

10069- Organophosphorus Cumulative Assessment: A Comparison of Model Predictions
Price, PS, Chaisson, CF
The LifeLine Group

In early summer of 2002, EPA will release the final version of an assessment of the cumulative risks associated with organophosphorus pesticides. This assessment will include an evaluation of cumulative exposures to approximately 27 different compounds from residues in food, water supplies, residences, and golf courses. The cumulative risk assessment is being performed using two probabilistic models, CALENDEX™ and LifeLine™ Version 1.2. The two models differ in design, reliance on the use of preexisting survey data, and characterizations of exposure. The CALENDEX™ based analysis has an emphasis on geographical and seasonal linkage of pesticide use and the potential for surface water contamination while LifeLine uses a survey-based approach for residential exposures that results in internally consistent model of daily activities, residential pesticide use, and source of tapwater. This presentation will discuss the differences in the risk predictions from the two models and the implications of these differences for risk management decisions. The talk will be based on the published results of the CALENDEX™ and LifeLine™ models provided by EPA and additional analyses performed using LifeLine™.

10087- Physiologically based pharmacokinetic/pharmacodynamic modeling of organophosphate pesticides for biologically based risk assessments.

Poet, T.S., Kousba, A., Wu, H. and Timchalk, C. Battelle, Pacific Northwest Division, Richland, WA, USA.

Organophosphate (OP) insecticides like diazinon (DZN) and chlorpyrifos (CPF) constitute a large class of chemical insecticides that are widely utilized in the agricultural industry and in home applications. The potential exists for significant exposures to a combination of OP pesticides from multiple routes. The toxic effects of OP insecticides are associated with the capacity of the parent chemical or active metabolite to inhibit acetylcholinesterase (AChE). CYP450 enzymes are responsible for both the bioactivation and the detoxification of OPs. Metabolism to the active oxon leads to inhibition of AChE at nerve endings. In addition, A-esterase detoxifies the oxon to the pyrimidinol. The balance between OP activation and detoxification is of critical importance in determining the toxicological response. The objective of this research was to develop a first generation physiologically based pharmacokinetic/pharmacodynamic (PBPK/PD) model capable of predicting the relationships between exposure route, bioactivation, detoxification, and AChE inhibition. The ultimate goal is to utilize this model to quantify systemic dosimetry and biological response utilizing available environmental and personal exposure data. The model structure integrates CYP450 and esterase metabolism, route-dependent absorption, target tissue dosimetry, and dynamic response (esterase inhibition) to predict circulating blood levels of CPF or DZN and esterase inhibition in target organs (blood, brain, and diaphragm). The model developed thus far includes descriptions for tissues that are important for route of exposure (skin, intestine), and tissues involved in the pharmacokinetics (blood, liver, intestine) or pharmacodynamic (blood, brain, diaphragm) responses. Partition coefficients have been determined and used in the model to estimate the solubility of CPF, DZN, and their active oxon metabolites. Metabolic rate constants for the CYP450-mediated conversion to the oxon and the inactive pyrimidinol and the esterase-mediated deactivation of the oxon to the pyrimidinol have been measured in vitro. Allometric scaling was used to extrapolate the in vitro metabolic rate constants to estimate in vivo bioactivation/detoxification. Biochemical parameters (metabolic rate constants and partition coefficients) and physiological constants (blood flows, tissue volumes, and tissue-specific basal enzyme activities) have been integrated into the PBPK/PD model and exposure data from the literature used to validate the model predictions. The inhibition of AChE activity is a sensitive and relatively easy measure of exposure and is therefore the preferred descriptive endpoint. Esterase inhibition and regeneration rates have been described using in vitro calculations and parameter optimization to fit the model to AChE inhibition data. Individual descriptive models for CPF and DZN have been developed and have been shown to predict blood levels of the parent chemicals and AChE inhibition in animal models. The PBPK/PD models will be linked together to estimate the effects of exposures to a mixture of OPs and to describe target tissue dosimetry and effects in humans. These biologically relevant PBPK models will be integral to risk assessments for DZN, CPF, and mixture exposures under a variety of scenarios. (Sponsored by CDC/NIOSH Grant R01 OH03629-01A2).

