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Lubricin, the basis of faulty biolubrication and immunological consequences

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Biolubrication is key for sustaining the mobility of joints. The consequence of faulty biolubrication is pronounced in pathological conditions such as osteoarthritis (OA) and rheumatoid arthritis (RA), where degradation of the joint is exacerbated by defect of the lubricating superficial layer on the cartilage. We have identified how a lubricating glycoprotein (lubricin) in the synovial fluid associates with joint surfaces using proteomic techniques and mass spectrometric identification of lubricin associated protein macrocomplexes in synovial fluid. Our data suggest that part of lubricin becomes covalently linked to proteins in the extracellular matrix (ECM) of joint tissue. This explains how lubricin can provide efficient lubrication even under high stress conditions in a healthy joint. The association of lubricin to joint surfaces allows oligosaccharides attached to lubricin to generate a friction free joint surface. In pathological conditions such as RA, the association of lubricin to joint surfaces is lost. Instead, lubricin will interact with synovial neutrophils accumulated in the synovial fluid due to the inflammation. We hypothesize that lubricin glycosylation, while necessary for the biolubrication, is responsible for inappropriate activation of synovial neutrophils present in high amounts in RA synovial fluid. This is partly due to binding to the subpopulation of L-selectin positive neutrophils in the synovial fluid with sialylated and sulfated oligosaccharides present on lubricin. The data suggest that the mechanisms for localization of surface active biolubricating molecules to synovial surfaces provide insight into transformation from a healthy state to pathological state.

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