Swedish position on risks to human health posed by cadmium

Sweden strongly support the Commission’s proposal on regulation of cadmium content in fertilisers, which is further discussed in this paper.

The various assessments of risks conducted within the EU and internationally, underlying the Commission’s proposal, build on the scientific knowledge around 2008/09. At that point in time, kidneys were considered to be the most sensitive organ to cadmium exposure, and the effect levels for renal dysfunction correspondingly were used as the so called “endpoint” in the risk assessment for human health.

However, since then, much progress has been made in investigating various other adverse effects of cadmium at very low exposure, i.e. at levels found in the general population. Not least have several scientific studies found an association between low concentrations of cadmium in the urine (a measure of an individual’s body burden of cadmium) of the general population and increased risk of osteoporosis – a disease where decreased bone strength increases the risk of a broken bone. Osteoporosis is the most common reason for a broken bone among the elderly. The association has been found for both men and women that never have been smokers. In summary, these studies indicate that the critical adverse effect for human health, at low cadmium exposure through the diet, would be osteoporosis, and at even lower levels than those leading to renal dysfunctions.

The risks for effects on the cardio-vascular system have also been more thoroughly investigated in recent years. Associations have been found between blood cadmium and higher incidents for cardiovascular disease and mortality among never smokers. In other studies, correlations have been shown between cadmium exposure and increased risk of cancer, diabetes, mortality, reproductive toxicity and neurotoxicity.
Apart from the suffering of those who are affected, osteoporosis as well as cardio-vascular diseases and mortality entail high economic cost to society. In a recent report from the Swedish Chemicals Agency, it was calculated that the socio-economic cost of fractures caused by cadmium in food amounts to approximately 4.2 billion SEK (approx. 400 million Euros) per year in Sweden. The costs are health care costs in the short and long term, costs of lower quality of life and shortened life for those suffering from fractures, mostly the elderly. An important conclusion is therefore that increased intake of cadmium in food is likely to increase the socio-economic costs of osteoporosis and other diseases to which cadmium exposure contributes.

The existence of a vast number of studies showing adverse effects in the general public makes the risks posed by cadmium and cadmium compounds quite unique. Conclusions as regards risks posed by hazardous chemicals are normally based exclusively on results of studies performed on animals, extrapolated to humans. It should further be noted that the half life of cadmium in our bodies is several decades.

As mentioned in the introduction to the Impact Assessment, the concerns at EU level for the adverse effects of cadmium on human health dates back to a Council resolution adopted almost 30 years ago. Since then, various initiatives have been taken at EU level to restrict the use of cadmium as well as limit emissions from production. The use of cadmium is now restricted in almost all relevant applications, e.g. in some batteries, electric and electronic devices, as stabiliser and pigment in plastic and most paints, etc.

It should further be noted that cadmium and most cadmium compounds are classified as carcinogenic (Carc. 1B or 2), mutagenic (Muta. 1B or 2), toxic for reproduction (Repr. 1B or 2) and toxic to specific organs, in particular to bone and kidneys, after repeated exposure (STOT RE 1 or 2). They are also classified as toxic to the environment (Aquatic Acute 1, Aquatic Chronic 1).

So far, cadmium metal and five cadmium compounds have been identified as substances of very high concern within Reach and included in the candidate list due to their carcinogenic properties and the toxicity to bone and kidneys. This means that suppliers of articles have a duty to communicate the presence of cadmium and cadmium compounds in articles. Manufacturers, importers and users of these substances may furthermore have to apply for an authorisation of their use of cadmium.
In contrast to these efforts, the EU has so far not been able to agree on a regulation of cadmium for one of the most important source of contamination of the environment – phosphorus fertilisers. This failure may to a large extent explain why cadmium limits in soils still constitutes a risk to human health through exposure via the consumption of contaminated cereals, potatoes, carrots and other vegetables, as described in the Impact Assessment.

DISCUSSION: The associations between U-Cd and urinary proteins at very low exposure may not be due to Cd toxicity, and the clinical significance of slight proteinuria may also be limited. More importantly, other effects have been reported at very low Cd exposure. There is reason to challenge the basis of the existing health risk assessment for Cd. Our review of the literature found that exposure to low concentrations of Cd is associated with effects on bone, including increased risk of osteoporosis and fractures, and that this observation has implications for the health risk assessment of Cd. Other effects associated with Cd should also be considered, in particular cancer, although the information is still too limited for appropriate use in quantitative risk assessment.

CONCLUSION: Non-renal effects should be considered critical effects in the health risk assessment of Cd.


