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Editorial

Cellular Senescence

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EDITORIAL

The Musculoskeletal System (MSK) is the structural underpinning for the body and allows mobility. It includes bone, cartilaginous tissues, skeletal muscle, tendon, ligament, intervertebral disc, and others. Ligaments help to keep bones in place. The movement of bones is aided by skeletal muscles. Tendons connect the muscles to the bones. The articulating joints are made up of these tissues. MSK tissues perform endocrine roles and communicate with other tissues to alter organism homeostasis and general physiological health, in addition to providing physical support. The skeleton is a highly dynamic structure that evolves over time in terms of shape and composition. These three illnesses, which are common musculoskeletal problems, share genetic, endocrine, and mechanical risk factors, as well as being mechanically and metabolically linked. MSK structural deterioration, mechanical pain, decreased mobility, and limited function are all symptoms of these age-related musculoskeletal disorders. Cellular senescence was assumed to be an in vitro phenomena when it was first characterized by Hayflick in 1961 as restricted replicative capability of normal cultured human fibroblasts. Multiple groups have shown that these SnCs are abundant in vivo since the beginning of the twenty-first century. Cellular senescence is now viewed as a cell state triggered by stressful insults and certain physiologic processes, characterized by a prolonged—and generally irreversible—cell cycle arrest with secretory features, macromolecular damage, and altered metabolism, according to the International Cell Senescence Association's consensus. The SASP allows SnCs to influence their local tissue surroundings. Importantly, removing SnCs from adult mice resulted in significant gains in health and lifespan. Our understanding of the phenotypic traits and pathophysiology of SnCs, as well as their contributions to physiologic and pathologic processes, has advanced thanks to the recent emergence of senescence reporter/ablation mouse models and the introduction of senotherapies.

Despite its various features, irreversible cell cycle halt is a defining feature of cellular senescence. Earlier research into the mechanics of replicative senescence, which causes stable cell cycle arrest, discovered that telomere erosion following significant serial passaging produces replicative senescence in human cells. Senescence can also occur without telomere shortening, and it can be triggered by genotoxic agents such as reactive oxygen species, DNA-damaging agents, hypoxia, mitochondrial malfunction, certain activated oncogenes, and epigenetic changes. The growth stop of SnCs is caused by two tumour suppressor-mediated signalling pathways: p53/p21CIP1 and p16INK4a/pRB. After DNA double-strand breaks or uncapped telomeres cause DNA damage, the p53/p21CIP1 pathway is initiated. The p16INK4a/pRB signalling pathway is responsible for initiating and maintaining persistent cell cycle arrest. Polycomb repressive complexes generally mute the INK4A/ARF locus epigenetically. When the activities of polycomb repressive complexes are disrupted, p16INK4a is derepressed and senescence is induced. The growth plate at the ends of long bones, as well as the adjacent primary spongiosa, undergo significant modifications at the cellular level during late puberty in order to adapt to the considerably slower bone growth/accrual during this period. During the rapid bone-growth period, vascular endothelial cells that form invading blood arteries and mesenchymal stem/progenitor cells that replenish bone-forming osteoblasts are highly proliferative, although these cells likely stop proliferating or are replaced by other cell types. At this age, we discovered a cellular senescence linked to the halt of bone development.

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