= 3482) vs. rofecoxib (n = 6290); and, 1.31 (0.86, 2.01) when comparing non-naproxen NSAIDs or placebo (n = 6017) vs. rofecoxib (n = 7675).

Conclusion (1) The risk of sustaining a thrombotic cardiovascular event was similar in patients treated with rofecoxib, placebo, or non-selective NSAIDs without sustained effects on platelet function and, (2) the risk of sustaining a thrombotic cardiovascular event was reduced in patients treated with naproxen compared to rofecoxib. This reduction in events on naproxen is likely due to its ability to maintain near complete inhibition of platelet function throughout its dosing interval.

SAT0047 PURINE METABOLISM AND ANTIOXIDANT SYSTEM IN OSTEOARTHRITIS

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Background Till now there are few of information on a Purine metabolism (PM) and Antioxidant system (AS) condition of blood of osteoarthritis (OA) patients.

Objectives To study the enzymatic profile of PM and AS in blood of OA patients.

Methods The activities of Xanthine Oxidase (XO), Xanthine dehydrogenase (XDG), 5'-Nucleotidase (5'-NT), Adenosindesaminase (ADA), Guanindesaminase (Guanase), Superoxid-dismutase (SOD), Glutathion reductase (GP), the contents of Uric acid (UA) were measured with Spectrophotometer. XDG, ADA, SOD isoenzymes were examined with method of electrophoresis in agarose gel and obtained enzymograms were evaluated at Densitometry.

Results 44 patients with OA and 30 healthy controls (HC) were under observation. The mean age of patients – 53 ± 2.6 years. The mean duration of disease – 10.2 ± 2.7 years. We revealed the increase of XO, ADA, Guanasa, SOD, GP, GR activity, XDG-2, SOD-1 isoenzymes, contents of UA in OA patients with synovitis in comparison with HC. The increase of XDG, SOD, GP activity, SOD-1 isoenzymes were observed in OA patients without synovitis in comparison with HC and the decrease of UA, XDG-2 isoenzymes, also the increase of XDG, GP activity were revealed in comparison with OA patients with synovitis.

Conclusion PM and AS are essentially changed in OA. These changes depended on the clinical form of OA. The enzyme indices can be used for early activity of disease diagnostic and differentiation of OA clinical form.

SAT0048 A NEW ACID ATTACK AND CATHEPSIN K MEDIATED OSTEOLYTIC MECHANISM RECOGNISED AROUND LOOSENING THR IMPLANTS

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Background Normal bone remodelling and pathological bone destruction have been considered to be osteoclast-driven. Osteoclasts are able to attach to bare bone surface and produce an acidic subcellular compartment. This leads to acid dissolution of hydroxyapatite allowing osteoclast-derived cathepsin K to degrade the organic type I collagen-rich osteoid matrix.

Objectives We were interested to find out the pH prevailing in the interface membrane, which develops around aseptically loosening total hip replacement (THR) implants. Our hypothesis was, that this hypovascular tissue, containing activated macrophages and subjected to ischemia-reperfusion, with an avascular implant embedded in it, is acidic. This could lead to demineralization and degradation of the demineralized bone matrix by acidic endoproteases.

Methods We used a special sting electrode for peroperative pH measurements during revision THR operations performed for loosening. This electrode was sterilised using hydrogen peroxide plasma produced in a vacuum chamber and an electrical field generated using radiowave frequencies. Demineralization was assessed using Villanueva bone mineral stain analysed using confocal laser scanning microscopy. mRNA was isolated from interface tissue and measured using a quantitative RT-PCR method. Finally, cathepsin K enzyme protein was stained using biotin-streptavidin-peroxidase method and a staining robot and in double staining using manual double immunofluorescence method.

Results The pH at the gluteus medius control measure point was 7.39 ± 0.05 . The value recorded at the interface tissue facing the periprosthetic bone was 6.1 ± 0.2 ($p < 0.001$). Confocal laser scanning of the adjacent bone disclosed high proportions of non-/red ($p < 0.01$) and low-mineralized/yellow ($p < 0.01$) bone compared to mature, mineralized bone/green around well-fixed, but mechanically broken cups. Cathepsin K mRNA copy number per thousand beta-actin copies was 1875 ± 302 in interface compared to 268 ± 61 in control samples ($p < 0.001$). Interface tissue contained many cathepsin K enzyme protein positive cells, more than 95% of them being mononuclear, TRAP-positive cells of the monocyte/macrophage lineage. Furthermore, extracts of periprosthetic tissues contained both the 42 kD pro- and the 27 kD activated-form of the cathepsin K enzyme, whereas control samples contained only the 42 kD proenzyme.

Conclusion These observations suggest that the low pH prevailing in the interface around loosening THR implants leads to demineralization of the periprosthetic bone. Under these acidic conditions, cathepsin K mRNA and enzyme protein production are induced in mononuclear cells. These cells release cathepsin K to the extracellular space, where it undergoes acid-induced autocatalytic activation and degrades demineralized periprosthetic bone matrix. These observations have therapeutic implications, because cathepsin K inhibitors are being developed for the treatment of metabolic bone diseases like osteoporosis.

SAT0049 ANTERIOR KNEE PAIN AND HYPERLAXITY

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Background Patellar malalignment is one of the most important reason of anterior knee pain and is determined by Q angle measurement. Q angle is defined as the angle between quadriceps muscle and patellar tendon. It is approximately 13 degrees in male and 18 degrees in female.

Objectives The first aim of this study is to determine Q angle differences in supine and upright position. The second aim is to evaluate Q angle differences in person who have hyperlaxity compared to healthy ones.