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Review

Age-related degeneration of articular cartilage in the pathogenesis of osteoarthritis: molecular markers of senescent chondrocytes

Giuseppe Musumeci[1], Marta Anna Szychlinska[1] and Ali Mobasheri[2,3,4]

[1]Department of Bio-medical Sciences, Human Anatomy and Histology Section, School of Medicine, University of Catania, Catania, Italy, [2]School of Veterinary Medicine, Faculty of Health and Medical Sciences, University of Surrey, Duke of Kent Building, Guildford, Surrey, United Kingdom, [3]Medical Research Council and Arthritis Research UK Centre for Musculoskeletal Ageing Research, Arthritis Research UK Centre for Sport, Exercise and Osteoarthritis, Arthritis Research UK Pain Centre, University of Nottingham, Queen's Medical Centre, Nottingham, United Kingdom and [4]Center of Excellence in Genomic Medicine Research (CEGMR), King Fahd Medical Research Center (KFMRC), King AbdulAziz University, Jeddah,Kingdom of Saudi Arabia

Offprint requests to: Dr. G. Musumeci Ph.D., Department of Bio-medical Sciences, Human Anatomy and Histology Section, School of Medicine, University of Catania, Via S. Sofia 87, 95125 Catania, Italy. e-mail: g.musumeci@unict.it

Summary. Aging is a natural process by which every single living organism approaches its twilight of existence in a natural way. However, aging is also linked to the pathogenesis of a number of complex diseases. This is the case for osteoarthritis (OA), where age is considered to be a major risk factor of this important and increasingly common joint disorder. Half of the world's population, aged 65 and older, suffers from OA. Although the relationship between the development of OA and aging has not yet been completely understood, it is thought that age-related changes correlate with other risk factors. The most prominent hypothesis linking aging and OA is that chondrocytes undergo premature aging due to several factors, such as excessive mechanical load or oxidative stress, which induce the so called “stress-induced senescent state”, which is ultimately responsible for the onset of OA. This review focuses on molecular markers and mechanisms implicated in chondrocyte aging and the pathogenesis of OA. We discuss the most important age-related morphological and biological changes that affect articular cartilage and chondrocytes. We also identify the main senescence markers that may be used to recognize molecular alterations in the extracellular matrix of cartilage as related to senescence. Since the aging process is strongly associated with the onset of osteoarthritis, we believe that strategies aimed at preventing chondrocyte senescence, as well as the identification of new increasingly sensitive senescent markers, could have a positive impact on the development of new therapies for this severe disease. **Histol Histopathol 30, 1-12 (2015)**

Key words: Osteoarthritis, Aging, Cartilage, Chondrocyte, Lubricin, Senescence, Senescence markers

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