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Molecular Mechanisms Underlying an Aging Skeleton

Guest Editor:

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Deadline for manuscript submissions:
closed (31 August 2021)

Message from the Guest Editor

Bone is a dynamic organ that is under a balanced homeostasis during normal conditions. Aging, menopause in women, and several diseases tilt this balance, resulting in bone loss. Since bone is also an endocrine organ, changes in the bone environment may cause systemic changes. DNA damage, apoptosis, and cellular senescence together with a pro-inflammatory secretome drive age-related osteoporosis during normal physiological and pathological conditions. Molecular signatures that regulate these pathways in bone, and the autocrine, paracrine, or endocrine secretions that work locally and systemically are still being explored. This Special Issue will encompass studies that help us to understand these molecular signatures, focusing on cell fates to understand age- and disease-related bone loss. Several cellular events such as telomere shortening, epigenetic changes, proteostasis, mitochondrial dysfunction, and extracellular and intranuclear chromatin remodeling are all linked to cellular senescence. Research articles or reviews exploring the roles of these cellular mechanisms are solicited to understand the pathophysiology of an aging or diseased bone.

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Editor-in-Chief

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Message from the Editor-in-Chief

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