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Intake Fraction for Multimedia Pollutants: A Tool for Life Cycle Analysis and Comparative Risk Assessment

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We employ the intake fraction (*iF*) as an effective tool for expressing the source-to-intake relationship for pollutant emissions in life cycle analysis (LCA) or comparative risk assessment. Intake fraction is the fraction of chemical mass emitted into the environment that eventually passes into a member of the population through inhalation, ingestion, or dermal exposure. To date, this concept has been primarily applied to pollutants whose primary route of exposure is inhalation. Here we extend the use of *iF* to multimedia pollutants with multiple exposure pathways. We use a level III multimedia model to calculate *iF* for TCDD and compare the result to one calculated from measured levels of dioxin toxic equivalents in the environment. We calculate *iF* for emissions to air and surface water for 308 chemicals. We correlate the primary exposure route with the magnitudes of the octanol-water partition coefficient, K_{ow}, and of the air-water partitioning coefficient (dimensionless Henry constant), K_{aw}. This results in value ranges of K_{ow} and K_{aw} where the chemical exposure route can be classified with limited input data requirements as primarily inhalation, primarily ingestion, or multipathway. For the inhalation and ingestion dominant pollutants, we also define empirical relationships based on chemical properties for quantifying the intake fraction. The empirical relationships facilitate rapid evaluation of many chemicals in terms of the intake. By defining a theoretical upper limit for *iF* in a multimedia environment we find that *iF* calculations provide insight into the multimedia model algorithms and help identify unusual patterns of exposure and questionable exposure model results.

KEY WORDS: Multimedia modeling; life cycle analysis; comparative risk assessment; exposure; intake fraction

1. INTRODUCTION

A large number of industrial pollutants are released into the environment through a number of release/use scenarios. Managing these releases calls for an efficient process to identify pollutants that will result in potential adverse effects. Understanding the potential risk to humans for a pollutant release during the production, use, and disposal of a product is one of the goals in a life cycle impact assessment (LCIA). Knowing the potential adverse effect of a chemical per unit release is also important for differentiating between chemicals in a toxic release inventory database—the self-reported releases from manufacturing facilities.

The potential adverse effects from a pollutant release depend on the intake to all exposed individuals and the likelihood of adverse effects with increasing intake. Here, we focus only on the intake, which depends on the fate and transport of the chemical in

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the environment (including air, soil, vegetation, surface water, and sediment) and the human exposure through a comprehensive set of exposure pathways (including those leading to inhalation, ingestion, and dermal uptake). An ideal measure of these multiple source-to-intake pathways is a measure that is concise but incorporates fate and transport as well as measures of human exposure into a single metric.

In the recent environmental health literature, the concept of intake fraction has been used over the years with various names to express the fraction of a pollutant release that is inhaled by a receptor population.[1–3] This concept has been applied to determine the potential risk to a population or to better understand tradeoffs among different types of pollutant releases (for example, reductions in pollution sources to the indoor environment more effectively reduce exposure than reductions to the outdoor environment).[1–3]

A working group recently formed to develop consistent terminology and definitions proposed the term "intake fraction" as a common term for this concept.[4] The intake fraction (*iF*) is defined as the integrated incremental intake of a pollutant, summed over all exposed individuals, and occurring at any time, released from a specified source or source class, per unit of pollutant emitted. Intake through inhalation, ingestion, or dermal uptake are all considered. *iF* maintains many of the desirable properties of a population-based source-to-intake measure, such as accounting for persistence, long range transport, and bioaccumulation. When any of these factors increase, *iF* also tends to increase.

Over the last several years, the LCA literature has clearly expressed the need for simple measures that link source to effect. But while early work focused primarily on toxicity as a measure of potential impact,[5] more recent work stresses the need to include fate, transport, and exposure along with toxicity in measures of potential impact.[6–8] In addition to the literature noted above, there are many other examples of concepts similar to *iF*, such as the intake of a population from a unit source emission for multimedia intakes to populations.[9–11]

In this work, we determine the *iF* for a comprehensive set of chemicals using multimedia, multipathway exposure assessments. The benefit of *iF* in LCA and comparative risk is that it provides a simple, transparent, and potentially comprehensive measure of the relationship between emission and human exposure. It has no units and thus avoids the pitfalls of unit conversions, facilitating the comparison of results between researchers in a clear and understandable way. By adding a measure of toxicity, one captures two key elements of the potential health effect from an environmental release—exposure and toxicity. Because it is expressed as a fraction, it is more easily inspected for plausibility, such that the potential maximum value can be quantified to determine if the calculated value is within realistic bounds. Also, the intake fraction can be calculated based on either measured data or model results.

In the sections below, we discuss the use of intake fraction and propose the CalTOX multimedia fate and exposure model for calculating *iF*. An example with TCDD is presented and compared to empirical results to provide a screening-level evaluation of the model. We calculate *iF* for releases to air and surface water for a set of 308 pollutants that are currently in use and reported under the U.S. Toxic Release Inventory (TRI)[12] and for which we have chemical properties data, that is, partition factors and degradation rates. The resulting set of *iF* values provides a resource for making choices or comparisons among pollutants. Additionally, we use *iF* to identify how both the primary exposure pathway and the magnitude of the intake fraction depend on chemical properties. Where applicable, we develop relationships between *iF* and the chemical properties of a pollutant. By identifying exposure routes and chemical properties that result in high levels of exposure, we can better allocate efforts to more thoroughly quantify a particular pathway for a given class of chemicals. The relationship between *iF* and chemical properties provides predictive power for understanding the potential intake of new chemicals under development and chemicals for which there is currently inadequate exposure data.

2. METHODS

2.1. Intake Fraction

As stated above, the intake fraction (*iF*) is defined as the integrated incremental intake of a pollutant, summed over all exposed individuals, and occurring at any time, released from a specified source or source class, per unit of pollutant emitted. The individual intake fraction (iF_i) is defined as that component of the intake fraction associated with intake by a specified individual. Thus, the intake fraction can be represented as the individual intake fractions summed over

all members of the potentially exposed population, including current and future generations. In reality, there is both spatial variation in concentration and interindividual variability in exposure patterns that one may want to account for when summing over the various subpopulations of the total population. However, in this analysis, we calculate the intake by a representative, or per caput, individual from a spatially averaged concentration. This is a common approach in multimedia modeling. An additional simplification can be made by basing calculations on an environmental system that has reached steady state, at which time the intake fraction is the ratio between the rate of intake (kg/d) by the population and the release rate (kg/d) to the environment. One advantage of a population-based measure is that it accounts for the number of exposed individuals, which may be large if the pollutant travels a long way in the environment.

When using a multimedia model, we need to consider intake through all routes and it is desirable to specify both the route of exposure and the medium or media through which the pollutant is released. These are defining attributes of the intake fraction and are specified in parentheses after the intake fraction symbol. For example, if we calculate the intake fraction through the ingestion pathway for a chemical released to the air, we write *iF* (ingestion, air). In this article, we assume the intake response functions are the same for each route and thus sum intake across all exposure routes (inhalation, ingestion, and dermal uptake). However, intake-response relationships can vary by exposure route and should not necessarily be summed among these different routes. If summed, the mathematical expression for exposure to an air release through all exposure pathways then becomes:

$$iF\,(\text{total, air}) = iF\,(\text{inhalation, air}) + iF\,(\text{ingestion, air}) + iF\,(\text{dermal, air}) \quad (1)$$

where the term "total" indicates that intake is summed across all exposure routes.

