Biography

Dr. Judith Campisi received a PhD in Biochemistry from the State University New York at Stony Brook and completed postdoctoral training at the Harvard Medical School. As an assistant professor at the Boston University Medical School, she became interested in the control of cellular senescence and its role in tumor suppression and aging. She joined the Lawrence Berkeley National Laboratory as a Senior Scientist in 1991. She established a second laboratory at the Buck Institute in 2002. At both institutions, she established a broad program to understand various aspects of aging, with an emphasis on the interface between cancer and aging. The Campisi laboratory has made several pioneering discoveries in these areas, and her research continues to challenge and alter existing paradigms. In recognition of the quality of her research and leadership in the field, she has received numerous awards, including two MERIT awards from the US National Institute on Aging, and awards from the AlliedSignal Corporation, Gerontological Society of America, American Federation for Aging Research, and, most recently, the Longevity prize from the IPSEN Foundation. She currently serves on numerous national and international editorial and advisory boards.

Abstract

“Cancer and aging: Rival demons?”

Aging causes a decline in the structure and function of most, if not all, mammalian tissues. The prime causes of aging remain debatable, but one process has been gaining increasing credibility as major driver of aging phenotypes and pathologies: the complex cellular stress response and potent tumor suppressive mechanism known as cellular senescence. Cells undergo a senescence response upon many challenges, including genomic or epigenomic damage, activation of an oncogene, and metabolic deficits such as mitochondrial dysfunction. Senescent cells increase with age in many tissues, and acquire three main phenotypes: 1) an essentially irreversible arrest of cell proliferation; 2) a multi-component senescence-associated secretory phenotype (SASP); and 3) resistance to cell death. The SASP includes numerous pro-inflammatory cytokines and chemokines, and is thought to drive aging phenotypes and pathologies by creating local and systemic chronic inflammation. Recent findings using mouse models in which senescent cells can be selectively eliminated support the idea that senescent cells, and particularly the SASP, are indeed important causes of aging. On the other hand, senescent cells can be beneficial in certain physiological contexts. These contexts include certain stages of embryogenesis and instances of tissue remodeling and repair. Thus, the senescence response is likely an evolutionary trade-off between optimizing fitness during young ages, but driving pathology at later ages. In order to harness the beneficial effects of eliminating senescent cells, which will likely be possible by pharmacological approaches, it will be important to understand both the beneficial and deleterious effects of these cells.