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Physiologic changes of the musculoskeletal system with aging

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Description

Aging is one of the important challenges of modern society. Advanced adult age is associated with changes in many physiologic systems. Particular interest is the musculoskeletal system because it directly contributes to mobility and functional independence. Skeletal muscle mass and strength decline with age. These changes are mostly due to a reduction in the number of muscle fibers and cellular and molecular changes that reduce the force-generation process. Bone mass and architecture are compromised and may result in fractures. Tendons and ligaments undergo significant biochemical alterations that directly compromise their biomechanical function.

Neuropsychological Testing

To examine the effect of an aging environment on Sister Chromatid Exchange (SCE) induction, Ehrlich Ascites Tumor (EAT) cells were introduced into young and old C57BL/6J mice. Background SCE levels were not significantly different in either EAT cell or normal bone marrow cell populations between young and old animals. Despite a decline in SCE induction in bone marrow cells in older mice at high mitomycin C concentrations, SCE induction in EAT cells was not significantly affected by the age of the animal. These findings suggest that the aging environment may not play a major role in the diminished SCE induction observed in old cell populations. This is a brief review of some literature on practice effects and age on neuropsychological testing. Research suggests that younger subjects show greater improvement when retested on intelligence tests than older persons. The implication is that the effects of neuropsychological practice may vary with the age of the person assessed. Suggestions for further research are discussed.

Survival of human lymphocytes after X-ray and ultraviolet irradiation was studied in individuals of various ages using a technique of cloning in soft agar. There was no effect of age on survival after ultraviolet irradiation but lymphocytes from elderly individuals were approximately twice as sensitive to X-radiation as lymphocytes from young individuals. The results are relevant to the possible importance of DNA damage and repair in cellular ageing.

The geroscience hypothesis states that targeting fundamental aging processes will delay, prevent, alleviate, or reverse multiple geriatric syndromes, chronic diseases, and loss of resilience. In other words, by mitigating the aging process, it should be possible to delay not just one but most chronic diseases affecting the elderly. In linking aging to chronic disease and multimorbidity,1 there is a strong rationale for targeting aging to treat the conditions of older age. There may also be substantial health and economic returns with delayed aging.2

Aging-related cellular and molecular changes are generally thought to be caused by 1 or more of a few deteriorative mechanisms, including chronic inflammation, cellular senescence, damaged macromolecules, and progenitor cell dysfunction.3 These fundamental aging processes result in a plethora of pathophysiological alterations that are the basis for organ system declines over time. These pathophysiological alterations include tissue atrophy/loss in many tissues, hypertrophy in a select few tissues (e.g., cardiac ventricles), denervation, decreased tissue perfusion, decreased responsiveness to external stimuli/challenges, decreased regenerative responses to injury, impaired induction of cytoprotective pathways, fibrosis, decreased cell turnover, fatty infiltration of tissues, and changes in homeostatic set points.4 The organismal manifestations of these alterations are reported clinically as decreased resilience, frailty, increased vulnerability, functional decline, and multimorbidity.

As yet, there are limitations and great complexity to studying human aging physiology,4 including confounders such as concomitant disease, as well as the role of environmental factors and interactions with the physical world. Each organ system has its own trajectory for age-related dysfunction with system-specific declines. Furthermore, aging physiology in humans is heterogeneous and interdependent with other systems and extrinsic influences. Superimposed on these considerations are factors that regulate resilient responses to stressors and lessen potential adverse physiological consequences.

Over the past several years, a renaissance has occurred in the understanding of fundamental aging processes, especially with respect to emerging perspectives on cell senescence. In 1965, Hayflick and colleagues described replicative (cell) senescence in vitro as a reflection of aging in vivo and put forward the idea that proliferative exhaustion contributes to the aging phenomenon.5 Later, Cristofalo and others6,7 reported that the lifespan of cells can be predicted and that cell cycle arrest in senescence occurs at the Gap1/DNA synthesis checkpoint. The ideas that senescent cells are not quiescent cells and that cell senescence manifests as a failure to respond to environmental cues were promulgated.6,7 Campisi suggested that cell senescence is a defense mechanism against unchecked proliferation and neoplastic transformation8 and, more recently, that the deleterious effects of senescence are mediated by an inflammatory secretome, the senescence-associated secretory phenotype.9 In 2015, Kirkland and colleagues10 published a landmark article describing cell senescence as a precancerous state with extreme resistance to apoptosis. Kirkland and others11,12 have reported that senescent cells can be cleared genetically by exploiting checkpoint arrest or pharmacologically by targeting apoptosis pathways. The latter observation has made cell senescence (and potentially aging) a druggable target. Another notable advance in the biology of aging, also related to cell senescence, was the recognition that shortening of telomeres (and dysfunction of the telomere protein caps that safeguard the ends of chromosomes) promoted aging; in 2009, the Nobel Prize in Physiology or Medicine was awarded to a group of scientists for the discovery of telomeres, the enzyme telomerase, and their significance. Many of the behavioral changes that promote longevity—exercise, diet, weight reduction, and mitigation of stress—are associated with the preservation of telomere length.

Advances in aging research as exemplified by the recent strides made in understanding the roles of cell senescence in aging and disease, as well as the potential for clinical translation of these findings, have sparked renewed appreciation for aspects of geriatric medicine, including the focus on independence and function, altered presentations, geriatric syndromes, and the challenges of caring for complex patients with multimorbidity and acute-on-chronic conditions.

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