In a multimedia, multipathway model, a source-to-intake relationship is typically expressed as an intake rate (mg/kg-BW/d) per unit emission rate (mg/d). To convert from a source-to-intake relationship to an intake fraction, the following conversion must be made:

$$iF = Source\ to\ Dose\left(\frac{mg/kg/day}{mg/day}\right) \times BW(kg) \times Population \quad (2)$$

where BW is the population average body weight (kg) and *Population* is the size of the exposed population.

2.2. Fate and Transport

Multimedia fate and exposure modeling has been recognized as an appropriate tool to provide a screening-level assessment of source-to-intake relationships.[13] Often, Mackay-type level III fugacity models are used to account for the intermedia transfers to predict environmental distributions.[8,14–16] For the *iF* calculations, we have selected the CalTOX model, a fugacity-based multimedia model that includes air, three soil layers, vegetation, surface water, and sediment. The model recently has been revised to reflect new information on the transport to and from vegetation and to account for a chemical-specific soil depth.[17–20] Some of the key assumptions in the CalTOX model are the use of a uniform volume source in the release medium, a single compartment atmosphere, uniform landscape parameters, and uniform population density. Additional information on the model and the associated assumptions can be found in Hertwich *et al.*[14] and the model and technical support documents can be downloaded from the CalTOX website.[21]

2.3. Intake

Individuals are exposed to pollutants in the air, water, food, and soil that they contact in their day-to-day activities. Once contacted, these pollutants enter the human receptor through inhalation, ingestion, and/or dermal uptake. The CalTOX model includes multiple pathways that link a chemical concentration in atmosphere, soil, surface water, and vegetation to chemical uptake though the inhalation, ingestion, and dermal contact routes. Inhalation pathways include contact with both indoor and outdoor air. Ingestion pathways include tap water consumption; incidental soil ingestion; and intake of fruits, vegetables, grains, and animal products, such as meat, poultry, eggs, fish, and dairy. Dermal exposure pathways include contact with contaminated water during bathing and recreation, as well as contact with soil and house dust. Exposure media concentrations may differ from the ambient environmental media concentrations and can be calculated from the ambient air, soil, vegetation, and surface water concentrations (e.g., the concentration in vegetation can be affected by irrigation water).

Potential intake is calculated from the contact rate with the exposure media and the pollutant concentrations in these exposure media (i.e., tap water, indoor air, etc.). One must consider the concentration in the environmental medium, the relationship between the exposure medium concentration and environmental medium concentration, intake rate, body weight, activity patterns, and exposure duration.[22] The equations used for each exposure pathway are taken from the CalTOX model.[20] The risk to an individual due to exposure to a carcinogen can be calculated by multiplying the average daily intake by a cancer potency factor (CPF). Hazard due to exposure to a noncarcinogenic compound can be calculated from the allowable daily dose. However, we do not calculate health outcomes in this article.

2.4. Scenario Definition

The scenario we use in these calculations is an open environmental system with landscape and climate parameters that reflect U.S. averages and population-based lifetime average exposure parameters, that is, breathing rates, diet, activity patterns, etc. The atmospheric mixing height is assumed to be 700 m.[23] The simulated continuous release and multimedia dispersion lasts for 100 years, with exposure occurring during the last 70 years of the release. The initial 30 years brings the model to steady-state conditions for all chemicals. The choice of the average U.S. landscape parameters is arbitrary and a similar analysis could be completed using the landscape parameters in any location.

The choice of scenario imposes some notable limitations on the analysis. Because our multimedia model is an open system, it does not account for the eventual fate of pollutants carried out of the system by advection, a potential problem for persistent substances. However, because we use a continental scale multimedia model, the advective losses from the system are minimal and the majority of mass carried out of the system goes over an ocean where there is no human exposure. Additionally, we do not account for the variation of either pollutant concentrations or population densities. Neither the relationship between releases and food production regions, nor the movement of food from one region to another, are taken into consideration. However, for this case study, the choice of a large region makes it reasonable to assume that agricultural production equals consumption and that the food concentrations are uniform. Fish consumption rates are based on the U.S. inland fish production rates.[24]

3. RESULTS

3.1. TCDD Example

We carried out a case study for dioxin-like compounds, a chemical class for which there is widely available emissions and environmental concentration data.[25] These compounds are used to determine if the model yields reasonable results. The U.S. EPA dioxin risk assessment reports that over 80% of the releases of dioxin toxic equivalents (TEQ) are to air with total releases from all sources in the United States on the order of 3,300 g TEQ_{DF} in 1995.[25] This is down from 14,000 g TEQ_{DF} reported for 1987 (there may also be a number of dioxin emissions that are not recorded). Based on measured concentrations in air, vegetable fat, meat, dairy, milk, eggs, poultry, pork, fish, and soil, the estimated human intake is 63 picograms TEQ_{DF} per day.[25] Combining these 1995 values leads to an iF_i of 7×10^{-12} and a iF of 2×10^{-3} for the United States. Since the sources of dioxins have been declining in recent years, emissions from environmental reservoirs could be acting as an additional potential source. If accounted for, the environmental reservoirs would act to decrease the measured iF.

The iF based on the dioxin/furan TEQ measurements are higher by a factor of three than the value calculated with the CalTOX model. We base the CalTOX calculations on 2,3,7,8 TCDD for an emission to air. The CalTOX-calculated iF_i is 2.1×10^{-12} and iF is 6×10^{-4}.

3.2. Multiple Chemical Case Study

A set of 308 organic chemicals with a wide range of physicochemical parameters was assembled from the U.S. EPA Toxic Release Inventory List. Chemical property values for these chemicals are primarily those compiled by Hertwich *et al.*[14] and can be found on the CalTOX website.[21] We use this analysis to illustrate trends for various chemical properties. Table I lists iF values calculated for each chemical based on multiple exposure pathways and for releases to both air and surface water.

