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Authors

MacLeod, Matthew
McKone, Thomas E.
Foster, Karen L.
et al.

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**Applications of Contaminant Fate and Bioaccumulation Models in Assessing
Ecological Risks of Chemicals: A Case Study for Gasoline Hydrocarbons**

Matthew MacLeod¹, Thomas E. McKone², Karen L. Foster³, Randy L. Maddalena¹,
Thomas F. Parkerton⁴ and Don Mackay^{3*}

*-Corresponding author

1 – Lawrence Berkeley National Laboratory, One Cyclotron Road 90R-3058, Berkeley,
CA, 94720-8132

2 - University of California School of Public Health and Lawrence Berkeley National
Laboratory, One Cyclotron Road, 90R-3058, Berkeley, CA 94720-8132

3 – Trent University Canadian Environmental Modelling Centre, 1600 West Bank Drive,
Peterborough, ON, K9J 7B8. Phone: (705) 748-1011 x 1489 FAX: (705) 748-1080
Email: dmackay@trentu.ca

4 - ExxonMobil Biomedical Sciences, Inc., 1545 Route 22 East, Annandale, NJ,
08801-0971

Abstract

Mass balance models of chemical fate and transport can be applied in ecological risk assessments for quantitative estimation of concentrations in air, water, soil and sediment. These concentrations can, in turn, be used to estimate organism exposures and ultimately internal tissue concentrations that can be compared to mode-of-action-based critical body residues that correspond to toxic effects. From this comparison, risks to the exposed organism can be evaluated. To illustrate the practical utility of fate models in ecological risk assessments of commercial products, the EQC model and a simple screening level biouptake model including three organisms, (a bird, a mammal and a fish) is applied to gasoline. In this analysis, gasoline is divided into 24 components or "blocks" with similar environmental fate properties that are assumed to elicit ecotoxicity via a narcotic mode of action. Results demonstrate that differences in chemical properties and mode of entry into the environment lead to profound differences in the efficiency of transport from emission to target biota. We discuss the implications of these results and insights gained into the regional fate and ecological risks associated with gasoline. This approach is particularly suitable for assessing mixtures of components that have similar modes of action. We conclude that the model-based methodologies presented are widely applicable for screening level ecological risk assessments that support effective chemicals management.

Introduction

Reliable assessment of the potential impact of chemical releases on ecosystems is essential in fields such as ecological risk assessment (1), life-cycle impact assessment (2), pre-market chemical analysis, and green engineering (3). In these applications, the potential ecological impact of chemicals must be evaluated by assessing the likelihood that adverse effects may result from environmental exposure to the chemical. A comprehensive treatment requires an assessment that links chemical releases, environmental concentrations, target organism exposures, tissue concentrations, and likelihood of adverse effects. Constructing these linkages requires information from a variety of disciplines, including chemical fate modeling, toxicology, and aquatic and terrestrial ecology.

Faced with such a complex challenge, environmental scientists must develop, test and apply transparent, quantitative tools that describe the essential features of the interactions between chemicals and the abiotic and biotic environment. Transparent and rapid assessment methods are particularly required for conducting comparative screening-level risk assessments of large groups of chemicals, such as those listed on Pollutant Release and Transfer Registries (PRTRs). In these applications the goal of the assessment is usually to identify chemicals that pose the highest potential ecological risk so that resources can be effectively prioritized on substances that warrant further study and possible risk reduction measures.

Differences in potential impacts among a large set of chemical contaminants depend on how much and where the chemicals are released, how they are transported in the environment, how long they survive or persist, and how much toxic stress they place on ecosystems. Multimedia transport and transformation models can be used to evaluate (i) how and where chemicals will partition in the environment, (ii) how long they persist, and (iii) estimated concentrations in the air, water and food that directly contact organisms. These concentrations can be used to estimate exposure or dose with subsequent evaluation of the likelihood of toxic effects. In human health risk

assessments, exposure and dose-response are often evaluated separately and then combined to determine risk. MacLeod and McKone (4) have shown that for human populations the source-to-dose relationship expressed as the fraction of the emitted molecules contacting a target population (the “intake fraction”, iF) is strongly correlated with overall multimedia persistence (Pov). Pov can be deduced from chemical properties and media-specific degradation half-lives using a multimedia fate model, providing a quantitative link between releases and exposure. Unfortunately, analogs to the iF approach are not currently available for assessing aquatic or terrestrial ecosystem impacts.

Much of ecological risk assessment has been focused on defining environmental concentrations that protect the majority of individuals or species (5, 6). This process requires significant input of chemical- and species-specific concentration-response data. These data are available for many chemicals for some aquatic species, but for only a very limited number of terrestrial species. One common approach to overcome this lack of data is the use of a species sensitivity distribution (SSD) that assumes the dose-response function follows a logistic shape with respect to variation in species sensitivity (1).

This allows laboratory or ecosystem-scale dose-response functions to be constructed from species-specific toxicity data. The resulting relationship extrapolates empirical information about variations in species sensitivity, but it does not consider the underlying mechanistic relationship between exposure and internal dose that may help to explain the shape and spread of the distribution.

