Hotet från kadmium – en introduktion

Kadmium hotar att på bred front skada vår njurfunktion. Så här ligger det till.

Människokroppen har en förmåga att göra sig av med överskott på en rad ämnen. De kan lämna kroppen via gallan eller njurarna/urinen. Men skapelsen kunde inte förutse att tungmetallen kadmium skulle spridas av industrisamhället till den grad, att njurarna behövde avbörda också detta ämne.

Istället för att följa urinen ut så stannar en del i njuren, som med tiden får allt högre kadmiumhalter. De flesta människor idag har flera gånger högre kadmiumhalt i njurarna än våra förfäder. Denna har successivt ökat i njurarna eftersom kadmiumhalten i vår mat har ökat. Detta beror i sin tur på att halten i jordbruksmarken har ökat de senaste 100 åren. De stora källorna har varit *konstgödsel, avloppsslam, luftnedfall och kalk*. Dessutom finns en naturlig nivå i åkerjorden som kan variera från plats till plats beroende på vilken berggrund det är. Områden i Sverige med hög naturlig halt i jorden finns i Österlen, Östergötland, Västergötland och runt Storsjön i Jämtland.

Njurarna läcker äggviteämnen och kalk

Idag är kadmiumhalten så hög i våra livsmedel och njurar att njurskador börjar uppträda. Dessa ger sig till känna på två sätt, dels läcker njurarna vissa äggviteämnen och dels läcker de kalcium, dvs kroppen förlorar kalk och skelettet urkalkas.

Det finns vissa riskgrupper. Först och främst gäller det rökare som förutom genom maten får in kadmium med tobaksröken. En rökare har ungefär dubbelt så mycket kadmium i njurarna som en icke-rökare. En annan riskgrupp är alla kvinnor i barnafödande ålder. De förlorar järn vid sina menstruationer och kan få järnbrist. Då ökar kroppens förmåga att ta upp järn, men då tas också mer kadmium upp.

Kvinnor och vegetarianer utgör riskgrupper

En annan riskgrupp är vegetarianer. Generellt har animaliska livsmedel lägre kadmiumhalter än vegetabilier. Det kadmium våra husdjur får i sig försöker de då avbörda genom sina njurar – men det stannar där - medan köttet får lägre halter, liksom mjölk och ägg. Men man skall däremot inte alltför ofta äta njure. Vegetarianen som inte hämtar sin energi från animaliska livsmedel, måste i stället kompensera detta med ökat intag av vegetabilier och får på så sätt ett högre intag av kadmium. Man kan uppskatta att vegetarianer har ett ca 50 procent högre intag av kadmium än en vanlig blandkostare. En tredje riskgrupp är de som har ökad känslighet, t ex de med redan nedsatt njurfunktion etc.

I tabellen nedan anges hur många procent i riskgrupperna bland den svenska befolkningen som redan beräknas ha begynnande njurskada. Det normala intaget idag är 15 mikrogram per person och dag. Vi ser att 0,5 procent av rökarna beräknas ha njurskador. Denna grupp anges bestå av 2,2 milj personer. Då är det frågan om cirka 10.000 svenska rökare som beräknas ha skador.

Bland dem som har ökad känslighet beräknas ca 0,2 procent ha skador, medan storleken på gruppen är okänd. Gruppen kvinnor med låg järndepå uppskattas till 1 miljon och 1 procent av dem, dvs 10.000 beräknas ha njurskador.