3.2.1. Air Release

The iF_i (total, air) includes all the exposure routes for a release to air and ranges from 3.1×10^{-17} (for

Table I. *if* Values Calculated for Each Chemical Based on Multiple Exposure Pathways and for Releases to Both Air and Surface Water

Chemical Name	*iF*(tot,air)	*iF*(tot,water)	Chemical Name	*iF*(tot,air)	*iF*(tot,water)
Acenaphthene	2.86E-07	8.18E-06	Bromoform	2.42E-05	3.49E-05
Acephate	8.65E-05	1.16E-05	Bromoxynil	1.10E-05	1.57E-05
Acetaldehyde	2.88E-07	1.42E-06	1,3-Butadiene	8.00E-08	4.97E-06
Acetamide	4.64E-06	4.43E-07	Butanol	2.58E-06	1.59E-06
Acetone	1.82E-05	5.54E-06	2-Butenal	2.87E-07	1.36E-06
Acetonitrile	2.05E-05	1.22E-05	Butyl benzyl phthalate	9.64E-06	1.31E-06
Acetophenone	1.16E-05	4.61E-06	Butyric acid, 4-(2,4-Dichlorophenoxy)	6.83E-06	3.19E-06
Acrolein	3.57E-07	5.03E-06	Captafol	6.91E-06	8.42E-06
Acrylamide	1.66E-05	3.32E-07	Captan	3.58E-07	4.65E-08
Acrylic acid	1.03E-06	4.54E-07	Carbazole	6.01E-08	7.86E-06
Acrylonitrile	1.38E-06	1.65E-06	Carbendazim	6.51E-05	1.40E-05
Aldicarb	6.36E-05	3.90E-05	Carbofuran	2.00E-05	7.60E-06
Aldrin	4.40E-05	1.05E-03	Carbon disulfide	1.32E-05	2.64E-05
Allyl alcohol	2.51E-07	5.82E-07	Carbon tetrachloride	2.55E-05	3.63E-05
Allyl chloride	3.14E-07	1.35E-06	Carbonyl chloride	2.50E-05	9.80E-09
Allyl trichloride	1.12E-05	2.25E-05	Catechol	9.78E-06	4.51E-07
4-Aminobiphenyl	3.65E-06	7.45E-07	Cellosolve	1.87E-06	2.78E-06
2-Aminonaphthalene	3.14E-07	2.49E-06	CFC-11	2.56E-05	3.65E-05
Anilazine	7.07E-07	1.19E-06	CFC-12	2.30E-05	2.57E-05
Aniline	3.57E-07	1.92E-06	Chlordane	7.51E-05	3.06E-04
O-Anisidine	7.78E-08	1.45E-06	Chlorfenvinphos	7.95E-06	7.21E-06
Anthracene	1.74E-07	1.07E-07	Chlorinated fluorocarbon	2.57E-05	3.89E-05
Aroclor 1016	8.11E-06	6.26E-05	2-Chloro-1,3-butadiene	3.01E-07	8.91E-06
Atrazine	1.64E-05	5.37E-08	1-Chloro-2,3-epoxypropane	9.42E-06	5.95E-06
Azinphos-methyl	1.41E-05	7.72E-07	1-chloro-4-nitrobenzene	2.45E-05	3.28E-05
Aziridine	1.73E-06	1.36E-05	Chloroacetic acid	1.84E-05	4.48E-07
Baygon	2.18E-05	4.13E-06	P-Chloroaniline	6.57E-07	1.98E-06
Benomyl	2.37E-05	1.67E-06	Chlorobenzene	1.21E-05	2.21E-05
Bentazone	9.81E-05	9.54E-05	1-Chlorobutane	1.17E-05	3.04E-05
Benzene	6.22E-06	8.75E-06	Chlorodibromomethane	2.38E-05	3.08E-05
Benzene, M-Dimethyl	2.71E-07	6.48E-06	Chlorodifluoromethane	2.56E-05	2.98E-07
Benzene, O-Dimethyl	4.55E-07	6.60E-06	Chloroethane	1.02E-05	1.54E-05
Benzene, P-Dimethyl	4.35E-07	6.68E-06	Chloroform	2.27E-05	2.98E-05
Benzenethiol	2.62E-06	1.97E-05	Chloromethyl methyl ether	2.18E-06	1.04E-09
Benzidine	2.40E-05	1.96E-06	2-Chlorophenol	1.08E-05	1.12E-05
Benzo(a)anthracene	7.52E-06	4.36E-08	2-Chloropropane	1.48E-05	2.65E-05
Benzo(a)pyrene	2.43E-05	8.30E-08	Chlorothalonil	3.39E-06	7.33E-07
Benzo(b)fluoranthene	6.41E-05	1.29E-05	Chlorpropham	8.81E-06	1.34E-05
Benzo(b)pyridine	1.17E-06	1.52E-06	Chlorpyrifos	1.38E-05	1.76E-05
11,12-Benzofluoranthene	7.93E-06	2.09E-04	Chrysene	3.05E-05	3.27E-07
Benzoic acid	2.82E-06	9.33E-07	Coumaphos	2.54E-06	2.76E-06
Benzoic trichloride	9.19E-06	1.01E-09	M-cresol	1.89E-07	1.13E-06
Benzyl Chloride	2.12E-06	5.29E-07	O-Cresol (2)	2.34E-07	5.36E-07
N,N′-Bianiline	2.56E-06	2.66E-06	P-cresol	2.33E-07	2.38E-08
Bifenthrin	2.56E-05	7.70E-06	Cumene	9.85E-07	3.85E-06
Biphenyl	8.42E-07	4.35E-06	Cyanazine	3.72E-05	1.30E-05
Bis(2-chloro-1-methylethyl)ether	4.85E-07	7.38E-06	Cyclohexane	8.88E-07	1.78E-05
Bis(2-chloroethyl)ether	2.93E-06	1.06E-05	Cyclohexanone	2.87E-06	3.35E-06
Bis(2-ethylhexyl)phthalate	1.21E-05	7.60E-06	Cygon	5.59E-05	1.23E-05
Bis(tributyltin) oxide	9.19E-06	2.27E-05	Cypermethrin	5.90E-05	2.02E-05
Bromodichloromethane	2.50E-05	2.72E-05	Cyromazine	1.08E-04	2.83E-05
2,4-D [Acetic acid (2,4-Dichlorophenoxy)-]	2.07E-05	1.35E-06	2,4-Dinitrophenol	1.12E-05	2.51E-06
DDD	1.98E-04	2.24E-04	2,4-Dinitrotoluene	5.96E-06	2.40E-07
DDE	4.06E-04	5.33E-05	2,6-Dinitrotoluene	5.52E-06	1.28E-07
DDT	1.54E-04	1.03E-04	Di-n-octyl phthalate	4.57E-05	1.37E-05

(continues)