Adverse impacts that may result from chemical exposure concentrations in water, sediment or soil show significant variation among chemicals and species. These variations depend on a number of factors, notably, dose to the organism, the relationship between dose and tissue concentrations, and the target tissue-specific toxic impact. The dose of chemical derived from ingested food and water depends on (i) the quantity of chemical ingested by the organism, (ii) retention time of food in the gut of the organism (iii) rate of uptake from the gut, and (iv) the reverse rate of elimination of chemical

across the gut. Analogous factors determine the dose obtained from respired air, or in the case of fish, respired water. The linkage between dose and tissue concentrations depends on (i) the relative solubility of the chemical in the target tissue (ii) the kinetics of delivery of the chemical to the target tissue in the body, and (iii) rates of metabolism. It is important to recognize that the dose into the body and concentration at a particular target tissue depend on chemical properties that also determine environmental fate. With the exception of metabolism rates, all of the above processes can be generically estimated from fugacity-type mass balance models and physico-chemical properties that are required for the environmental fate portion of the risk assessment.

Toxicologists recognize that the concentration of chemical at the specific target site and the mode of action at that site are what combine to determine the likelihood of toxic effects on an organism. A major effort has been made to interpret environmental toxicology data in terms of internal “critical residue concentrations” that induce toxic effects by various modes of action (7-10). This approach offers several practical advantages over assessments based on external concentrations in exposure media. Critical residue concentrations provide an intensive metric of toxicity that can be used in comparative ecological risk assessments to translate exposures into risk estimates (11), as well as in the evaluation of chemical mixtures that are comprised of components sharing a similar mode of toxicological action (12). At present the whole-body “critical body residue” (CBR) for lethal effects of non-specific acting narcotics (~2 mmol/kg) is the most well established and agreed upon example of a toxicological endpoint based on an internal dose (11).

Goals of this paper

Human activities not directly related to chemical exposure can also impact plant and animal species (1, 13). For example, changing land-use patterns disrupt or destroy habitat. Although interactions between chemical and non-chemical stressors are likely, here we consider only direct impacts of chemical emissions on ecosystem protection.

Specifically, we address methods for developing combined source-to-dose and dose-response models for ecosystem food webs.

Our goal is to illustrate how multimedia contaminant fate models can be coupled to evaluative bioaccumulation models to estimate internal concentrations that serve as input to screening ecological risk assessments.

We do this using a case study of gasoline discharged into various environmental compartments. Gasoline has been selected since the component hydrocarbons comprising this complex substance exhibit a common ecotoxicological endpoint (narcosis) that can be assessed relative to effects-based critical body residues. Given this common mode of toxic action, our premise is that environmental fate, bioaccumulation, and metabolism are the key factors that distinguish potential impacts among these components. Further we assume that the component hydrocarbons comprising this complex substance additively contribute to toxicity. Ecological risk assessments based on source-to-target models using chemical properties data have the potential to account for variations in tissue concentrations across chemicals and species.

Methods and Data

Our proposed risk assessment methodology requires sequentially modeling the relationships between (i) emissions and environmental concentrations, (ii) environmental concentrations and intake/uptake by organisms, and (iii) chemical uptake and concentration for a specific target tissue in the body. This tissue concentration can be compared to critical tissue residue values to assess risk. Models and data used in the case study to assemble these linkages are discussed below.

Our illustrative case study considers gasoline released into a generic regional environment. Modern industrialized economies depend on efficient production and distribution of gasoline. In the United States approximately 1.4×10^9 liters of gasoline are consumed per day (14), and discharges to the environment are possible at every step

of the supply line from refinery to consumer. Gasoline is a mixture of many individual chemicals. Our assessment strategy relies on grouping this mixture into a set of 24 “blocks” of hydrocarbon compounds that have similar physico-chemical properties and degradation rates, as described by Foster et al. (15) and illustrated in Table 1. These blocks were selected on the basis of carbon number, chemical structure (i.e., alkanes, aromatics, alkenes) and properties. In some cases, such as benzene and toluene, the block consists of a single substance. Gasoline additives are not considered in the assessment.

1. Estimation of environmental concentrations from emissions

A wide variety of multimedia fate models are currently available that treat the environment-chemical system on different spatial and temporal scales, and at different levels of complexity. For our illustrative case study we selected the Level III Equilibrium Criterion (EQC) model (16) to represent the fate and transport of gasoline hydrocarbons on a regional scale. The EQC model was developed to provide a standard reference model for conducting multimedia assessments of new and existing chemicals. The model calculates chemical inventories, fugacities and concentrations in air, water, soil and sediment under steady-state conditions for a defined emission scenario under a set of standard reference environmental conditions.

The physico-chemical properties required by the EQC model include partition coefficients between air, water and octanol and estimated degradation rate constants in the primary environmental media. Properties used as inputs to the model to represent the 24 hydrocarbon blocks are shown in Table 2 (15). Details of the data sources and methods used to compile this data are provided in the supporting information.

Emissions to air, water and soil are treated separately but the results can be scaled and combined later to evaluate the total effect. For this illustrative case study we arbitrarily assume that each “unit” emission rate of the gasoline mixture in the EQC model region is 100 kg/h. The releases are treated as area sources that are evenly distributed throughout the region. Evaluation of localized sources and impacts are beyond the scope of this

study. Figure 1 shows the representative molar composition of gasoline used to calculate emissions of each hydrocarbon block. Gasoline formulations vary between locations and with season and octane rating. The gasoline mixture composition used here is based on formulations used in Western Europe and is assumed to be broadly illustrative of gasoline used in most industrialized countries (15). We have further made the simplifying assumption that gasoline entering air, water or soil has the same composition. In reality, gasoline vapor lost to air during transfer and storage is likely to have a higher fraction of the more volatile components.