Table 1. The frequency of cadmium induced tubular dysfunction in different groups in Sweden with two levels of average intake of cadmium with food, and the size of these groups

	Tubular dysfunction, %		
	15 µg Cd/day	30 µg Cd/day	Population in thousands
Average population, non-smokers	0	1	3,050
Smokers	0.5	2	2,200
Increased susceptibility	0.2	2	?
Women, low iron-stores	1	5	1,000
Women, low iron-stores and smokers			250
The general population	?	1.6	

Comments. The number of adult persons is assumed to be 6.25 millions. The size of the average population non-smokers (3.05 millions) equals 6.25 millions minus average population smokers (2.2 millions) minus non-smoking women with low iron-stores (1,0 million). Current and previous smokers are assumed to be 35% of 6.25 millions. The number of persons with increased susceptibility is unknown, thus here they are included in the average population non-smokers. Smoking women with low iron-stores are included in the group of smokers. The figures in the table concerning the frequency of cadmium induced tubular dysfunction interpreted from figure 10.2 in Järup et al. (1997).

(The Table 1 originates from *"The Economics of the Swedish Policy to Reduce Cadmium in Fertilisers"*, Lars Drake/Stefan Hellstrand, Oct 1997, KEMI, page 12)

Antalet skadade växer snabbt om kadmiumintaget ökar

Vidare ser vi att antalet skadade växer snabbt om intaget av kadmium ökar. Om detta fördubblas beräknas antalet skadade rökare fyrdubblas, de med ökad känslighet tiofaldigas och antalet skadade kvinnor med låga järndepåer femdubblas.

Idag beräknas 10 procent av befolkningen ha fördubblat intag (30 mikrogram per dygn), medan 3 procent beräknas ha ett tredubbelt intag (45 mikrogram per dygn). Mer om de medicinska aspekterna på kadmiumfrågan finns i följande artikel.

Supplement

Health effects of cadmium exposure -- a review of the literature and a risk estimate.

Järup L (editor), Berglund M, Elinder CG, Nordberg G, Vahter M

Scand J Work Environ Health 1998;24 suppl 1:1--51.

This report provides a review of the cadmium exposure situation in Sweden and updates the information on health risk assessment according to recent studies on the health effects of cadmium. The report focuses on the health effects of low cadmium doses and the identification of high-risk groups. The diet is the main source of cadmium exposure in the Swedish non-smoking general population. The average daily dietary intake is about 15 µg /day, but there are great individual variations due to differences in energy intake and dietary habits. It has been shown that a high fiber diet and a diet rich in shellfish increase the dietary cadmium intake substantially. Cadmium concentrations in agricultural soil and wheat have increased continuously during the last century. At present, soil cadmium concentrations increase by about 0.2% per year. Cadmium accumulates in the kidneys. Human kidney concentrations of cadmium have increased several fold during the last century. Cadmium in pig kidney has been shown to have increased by about 2% per year from 1984--1992. There is no tendency towards decreasing cadmium exposure among the general non-smoking population. The absorption of cadmium in the lungs is 10--50%, while the absorption in the gastrointestinal tract is only a few percent. Smokers have about 4--5 times higher blood cadmium concentrations (about 1.5 µg/l), and twice as high kidney cortex cadmium concentrations (about 20--30 µg/g wet weight) as non-smokers. Similarly, the blood cadmium concentrations are substantially elevated in persons with low body iron stores, indicating increased gastrointestinal absorption. About 10--40% of Swedish women of child-bearing age are reported to have empty iron stores (S-ferritin <12 µg/l). In general, women have higher concentrations of cadmium in blood, urine, and kidney than men. The population groups at highest risk are probably smokers, women with low body iron stores, and people habitually eating a diet rich in cadmium. According to current knowledge, renal tubular damage is probably the critical health effect of cadmium exposure, both in the general population and in occupationally exposed workers. Tubular damage may develop at much lower levels than previously estimated, as shown in this report. Data from several recent reports from different countries indicate that an average urinary cadmium excretion of 2.5 µg/g creatinine is related to an excess prevalence of renal tubular damage of 4%. An average urinary excretion of 2.5 µg/g creatinine corresponds to an average concentration of cadmium in renal cortex of 50 µg/g, which would be the result of long-term (decades) intake of 50 µg per day. When the critical concentrations for adverse effects due to cadmium accumulation are being evaluated, it is crucial to consider both the individual variation in kidney cadmium concentrations and the variations in sensitivity within the general population. Even if the population average kidney concentration is relatively low for the general population, a certain proportion will have values exceeding the concentration where renal tubular damage can occur. It can be estimated that, at the present average daily intake of cadmium in Sweden, about 1% of women with low body iron stores and smokers may experience adverse renal effects related to cadmium. If the average daily intake of cadmium would increase to 30 µg/day, about 1% of the entire population would have cadmium-induced tubular damage. In risk groups, for example, women with low iron stores, the percentage would be higher, up to 5%. Both human and animal studies indicate that skeletal damage (osteoporosis) may be a critical effect of cadmium exposure. We conclude, however, that the present evidence is not sufficient to permit such a conclusion for humans. We would like to stress, however, that osteoporosis is a very important public health problem worldwide, but especially in the Scandinavian countries. Studies assessing the potential role of cadmium as a risk factor for osteoporosis are already in progress. The result studies, including a limited number of lung cancer cases, and that the control of confounding was insufficient. Recent revaluation of these studies, as well as new data from a Swedish cohort, indicate that a classification of cadmium as a "probable human carcinogen, group 2A" would be more appropriate. This conclusion is also in agreement with the existing classification of some cadmium compounds in EC countries in the European Union (carcinogen category 2; annex 1 to directive 67/548/EEC). Although an increased risk for cardiovascular disease was found in a Japanese population with evidence of tubular damage, there is presently not sufficient evidence for cadmium as a risk factor for cardiovascular disease. Studies on experimental animals have indicated that a single high dose of cadmium can give rise to necrosis of the

