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Interleukin-1 and interleukin-18 as mediators of inflammation and the aging process

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Oxidative stress and low-grade inflammation are hallmarks of aging process as mitochondrial dysfunction and oxidative stress induce multi-protein inflammatory complexes called inflammasomes. Nod like receptor protein 3 (NLRP3) major immune sensors for cellular stress signals, e.g. reactive oxygen species cathepsin B NLRP3 activation triggers caspase1 mediated maturation of IL-1b and IL-18. IL-1b and IL-18 are of the same family closely related similar 3-dimensional structure their precursors are inactive until cleaved by intracellular cysteine protease caspase. NLRP3 gene control activity of caspase1 as NLRP3 gene participate in conversion of procaspase-1 to active caspase1 so mutation in NLRP3 gene will affect IL-1b and IL-18 secretion during aging. Autophagic clearance of mitochondria declines and dysfunctional mitochondria provoke oxidative stress disturb cellular redox balance. During aging increased NF-Kb potentiate expression of NLRP3 and cytokines inflammasomes play role in proinflammatory phenotype during aging process and IL-1b and IL-18 participate in fundamental inflammatory process that increased during aging process.

Biography

Dermatologist and cosmetologist (Elsheikh Zaied Aal Nahyan Hospital), Member of AAD (American Academy of Dermatology and Cosmetology), Laser specialist (National Institute of Laser Science Cairo University) Graduated from kasr al-ainy faculty of medicine Cairo University.

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