The results from the EQC model include three sets of predicted regional concentrations of each gasoline hydrocarbon block in air, water, soil and sediments resulting from emission to air, water and soil.

2. Estimation of intake of environmental contaminants by organisms

Wildlife are exposed to chemicals in the environment through contact with air, water and food. Dermal exposure is not treated. Intake of chemicals into an organism is therefore based on concentrations in relevant exposure media including respired air (or in the case of fish, respired water) and ingested food. The resulting chemical intake rate or dose is obtained by multiplying the contact concentrations in air, water or food by the corresponding intake rates for respiration, water and food ingestion.

In our case study, exposure concentrations in air and water are taken directly from the EQC model results for these media. Exposure concentrations in foods are calculated from a specified environment-to-food accumulation factor. For initial screening purposes, we assume equilibrium partitioning between foods and a selected reference environmental medium (Table 3).

Generalized respiration and feeding rates for the three species selected for the case study are calculated from allometric equations. Allometric models are widely available for estimating a variety of physiological parameters for various taxa, usually as a function of

body mass. The text by Peters (17) includes a compilation of such relationships for rates of metabolism, feeding, respiration, locomotion and reproduction. The relationships are usually expressed in the form

$$\text{Log}(P) = A + B \text{Log}(\text{Body Mass}).$$

Where P is the physiological parameter of interest and A and B are empirical constants. Specific parameters for describing breathing rates of mammals and birds in the case study were taken from Frappell et al. (18). Details of parameters and equations used are given in Table 3 and the supporting information.

These calculations relate environmental concentrations to exposure media concentrations in air, water and food, and estimate the resulting chemical exposure and intake by the organism through respiration and ingestion routes.

3. Estimation of uptake and target tissue concentrations

While intake brings contaminants into the gut or lung, uptake by the organism across the biological barriers into the body is determined by the efficiency of chemical assimilation or absorption across these barriers. A considerable literature has developed in recent years on models describing the bioconcentration, bioaccumulation and biomagnification of contaminants by a variety of organisms and in food webs comprising several trophic levels (19-23). Although most models apply to fish, recent models include birds and mammals (24). These models have the common feature that they calculate uptake from food and respired air or water and loss by respiration, metabolism, egestion, growth dilution and possibly reproduction. A steady-state concentration can be calculated by balancing input and loss rates in the organism. More complex dynamic models can be used when appropriate.

For this illustrative case study, a generalized two-compartment evaluative model of chemical uptake was developed based on the FISH model of Mackay (25) and

parameterized for three generic species by specifying the respired medium (air or water) and the composition of the organism's diet. The two-compartment model represents the gastrointestinal tract and the entire remaining internal body volume of the organism.

The 24 gasoline hydrocarbon blocks in our case study are assumed to have a narcotic mode of action. Narcotics cause depression of locomotion and sensory functions by non-specific interactions with cellular proteins and lipids (26). Because the target tissues for narcotics are located throughout the body, the whole-body internal concentration calculated by the model is appropriate for comparison with the critical concentration of 2 mmol/kg, which has been estimated as the approximate toxic threshold for narcotics (7, 8). Critical concentrations corresponding to chronic effects for narcotic chemicals are expected to be less than an order of magnitude below this value (27).

In cases where chemicals of interest have other tissue-specific modes of action, physiologically based pharmacokinetic (PBPK) models can be applied to estimate the distribution of contaminants within the body, and to calculate tissue concentrations at the site of toxic action. For example, Cahill et al. (28) recently described a general PBPK model that can be parameterized to represent a variety of species and calculates contaminant concentrations in different tissues either under steady-state conditions or as a function of changing physiological and uptake parameters.

In addition to being a recognized narcotic, benzene is also a human carcinogen (29). However, because cancer is typically not a population relevant endpoint used in ecological risk assessments we consider benzene as contributing only to the total narcotic tissue burden of the organism.

For some organisms and chemicals, metabolism is an important process for transformation and subsequent removal of chemicals from the body following uptake. The current dearth of data on species- and chemical-specific metabolism rates can introduce uncertainty at this stage of the assessment. In the current case study we ignore metabolism as a mechanism for removal of chemical from the body. We justify this by

noting that ignoring metabolic losses will conservatively over-estimate the concentration of narcotic substances in the whole body of the organism. If metabolic rate data or empirical bioaccumulation factors are available they can be used in preference to the conservative assumption of zero metabolism. We also do not explicitly account for metabolized dose or metabolite concentrations. Instead, we estimate the risk of toxic effects by comparing total organism concentrations of narcotic molecules to the critical body residue value. Including metabolism will significantly alter the results of the assessment if metabolism followed by excretion of metabolites is a dominant loss mechanism relative to the rates of egestion, respiration losses and growth dilution.

Results

The results of the environmental fate modeling include concentrations and fugacities calculated for each of the 24 hydrocarbon blocks in air, water, soil and sediment for each of the modes of release to the environment, i.e., emissions to air, water and soil. The results from the organism exposure and biouptake modeling include concentrations and fugacities in the three species, expressed as whole-body internal concentration in units of mmol/kg (wet weight). These internal body concentrations are a function of the emission rate for each hydrocarbon block, which depend on the total emission rate of gasoline (100 kg/h) and the fraction of each block in the gasoline mixture (Figure 1). The full results are presented in the Supporting Information.