10195- Application of an Individual Human Exposure Model to the CHS Cohort Children in Southern California

Jun Wu, Environmental Health Sciences Department, School of Public Health,
University of California, Los Angeles, 90095 junwu@ucla.edu

Fred Lurmann, Sonoma Technology Inc., Petaluma, CA fred@sonomatech.com

Arthur Winer, Environmental Health Sciences Department, School of Public Health,
University of California, Los Angeles, 90095 amwiner@ucla.edu

Steve Colome, Environmental Health Sciences Department, School of Public Health,
University of California, Los Angeles, 90095 scolome@pacbell.net

An individual-level human exposure model has been developed to assess individual exposure to vehicle-related pollutants (NO₂, O₃, PM₁₀, PM_{2.5}, and elemental carbon) for children recruited by investigators from the University of Southern California in the Children's Health Study (CHS) beginning in 1993. The objective of the present study is to quantify intra-community variability in exposures of selected CHS children, and to facilitate evaluation of relationships between exposure and health outcomes for individual children. Baseline questionnaire and time-activity surveys for the CHS children were analyzed and applied in the model. The CALINE4 model developed by the California Department of Transportation and the U.S. Federal Highways' Agency was used to calculate ambient pollutant concentrations near freeways and major roadways for geo-coded homes and schools of each children cohort. In the exposure model, four microenvironments (outdoor, residential indoor, school indoor, and in-vehicle) were studied, and Monte Carlo simulations were applied for variability and uncertainty analysis. Using the model, we investigated children's exposures to motor vehicle emissions, including differentiating between diesel and gasoline vehicles. We also studied intra-community variability in children's exposure to particulate matter and other vehicle-related pollutants for several of the 12 CHS communities. Better characterization of the exposure of individual children in the CHS cohort is expected to increase the robustness of observed health outcomes, and to provide more accurate information on the causative agents of those outcomes.

10238- REVIEW OF DATA AVAILABLE FOR THE DEVELOPMENT OF PREDICTIVE OP PESTICIDE QSARS AND PBPK/PD MODELS FOR HUMAN RISK ASSESSMENT.

C.C. Dary¹, J.B. Knaak², F. Power³, C. Thompson³, and J.N. Blancato¹. ¹ National Exposure Research Laboratory, U.S. Environmental Protection Agency, Las Vegas, NV; ² Department of Pharmacology and Toxicology, School of Medicine and Biomedical Sciences, SUNYAB, Buffalo, NY; ³ Anteon Corporation, Las Vegas, NV.

The successful use of Exposure Related Dose Estimating Model (ERDEM) involving the OP pesticides, parathion, isofenphos and chlorpyrifos prompted us to search the literature for additional rat and human PBPK/PD data to build new OP models. Central to the approach of building PBPK models was the development and use of predictive Quantitative Structure Activity Relationship (QSAR) models for obtaining the needed PBPK model parameters (i.e., K_p (cm/h) for percutaneous absorption, skin/water and tissue/blood partition coefficients, metabolic parameter such as V_{max} and K_m). A review of the available physicochemical and biological data on thirty-one selected OP pesticides (thionates and dithioates) was made using on-line database services provided by the National Library of Medicine, NIH, and American Chemical Abstracts Services, ACS. A few partition coefficients (skin/air, skin/water, skin/blood, blood/air and tissue/blood) and metabolic parameters (V_{max} , K_m) were found, but were insufficient in number and quality to develop predictive QSAR models. Published OP rat liver P450 microsomal V_{max} , K_m values on oxon formation were reviewed and compared with current studies with human liver microsomal P450 CYPs. Information on the inhibition of B-esterases (k_{is}) by oxons, hydrolysis of the oxons (V_{max} , K_m) by plasma and liver A-esterases were reviewed along with conjugation reactions involving leaving groups. New research is needed to obtain the necessary data for model construction and risk assessment. *This work has been funded (wholly) or (in part) by the United States Environmental Protection Agency under Interagency Assistance Agreement (DW 47944301) to GSA. It has been subjected to Agency review and approved for publication.*

