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# Alterations of the Cell Nucleus as a Driver of Chronic Inflammation in Aging

Guest Editor:

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Deadline for manuscript submissions:
**closed (1 December 2022)**

## Message from the Guest Editor

Dear Colleagues,

Changes to the nucleus homeostasis can occur due to exposure to oxidative stress; agents causing chromatin disruptions; and DNA damage resulting from losing repair factors or mutations to genes encoding nuclear proteins including nuclear envelope proteins, lamina components, chromatin remodeling, protein kinase, phosphatase, or other modifiers. These modifications in the nucleus can drive cells to enter permanent cell cycle arrest and senescence. Senescent cells, beside exhibiting nuclear, cytoplasmic, and morphological changes, are also known to produce the senescence-associated secretary factors that include proinflammatory factors. These molecules can propagate to neighboring cells and cause them to enter senescence and secrete similar sets of proinflammatory molecules. This senescence cycle can eventually affect the entire tissue or organ where the senescent cells reside and cause tissue aging, organ dysfunction, and the development of the tissue- or organ-related pathology. This Special issue seeks to expand our understanding of the mechanisms that are at play in the setting of this cycle of chronic inflammation and starts from the nucleus.

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