In addition to calculating the concentration of the individual blocks, the total concentration of the mixture of hydrocarbons is also calculated for the environmental media and the organisms. Assuming additive toxicity of narcotics, the total body or lipid concentration can be compared with critical values to provide an estimate of the ratio of the calculated levels to those that are likely to cause effects, in a manner analogous to a Predicted Environmental Concentration to Predicted No Effect Concentration or PEC/PNEC ratio. This also shows which components of the mixture contribute most to the toxic burden. It would not be meaningful to add the concentrations for the different individual chemicals or blocks if the modes of toxic action differed.

Figures 2, 3 and 4 summarize the results for emissions to air, water, and soil, respectively. Displaying the results of these calculations succinctly is challenging, but these figures illustrate where gasoline partitions, the regional inventory, the overall residence time (which depends on the degradation half-lives, partitioning, and advection rates of air and water), the uptake routes for the three organisms and the corresponding internal body residues.

Discussion

The results of the case study clearly demonstrate that environmental concentrations and body residues for both the individual blocks and the overall gasoline mixture differ considerably depending on target species and the mode of release to the environment. Differences in transfer efficiencies for the individual blocks result in significant differences between the compositions of the gasoline hydrocarbons in the target organism tissue and that of the emitted mixture. In the following paragraphs, we examine in sequence the transfer pathways from emission to internal tissue concentration for each mode of release.

Emissions to air

At steady-state, almost all of the gasoline components that are released to air remain in the air compartment (Figure 2). As a result, exposure pathways for birds and mammals are dominated by inhalation, and the body residues in these species represent near-equilibrium partitioning between the atmosphere and the animal. The internal body burden for birds and mammals is dominated by components of the mixture such as xylenes (Block 19) and other alkylated aromatic compounds (Blocks 21 - 24) that have relatively high octanol-air partition coefficients (K_{OA}). The composition of hydrocarbons in the water compartment is skewed toward those with low air-water partition coefficients (K_{AW} or Henry's Law constant), which partition in higher proportion from the atmosphere to water. Fish have a lower body burden than birds or mammals under this release scenario. The internal concentration is determined by near equilibrium

partitioning across the gills between water and lipids of the fish, and thus the tissue concentration is dominated by components of the mixture that exhibit both low K_{AW} and high octanol-water partition coefficient (K_{OW}).

Emissions to water

Releases of gasoline to water (Figure 3) result in fish accumulating the highest calculated internal body residue concentration of any of the release scenarios examined. Under this scenario the composition of the gasoline mixture in water is very similar to that of the emitted mixture. Linear and branched paraffins (Blocks 3 – 7), which are hydrophobic but did not partition to water in the emission to air scenario because of relatively high K_{AW} , are now present in water and bioconcentrate into fish across the gills. Exposure pathways and composition of internal residue for birds and mammals are similar to the results from the emissions to air scenario, but are reduced by approximately a factor of three due to resistance to volatilization from water to air.

Emissions to soil

Gasoline emissions to soil (Figure 4) result in relatively high internal body residue concentrations in birds because 50% of their diet is comprised of soil-dwelling insects and worms. Thus under this scenario, ingestion is the dominant route of exposure and intake for birds, and composition of the internal body burden is highly skewed toward hydrocarbon blocks that exhibit high K_{OW} and low K_{AW} or high K_{OA} and thereby accumulating in soil and soil-dwelling organisms. In contrast, the mammal is an herbivore that consumes vegetation assumed to be in equilibrium with atmospheric concentrations of the gasoline components. Concentrations and fugacities in air under this release scenario are a fraction of those for direct emissions to air, however inhalation is again the most important exposure route for mammals and the composition of the internal dose represents near-equilibrium partitioning between the animal and air. As compared to the emission to air scenario, the internal body burden is lower by a factor of approximately four and composed of a lower fraction of naphthalenes (Blocks 23 and 24), which are not efficiently volatilized from the soil. Body burdens in fish under this scenario are higher than for releases to air because run-off is more efficient at transferring the intermediate K_{OW} components of the mixture into water than air-water exchange. As

in the other scenarios, uptake from water by fish is dominated by exchange across the gills.

Considering the fate, transport and accumulation pathways illustrated in Figures 2-4 it is clear that understanding and evaluating the possible ecological impacts of individual chemicals and chemical mixtures is a complex and demanding task. In this case study the same emission rate of 100 kg/h translates into internal body residues that vary over several orders of magnitude depending on whether emissions are to air, water or soil and on the characteristics of the receptor. Internal tissue concentrations are often dominated by a relatively small number of blocks that comprise the original substance (i.e. gasoline). The composition and total concentration depend on the release scenario and characteristics of the receptor species. Quantitative modeling frameworks illustrated in this study are necessary to explore and gain insight into the complex relationships between chemical releases into the environment and target concentrations in ecological receptor populations.

We emphasize that this is a screening level model and there is considerable uncertainty about many of the parameters used in the calculations. The model calculations presented here are not designed to describe local effects caused by point releases to a particular environmental medium. Given the generic description of environmental conditions and the ecological species, the present model is unlikely to yield concentrations that can be meaningfully compared with monitoring data. However, model insights can be valuable in guiding the development of monitoring strategies to refine model predictions for key pathways/receptor populations. Further definition of site-specific, emission, chemical and ecosystem property data should allow future work to evaluate the reliability of the model predictions.

