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Dietary, occupational, and ecological risk assessment of carbaryl and dimethoate

The application of pesticides may cause adverse impacts on human users and environmental receptors. We carried out consumers', occupational, and ecological risk assessment of two commonly-used pesticides, carbaryl (carbofosphate) or dimethoate (organophosphate). For consumers' health risk assessment, based on the current tolerance and highest residue levels in crops, fruits and vegetables, the non-carcinogenic risk index (hazard indexes, HI(s)) of carbaryl but not dimethoate was less than 1. Further analysis using Monte Carlo Simulation method showed that the means and upper 95% confidence limits of total HIs for carbaryl and dimethoate in different crops did not exceed one. For occupational exposure risk assessment, the distributions of pesticide exposure were assessed by HPLC analysis of personal air sampling tubes and patches from 27 workers and 16 farmers. Some of 15% confidence limits of total HIs for carbaryl and dimethoate were larger than 1, suggesting the importance of strengthening personal protective measures at work. The results from ecological risk assessment show that carbaryl possesses potential risk to aquatic insects, but not to fishes and frogs, whereas, dimethoate has significant ecological hazard effects on minnows, stoneflies, and frogs. The results can be regarded as the reference of government's pesticide administrative decision.

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Are epidemiological associations of higher chemical concentrations in blood with health effects meaningful?

A number of epidemiological studies have reported associations of higher blood concentrations of environmental chemicals such as perfluorinated acids (PFAAs) and PCBs with a variety of health effects including low birthweight, delayed onset of menarche or early onset of menopause. However, the effect of physiological changes during these life stages on the kinetics of a particular chemical are complex and difficult to elucidate without the quantitative structure provided by a physiologically-based pharmacokinetic (PBPK) model. To address the question of whether associations between age of menarche/menopause and chemical concentration can be explained by pharmacokinetics, we have developed human PBPK models that incorporate age-dependent physiological changes. We present two examples of how PBPK models can be used to evaluate associations in epidemiological studies between concentrations of a chemical in blood and physiological outcomes: (1) PFAAs and age at menarche, and (2) PCBs and birthweight. The models indicate that the relationships between blood levels and health outcomes can be explained on the basis of pharmacokinetics rather than toxic effects. In both cases the internal chemical exposures driven by physiological changes, in the case of PFAAs, menarche is an important route of excretion; therefore, onset of menstruation links chemical concentration in blood to age at menarche. In the case of PCBs, differing degrees of maternal weight gain during pregnancy, and resulting variation in the fat volumes in the pregnant women, serves as a hidden variable underlying the apparent relationship between birthweight and chemical concentration in blood. Many other examples exist of epidemiologic associations between exposure biomarker concentrations and outcomes that, by using PBPK models, may be explainable on the basis of chemical kinetics rather than causality.

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Exploring the concept of transportation systems risks

In present-day society, transport modes such as transit and automobiles are indispensable tools that are required to maintain a minimum level of wellbeing. Their importance spans the urban-rural dimension. In the U.S., national, state and local governments have generally assumed the responsibility of developing and maintaining infrastructure of transportation systems (e.g., highways and mass transit rail). The balance between mass transit availability and private vehicle use varies dramatically. In areas with effective mass transit systems, personal vehicle use can, in many cases, be considered voluntary thus making the associated risk voluntary as well. However, in areas without mass transit, personal vehicle use is a necessity and a large portion of the associated risks is essentially involuntary. Despite the varying characteristics of personal vehicle risks, most traffic risk studies have focused solely on personal vehicle risks (e.g., a number of fatalities or injuries per unit exposure of vehicle travel). In this study, we first propose an alternative transportation risk measure that focuses on the accident risks of the entire system. We particularly argue that the proposed system-risk measure is much more appropriate for policy discussions over allocation of scarce resources to improve safety. Understanding the impact of shifting personal vehicle risks from being involuntary to the voluntary by making mass transit more readily available changes the framing of the problem. In this study we compare differences in vehicle and system risks across multiple exposure measures (i.e., per mile, per trip, per driver) for urban, suburban, and rural areas using data from National Household Travel Survey, Fatality Analysis Reporting System, and American Community Survey.

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A risk assessment approach for inorganic arsenic that considers its mode of action

Despite the absence of a complete understanding of mechanism(s) underlying the carcinogenicity and toxicity of inorganic arsenic, an alternative to the default linear extrapolation approach is needed that is more consistent with the available evidence suggesting a non-linear dose-response. Contributory factors to the mode of action for arsenic carcinogenesis include DNA repair inhibition under conditions of oxidative stress, inflammatory and proliferative signaling, leading to a situation in which the cell is no longer able to maintain the integrity of its DNA during replication. It has been suggested that the dose-response for cancer risk assessments could be based on quantitation of molecular endpoints, or "biomarkers" of response, selected on the basis of their association with obligatory precursor events for tumorigenesis (Freston, 2002). We have applied this approach to inorganic arsenic using benchmark dose (BMD) modeling of gene expression changes in human target cells (urcepthelial cells) treated with arsenic in vitro. The BMDs for cellular gene expression changes related to the carcinogenic mode of action for arsenic were used to define the point of departure (POD) for the risk assessment. The POD was then adjusted based on data for pharmacokinetic and pharmacodynamic variability in the human population.