Research Lectures at Nobel Forum 11 October, 16.30

The two faces of cellular senescence in tissue repair and in multiple diseases

Manuel Serrano

Manuel Serrano, PhD
Institute for Research in Biomedicine (IRB Barcelona)
Barcelona, Spain
+34 934 020 287
manuel.serrano@irbbarcelona.org

Upon tissue damage or stress, a substantial fraction of cells respond by adopting a cellular state known as "senescence". Regardless of their initial cell identity, senescent cells share key properties; namely, global chromatin remodelling, robust proliferation blockade, and a massive pro-inflammatory secretome. Major advances in recent years indicate that the biologic purpose of senescent cells is to orchestrate tissue repair, ultimately leading to their own disposal by the immune system and to their replacement by new, functional cells. This is the favourable, beneficial, face of cellular senescence. However, in certain contexts that are generally associated with chronic damage, degenerative processes, or organismal ageing, tissue repair is inefficient and senescent cells are not cleared. Indeed, senescent cells accumulate in many human pathologies including various fibrotic diseases, atherosclerosis, and neurodegenerative diseases. This is the detrimental, pathological, face of cellular senescence. Importantly, the last few years have witnessed the identification of pharmacologic interventions that preferentially kill senescent cells, termed senolytics. Such senolytic treatments in mice show an unprecedented therapeutic effect on animal models of the aforementioned diseases including lung fibrosis, atherosclerosis, and Parkinson's disease. I will present our contributions to the understanding of cellular senescence both in tissue repair and in pathological contexts.

Venue: Nobel Forum, Nobels väg 1.

Host: Staffan Strömblad