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Editorial
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Why Orthopedic Surgeons Have to Rethink – Current Changes in Understanding Osteoarthritis and Implications for Rationale Treatment

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Editorial

As an orthopedic surgeon, surgical procedures for joint degeneration were my daily business and osteoarthritis was not a personal challenge. I could either patch initial cartilage lesions or replace severely destructed tissue by implanting the latest prosthesis. Patient education about the nature, progression and suitable therapies for osteoarthritis was truly simple. Neither patients nor physicians seriously had any doubt about the "tear and wear" – nature of OA. We as orthopedic surgeons could draw an illustrative comparison to a flat, worn tire with showing X-rays with bony deformation and a tiny, narrowed joint space. Apart from analgetic medication, I could not consider any reasonable pharmaceutical for treating this disease. A mechanical condition needs a mechanical solution, I thought. But I was disabused by modern life science.

As I began to engage in basic osteoarthritis research, I found a review with a colorful title which definitely changed my mind and my treatment algorithms for joint degeneration. "Osteoarthritis is not osteoarthrosis", written by Berenbaum [1], is an illustrative article about the inflammatory nature of OA and its implications for a rationale approach to treatment. Berenbaums [1] review is just one example for the demanding and fascinating research about the principles of joint degeneration. A variety of studies about the underlying molecular mechanisms using current techniques in biomedicine have been published during the last years [2,3].

The number of interesting studies, reports and discoveries increases every week, adding a new small block to the versatile entity of osteoarthritis. Like in other areas of clinical medicine, sustainable therapies require a fundamental and broad knowledge of disease mechanism. The identification of TNF-alpha or IL-1beta as deteriorating cytokines [4] and the antagonistic, anti-inflammatory effects of PDGF or HGF [5], for example, supported clinical investigations of growth-factors on joint inflammation and could successfully be transferred to established therapies by intra-articular application of platelet-rich plasma formulations [6,7]. Signaling pathways as possible working points for pharmaceuticals provide new opportunities. By exploring the proteome of the OA triad cartilage, subchondral bone and synovial tissue, serum and synovial fluid biomarkers may help to monitor disease progress and severity beyond radiographs or MR-imaging [8,9].

Known for generations, but misunderstood until the turn of the millennium: cyclic, moderate exercise is beneficial in OA therapy. Researches could identify mechanoreceptors regulation cartilage metabolism [10] and hereby help us to apprehend OA as a complex, molecular disease entity.

So, what does it mean for us orthopedic surgeons? Trade scalpel and saw against immunoassays and cytokine-compositions?

Not at al. There will always be a role for surgical techniques in OA treatment. Identifying Crohn's disease as an inflammatory process, did not supersede operative procedures, but led to a differentiated treatment regime.

We orthopedic surgeons have to take advantage of modern OA research. A profound understanding may help us to adapt and revise existing procedure and improve surgical outcome. By affecting the inflammatory milieu in OA joints and enhance the intra articular environment, autologous chondrocyte transplantation could succeed despite today's disappointing results in advanced disease [11]. By addressing altered tissue also joint replacement may benefit from contemporary non-surgical research efforts by enabling better implant endurance and less postoperative complications. Thus we can use this fundamental research to achieve better results,

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thereby, to get better surgeons, and in the end, to get pleased and healthy patients.

Let's rethink and augment our inventory with these demanding and fascinating findings without dropping our knives.

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