Although the current screening-level results do not explicitly apply to any real environmental conditions, the combined fate, exposure and biouptake models can reveal the dominant pathways for exposure and uptake and identify the most sensitive parameters. These results can therefore assist efforts to conduct the ecological risk

assessment with greater accuracy by guiding further studies that incorporate realistic estimates of emission scenarios and environmental conditions to allow informative comparisons between modeled concentrations and observations of contaminant concentrations in the environment and wildlife.

Ecological risk assessments are often plagued by lack of emission data. This is unfortunate, but it does not preclude model application. The model can be run for unit emissions and corresponding environmental and organism concentrations can be deduced. The relative efficiency of transport from emission to target concentration in the ecological receptor (i.e. fish, bird, mammal) for different chemicals can be assessed. As emission data become available the results can be scaled to include these data since the model equations are linear, i.e., a factor of 100 increase in emission rate to a given environmental compartment causes a corresponding factor of 100 increase in the body residue attributable to that source. The linear relationship between source and internal tissue concentration is valid as long as the emissions do not saturate the environmental system, i.e., the fugacity of the chemical in the environment is lower than its vapor pressure. The unit emission assessment facilitates comparison of the relative risks associated with emissions to air, water and soil.

It can be informative to deduce the body or tissue residue resulting from a unit emission, and then determine the factor by which the residue is lower than the critical level. This factor can then be used to estimate a “critical emission rate” that produces the corresponding critical tissue level. The critical emission rate can then be compared to order-of-magnitude estimates of actual emissions to identify situations that warrant more detailed analysis. While production, usage and emission data are often unavailable for a specific region or industrial facility for reasons of commercial confidentiality, estimates of aggregate national or per capita are often available.

Similarly, when there is a lack of empirical toxicity data but the molecular structure suggests a specific mode of action, it is possible to assign a target tissue or whole body concentration to that mode of action, considering appropriate uncertainty limits. While

toxicity classifications that take mode of action into consideration are available (30, 31), future work is needed for translating these framework schemes into critical internal concentrations that are protective of adverse effects.

In the extreme case in which the mode of action is uncertain or unknown, a worst plausible case scenario can be assumed. The corresponding critical emission rate that will produce that concentration can be deduced and compared with likely ranges of actual emissions. If these rates or ranges in rates are comparable there is an incentive to conduct experimental tests or monitoring programs to better characterize the substance's toxicity and/or environmental exposure.

We believe that it is preferable to use internal tissue concentrations representing the "delivered dose" to a target site rather than external concentrations when assessing the likelihood of an adverse effect. When risks of toxic effects are assessed using external concentrations the relationships are confounded by factors that influence the efficiency or rate of uptake. While future research is needed to better define tissue concentrations that correspond to adverse effects for various modes of toxic action, addressing the uptake process separately makes it possible to obtain more generalized relationships between toxicity and molecular structure. It is probable that at least some of the variability among species susceptibility is attributable to the predictable differences in uptake sources and rates as influenced by chemical properties.

The models described in this study are best suited to screening level assessments such as comparative assessments between the same chemicals released in different environments or with different species, or between chemicals with different modes of toxic action. These models are valuable to make estimates of expected chemical concentrations and associated risks and to develop an understanding of key processes, but there are limitations and potential pitfalls in extrapolating the results across chemicals and chemical classes or to specific regions or sites. Modeling tools are constantly being improved and evaluated against monitoring and exposure data, and new models are likely to emerge with enhanced capability to track chemicals quantitatively from point of

release through partitioning and fate in the environment, entrance into exposure pathways and migration into and within the receptor organism. As models improve, are tested against monitoring data and become more credible they can be applied with greater confidence to site specific and region specific situations resulting in more accurate and detailed ecological risk assessments.

Whereas the present focus has been on ecological risk assessment using critical body residues as the endpoint, an analogous methodology could in principle be applied to human health risk assessment. Not only can individual or population-level intakes of chemical, in units such as milligrams per kilogram body weight per day, be assessed but it should also be possible to calculate “biomarkers of exposure” such as internal concentrations of parent substances or metabolites in specific body fluids or tissues. The use of such a model framework would allow the linkages between emissions and human population exposures to be quantitatively assessed and reconciled with measured human biomonitoring data.

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Table 1. Gasoline hydrocarbon blocks 1 through 24 defined by structural class and number of carbon atoms.

Structural Class	Number of Carbon Atoms									
	3	4	5	6	7	8	9	10	11	12
n-alkane	1		2	3	4		7			
iso-alkane			5		6					
n-olefins			9			20				
iso-olefins	8		10							
cyclic-alkanes			11		13		14			
cyclic-olefins			15							
mono-aromatics			16	17	19		21		22	
di-aromatics								23	24	
cyclohexane			12							
ethyl benzene						18				

Table 2. Estimated physico-chemical properties, partition coefficients and degradation half-lives at 12°C for the 24 hydrocarbon blocks.