10315- DEVELOPMENT OF AVERAGING TIME MODELS FOR PARTICULATE AIR POLLUTANTS

Neil Klepeis, Paul Switzer, and Wayne Ott, School of Public Health, University of California, Berkeley, and Department of Statistics, Stanford University, Stanford, CA

The national ambient air quality standards designed to protect public health specify averaging times over which ambient concentration measurements are to be averaged, typically 1 hour, 8 hours, 24 hours, or 1 year. When measurements of air quality are made using continuous monitoring instruments, it is possible to compute concentrations with time averages of 1 hour or multiples of 1 hour, overlapping or nonoverlapping, that can be compared directly with health-based standards. However, when ambient or personal monitoring measurement methods collect “integrated” samples with averaging times of 12 hours or 24 hours or longer, it is difficult to predict the average concentrations over shorter time periods. An averaging time model is designed to predict the concentrations at different averaging times based on statistical relationships between the concentrations and averaging times. For example, it may be possible to predict the sample variance of PM_{10} concentrations for different averaging periods (1,2,3,...hours, etc.) using an averaging time model. Despite the importance of averaging times for complying with health-based standards and for developing exposure models, there has been relatively little theoretical development of averaging time models and few applications of these models to measured concentrations in indoor or outdoor microenvironments.

Our prior research derived a theoretical averaging time model from the mass balance equation for indoor and outdoor concentration time series. The present study analyzes indoor and outdoor particulate concentration measurements to understand and describe their averaging time characteristics. Two data sources are investigated: (1) indoor and outdoor hourly average measurements of fine particulate polycyclic aromatic hydrocarbons (PPAH) from a photoionization aerosol sensor (PAS) monitor at a California home, and (2) hourly average ambient measurements of PM_{10} from the Tapered Element Oscillating Method (TEOM) monitors operated throughout the U.S. The PPAH data come from a field study of residential indoor-outdoor time series used for exposure modeling, and the TEOM hourly PM_{10} come from EPA’s Aerometric Information Retrieval System (AIRS) in 25 states over 1995-2000.

The ambient concentrations in these data sets show cyclical patterns due to seasonal effects, weekend/weekday differences in human activities, and diurnal effects such as daily traffic patterns. We simultaneously estimate the cyclical components and the residual serial autocorrelation of the cyclically-corrected concentrations. Using the estimated cyclical structure and serial residual autocorrelation, we develop an averaging time model that relates statistical variability to the length of the averaging period. We examine the validity of the averaging time model using the data sets described above.

A goal of this research is to provide human exposure researchers with basic models for designing and developing useful, practical algorithms for human exposure and indoor air quality models.

10333- A Probabilistic Modeling Framework for Predicting Population Exposures to Benzene

Stephen E. Graham, Janet M. Burke, Halûk Özkaynak

National Exposure Research Laboratory, US EPA, MD-56, Research Triangle Park, NC 27711

The US Environmental Protection Agency (EPA) is modifying their probabilistic Stochastic Human Exposure Dose Simulation (SHEDS) model to assess aggregate exposures to air toxics. Air toxics include urban Hazardous Air Pollutants (HAPS) such as benzene from mobile sources, particulate organic matter and metals from industrial point sources, and formaldehyde from indoor sources. However, in addition to these and other air emission sources, many of these HAPS have exposure pathways other than through inhalation. Therefore, a model needs to be developed that allows for prediction of the population distribution of exposure to air toxics through both single or multiple exposure pathways. As part of a preliminary case study, benzene was selected for investigation and initial model development. Data for exposure factors, ambient air concentrations, and other relevant concentrations were compiled from multiple sources, including EPA's Aerometric Information Retrieval System (AIRS) and Consolidated Human Activity Database (CHAD), as well as measurement data obtained in the literature. The selection of critical microenvironments, the use of mass balance equations or linear models in generating specific exposure concentrations such as in-vehicle or indoor air concentrations, and the development of other microenvironmental exposure concentration distributions were based on human activity patterns, data quality, and data availability. The results of this research serve as inputs to a newly constructed SHEDS-Air Toxics model to estimate population distributions of benzene exposure and absorbed dose.

