Chlordecone exposure and risk of prostate cancer

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In an article to be published on 21 June 2010 in the Journal of Clinical Oncology, researchers from Inserm (Inserm unit 625 - Research Group on Human and Mammalian Reproduction, University of Rennes 1), the CHU (University Hospital Centre) in Pointe à Pitre (urology department, University of the French West Indies and Guiana) and from the Center for Analytical Research and Technology (University of Ličge, Belgique), show that exposure to chlordecone (also named Kepone), an organochlorine chemical with well defined estrogenic properties used in the French West Indies until 1993, is associated to a significant increased risk of prostate cancer.

Chlordecone is an organochlorine insecticide used in the French West Indies from 1973 to 1993 to control the banana root borer. Permanently polluted soils and waters have remained the primary source of foodstuff contamination, and humans being continue to be exposed to this chemical. Chlordecone is recognized as endocrine disruptor, and is classified by IARC/WHO as possibly carcinogenic to humans.

Research results to be published in the Journal of Clinical Oncology come from an interdisciplinary prostate cancer epidemiology program named Karuprostate (from Karukera, the original Caribbean name of Guadeloupe). A case control study compared the characteristics of 709 consecutive incident cases of prostate cancer and 723 controls without prostate cancer. One of the main objectives of the research programme was to test the hypothesis that chlordecone exposure favors the development of prostate cancer in the French West Indies. Chlordecone
exposure was evaluated by measuring its concentration in the blood.

The analysis of the results by the researchers shows that chlordecone exposure is associated to a significant increase in the risk of prostate cancer with increasing plasma chlordecone concentration. These results are supported by the fact that men, presenting genetic variations which reduce their ability to eliminate the molecule, have higher risk of developing the disease.

The prostate cancer risk associated with chlordecone exposure was higher in subjects with a family history of prostate cancer first-degree relatives. Moreover, the prostate cancer risk associated with chlordecone exposure was particularly marked in subjects who had spent some time living in a Western country. According to the authors, several explanations may be given:

"The interaction of family history with prostate cancer may be explained by the presence of genetic susceptibility factors which are common both to the disease and to the chlordecone metabolic pathway but also by similar patterns of exposure, shared by members of a same family".

"Migration constitutes a period of exposure to specific environmental risk factors, including hazardous chemicals or nutritional agents. Residing in Western countries may induce significant changes in an individual, due, for example, to the adoption of a Western lifestyle, including, in particular, eating habits that may be risk factors for prostate cancer"

These results are the first to suggest that there is a causal relationship between chlordecone exposure and prostate cancer risk, and support the hypothesis that environmental estrogens may be involved in the development of prostate cancer. Such a relationship may be affected by genetic background, together with environmental agents related to diet or
lifestyle.

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