Hydrocarbon Block #	Molecular Weight (g/mol)	Density (g/mL)	K _{OW}	K _{AW}	K _{OA}	Estimated Degradation Half-lives (h) in:			
						Air	Water	Soil	Sediment
1	57.68	0.5640	1000	81.78	13	35	300	900	2700
2	72.15	0.6214	3981	32.95	124	30	150	450	1350
3	86.17	0.6548	19953	44.7	417	30	150	450	1350
4	102.88	0.6812	158489	50.26	3071	30	150	450	1350
5	78.62	0.6300	501	38.83	13	40	150	450	1350
6	107.55	0.6774	12589	82.99	161	40	300	900	2700
7	136.55	0.7094	158489	74.46	2150	35	300	900	2700
8	56.05	0.5879	316	19.59	18	10	225	675	2025
9	78.52	0.6613	1000	6.86	145	10	150	450	1350
10	84.22	0.6747	1995	7.12	303	10	300	900	2700
11	81.26	0.7419	3162	7.13	418	30	150	450	1350
12	84.16	0.7739	3981	4.65	856	30	300	900	2700
13	100.79	0.7618	12589	11.55	993	30	300	900	2700
14	126.24	0.7664	39811	23.1	1805	30	300	900	2700
15	75.83	0.7883	631	1.53	458	10	150	450	1350
16	78.11	0.8765	200	0.13	1460	100	180	540	1620
17	92.14	0.8669	631	0.16	4564	50	180	540	1620
18	106.2	0.8670	1995	0.19	10034	50	180	540	1620
19	106.2	0.8611	1995	0.13	17335	50	180	540	1620
20	112.2	0.7104	31623	10.31	2844	10	300	900	2700
21	126.34	0.8659	7943	0.19	46383	50	300	900	2700
22	155.51	0.8723	158489	0.34	459394	50	300	900	2700
23	128.17	0.8684	3162	0.01	410174	20	300	900	2700
24	142.2	0.8375	10000	0.01	1160886	20	300	900	2700

Table 3. Organism input data used in the evaluative biouptake model.

	Bird	Mammal	Fish
Respired Medium	Air	Air	Water
Bodyweight (kg)	1	1	1
Density (g/cc)	1	1	1
Volume Fraction Lipid	0.05	0.05	0.05
Gill exterior resistance time constant (h)	N/A	N/A	0.001
Gill interior resistance time constant (h)	N/A	N/A	300
Gut absorption exterior resistance	1.E-07	1.E-07	1.E-07
Gut absorption interior resistance	2	2	2
Maximum biomagnification factor - Q	18	40	3
Growth rate as a fraction of volume per day	0.001	0.001	0.001
Respiration rate (m ³ /h)	0.023	0.031	N/A
Diet description	insects	vegetation and seeds	aquatic invertebrates
Reference environmental medium for diet	50% Air, 50% Soil	Air	Water
Reference medium to diet accumulation factor	1	1	1
Feeding rate as a fraction of bodyweight per day	0.15	0.055	0.015

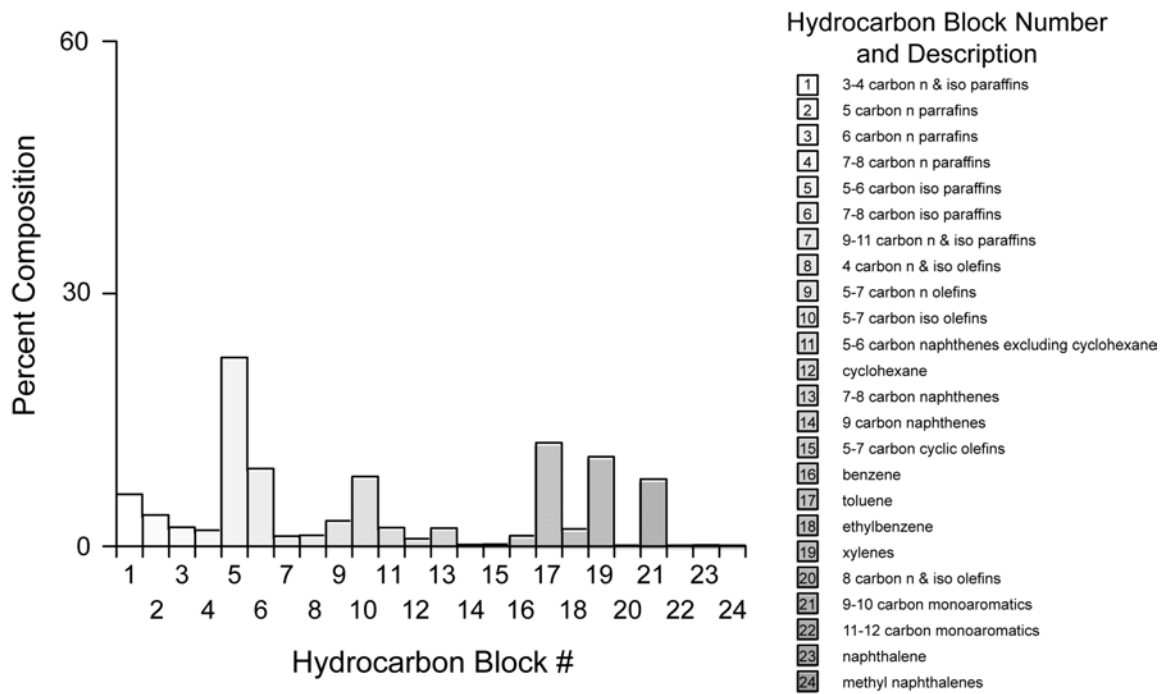


Figure 1. Representative molar composition of gasoline assumed for releases to air, water and soil.