Disclaimer: This work has been subjected to United States Environmental Protection Agency review and approved for presentation and publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

10457- Modeling of urban population exposure to Carbon Monoxide using the EXPOLIS-Milan personal CO exposure and Time Activity data

Yuri Bruinen de Bruin(1), Otto Hänninen(2), Paolo Carrer(1), Greta Scotto-Di-Marco(3), Stylianos Kephelopoulos(3), Domenico Cavallo(1), Matti Jantunen (2), Marco Maroni(1)

- (1) University of Milan, Department of Occupational Health, Via San Barnaba 8, 20122 Milan, Italy
- (2) KTL, Department of Environmental Health, P.O. Box 95, 70701 Kuopio, Finland.
- (3) EU Joint Research Center, Environment Institute, Air Quality Unit, I-21020 Ispra (VA), Italy

EXPOLIS (Air Pollution Exposure Distributions of Adult Urban Populations in Europe) is a multicenter urban population exposure study. In the study comparable population exposure distributions were created for six European cities. In Milan, majority (over 75%) of the working population work in offices or similar environments. During fall 1996 to winter 1997-98, personal CO exposures of 50 office workers were monitored using Langan Model T15 personal monitors. During the measurements, the subjects completed a 15-min. time resolution Time Microenvironmental Activity Diary differentiating among the indoor environments “home-“, “work-“ and “other indoor”. Additionally, the participants were asked to indicate whether they were exposed to Environmental Tobacco Smoke (ETS) or GAS-cooking (GAS).

The EXPOLIS data is stored in a relational ACCESS database (software version 7, alias 95). Within EXPOLIS, the RIVM (The Netherlands) and KTL (Finland) developed a simulation framework to create population exposure distributions for a selected air pollutant for the total population or sub-population. The simulation framework supports Monte Carlo and Latin Hypercube sampling. The simulation framework has been applied to PM_{2.5} data in Helsinki and the simulated 48-h average population exposure distributions were shown to match observed corresponding distributions reasonably well.

The Milanese CO measurements were used to create concentration distributions for the home indoor, work indoor and other microenvironments. Concentration distributions of sub populations exposed to ETS and gas cooking are compared to the non-exposed population.

These concentration distributions were used together with Milanese time activity diaries to create simulation model inputs.

Using these inputs, 48h and 24h Time Weighted Averaged population exposure distributions were simulated. The exposures were calculated using the model $E = f_{HI} * C_{HI} + f_{WI} * C_{WI} + f_{OI} * C_{OI}$ in which E is the exposure, f is the fraction of time and C is the concentration within that specific microenvironment. Descriptive analyses of the simulated exposures are tabulated and shown graphically on a lognormal plot.

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10484- APPLICATION OF SWARM DYNAMICS TO DOSIMETRY OF CIGARETTE SMOKE. David Broday*, Agricultural Engineering Department, Technion, Haifa 32000, Israel, and Risa J. Robinson, Department of Mechanical Engineering, Rochester Institute of Technology, 76 Lomb Memorial Drive, Rochester, NY 14623.

The distribution of tumors resulting from inhalation of cigarette smoke particles (CSP) depends on the deposition profile of the smoke particles along the respiratory tract. CSP deposition data vary over a wide range, 22-89%, and are inconsistent with typical deposition theory based on single particle dynamics. Regional deposition measurements indicate that CSP collect in the human airways mainly in the tracheobronchial region rather than in the pulmonary region. Inter-subject variability in breathing patterns and accounting for the processes of coagulation, electrostatic deposition, and hygroscopic growth are not sufficient to explain the enhanced regional deposition in the upper proximal airways of the respiratory tract and the wide variation in deposition data (Robinson and Yu, 2001). A plausible cause for this behavior is particle interactions within the concentrated CSP cloud, here termed the colligative effect. Previous studies addressing this effect considered the cloud to be a solid sphere and assumed that the air flows around it (Hinds, 1982, Martonen and Musante, 2000). Application of this theory to CSP dosimetry yields 99% deposition efficiency, which is in disagreement with experimental evidence. In the present study we let some of the air to pass through the cloud. The basic approach is to consider the cloud to be a porous medium, and allow for weak hydrodynamic interactions among the particles. In dilute clouds CSP settle independent of each other while moving within the porous medium. For dense clouds, the porous medium settles like a rigid body. Concentration dependent settling velocities, calculated via this approach, are incorporated into a lung deposition model along with coagulation, hygroscopic growth and electrostatic deposition. The model is evaluated by comparing predictions of CSP deposition with published data.