Table I. *(Continued)*

Chemical Name	*iF*(tot,air)	*iF*(tot,water)	Chemical Name	*iF*(tot,air)	*iF*(tot,water)
DDVP (Dichlorvos)	1.01E-06	2.46E-06	1,4-Dioxane	9.40E-07	4.35E-06
Deltamethrin	1.90E-05	3.59E-08	Diphenylamine	1.85E-06	1.89E-05
Demeton	1.87E-05	3.80E-06	Disulfoton	4.58E-06	1.25E-05
4,4′-Diamino ditan	1.92E-05	4.72E-07	Diuron	1.83E-05	1.69E-05
2,4-Diaminotoluene	2.24E-05	6.46E-07	Endosulfan	9.38E-07	1.93E-06
Diazinon	2.06E-05	3.04E-05	Endrin	1.64E-05	7.14E-05
Dibenz(a,h)anthracene	1.16E-05	1.45E-05	Ethoprop	1.92E-05	8.22E-05
1,2-Dibromoethane	1.32E-05	1.85E-05	Ethyl acetate	2.83E-06	1.80E-06
Dibromomethane	2.00E-05	2.88E-05	Ethyl acrylate	2.47E-07	1.02E-06
Dicamba	1.13E-05	1.30E-05	Ethyl dipropylthiocarbamate	9.94E-07	3.97E-06
1,3-Dichlorobenzene	1.40E-05	2.62E-05	Ethyl ether (diethyl ether)	9.50E-07	6.79E-06
1,2-Dichlorobenzene (o)	1.68E-05	3.22E-05	Ethyl methacrylate	1.53E-06	1.35E-05
1,4-Dichlorobenzene (p)	1.74E-05	1.55E-05	Ethylbenzene	2.46E-06	5.25E-06
3,3-Dichlorobenzidine	2.80E-07	1.84E-09	Ethylene glycol	2.95E-06	8.69E-07
Dichlorobenzene (mixed isomers)	1.11E-05	4.60E-05	Ethylene oxide	1.99E-05	1.32E-05
1,1-Dichloroethane	1.93E-05	2.64E-05	Ethylenethiourea	3.13E-05	2.75E-06
1,2-Dichloroethane	1.82E-05	2.88E-05	Fenitrothion	7.66E-06	8.47E-06
1,1-Dichloroethylene	1.31E-06	1.57E-05	Fenthion	1.65E-05	2.48E-04
1,2-Dichloroethylene	2.43E-06	1.07E-05	Fentin acetate	1.05E-05	1.72E-06
cis-1,2-Dichloroethylene	6.73E-06	1.66E-05	Fluoranthene	1.47E-05	2.63E-06
trans-1,2-Dichloroethylene	6.72E-06	1.68E-05	Fluorene	2.32E-06	2.88E-05
2,4-Dichlorophenol	2.79E-06	3.35E-08	Folpet	1.03E-05	5.83E-08
1,2-Dichloropropane	1.15E-05	2.38E-05	Formaldehyde	2.56E-07	4.66E-07
1,3-Dichloropropene	2.28E-06	5.02E-06	Formic Acid	5.99E-06	4.69E-07
cis-1,3-Dichloropropene	2.51E-06	5.28E-06	Furan	9.48E-07	3.83E-06
trans-1,3-Dichloropropene	1.57E-06	4.94E-06	Glyphosate	8.65E-05	1.17E-0
Dichlorprop	8.02E-05	2.61E-06	alpha-HCH (alpha-BHC)	5.62E-06	1.36E-05
Dicofol	1.47E-05	5.30E-05	beta-HCH (beta-BHC)	2.53E-05	1.98E-05
Dieldrin	3.24E-05	1.99E-04	Gamma HCH (Lindane)	6.42E-06	2.06E-05
Diethanolamine	4.47E-05	2.79E-07	Heptachlor	1.77E-06	4.84E-06
Diethyl phthalate	6.33E-06	9.42E-06	Heptachlor epoxide	3.03E-06	3.26E-05
Diethyl sulfate	1.69E-07	2.88E-08	1,2,3,4,6,7,8-Heptachlorodibenzofuran	9.07E-04	4.74E-05
1,1-Difluoro-1-Chloroethane	2.51E-05	3.14E-07	Hexachloro-1,3-butadiene	2.47E-05	5.09E-05
2,2-(4,4′-Dihydroxydiphenyl) propane	9.61E-06	1.29E-06	Hexachlorobenzene	8.44E-05	1.33E-04
Dimethyl phthalate	6.56E-06	1.37E-06	Hexachlorocyclopentadiene	2.94E-07	4.38E-06
Dimethyl sulfate	3.04E-06	1.94E-08	Hexachloroethane	2.55E-05	4.71E-05
Dimethylamine	2.94E-07	1.04E-06	Hexane	9.49E-07	2.02E-05
1,1-Dimethylhydrazine	9.61E-06	2.89E-06	Hexone	4.77E-07	9.94E-07
2,4-Dimethylphenol	1.63E-07	6.45E-07	Hydrocyanic acid	2.50E-05	3.25E-05
2,6-Dimethylphenol	8.52E-07	3.35E-05	Hydrogen Sulfide (H2S)	3.80E-06	1.33E-05
Dimethylphylamine	2.76E-07	9.84E-07	Hydroquinone	4.23E-05	8.23E-09
Di-n-butyl phthalate	3.01E-05	1.42E-05	Indeno(1,2,3-c,d)pyrene	2.12E-05	9.41E-05
1,2-Dinitrobenzene	6.85E-06	9.50E-06	Iprodione	1.11E-05	1.22E-06
1,4-Dinitrobenzene	2.25E-05	2.18E-05	Isobutanol	2.82E-06	1.16E-06
M-Dinitrobenzene	5.15E-06	6.59E-06	Isophorone	1.16E-07	4.30E-06
Dinitrobutyl phenol	3.01E-05	1.28E-04	Isopropyl alcohol	6.68E-07	6.81E-07
4,6-Dinitro-o-cresol	6.14E-06	1.73E-06	Kepone	4.75E-05	1.12E-04
Linuron	1.27E-05	2.86E-05	Pirimicarb	5.40E-05	2.74E-07
Malathion	9.00E-06	6.69E-06	Pronamide	9.98E-06	2.32E-05
Maleic anhydride	5.41E-05	1.90E-10	Propachlor	1.44E-05	2.31E-06
Mecoprop	8.05E-05	1.75E-06	Propylene (Propene)	1.74E-07	5.02E-06
Methanol	6.18E-06	1.22E-06	Propylene oxide	1.29E-05	1.09E-05
Methomyl	9.16E-05	3.99E-05	Pyrazophos	6.97E-06	2.79E-06
Methoxone	8.22E-05	1.32E-06	Pyrene	1.64E-06	5.94E-08
Methoxychlor	4.72E-06	3.78E-08	Pyridine	7.95E-06	1.14E-06

(continues)

