Glucosamine in the treatment of osteoarthritis

Glucosamine trials are inconsistent, but to dismiss glucosamine as Roland and co-workers do in their article in Tidsskriftet no. 16/2007 (1), reveals the ignorance of pharmacoeconomics (the therapeutic application of nutritional biochemistry) that pervades conservative medical institutions.

Like every living tissue cartilage is constantly degraded and replaced, and changes in health or disease must be considered in terms of cartilage economics. During the first two decades of life weight-bearing cartilage plates thicken because cartilage formation exceeds wear. They achieve optimal thickness and then remain in dynamic equilibrium, with cartilage wear and replacement in balance. After 40, cartilage plates slowly thin during a pre-clinical osteoarthritis until clinical symptoms emerge.

With age we become catabolically dominant. New cartilage grows more slowly, while breakdown rates increase. Anabolic slowdown explains cartilage thinning and the accumulation of age-related biochemical changes in ground substance. We become catabolically dominant for lifestyle/nutritional reasons.

Due to historically low levels of physical activity and calorific throughputs, multiple micronutrient depletion is prevalent. Most people are depleted in most micro- and probably most phytonutrients (2). With age, calorific throughput falls further and dysnutrition worsens, with progressive depletion in vitamins and minerals (primarily anabolic co-factors), and phytonutrients like flavonoids (primarily anti-catabolic co-factors). With anabolic processes handicapped and catabolic processes (nitro-sylation, inflammation etc) unbraked, we become catabolically dominant.

Glucosamine is a precursor for glycosaminoglycans and proteoglycans in cartilage. As glucosamine availability is rate-determining for subsequent polymerisation, glucosamine supplements should affect cartilage formation. Despite negative meta-analyses, there are too many trials – and patients – with positive results to dismiss glucosamine. Its benefits (in responders) outstrip placebo time-course; and cartilage biochemistry explains the clinical trials’ inconsistencies.

Cartilage contains proteins like collagen; and protein synthesis is unaffected by glucosamine. Protein synthesis damped by dysnutrition can only be enhanced by relevant amino acids and co-factors, explaining the patchily positive results obtained with collagen extracts (3).

Hydrolysed collagen can only help when shortage of key amino acids is rate-limiting cartilage synthesis, and glucosamine can only be effective when glucosamine is rate-limiting. Using glucosamine or collagen as sole agents is irrational, because most patients are sufficiently nutritionally compromised to need both; together with co-factors required for matrix synthesis, and anti-catabolic agents such as flavonoids.

The effects of Mediterranean diet on morbidity including arthritis indicate what can be achieved with pharmacoeconomic support. Glucosamine is not particularly effective when used as monotherapy, but is an important element in any rational approach to osteoarthritis.

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Litteratur

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and medarbeidere svarer:
Vi ser ikke helt relevansen i Paul Claytons innlegg i forhold til vår kritikk av markedsføringen av glukosamin i behandling av artrose (1). Vi konstaterer også at Clayton ikke presenterer fakta som rokker ved vårt syn. Interesserte som går inn på Claytons hjemmeside, ser at han bl.a. samarbeider med Uni-Vite Healthcare Ltd. (2), som produserer et glukosaminholdig produkt kalt Nutrishield. På produktets nettsted informerer vi om at det er oppfunnet av – er det virkelig mulig? – Paul Clayton (3). Tidsskriftet står naturaligvis fritt til å trykke Claytons egne reklame. For egen del forundres vi over Claytons, Legemiddelverkets og diverse «ekspert» vilje – for ikke å si evne – til å ignorere data fra metaana-