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Matonen, T. B., Musante, C. J. (2000). Importance of Cloud Motion of Cigarette Smoke Deposition in the Lung, *Inhalation Toxicology* 12 (Supplement 4): 261-280.

Robinson, R. J., Yu, C. P. (2001). Deposition of Cigarette Smoke Particles in the Human Respiratory Tract, *J. Aerosol Sci. Tech.* 34: 202-215.

10537- EVALUATION OF GEE AND BAYESIAN APPROACHES TO ASSESSING THE RELATIONSHIP BETWEEN ENVIRONMENTAL FACTORS AND THE GEOSPATIAL DISTRIBUTION OF *WUCHERERIA BANCROFTI* INFECTION IN LEOGANE COMMUNE, HAITI

HEATHER A. BOYD^{1,3}, WALLER LA², ADDISS DG³, FLANDERS WD¹.

Departments of Epidemiology¹ and Biostatistics², Emory University, and Division of Parasitic Diseases³, Centers for Disease Control and Prevention, Atlanta, GA. Email: hboyd@emory.edu

Analytic methods commonly used in epidemiology often ignore the spatial nature of the distributions of many infectious diseases. However, ignoring spatial correlation between outcomes in regression analyses can result in incorrect estimates of regression parameter standard errors, potentially leading to invalid inferences. One method of accounting for spatial correlation first uses a variogram to model the spatial correlation between responses as a function of distance between observation sites, and then uses this variogram to define the variance-covariance matrix for a regression model fitted using generalized estimating equations (GEE). However, defining the correlation structure and fitting such models become difficult or even impossible if other types of correlation are also present, both for theoretical reasons and because standard statistical software packages (SAS, S-Plus) require the user to specify numerical values for all terms in potentially large user-defined variance-covariance matrices. In addition, regression parameter estimates may not be robust to the choice of correlation structure. Bayesian hierarchical models, fitted using software such as WinBUGS, are more accommodating of non-standard variance-covariance structures than are GEE methods and also allow an assessment of the extent to which regression parameter estimates depend on the choice of correlation structure, but appear relatively infrequently in the epidemiologic literature. This paper uses a study of lymphatic filariasis, a mosquito-borne parasitic disease caused by the worm *Wuchereria bancrofti*, to illustrate these methodologic issues. The non-uniform geospatial distribution of this highly focal disease has not been adequately explained. *Wuchereria bancrofti* infection prevalence data collected at 57 schools from across the 500 km² Leogane Commune in Haiti, and used to estimate prevalence of infection in the surrounding communities, exhibit a clear spatial pattern. Infection prevalences of 35-45% predominate in the northwestern portion of the 170 km² coastal plain, but within 13 km prevalences fall to 20-25% in the south and to below 10% in the east. Infection in the foothills and mountains is limited to sporadic cases, with the exception of three areas: a high mountain valley, a district where the mountains drop to a narrow stretch of coast, and a district overlooking a suburb of Port au Prince. Unmodified and modified GEE and Bayesian hierarchical models are used to assess the relationship between environmental and geographic factors – the variables for which are derived from Landsat 5 and 7 satellite data – and the geospatial distribution of *W. bancrofti* infection in Leogane. The three methods are discussed with respect to the issues outlined above; the ultimate goal is to predict community risk of infection from easily observable data, such as Landsat data.

