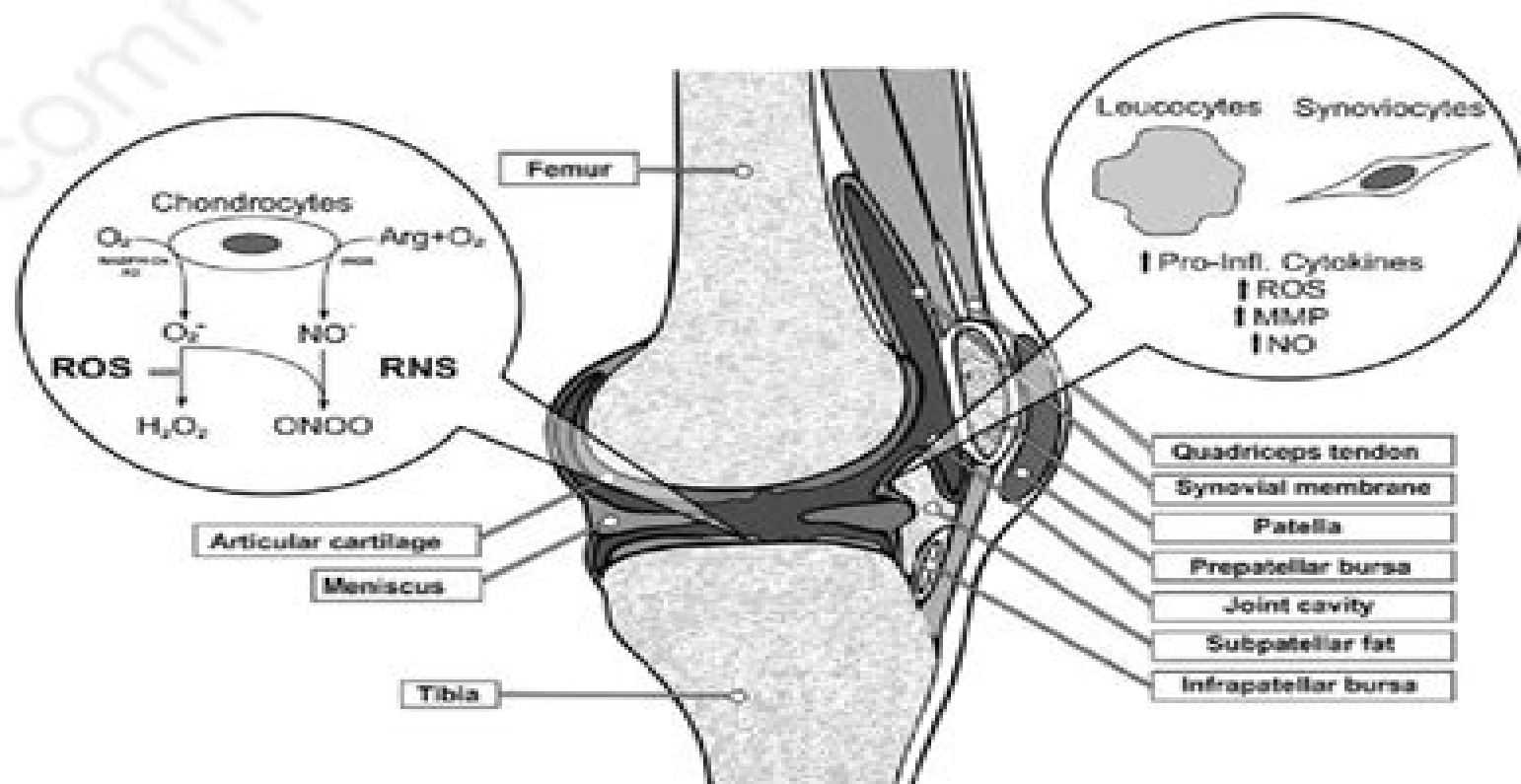


use the sampling average presuppose which were math- after BCA assay. In that the sampling for the decreased by ELISA for both, ive, biomarkers. measurements of be compared to a he future to illus- tential. However, osine in synovial resting biomarker associated joint disor- ticular sensitivity

obtained by meas- in human serum. s reported about concentrations of 3- in OA patients ects,³⁰⁻³² the pres- hand much lower ther hand not dif- fered groups. Within erent established thresholds, our with findings by were additionally easurable concen- RA were signifi- cants with OA.³³ e to support our biomarker for osedly originated to greater concen-

oxidation in OA patients compared to a healthy control group and RA patients, independently from a rise in NO production as a source for nitrosative stress.³⁵ Based on the significant correlation between the histopathological synovitis and oxidative stress ($r=0.728$, $P<0.01$), we suggest a considerable pathophysiological impact of the processes in the synovial membrane on the pro-inflammatory environment. Therefore, it seems reasonable that next to the cartilage and bone tissue also the synovial membrane plays a key role in the pathophysiological formation of OA (Figure 1).^{16,36,37}

as anti-inflammatory drug intake of the patient, the small sample size or different applied laboratory methods, could be responsible for the low correspondence between our current and former findings in literature. Summarized, this study was able to show an indirect increase of oxidative stress in all three investigated groups which was evident in synovial biopsies, synovial fluid and human serum. Consequently, enhanced non-enzymatically oxidative processes induced by ROS seem to take an important part in formation of chronic diseases (OA, RA) as well as in acute lesions



Pathology Of Oxygen

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