Table I. *(Continued)*

Chemical Name	*iF*(tot,air)	*iF*(tot,water)	Chemical Name	*iF*(tot,air)	*iF*(tot,water)
2-Methoxyethanol	5.87E-07	4.73E-06	Safrole	7.58E-08	6.96E-06
Methyl acetate	2.83E-06	1.69E-06	Sec-butyl alcohol	1.66E-06	1.20E-06
Methyl acrylate	2.82E-07	1.04E-06	Silvex [2-(2,4,5-trichlorophenoxy Propanoic acid]	6.63E-07	1.36E-06
Methyl bromide	2.44E-05	2.05E-05	Simazine	3.67E-05	6.30E-06
Methyl chloride	2.43E-05	2.06E-05	Styrene	2.36E-07	8.05E-06
Methyl ethyl ketone	6.90E-06	1.89E-06	Styrene oxide	1.22E-06	8.44E-07
Methyl hydrazine	2.56E-07	3.31E-06	2,4,5-T	1.44E-05	4.46E-06
Methyl iodide	1.76E-05	1.27E-05	2,3,7,8-TCDD	5.99E-04	1.32E-05
Methyl methacrylate	1.14E-07	4.44E-06	Tert-butyl alcohol	1.08E-05	1.36E-05
Methyl parathion	3.38E-06	2.53E-05	1,2,4,5-tetrachlorobenzene	2.17E-05	4.99E-05
Methyl tert-butyl ether	2.05E-06	8.64E-06	1,1,1,2-Tetrachloroethane	2.31E-05	3.37E-06
Methylacrylonitrile	3.37E-06	9.95E-06	1,1,2,2-Tetrachloroethane	1.85E-05	1.80E-05
Methylene chloride	2.18E-05	1.85E-05	Tetrachloroethylene	1.89E-05	2.07E-05
Metolachlor	7.87E-06	1.46E-05	2,3,4,6-Tetrachlorophenol	1.23E-05	2.91E-05
Metribuzin	7.87E-06	1.47E-05	2,3,7,8-Tetraochlorodibenzofuran	1.65E-04	1.94E-05
Mevinphos	6.10E-05	1.25E-06	Thiourea	3.82E-05	4.41E-07
Mirex	7.90E-05	9.33E-05	Thiram	5.44E-06	6.19E-07
Naphthalene	9.18E-07	7.93E-06	Tolclofos-methyl	4.72E-05	1.55E-05
1-Naphtyl-n-methylcarbamate	8.61E-09	5.25E-06	Toluene	2.93E-06	7.42E-06
2-Nitroaniline	8.81E-07	2.44E-06	O-Toluidine	4.14E-08	9.68E-07
Nitrobenzene	3.74E-06	1.16E-05	P-Toluidine	3.22E-07	1.58E-05
Nitrogen dioxide	1.90E-06	1.03E-05	Toxaphene	1.40E-05	3.20E-05
Nitroglycerin	1.69E-06	8.36E-07	Triallate	2.77E-05	6.34E-05
4-Nitrophenol	3.47E-07	9.33E-07	Triazophos	1.08E-05	1.11E-05
2-Nitropropane	5.11E-07	8.21E-06	S,S,S-Tributyltrithiophosphate	1.18E-05	2.40E-05
N-Nitrosodiphenylamine	1.03E-07	6.95E-06	Trichlorfon	6.64E-05	1.68E-06
2-Nitrotoluene	9.64E-07	1.27E-06	1,2,4-Trichlorobenzene	1.57E-05	4.06E-05
3-Nitrotoluene	1.53E-05	3.09E-05	1,1,1-Trichloroethane	2.53E-05	3.46E-05
Oxamyl	8.00E-05	1.98E-06	1,1,2-Trichloroethane	1.82E-05	2.80E-05
Oxydemeton methyl	8.26E-05	1.04E-05	Trichloroethylene	4.05E-06	1.76E-05
Parathion	6.27E-06	4.50E-06	2,4,5-Trichlorophenol	8.40E-06	8.12E-06
PCB-1254	8.04E-04	8.78E-05	2,4,6-Trichlorophenol	1.25E-05	3.04E-07
Pentachlorobenzene	4.17E-05	8.32E-05	Triethylamine	2.58E-07	2.28E-07
2,3,4,7,8-Pentachlorodibenzofuran	4.10E-04	1.87E-05	Trifluralin	1.29E-05	6.05E-07
Pentachloronitrobenzene	2.51E-05	3.55E-05	1,2,4-Trimethylbenzene	1.68E-07	9.92E-06
Pentachlorophenol	5.28E-05	5.48E-07	2,4,6-Trinitrophenol	7.82E-05	5.68E-06
Permethrin	1.71E-05	7.50E-06	2,4,6-Trinitrotoluene	1.73E-05	2.10E-07
Phenol	4.84E-07	1.13E-07	Triphenyltin chloride	2.58E-05	1.83E-06
1,3-Phenylenediamine	4.34E-05	6.64E-06	Vinyl acetate	2.83E-06	4.17E-06
P-Phenylenediamine	2.10E-05	6.23E-07	Vinyl bromide	9.53E-07	9.05E-06
2-Phenylphenol	1.18E-08	1.28E-06	Vinyl chloride	3.82E-06	2.14E-05
Phoxim	4.80E-07	2.27E-06	Xylenes (total)	1.28E-06	8.57E-06
Phthalic anhydride	6.66E-06	5.13E-09	Zineb	5.89E-05	8.61E-06

1-Naphtyl-n-methylcarbamate) to 3.2×10^{-12} (for Heptachlorodibenzofuran). When multiplied by the U.S. population (280 million), these values yield *iF* (total, air) values ranging from 8.6×10^{-9} to 9.1×10^{-4}. One goal of this article is to understand to what extent exposure route can be linked to chemical properties. Because exposure to many of the pollutants is dominated by one route, we classify the chemicals by exposure route. If more than 90% of the total intake is through inhalation, the chemical is classified as *inhalation dominant*. If ingestion intake exceeds 90% of total intake, it is classified as *ingestion dominant*. If the exposure results from multiple pathways, it is defined as *multipathway*. The dermal exposure route is only relevant (greater than 10%) for fenthion and ethoprop with a contribution to the *iF* of 34% and 20%, respectively. Because there are no dermal-dominant intakes, we did not develop a separate dermal-dominant category. Fig. 1 is a histogram of the number of chemicals that are inhalation dominant,

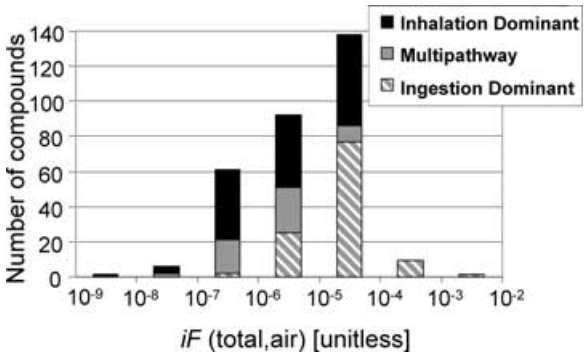

Fig. 1. Histogram of the number of chemicals that are inhalation dominant, ingestion dominant, or multipathway sorted by the value of *iF* (total, air) for an emission to air.

ingestion dominant, or multipathway, sorted by the value of *iF* (total, air). For compounds released to air, the largest overall *iF* values are associated with ingestion exposure (e.g., *iF* values over 10^{-4} all have exposure through ingestion), while for compounds where total exposure is dominated by inhalation, the overall *iF* values tend to be much smaller (e.g., below 10^{-5}).

In an effort to explore the relationship of environmental partitioning to chemical properties, Gouin *et al.*[26] used the octanol-water partition coefficient (K_{ow}) and the air-water partitioning coefficient (K_{aw}) to make a plot that includes regions that contain chemicals that are found primarily in the air phase, primarily in the water phase, primarily in the soil phase, or true multimedia chemicals. Whereas Gouin *et al.*[26] applied this approach to multimedia fate, we extend this approach to multipathway exposure and relate dominant exposure route to the K_{ow} and K_{aw} values. The result is illustrated in Fig. 2. For ingestion-dominant pollutants, we also indicate which chemicals have over 50% of exposure through the produce and grain pathway (those with values of K_{ow} less than 10^6, indicated in Fig. 2 as the lined square) and which have over 50% of the exposure through the combined meat and milk pathways (those with values of K_{ow} greater than 10^6, indicated in Fig. 2 as the crossed square).