10560- Modeling exposure -sensitization response curve among bakers

Chava Peretz¹ Nettie de Pater² Dick Heederik²

¹Department of Health Professions, Sackler School of Medicine, Tel Aviv University, Israel; ²Department of Environmental Sciences, Occup.and Environ. Health Group, Utrecht University, The Netherlands.

Introduction: Most epidemiological studies among bakers were based on (a) simple exposure categorizations instead of continuous exposure levels or (b) were limited to low and medium exposure levels, (c) did not consider the shape of the exposure-response relationship or assumed a linear one. We studied the shape of relationship (either linear or not) between exposure and sensitization, based on a wide exposure range, by using advanced statistical tools; controlling for sector of industry and atopic status.

Methods: About 250 Dutch bakers from 4 industry-sectors: traditional and industrial bakeries, flour mills and bakery product industries were included in this study. Exposure to inhalable dust and wheat allergens were measured. For all workers information about sensitization to wheat and common allergens (atopy) (no/yes-class 1 or higher) was available.

Data analysis: The shape of the relationship between sensitization (dependent variable) and exposure, sector and atopy (covariates) was studied in a 2-stage process:

- (1) We fitted a semi-parametric generalized additive model where atopy and sector were the parametric part of the model and log-concentration was the non-parametric part. The term for the additive predictor - log-concentration, was fitted using a spline smoother. The degrees of freedom (DF) for the additive predictor were selected by a generalized cross-validation method and they indicated the degree of the polynomial representing the data best (SAS-Proc Gam).
- (2) We applied a parametric model – a quadratic (based on the previously found degree of the polynomial) logistic model with linear and quadratic terms of logged concentration. (SAS- Proc Genmod).

Results: The effect of exposure using a smoothing spline was found to be of borderline significance in exposure to both inhalable dust and to wheat allergens ($p=.1037, p=.1214$ accordingly). The DF was found to be 2, for both exposures, indicating a quadratic relationship. Consequently we applied a quadratic logistic regression. Fig 1 presents the curves for non-atopic workers. The probability of sensitizing increases with exposure up to a specific value: $\sim 3.7 \text{ mg/m}^3$ for inhalable dust and $\sim 33.1 \mu\text{gEQ/m}^3$ for wheat allergens. At higher exposures the risk decreases (probably a health worker effect). Odds ratios for the squared log concentrations were lower than 1 (OR=.84,.94 $p=.0121, .0731$ for inhalable dust and wheat allergens respectively). In all analyses, atopy was a highly significant risk factor for sensitization (OR= ~ 6 , $p<.0001$), and sector also affected risk significantly. The odds ratio in the industrialized bakeries was ~ 4.2 times higher than in flour mills.

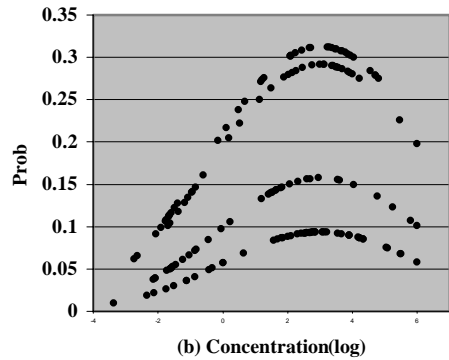
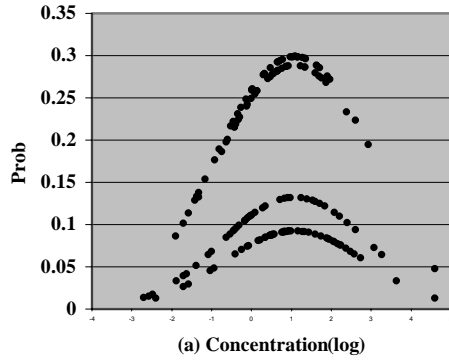
To conclude the exposure-response relationship among bakers may be non-linear and requires further exploration.

