

## Special Issue

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

### Message from the Guest Editor

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 secretory 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.

### Guest Editor

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### Deadline for manuscript submissions

closed (1 December 2022)



## Cells

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*Cells* has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. *Cells* encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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