From the results illustrated in Fig. 2, we were able to use values of K_{aw}, K_{ow}, and K_{oa} (the octanol-air partition coefficient, which is equal to K_{ow}/K_{aw}) to classify a pollutant that is inhalation dominant, ingestion dominant, or multipathway as follows:

- *Inhalation Dominant*: $K_{aw} > 1 \times 10^{-4}$ and $K_{oa} < 1 \times 10^6$
- *Ingestion Dominant*: $K_{aw} < 2 \times 10^{-6}$ or $K_{oa} > 2 \times 10^8$

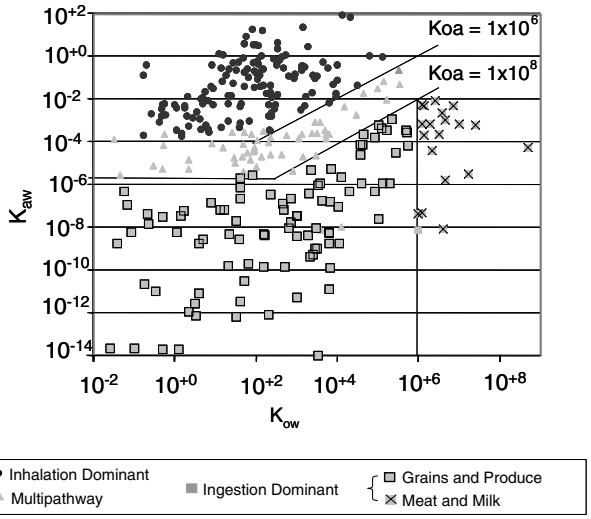

Fig. 2. Exposure classification as function of K_{ow} and K_{aw} parameters for 308 chemicals for a release to air. The sloped lines indicate the K_{ow} values.

- *Multimedia*: $1 \times 10^6 < K_{oa} < 2 \times 10^8$ and K_{ow} higher than 1×10^2; or $1 \times 10^{-4} < K_{aw} < 2 \times 10^{-6}$ and K_{ow} lower than 1×10^2.

The region containing inhalation-dominant chemicals is similar to that defined by Gouin *et al.*[26] for pollutants primarily partitioning into air. The ingestion-dominant pollutants partition strongly into the water and lipid phases of the environment, and thus are ingested primarily through food products.

One might like to use a limited set of chemical properties to predict iF_i (total, air) in place of running the model. To illustrate the feasibility of such predictions, Fig. 3 shows iF_i (total, air) versus the reaction half-life in air for inhalation-dominant pollutants. From this result, we derived the following relationship:

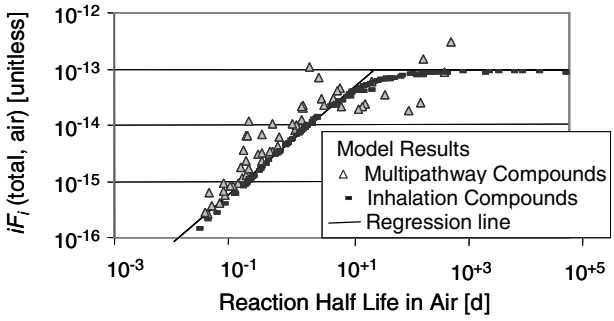

Fig. 3. Relationship between iF_i (total, air) and reaction half-life in air for inhalation-dominant and multipathway substances.

$\log [iF_i(\text{total, air})] = 0.87 \times \log (\text{reaction half-life in air}) - 14.4$, for reaction half-life in air $< \sim 35$(d)

$\log [iF_i(\text{total, air})] = -13$, for reaction half-life in air $> \sim 35$(d)

The maximum predicted value of iF_i in this scenario is 9.1×10^{-14}, and is determined by the advection rate out of the system. For those compounds with a reaction half-life of greater than 35 days in air, there could be additional exposure occurring outside the system boundaries. The average residual error (standard error of the estimation) in the log *iF* for this predicted relationship is 0.09 for inhalation-dominant compounds. We also plotted the multipathway pollutants on this graph and found they all fall slightly above the line. Because multipathway pollutants generate additional exposure through ingestion, it is not at all surprising that for most, the total *iF* values tend to be greater than those for pollutants for which exposure is through inhalation alone.[4] The standard error of the estimator for log (*iF*) is 0.40 for the multipathway compounds.

A correlation for a similar relationship for the ingestion-dominant pollutants is not strong enough and the residual error is too large to propose a sufficiently reliable prediction algorithm.

3.2.2. *Water Release*

For a release to water, the iF_i (total, water) ranges from 6.8×10^{-19} (for maleic anhydride) to 3.8×10^{-12} (for aldrin) and thus *iF* (total, water) ranges from 1.9×10^{-10} to 1.0×10^{-3}. Fig. 4 provides a histogram of ingestion-dominant or multipathway chemicals sorted by the intake fraction value (there were no inhalation-dominant or dermal-dominant chemicals for the water-release scenario). There is a mix between ingestion-dominant and multipathway compounds for all values of *iF*.

Fig. 5 shows the dependence of the ingestion-dominant and multipathway classification on K_{aw} and K_{ow}. The ingestion-dominant chemicals are characterized by a $K_{ow} > 1 \times 10^5$ or a $K_{ow} < 1 \times 10^2$ and a $K_{aw} < 5 \times 10^{-5}$. The regions of dominant exposure pathways are not as clearly defined as they were in the case of releases to air. The ingestion-dominant chemicals occur in two distinct regions, and are characterized by either a $K_{ow} > 5 \times 10^5$, or a $K_{ow} < 1 \times 10^2$ and a $K_{aw} < 5 \times 10^{-5}$. The compounds with a $K_{ow} > 5 \times 10^4$ reach humans primarily through fish consumption and have the highest *iF* (total, water) values with a few exceptions.[5] Those with a $K_{ow} < 1 \times 10^2$ and a $K_{aw} < 5 \times 10^{-5}$ reach humans through tap water ingestion. The

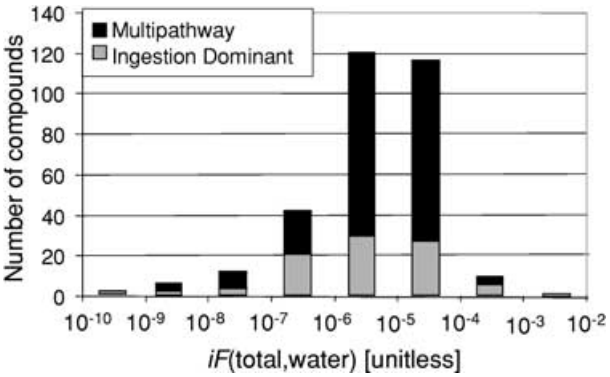

Fig. 4. Histogram of the number of chemicals that are ingestion dominant or multipathway sorted by the value of *iF* (total, water) for each substance for an emission to water. The multipathway compounds for which the dermal pathway is significant are indicated.