Fig1: Probability for sensitization (Prob.) as a function of exposure to

(a) inhalable dust in logged concentration mg/m^3 (n=193)

(b) wheat allergens in logged concentration $\mu\text{gEQ}/\text{m}^3$ (n=168)

among non-atopic workers in 4 sectors: industrialized bakeries, traditional bakeries, enzyme processing and flour-mills. (from top down)



10806- Modeling the Evaporative Loss of Organophosphorus pesticides from Skin using The Exposure Related Dose Estimating Model (ERDEM). J.B. Knaak¹, C.C. Dary², F. Power³, E.J. Furtaw, Jr², and J.N. Blancato². ¹Department of Pharmacology and Toxicology, School of Medicine and Biomedical Sciences, SUNYAB, Buffalo, NY; ² National Exposure Research Laboratory, U.S. Environmental Protection Agency, Las Vegas, NV; ³Anteon Corporation, Las Vegas, NV.

The ERDEM model was developed at EPA, Las Vegas and used to describe the multi-route absorption of parathion, isofenphos and chlorpyrifos in children. ERDEM was upgraded by adding an equation depicting the transfer of the pesticides to skin ($k_d R$) and their disappearance from the surface of the skin due to evaporative loss ($K_a A_{surf}$), penetration ($K_p a (C_{surf} - C_{sk})$) and wash-off ($K_w A_{surf}$):

$$dA_{surf} dt = K_p a (C_{sk} - C_{surf}) - K_a A_{surf} - K_w A_{surf} + k_d R$$

The model keeps track of skin surface residues during single and repeated periods of daily or weekly exposure. Factors such as concentration (C , $\mu\text{g}/\text{cm}^3$) of the topical dose, exposed skin area (a), vehicle, partition coefficients, pesticide vapor pressure, and air flow over the surface affect the availability and consequently the amount of pesticide absorbed from skin. Parathion and isofenphos are lost by evaporation from in vivo rat skin after application, with more parathion (vapor pressure = 0.89 mPa @ 20 C) being initially lost than isofenphos (vp = 0.22 mPa @ 20 C). We modeled the loss of parathion from the skin of field workers reentering pesticide-treated foliage and the loss of isofenphos from the skin of individuals coming in contact with treated turf, using parameter values estimated from rat studies. Approximately 3% of the transferred dosage was absorbed, 3% loss to air, 2% in tissues, 0.6% in urine and feces, leaving 94.4% as a residue on skin after eight hours of exposure. In an isofenphos dermal exposure study involving human volunteers, 3.6% of the applied dose was absorbed after 24 hours with the remaining surface dose <1% and no residual isofenphos in skin strippings. We adjusted the evaporation rate constant (K_a) in ERDEM from 0.0055 to 0.15 hr^{-1} in order to reproduce the human in vivo values. Evaporative losses are anticipated to be as great or greater from the skin of children compared to that from adults. *This work has been funded (wholly) or (in part) by the United States Environmental Protection Agency under Interagency Assistance Agreement (DW 47944301) to GSA. It has been subjected to Agency review and approved for publication.*

10898- The California Population Indoor Exposure Model, Version 2: A User-Friendly Assessment Tool for Population Exposure to Air Pollutants

Arlene Rosenbaum¹, Jonathan Cohen¹, Farzad Kavvoosi¹, Susan Lum², and Peggy Jenkins²

¹ ICF Consulting, San Francisco, CA

² California Air Resources Board, Sacramento, CA

This presentation will report on and demonstrate a new version of the California Population Indoor Exposure Model (CPIEM version 2.0). Enhancements include greatly improved ease of use through a Windows interface, superior graphic outputs, an updated default database, as well as enhanced and new calculation capabilities, including uncertainty analysis. The CPIEM is a software tool that combines:

- air pollutant concentration distributions for several microenvironments, including outdoors, and
- population activity patterns that specify time spent in each microenvironment

in a Monte Carlo framework to predict distributions of exposure concentrations for the California population. The default databases of microenvironment concentration distributions and activity patterns are specific to California, but the model allows the user to easily add his or her own data as well.

For many air pollutants, the indoor concentration data are either sparse or nonexistent. To address this limitation, and to provide a means of evaluating hypothetical exposure reduction activities, the CPIEM also includes a mass-balance algorithm so that the user can estimate indoor concentration distributions based on distributional information for parameters such as indoor source emission rates, building volumes, and air exchange rates.

