Biological markers of bone breakdown predict pain and pain change in osteoarthritis

Nwosu LN, Allen M, Wyatt L, Huebner JL, Chapman V, Walsh DA, Kraus VB.
Pain prediction by serum biomarkers of bone turnover in people with knee osteoarthritis: an observational study of TRAcP5b and cathepsin K in OA
http://doi.org/10.1016/j.joca.2017.01.002

Background

One of the challenges in osteoarthritis (OA) is trying to understand the cause of pain, how it originates and the mechanisms involved in its progression. OA affects not only the cartilage, but also other tissues within the joint including bone. Changes within the bone are key to the development of OA whilst also linked to knee pain. Osteoclasts are cells responsible for the breakdown of bone and in OA the activity of osteoclasts are increased. Osteoclasts release biological molecules, cathepsin K and TRAcp5b which aid in bone breakdown.

Aim

We aimed to study cathepsin K and TRAcp5b in the blood of people with OA to investigate the association of these molecules with bone breakdown and changes in knee pain.

How the study was carried out

Levels of bone breakdown markers were measured in blood collected from participants with knee OA from the Prediction of Osteoarthritis Progression (POP) study group. Clinical data including pain symptoms were also recorded in these participants at the beginning of the study and again after 3 years. Knee tissue donated after total knee replacement from participants who sought help for knee pain and at post mortem from participants who did not seek help for knee pain (Arthritis Research UK (ARUK) Pain Centre joint tissue repository) were used to measure the amount of osteoclasts involved in bone breakdown.

What the study found

We found that osteoclasts were higher in people who had painful OA compared to those that did not. The biomarker TRAcP5b measured in blood was linked to osteoclast density, changes in the bone and pain in OA. Interestingly, it was able to predict pain change in OA.

Significance of the study to Pain Centre's research

Our observations support a role for osteoclast activity within bone in generating and altering OA pain. This will aid in identifying people who might be particularly responsive to pain relief medication. It will also help discover the potential for anti-bone breakdown agents to modify OA.