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Miriam Capri, Ph.D
DIMES-Department of Experimental, Diagnostic and Specialty Medicine
CSR-Centro di Studio e Ricerca sull'Invecchiamento
CIG- Interdepartmental Centre "L.Galvani" for Bioinformatics, Biophysics and Biocomplexity
ALMA MATER STUDIORUM, University of Bologna, Bologna, Italy

Human ageing and microRNAs: the contribution of muscle tissue

Human aging is a lifelong process characterised by a continuum of response and adaptation to different type of stressors, both endogenous and exogenous, and longevity phenotype represents the best example of adaptation and remodelling. Thus, centenarians or super-centenarians and their offspring are the best model to investigate the protective factors and biomarkers likely predictive of healthy status. In fact, centenarians live 20-30 years more than their demographic cohort of birth and have avoided or postponed all the age-related diseases, suggesting a "decelerate" ageing process. Similarly, centenarians' offspring show a lower risk to undergo major age-related diseases, (cardio- and cerebral-vascular diseases, type 2 diabetes and cancer) in comparison with individuals of the same age but without long lifespan parents. On the other side, age-related diseases represent an "accelerate" ageing and the inflammatory status characterizing ageing, i.e. *inflammageing (1)*, is the common molecular contributor to all the age-related pathologies. All cells, organs and tissues can contribute to *inflammageing*, but skeletal muscle seems to have a major relevance due to its weight (about 40% of the body) and its metabolic, hormonal contribution. To this regard, the age-related muscle alteration (for example the increase of intra-fiber lipid droplets) and sarcopenia may have profound systemic effects. Recently, tissue-specific and circulating microRNAs (miRs) have been highlighted as relevant epigenetic modifications which affect not only tissue-miRs expression but also circulating miRs. Circulating and extracellular vesicles -shuttled miRs can be up-taken by cells far from the site of origin, thus communication among tissues and organs or cells may be modified during aging process. Skeletal muscle can largely contribute to these changes and ageing process may propagate (2) through tissue-specific miRs, such as miR-206 or other pro-inflammatory miRs.

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