The Windows platform of this new version of CPIEM greatly improves the software's efficiency and ease of use with standard, easily understood drop-down menus and dialogue boxes. The graphic outputs are presentation quality. Scenarios are easily saved and edited to facilitate sensitivity analysis. The default databases have been

updated with more recent data on indoor and outdoor pollutant concentrations, mass-balance parameters, and the demographic composition of California's population.

The exposure distributions predicted by CPIEM reflect the variability of exposure concentrations across population groups, but not our uncertainty about them. A new supplementary software program, designed to be used in conjunction with the new CPIEM, facilitates the estimation of the uncertainty of these exposure distributions. The uncertainty supplement creates alternative distributions for the CPIEM input variables with Monte Carlo sampling to reflect our uncertainty about the parameters of the input distributions. The user provides each alternative to CPIEM for iterative simulations. At the conclusion of the simulations of the alternatives the uncertainty supplement combines the resulting exposure distributions to estimate uncertainty distributions for selected percentile values.

Other enhancements to the CPIEM capabilities include refinement of the pollutant removal process calculation in the mass-balance algorithm, and additional output metrics.

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11108- THE NORD-COTENTIN CHEMICAL RISK ASSESSMENT MODELING

Catherine ROMMENS¹ and Caroline RINGEARD²

Institute for Nuclear Protection and Safety (IPSN)

¹CE Cadarache, Bt 153, F-13105 Saint-Paul-lez-Durance, France

²B.P. 6, F-92265 Fontenay-aux-Roses, France

e-mail: catherine.rommens@ipsn.fr, caroline.ringeard@ipsn.fr

After the assessment of the radiological risk due to past and recent discharges of the nuclear facilities of the Nord-Cotentin region of France, the Groupe Radioécologie Nord-Cotentin (GRNC) was commissioned to evaluate the risk due to the corresponding chemical releases. The first step of this assessment was to build up a tool to estimate the level of pollutants in the environment. Then, as a second step, this tool was used to calculate the exposure and risks for several scenarios relevant for the local population.

To perform the first step, engineers and experts from environmental toxicology/chemistry and radioecology organizations worked together, within the framework of the GRNC, to model environmental dispersion and transfers and exposures to pollutants. The modeling of the atmospheric discharges in the terrestrial environment was especially studied due to the strong differences between radiological and chemical sources which prevented from using existing codes. The three release sources of the La Hague site were taken into account : the central boiler house, the reprocessing units, UP2 and UP3, and the incinerator. About 30 chemical substances were studied, from heavy metals to inorganic and organic substances such as dioxins. A transfer model was, first, developed using radioecology modeling approach, available from the previous GRNC work, combined with knowledge of properties on chemicals of interest. The existing codes were reviewed and compared to select the most relevant algorithms for the Nord-Cotentin case. It appeared that no existing software was "ready to use" for the calculations required. That is why a specific tool, supported by EXCEL© package, was developed which accounts for atmospheric dispersion, deposition on the ground, interception of pollutants by leaves (retention and translocation), root transfer and uptake by animals. This tool enabled to estimate the concentrations of chemical pollutants, that can be attributed to La Hague releases and to compare them with natural level existing within the environment. The terrestrial compartments studied were the air, the soil, different kinds of plants (grass, hay, maize fodder, root vegetables, leaf vegetable, fruits) and animals or animals products (beef, sheep, pork, poultry and eggs).

Exposure, dose and risk calculations were performed based on the results of concentrations in the environment and on corresponding values for marine environment. Ingestion, including inadvertent ingestion of soil, and inhalation were the main exposure pathways studied. Three scenarios reflecting the local way of life were chosen. The first one, based on the average individual (infant, child or adult), gave reference values to compare with the other two scenarios which highlight respectively the consumption of terrestrial agricultural products and the consumption of seafood.

The development of the methodology and the tool for the Nord-Cotentin case study allowed us to explore the state-of-the-art of modeling applied to risk assessment. In this work, a particular attention was devoted to identify the main limitations and uncertainty of the whole calculation process and to make clear how they were treated at each step of the risk assessment.