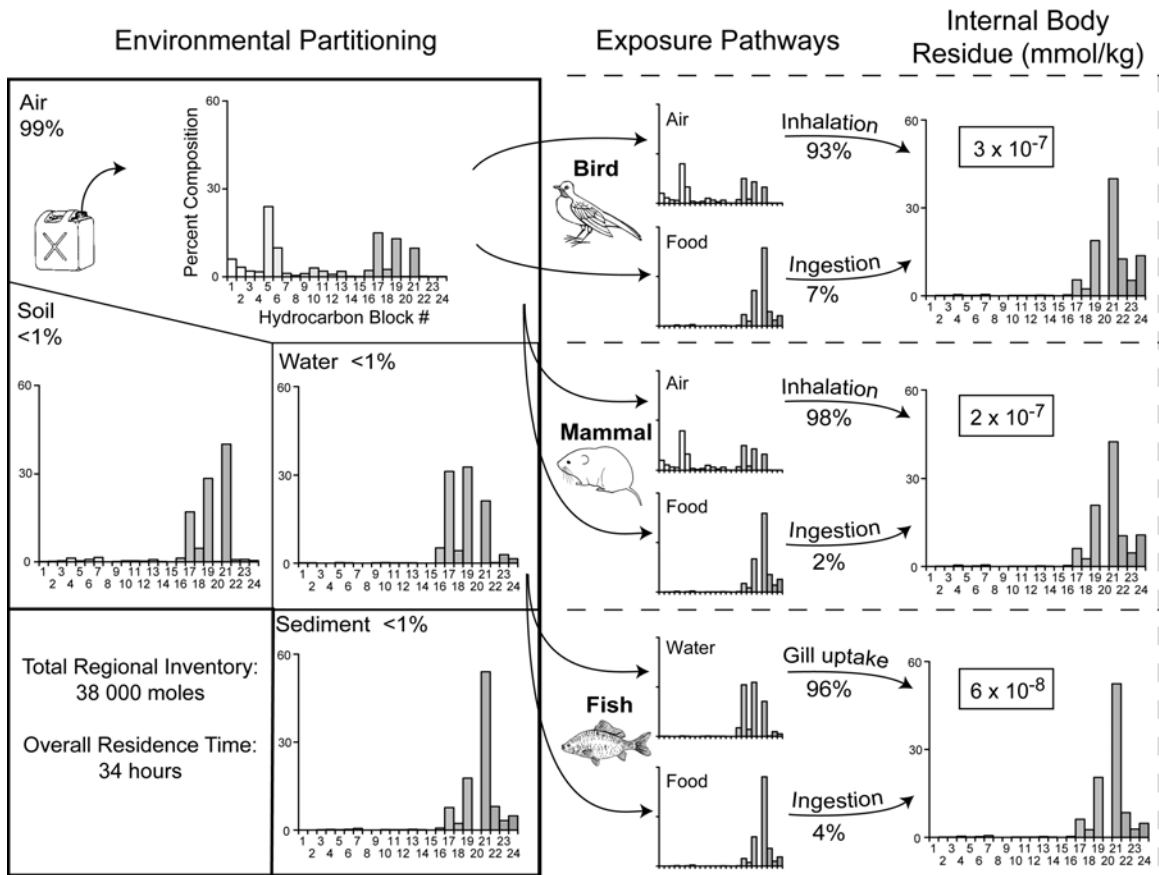


Figure 2: Modeled environmental partitioning, transfer to exposure pathways and accumulated internal body concentrations for gasoline released to air at an arbitrary rate of 100 kg/h. Bar charts illustrate the composition of the gasoline inventory, and can be compared to the composition of the original mixture shown in Figure 1. Horizontal and vertical scales are the same in all bar charts.

