

LETTERS TO THE EDITOR

Osteoarthritis' shifting paradigm – are we evolving?

Parente H¹, Tavares-Costa J¹, Teixeira F¹

Dear Editor,

To understand and act upon Osteoarthritis (OA) means being responsible and proactive about one of the most remarkable public health topics of today's society. This is a debilitating disease and a major source of pain and articular deformity, which affects 32.5 million adults on US only¹ – OA is the most common type of arthritis. It correlates with a heavy social and economic burden, explained by its chronic evolution, and subsequent therapeutic and diagnostic adjustments, and also by its contribution to absenteeism rates and loss of work productivity. Estimates on the US show total costs of \$136 billion, absenteeism costs of \$10.3 billion and an average direct cost of \$11.000 person/year¹.

Historically, OA is recognized as an inevitable condition – a fatality. By means of continuous articular abrasion due to chronic overload phenomena and altered biomechanics, we have been repeatedly attributing it an inexorable progression, pair and twin of the also unavoidable aging process. Over the years, a narrow sight of a disease has been built with countless façades, relating it only with irresolvable ends. From this point of view, almost only contemplative, of a pathology merely with a local and mechanical behaviour that will come no matter what, some scientific stagnation has fallen upon us, which urges to be shaken. It is vital to break the chain of populational (and even scientific) transmission that OA is a universal doom. According to latest developments², some fatalistic notions of OA's physiopathology have been demystified. Discredit yourselves of the exclusivity of the wear and tear apparatus! OA has a systemic and inflammatory basis that strikes not just cartilage, but several other structures; it is a complete organ failure – the joint, which becomes inefficient in absorbing and distributing the weight through itself. An organ that bypasses the generalist notion of the noblest ones (like the heart and brain), but which endows us of a significant part of our daily functionality and, subsequently, enables us a greater and fuller quality of life. Hence, maybe it deserves a comparable relevance with that given to other organ failures, such as heart failure and renal failure.

Interpreting OA as a set of distinct causalities with a common destination is essential to its better categorization. There is not just one OA, there are several. In the phenotypic grouping of this syndrome³, lies the rationale for new and better therapeutic targets. Moreover, recognizing the modifiable risk factors⁴ allows us to refine preventive measures that are just as or more important than therapeutic weapons, as a way to ease its so haunting prevalence and incidence rates.

Thus, we know today that environmental factors related to the degrading lifestyle changes, such as physical inactivity, maladjusted diet, obesity and metabolic syndrome, are implied on the genesis of the osteoarthritic phenomena, by its contribution to the systemic low-grade inflammation that we call meta-inflammation^{5,6}. Together with its analogous local articular inflammation, mechano-inflammation⁷, both establish OA as an inflammatory disease, which pathophysiological mechanisms extend, by vast and heterogenous pathways, to the molecular and cellular detail, climaxing in articular catabolism predominance.

It is imperative to revive the faltering glow that has impaired the OA approach. To reckon OA as the silent epidemic that it is and instigate the irreplaceable multidisciplinary status and complementarity of health care provided by Rheumatology, Family Medicine, Physical Medicine and Rehabilitation and Orthopedics is of crucial significance. Early diagnosis is a current reality, based on clinical and imaging findings (with an excellent support by the emergent musculoskeletal ultrasonography), and if we push harder and break the stagnation in which we are emerged, targeted effective treatment may be just around the corner.

REFERENCES

1. United States Bone and Joint Initiative. The Burden of Musculoskeletal Diseases in the United States (BMUS). In: In. Fourth ed. Rosemont, IL. 2018: Available at <https://www.boneandjointburden.org/fourth-edition>. Accessed June 12, 2019.
2. Woodell-May JE, Sommerfeld SD. Role of Inflammation and the Immune System in the Progression of Osteoarthritis. *J Orthop Res.* 2020 Feb;38(2):253-257. doi: 10.1002/jor.24457. Epub 2019 Sep 12. PMID: 31469192.
3. Van Spil WE, Kubassova O, Boesen M, Bay-Jensen AC, Mobasheri A. Osteoarthritis phenotypes and novel therapeutic targets. *Biochem Pharmacol.* 2019 Jul;165:41-48. doi: 10.1016/j.bcp.2019.02.037. Epub 2019 Mar 1. PMID: 30831073.
4. Berenbaum F, Wallace IJ, Lieberman DE, Felson DT. Modern-day environmental factors in the pathogenesis of osteoarthritis. *Nat Rev Rheumatol.* 2018 Nov;14(11):674-681. doi: 10.1038/s41584-018-0073-x. PMID: 30209413.

¹ Reumatologia, Hospital Conde Bertiandos

Submitted: 26/01/2022

Accepted: 16/05/2022

Correspondence to: Hugo Parente

E-mail: hugoparente12@gmail.com

5. Ouchi, N.; Parker, J.L.; Lugus, J.J.; Walsh, K. Adipokines in inflammation and metabolic disease. *Nat. Rev. Immunol.* 2011, 11, 85
6. Gratal P, Lamuedra A, Medina JP, Bermejo-Álvarez I, Largo R, Herrero-Beaumont G, Mediero A. Purinergic System Signaling in Metainflammation-Associated Osteoarthritis. *Front Med (Lausanne)*. 2020 Aug 28;7:506. doi: 10.3389/fmed.2020.00506. PMID: 32984382; PMCID: PMC7485330.
7. Felson DT. Osteoarthritis as a disease of mechanics. *Osteoarthritis Cartilage*. 2013 Jan;21(1):10-5. doi: 10.1016/j.joca.2012.09.012. Epub 2012 Oct 4. PMID: 23041436; PMCID: PMC3538894.