testicles. Long-term, low-dose exposure to cadmium did not give rise to this effect, but may cause changes in male sex hormone levels in animals. Such effects have not been shown to occur in humans, but very few studies have been done on this topic. The available evidence does not allow any conclusions to be drawn about the effects on the male reproductive system at protracted low exposures. Elevated levels of cadmium in babies of smoking mothers have been associated with decreased birth weight. Since decreased birth weight was not observed in babies of mothers occupationally exposed to cadmium, the causal relationship is uncertain. However, pathological changes have been induced in human placentas perfused with cadmium after delivery. Since experimental animal studies have shown that cadmium may give rise to several adverse effects in ovaries and placentas and also to teratogenic and developmental effects, there is a need for more studies on humans. The present human evidence is not sufficient to consider female reproductive and developmental effects as critical effects in humans. In conclusion, recent data indicate that adverse health effects from cadmium exposure may develop in about 1% of the adult general population at an average daily intake of 30 µg over a life-span. In high-risk groups the percentage will be even higher (up to 5%). This intake is already exceeded by some population groups in Europe, and the margin is very narrow for large groups. Therefore, measures should be taken to reduce cadmium exposure in the general population to minimize the risk of adverse health effects. At an average daily intake of 70 µg/day [corresponding to the present PTWI (provisional torable weekly intake)], 7% of the adult general population would be expected to develop cadmium-induced kidney lesions. For high-risk groups the percentage would be even higher (up to 17%). Thus, in our opinion, the current PTWI is unacceptable and needs to be lowere.

Farliga och meningslösa gränsvärden

Det värsta med det smygande kadmiumproblemet är att det *saknas relevanta gränsvärden*. Enligt artikelns avslutning är författarna mycket oroade över det gällande provisoriska gränsvärdet vid 70 mikrogram per person och dag. Vi såg tidigare att redan 15 mikrogram per dygn ger skador hos riskgrupperna. Det felaktiga gränsvärdet kan i värsta fall leda till att hela 7 procent av vår befolkning får njurskador. Vi börjar då närma oss en miljon personer i Sverige.

Skulle relevanta gränsvärden komma till stånd, skulle sannolikt en del av dagens livsmedel behöva svartlistas. Hur jordbruksmarken skall användas för livsmedelsproduktion skulle påverkas, liksom innehållsdeklarationen på livsmedlens förpackningarna.