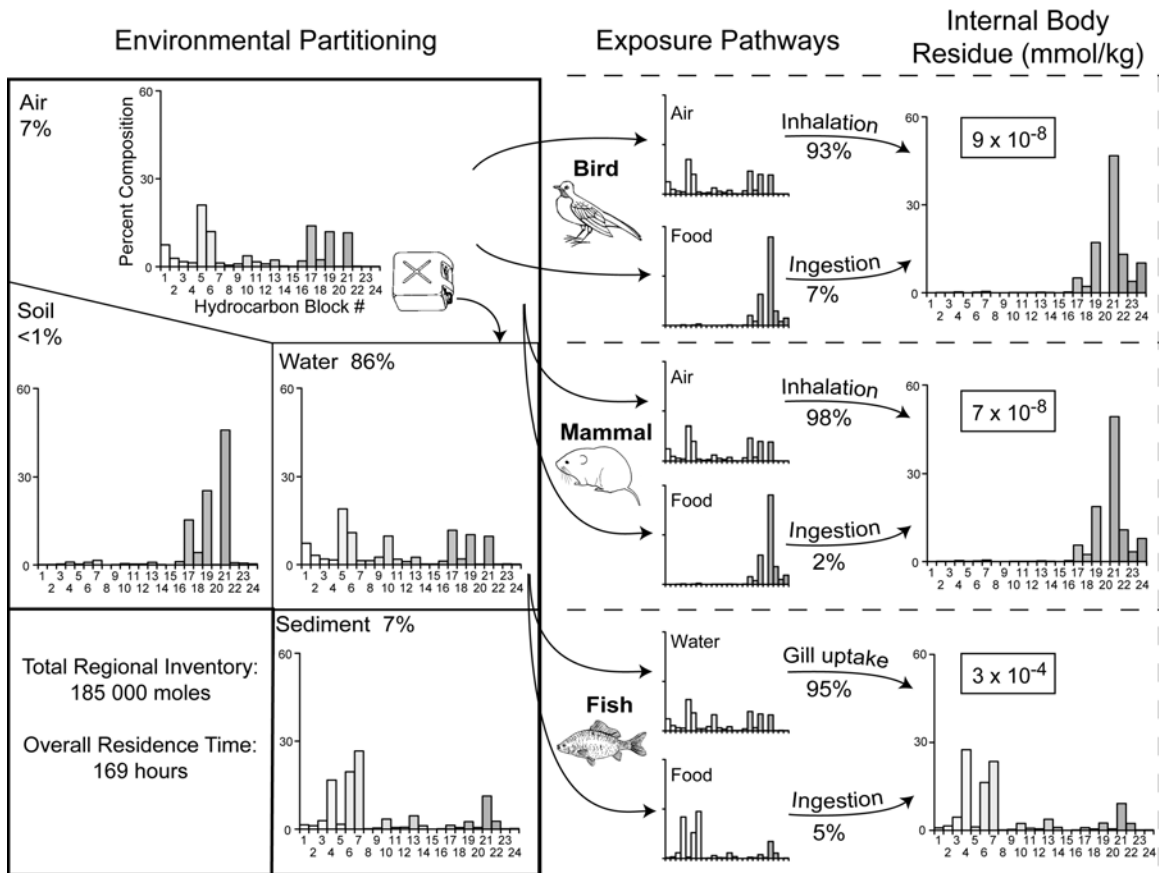


Figure 3: Modeled environmental partitioning, transfer to exposure pathways and accumulated internal body concentrations for gasoline released to water at an arbitrary rate of 100 kg/h. Bar charts illustrate the composition of the gasoline inventory, and can be compared to the composition of the original mixture shown in Figure 1. Horizontal and vertical scales are the same in all bar charts.

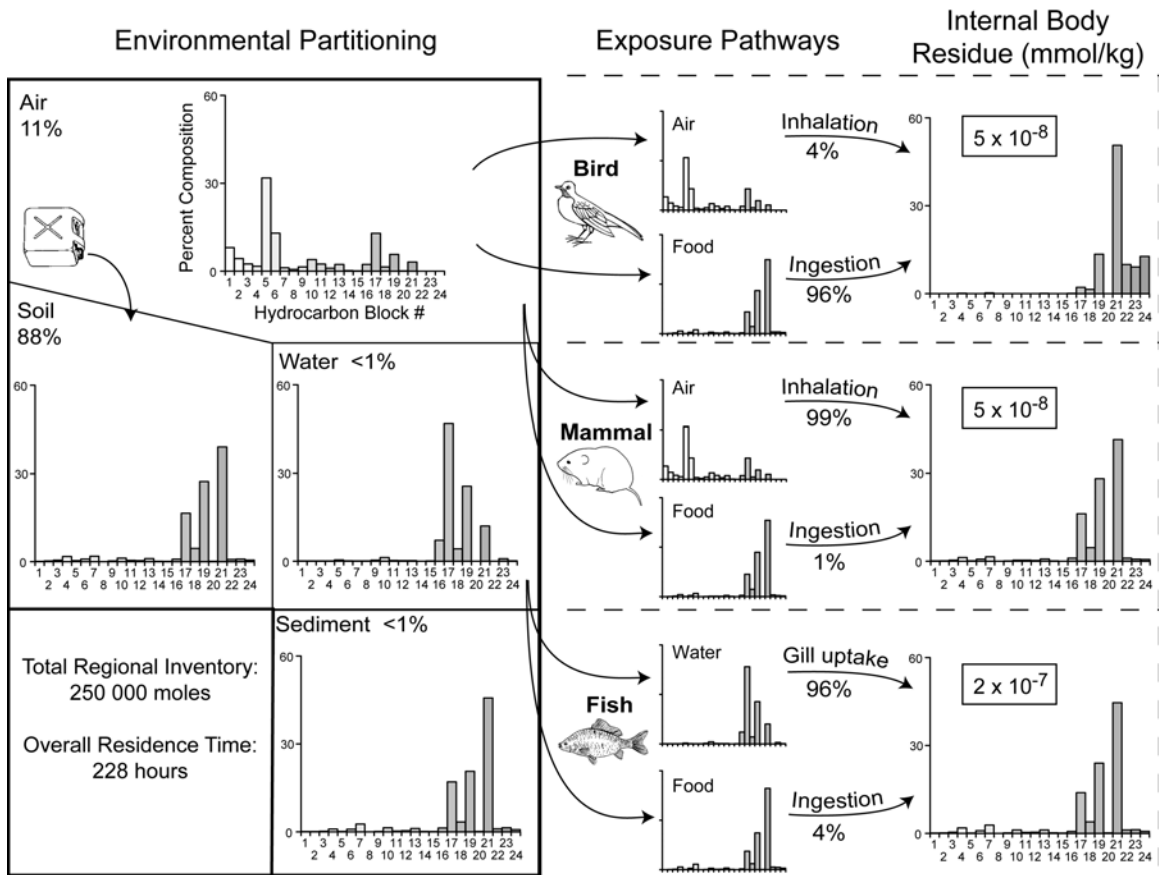


Figure 4: Modeled environmental partitioning, transfer to exposure pathways and accumulated internal body concentrations for gasoline released to soil at an arbitrary rate of 100 kg/h. Bar charts illustrate the composition of the gasoline inventory, and can be compared to the composition of the original mixture shown in Figure 1. Horizontal and vertical scales are the same in all bar charts.

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