Hutchinson-Gilford Progeria Syndrome

Message from the Guest Editor

Hutchinson–Gilford Progeria Syndrome is an ultra-rare "premature aging" disease. A de novo point mutation in the prelamin A gene results in an aberrant spicing and causes the accumulation of the spliced shortened form, progerin, at the nuclear envelope. Progerin is toxic and causes distorted nuclei and aging-related phenotypes, and death in the teenage years from cardiopulmonary arrest or cerebral infarction. Although they appear healthy at birth, most children with Progeria begin to display premature aging within the first year of life. It is a milestone for the field that Farnesyltransferase lonafarnib was approved as the first targeted therapy for children with HGPS, but the effect is modest. Therefore, it is urgent to understand the precise mechanisms of HGPS pathology and to develop better treatments for children suffering from HGPS. This Special Issue will examine new therapeutics for HGPS treatment, novel model mouse systems to help to prove progerin toxicity, methods to advance a better understanding of HGPS cells or progerin protein, organ-specific pathology of HGPS, modulation of cytokine signaling in HGPS, and genome structure in HGPS.

Guest Editor

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Message from the Editorial Board

Cells has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. *Cells* encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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