Cadmium, Dietary Exposure and Cancer

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Cadmium appears as a contaminant in most human foodstuffs because of its high rates of soil-to-plant transfer, rendering diet a primary source of exposure among non-smoking, non-occupationally exposed populations (Satarug et al. 2000, 2003). A "safe" intake limit of 70μg cadmium per day for a 70-kg person was set based on the critical renal cadmium concentration of between 100 and 200 μg/g wet weight which translates to a urinary threshold limit of 5-10 μg/g creatinine (World Health Organization, 1989, 1993). However, numerous studies have revealed adverse kidney effects at urinary cadmium levels below 0.5 μg/g creatinine (Satarug and Moore 2004). Further, bioavailability of ingested cadmium has been shown in studies on subjects consumed frequently high cadmium foods such as oysters, oilseeds and offal. The bioavailability of cadmium of the dietary origin is strengthened by the substantial amounts of cadmium accumulation in kidneys, eyes and other tissues and organs of environmentally exposed individuals. It is hypothesized that such accumulation results from the efficient absorption and systemic transport of cadmium, employing multiple transporters that are used for the body's acquisition of calcium, iron, zinc and manganese. Adverse effects of cadmium on kidney and bone have been observed in environmentally exposed populations at the frequencies higher than those predicted from models of exposure. There is increasing evidence implicating cadmium in the risk of diseases that involve other tissues and organ systems at cadmium concentrations not producing effects on bone or renal function.

Cadmium is classified as a cancer causing agent in humans based on an elevated incidence of lung cancer and mortality data derived from the occupational groups with evidence of elevated exposure to cadmium (IARC, 1993). The occupational exposures have historically been through inhalation of cadmium. A consequence of this initial association of inhaled cadmium with cancer in occupationally exposed workers is that a carcinogenic risk from exposure to dietary origin has long been ignored. There is accumulating evidence linking elevated dietary exposure and increased cancer incidence. Excess cancer mortality was found associated with environmental exposure to cadmium in Japan and the US prospective studies (Arisawa et al. 2007; Menke et al. 2009; Nishijo et al. 2006). Increased endometrial cancer risk was observed in a Swedish cohort among those consumed cadmium above 15μg/day, mainly from cereals and vegetables (Akesson et al. 2008). In this presentation, evidence from prospective studies will be summarized which reveal potential causal relationships of cadmium exposure with life-prognosis (all-cause mortality) and excess cancer mortality, including evidence that cadmium is at least a co-morbidity factor if not a causative factor. Specifically, cadmium-cancer associations will be summarized for the lung, pancreas, breast, endometrium, prostate and urinary bladder. These epidemiologic data argue strongly for public health measures for reducing exposure.

With the looming cancer and chronic disease epidemic worldwide, we encourage consideration given to cadmium exposure assessment, identification of potential exposure sources and determinant of cadmium body burden in future epidemiologic investigations to allow an estimate of total disease burden (cost) of the population exposure. There is a lack of therapeutically-effective chelating agents to enhance excretion of cadmium and this factor makes prevention of cadmium accumulation pivotal. The persistence of cadmium in the environment requires a long-term approach to minimize the food-chain transfer of cadmium and human exposure through environmental management and maintenance of lower cadmium levels wherever possible.