A meta-analysis providing summary estimates of several cohort studies on urinary cadmium and mortality. In an analysis restricted to six cohort studies conducted in populations with a mean urinary cadmium concentration of 1 mg/g creatinine, the hazard ratios were 1.38 (95% CI, 1.17–1.63) for all-cause mortality, 1.56 (95% CI, 0.98–2.47) for cancer mortality and 1.50 (95% CI, 1.18–1.91) for cardiovascular mortality (comparing highest vs lowest exposure category).

CONCLUSIONS: Even at low-level exposure, cadmium appears to be associated with increased mortality.


Maternal elevated urinary cadmium concentrations (≥0.8 μg/L) were inversely associated with children's general cognitive score [mean change: -6.1 points (95% CI -12; -0.33) per doubling of urinary
cadmium; corresponding to ~0.4 SD]. Stratifying by smoking status (p for interaction 0.014), the association was restricted to smokers.

In conclusion, elevated cadmium exposure in pregnancy of smoking women was inversely associated with the children’s cognitive function at pre-school age. The results indicate that cadmium may adversely affect neurodevelopment at doses commonly found in smokers, or that there is an interaction with other toxicants in tobacco smoke. Additionally, possible residual confounding cannot be ruled out.


RESULTS: Hazard ratios for all cardiovascular end points were consistently increased for participants in the 4th blood cadmium quartile (median, 0.99 μg/L). In models that also included sex, smoking, waist circumference, education, physical activity, alcohol intake, serum triglycerides, HbA1c, and C-reactive protein, the hazard ratios comparing the highest and lowest quartiles of exposure were 1.8 (95% CI: 1.2, 2.7) for acute coronary events, and 1.9 (1.3, 2.9) for stroke. Hazard ratios in never-smokers were consistent with these estimates.

CONCLUSIONS: Blood cadmium in the highest quartile was associated with incident cardiovascular disease and mortality in our population-based samples of Swedish adults. The consistent results among never-smokers are important because smoking is a strong confounder. Our findings suggest that measures to reduce cadmium exposures are warranted, even in populations without usual sources of exposure.


A 32% increased risk of osteoporosis (95% CI: 2-71%) and 31% increased risk for any first incident fracture (95% CI: 2-69%) were observed comparing high dietary cadmium exposure (≥13 μg/day, median) with lower exposures (<13 μg/day). By combining high dietary with high urinary cadmium (≥0.50 μg/g creatinine), odds ratios among never-smokers were 2.65 (95% CI: 1.43-4.91) for osteoporosis and 3.05 (95% CI: 1.66-5.59) for fractures. In conclusion, even low-level cadmium exposure from food is associated with low BMD and an increased risk of osteoporosis and fractures. In separate analyses, dietary and urinary cadmium underestimated the association with bone effects.

In linear regression, U-Cd was inversely associated with BMD at the total body (p < .001), femoral neck (p = .025), total hip (p = .004), lumbar spine (p = .088), and volumetric femoral neck (p = .013). In comparison with women with U-Cd < 0.50 µg/g of cr, those with U-Cd ≥ 0.75 µg/g of cr had odds ratios (ORs) of 2.45 [95% confidence interval (CI) 1.51-3.97] and 1.97 (95% CI 1.24-3.14) for osteoporosis at the femoral neck and lumbar spine, respectively. Among never-smokers, the corresponding ORs were 3.47 (95% CI 1.46-8.23) and 3.26 (95% CI 1.44-7.38). For any first fracture (n = 395), the OR was 1.16 (95% CI 0.89-1.50) comparing U-Cd ≥ 0.50 µg/g of cr with lower levels. Among never-smokers, the ORs (95% CIs) were 2.03 (1.33-3.09) for any first fracture, 2.06 (1.28-3.32) for first osteoporotic fracture, 2.18 (1.20-3.94) for first distal forearm fracture, and 1.89 (1.25-2.85) for multiple incident fractures. U-Cd at low environmental exposure from food in a general population of women showed modest but significant association with both BMD and fractures, especially in never-smokers, indicating a larger concern than previously know.


We found significant negative associations between U-Cd and BMD, with lower BMD (4% to 8%) for all sites in the fourth quartile of U-Cd, using the first quartile as the reference. In addition, we found positive associations between U-Cd and incident fractures, especially nonvertebral osteoporosis fractures in the fourth quartile of U-Cd, with hazard ratios of 1.8 to 3.3 in the various models. U-Cd as a continuous variable was significantly associated with nonvertebral osteoporosis fractures (adjusted hazard ratio 1.3 to 1.4 per µg Cd/g creatinine), also in never-smokers, but not with the other fracture groups (all fractures, hip fractures, vertebral fractures, and other fractures). Our results indicate that even relatively low cadmium exposure through diet and smoking increases the risk of low BMD and osteoporosis-related fractures in elderly men.