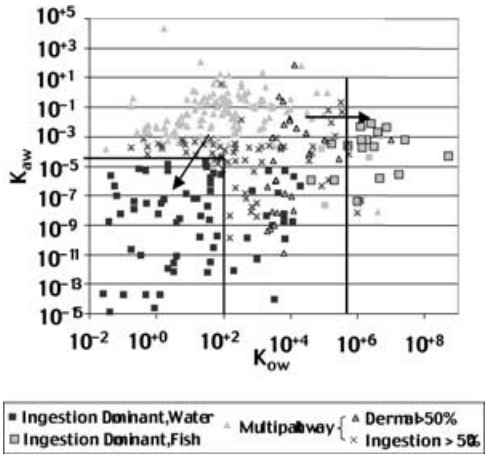

Fig. 5. Primary exposure routes as function of K_{ow} and K_{aw} parameters for 308 chemicals released to water. The arrows indicate the trends in K_{ow} and K_{aw} that lead to greater contributions from ingestion.

4 The few exceptions (see Fig. 3) can be primarily explained by (1) the lower measured bioconcentration factors compared to the value predicted from K_{ow} correlations (e.g., around two orders of magnitude for Permethrin and Cypermethrin, one order of magnitude for 2,3,7,8-TCDD, and a factor of four for DDT), (2) the relatively high dermal uptake fraction (11,12-Benzofluoranthene), or (3) a combination of both.

5 A few multimedia substances have an *iF* value less than that predicted by the curve due to a low Henry's law value causing a high degree of partitioning into water, which lowers the exposure through inhalation relative to other compounds with a similar half-life in air.

remaining pollutants are multipathway, characterized by a low K_{ow} and high K_{oa} value. We indicate multipathway compounds that have at least 50% of the exposure through the ingestion route. There is a region of K_{ow} values between 1×10^2 and 5×10^4 where the dermal pathway is potentially significant. The maximum contribution from the dermal route is 83%. However, there is low reliability associated with the algorithms for dermal uptake and one should carefully review the calculations if this route is important. When tap water consumption is the dominant exposure pathway, we expect the reaction half-life in water to have a strong impact on the intake fraction because the concentration/source ratio in water is proportional to the reaction half-life. When ingestion of fish is important, we expect both the reaction half-life in the water and the K_{ow}, which is an indicator of bioaccumulation, to have an impact on the intake fraction. In Fig. 6, plots a and b show iF_i (total, water) for tap-water-dominant compounds versus reaction half-life in water and iF_i (total, water) for fish-dominant compounds versus the product of the reaction half-life in water and the K_{ow}. From the relationships illustrated here, the following correlation functions are proposed:

$$\log [iF_i(\text{total, water})] = 0.98 \log (\text{reaction half-life in water}) - 15 \pm 0.05 \text{ (average residual error)},$$

$$\log [iF_i(\text{total, water})] = 0.56 \log (\text{reaction half-life in water} \times K_{ow}) - 17.5 \pm 0.63 \text{ (average residual error)}$$

The model value for deltamethrin (the labeled point with $iF_i = 1.25 \times 10^{-16}$) is much lower than the predicted value, primarily because the chemical has a lower K_{ow} (4×10^4), and consequently lower bioconcentration potential compared to the other ingestion-dominant compounds with $K_{ow} > 5 \times 10^5$. Excluding deltamethrin lowers the mean geometric residual error to 0.53. An effort to apply a similar analysis to multipathway substances revealed that they do not present any clear correlations with chemical properties and are thus not show on the figures.

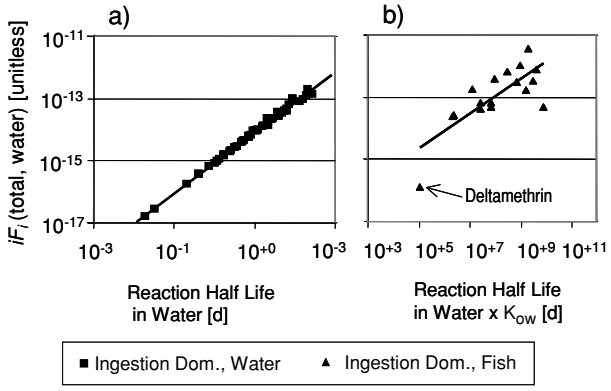

Fig. 6. a) Plot of iF_i (total, water) for the water pathway, ingestion-dominant chemicals versus the reaction half-life in water. b) Plot of iF_i (total, water) for the fish pathway, ingestion-dominant chemicals versus the product of K_{ow} and the reaction half-life in water.

3.2.3. Soil Release

The intake fractions resulting from a release to soil were calculated but, for brevity, are not presented here because for many compounds this is a less relevant release medium (this pathway is very relevant for pesticides but those are only a small subset of the total chemical set). In addition, the rapid exchange in our model between the thin surface soil layer and the air results in many of the trends for a release to soil being similar to those for a release to air.

4. DISCUSSION

4.1. Comparison with Theoretical Maximum

We conclude our assessment with a model evaluation. We focus our discussion on methods for determining if the results are reasonable and identify the corresponding advantages and limitations of the *iF* approach.

One advantage of expressing a source-to-intake relationship as an intake fraction is the ability to consider plausible and obvious limits for the intake fraction. To build confidence in the *iF* approach and the supporting calculations, we determine a plausible upper-bound value for *iF* values and compare the calculated values with these bounding values. In the cases where the calculated values exceed these limits, we explore the sources of the discrepancy.

We consider first the theoretical upper-bound *iF* value for compounds that are released to air and do not partition out of air such that transport and reaction in air determine fate and exposure. Among the compounds with this behavior, the maximum *iF* we calculated was 2.6×10^{-5}. We calculate the plausible upper-bound *iF* for this set of compounds based on the modeling assumptions used in our model application and some simple bounding calculations consistent with these assumptions. To achieve this, we assume that each person breathes 22 m^3/day, so that a population of 280 million breathes 6×10^9 m^3/d. With

an assumed average mixing height of 700 m,[23] the volume of air over the United States is 6.4×10^{15} m^3. The estimated advection loss rate from this volume is 4% a day based on an average windspeed and accounting for the fraction that might be blown back into the system. The residence time in the airshed is 27 days; thus the chemical mass in the air volume is 27 times the mass released each day. We use these numbers to determine the fraction of this air volume that is inhaled each day and multiply this by the chemical mass in air to estimate *iF* (inhalation, air) $= 2.67 \times 10^{-5}$, equal to the maximum from the model. We note that the maximum value is sensitive to system scale and could be greater under a different modeling scenario, that is, if we tracked a specific plume released close to the ground in a highly populated area. Additionally, values for *iF* (inhalation, air) could exceed this limit for pollutants that enter indoor air through contaminated tap water, where the *iF* is greater than for a release to outdoor air.

