Iodine, selenium, and joints: Kashin–Beck disease

In parts of China, Siberia, North Korea, and Tibet children and adolescents are prone to a strange joint disease called Kashin–Beck disease (named after those who described it in Siberia in the last century). It is a necrotising osteoarthropathy that affects the fingers, hands, elbows, ankles, and knees, usually starting at between 5 and 15 years of age. The regions in which it occurs have in common the fact that both iodine and selenium are in short supply. Now a study in Tibet (Rodrigo Moreno-Reyes and colleagues. New England Journal of Medicine 1998;339:1112–20; see also editorial by Robert D Utiger. Ibid, 1156–8) has suggested that this combined deficiency could be the cause of the problem.

They studied 575 5–15 year old children in 12 villages; only one village did not have endemic Kashin–Beck disease. Severe selenium deficiency was present in all the villages, mean serum concentrations being some 15–20% of the lower limit of normal. Almost half of the children studied had Kashin–Beck disease, a similar proportion were goitrous, and two thirds had very low urinary iodine concentrations. The findings that distinguished those with Kashin–Beck disease from those without were low urinary iodine, high serum thyrotropin, and low serum thyroxine binding globulin, but not serum selenium.

Selenium is an important component of two enzymes, glutathione peroxidase and iodothyronine deiodinase. It is postulated that combined deficiency of selenium and iodine could result in tissue oxidative damage because of lack of the first of these enzymes and this together with tissue thyroid hormone deficiency could produce joint damage. The hypothesis seems reasonable although it is as yet unproved.