We next consider chemicals released to air that partition into vegetation and soil, such that there can be exposure through the ingestion route. This increases the theoretical upper bound of the intake fraction. For example, the calculated *iF* (total, air) for 2,3,7,8-TCDD is 0.0006, indicating that six out of every 10,000 molecules released to the environment are ultimately ingested by the human population, a value that seems questionable. To examine this issue, we consider the value of *iF* for a pollutant that partitions entirely into vegetation with no degradation and no exchange with other media. The limit of the intake fraction in this case would be the fraction of annual production of biomass each year that is ingested by humans, or ingested by livestock that are used as a source of food products for humans. In the United States, for example, we estimate that approximately 1.5% of the annual increase in biomass results from agricultural growth.[6] Next, we must determine what percent of the pollutant is likely to be in the vegetation consumed. For TCDD, the multimedia model indicates that 2% of the total mass in the system is in the vegetation. We must also determine what percent of the mass enters the multimedia system. For TCDD, 6% of the mass undergoes chemical transformation in the air or is advected out of the system and is thus unavailable for multimedia partitioning. If we multiply 1.5% (agricultural vegetation production) by 2% (mass in vegetation) by 94% (the mass interacting with the multimedia system, 100–6%), we obtain an intake fraction of 0.0003, a factor of two lower than the model calculation and within a factor of six of the value calculated based on measurements. Given the complexity of this calculation, we are pleased that three different calculation methods yield values all within the same order of magnitude.

Among the 308 chemicals evaluated for air release, the highest predicted partitioning into vegetation was 7%.[7] If we assume this is approximately the maximum percentage that would partition into vegetation, and consider the fraction increase in agricultural biomass, this results in an upper bound on the intake fraction of 0.001. These evaluations reveal that one key advantage of the intake fraction approach is that it is a transparent way to dissect and evaluate the results.

It is of interest that when we initially calculated *iF* for the 308-chemical set, a few pollutants had an intake fraction exceeding 0.001, our proposed theoretical maximum. Careful examination revealed that the very large *iF* values could be traced to large biotransfer factors to meat and milk. The biotransfer factor was estimated from the empirical model of Travis and Arms,[27] which poses a linear relationship between log(biotransfer) and log K_{ow}. When K_{ow} is greater than 10^7, this model produces biotransfer values that require a pollutant mass taken in by the livestock to exceed the pollutant mass in the pasture they consume. We believe this results from a lack of fit between the linear model and the observations at high K_{ow} values. Fig. 7 shows the Travis and Arms relationship[27] and the data supporting this model. We note that the data appear to be limited to a maximum value for the biotransfer factor of approximately 0.1, so that using the Travis and Arms relationship for values of K_{ow} greater than 3×10^6 is not appropriate. When we limited biotransfer to 0.1, we decreased the intake fraction for the pollutants with the extremely high K_{ow} values to a result comparable to our theoretical limit. This example again emphasizes the value of intake fraction for confirming the results of a fate, transport, and exposure analysis.

6 We estimated the annual increase in biomass in the United States by estimating the land area of each ecosystem type and the corresponding primary production rate.[31,32] We estimated the rate of agricultural production both from USDA estimates[33] and by estimating vegetation consumption by people or by livestock based on the intake rates used in the model.[34] We found the two estimates to be consistent.

7 This result is for benzo[a]pyrene, which has a high plant-air partition coefficient.

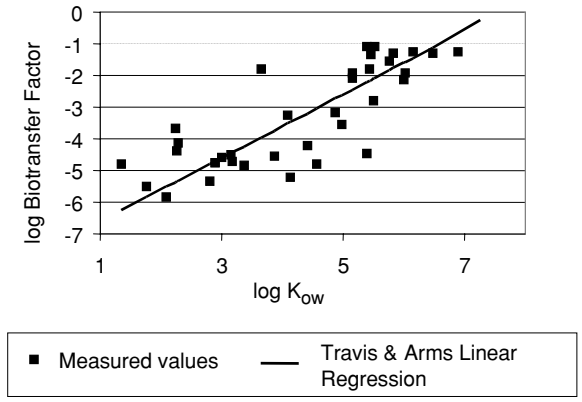

Fig. 7. The Travis and Arms[27] linear relationship between K_{ow} and the biotransfer factor, as well as measured data used to derive the relationship.

4.2. Relationship with Chemical Properties

Another goal of this article is to determine the primary exposure route based on a limited set of chemical properties and, when possible, develop a relationship between the parameter inputs and *iF* values. Substances with high K_{ow} values (greater than 10^4) are primarily ingestion dominant, with high concentrations in fish, meat, and milk. When released to air, chemicals with high K_{aw} values (greater than 10^4) tend to remain in the air and exposure is primarily through the inhalation pathway. As K_{aw} decreases, exposure through tap water, produce, and grain increases. For a release to water, exposure to chemicals with both a low K_{aw} value (less than 10^{-4}) and a low K_{ow} value (less than 10^2) is primarily through tap water consumption. If a chemical falls outside the expected region, one should examine the fate and transport of this chemical to make sure the equations used in describing the fate, transport, and exposure are applicable for such a chemical.

For compounds that remain primarily in the medium to which they were released, the modeled intake fraction is related to the reaction half-life in that medium, and a multimedia model is not needed to analyze the substance. For compounds released to air that remain in air, one might want to use an air model that takes into consideration stack height for typical use scenarios and population densities.[28] For those compounds that partition into lipids and are transferred through food webs, the intake fraction depends both on the K_{ow} and the reaction half-life of the medium into which the compound was released. For these compounds, a multimedia, multipathway analysis is needed. It is important to incorporate the transfer to the food pathway since this transfer tends to increase the intake fraction for a compound. Chemicals with an intake fraction over 5×10^{-4} are primarily ingestion dominant. However, modeling the transfer into the food pathway is still very uncertain and requires additional research.

Another key uncertainty in the *iF* approach is the lack of reliable chemical property values for many chemicals. As a result, the calculated values listed in Table I should be used only as screening-level values. Based on an uncertainty analysis completed by Hertwich *et al.*,[29] we expect the values to be accurate only within an order of magnitude. The uncertainty tends to be greater for pollutants that are transferred to humans through a food chain, as each cross-media transfer adds uncertainty. There is also uncertainty and variability in the landscape and exposure parameter values. These uncertainties may affect the classification by exposure route and the predictions of intake fraction based on chemical properties. However, the uncertainties from landscape and exposure factors do not impact on the results as much as the chemical parameter uncertainties.[29,30]

5. CONCLUSIONS

The intake fraction is a powerful concept that can be used to determine the relationship between pollutant emissions and the resulting intake to an individual or to a population. *iF* is a useful screening tool for both LCA and comparative risk assessments. Comparing *iF* values for a variety of chemicals between two environmental models can give insight on the similarities and differences of the models. Within a large set of industrial pollutants, the intake fractions can vary up to five orders of magnitude for a release to air and up to seven orders of magnitude for a release to surface water. Although we recognize the uncertainties in the results, with such a large range, clear differences among the chemicals are still apparent.

One of the useful features of the intake fraction is that one can determine plausible and defensible limits on the value for a specific environmental system. A intake fraction for the U.S. population of up to 0.001 is realistic, and values over 0.01 are not considered plausible. However, all values over 0.001 should be considered carefully to detect possible problems with the model algorithms.

We developed graphs that can be used to determine the primary exposure route or to determine if a chemical is likely to have multiple exposure routes

based on the K_{aw} and the K_{ow} values. The intake fraction for an air release of an inhalation-dominant chemical can be predicted based on the half-life in air. For a release to water, the intake fraction for a tap-water-dominant chemical can be predicted based on the half-life in water, and for a fish-dominant chemical, the intake fraction can be predicted based on the half-life in water and the K_{ow}.

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