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Receptor-oriented approaches in wildlife and human exposure modelling: a comparative study

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ABSTRACT

Five human and five wildlife receptor-oriented exposure models were compared with the aim to explore relevant differences and similarities, and to identify areas where the fields of human and wildlife exposure modelling can learn from each other. This comparison revealed differences in the effect endpoints, the stressors addressed, the exposure routes and media taken into account, and the simulation of the receptors’ activities (imposed from activity pattern databases vs. emerging from behavioural rules). These differences can largely be explained by differences in protection targets, dominant exposure routes and data availability. It is concluded that wildlife models can serve as a source of inspiration for human models when it comes to the simulation of activity patterns as emergent and the inclusion of non-chemical stressors. Human models can be as a source of inspiration for wildlife models when it comes to modelling exposure from multiple exposure routes and the compilation and use of data on activity patterns.
1. Introduction

Environmental risk assessment\(^1\) is an organized process which aims to describe and estimate the likelihood of adverse health outcomes after exposure to environmental stressors (US PCCRARM, 1997). Risk assessment traditionally focuses on single stressors, e.g., a particular chemical substance, biological agent or radiation. However, awareness is growing that exposure to single stressors is the exception rather than the rule (US EPA, 2003a; Hope, 2005; Callahan and Sexton, 2007; Sexton and Hattis, 2007). In practice, organisms are often exposed to multiple stressors, e.g. a chemical mixture or a combination of chemical, biological and physical agents. Exposure to multiple stressors may take place concurrently or sequentially, and the individual stressors may or may not interact, either directly or indirectly.

Traditional methods for risk assessment of single stressors fail to deal with the specific challenges posed by multiple stressors. On the effects side, novel scientific methods are required that can address the complexity of potential direct and indirect interactions between stressors in exposed organisms, and that can be used to produce reliable effect estimates (Ragas et al., 2008b). On the exposure side, there is a need for methods that can describe and quantify concurrent or sequential exposure of organisms to multiple stressors. Traditionally, source-to-dose models have been widely used to assess the exposure to single stressors (Price et al., 2003). These models focus on the stressor of concern and describe one or more pathways through which the receptor, either a human or another organism, can be exposed. However, exposure to multiple stressors requires a different approach because the activity pattern of the receptor plays an important role (Spurgeon et al., 2008). The receptor can be considered as an integrator of different exposures which it encounters while it moves through space and time. Therefore, exposure models for multiple stressors should primarily focus on the receptor, and not on the stressor(s).

Over recent years, a considerable number of models have been developed that aim to simulate the spatial and temporal behaviour of individual organisms. Some of these models have been specifically developed for exposure assessment, but most of them for other applications, e.g. forecasting of transport demand (Wang and Cheng, 2001), urban planning (Batty, 2005), behaviour of crowds (Willis et al., 2004), gaming (Arari et al., 2005), animation (Tu, 1996), and simulation of wildlife population dynamics (Reuter, 2005). The main characteristic feature of these models is the fact that each individual is tracked through time. The models typically consist of a number of individuals defined in terms of state (characteristic parameters) and behaviour (procedural rules), and an environment or framework with which these individuals interact. They are also known as individual-based, agent-based, person-oriented or receptor-oriented models. In the present paper, we use the term receptor-oriented model (ROM), because the term person-oriented model (POM) refers exclusively to humans and the terms agent-based model (ABM) or

\(^1\) Next to environmental risk assessment, the terms ecological and human risk assessment are regularly used. In the present paper, we use the term environmental risk assessment as an overarching term. The term ecological risk assessment is reserved for assessments which aim to assess risks to ecosystems, and the term human risk assessment for assessments which aim to assess human health risks.
individual-based model (IBM) are generally used in a very strict sense, i.e. to refer to models that simulate multiple individuals at the same time and include interactions between these individuals (Grimm and Railsback, 2005). Besides individual-based or agent-based modelling, other techniques commonly used for receptor-oriented exposure modelling include Monte Carlo simulation and random walk modelling. Monte Carlo simulation is a method for iteratively evaluating a deterministic model using sets of random numbers as inputs from a domain of possible inputs and aggregating the results of the individual computations into the final result. Random walk modelling here refers to a mathematical formalization of a trajectory that consists of taking successive random steps in order to simulate the search path of a foraging animal, thereby accounting for the spatial variability of the environment. The development and application of receptor-oriented models require a substantial amount of data, programming skills and computer capacity. The recent increase in the number of receptor-oriented models can be explained by the increasing availability of data, the development of object-oriented programming languages and faster processors in computers.

Several receptor-oriented models for exposure assessment of humans and wildlife have been developed. Examples of human models include CARES (CropLife America, 2002), LifeLine (The Lifeline Group, 2007), NORMTOX (Ragas et al., 2008a), SHEDS (Zartarian et al., 2007), and TRIM.Expo/APEX (US EPA, 2006a,b). Examples of wildlife models include ALMaSS (Heneghan et al., 2003), the Resource Selection Exposure Model (RSEM; Chow et al., 2005), SE4M (Hope, 2001), SpaCE (Schipper et al., 2008), and the Wading Bird Model (WBM; Wolff, 1994). Because ecological and human risk assessment have historically been separate fields, human and wildlife exposure models have largely been developed independently. This has led to differences between the two groups of models. However, there are also similarities, e.g. the use of similar modelling concepts and platforms. Exploration of these similarities and differences can help to identify areas where both fields can learn from each other. Ecological and human risk assessment could benefit from each other as integration can improve predictive capability, assessment quality, efficiency and cost effectiveness (Munns Jr et al., 2003).

The main aim of the present paper is to compare receptor-oriented models for human and wildlife exposure assessment in order to explore relevant differences and similarities, and to identify areas where both fields can learn from each other. This can be a first step towards integration of human and wildlife exposure assessment. It can help risk assessors and modellers from both fields to improve the quality of their exposure assessment by adopting successful approaches employed by the other field. Five human and five wildlife models are being compared. The paper starts with a general description of receptor-oriented exposure assessment (Section 2). The human and wildlife models included in the comparison are presented in two subsequent sections, i.e. Sections 3 and 4, respectively. Section 5 compares the selected models based on a number of characteristic features, e.g. exposure routes, endpoints, simulation of movement and handling of space and time. It further discusses the similarities, differences and, options for improvement of both human and wildlife exposure models. Finally, section 6 presents the conclusions.
2. Receptor-oriented exposure assessment

In risk assessment, exposure is usually defined as the contact between an organism and a stressor, which occurs when the organism and the stressor co-exist in space and time and interact with one another (US EPA, 1993; Suter, 1993; US EPA, 1997; Berglund et al., 2001). Four exposure routes can be distinguished: inhalation, dermal contact, ingestion, and maternal transfer (transplacental, lactational). The magnitude of exposure is commonly expressed either as a concentration, or as a dose (Smith et al., 2007). The exposure concentration of a chemical refers to the concentration in the carrier medium at the point of contact. The dose refers to the amount of a pollutant that enters the receptor’s body and is usually expressed in mg chemical per kg body weight per unit time (Berglund et al., 2001). Sometimes the dose is differentiated into intake and uptake dose. The intake refers to the amount of chemical entering the body via the skin, the gastrointestinal or the pulmonary system. The uptake comprises the amount of chemical that is actually absorbed from the carrier medium and reaches the circulating blood (IGHRC, 2004).

The likelihood, magnitude, duration and frequency of exposure are driven by both environmental factors and specific traits of the receptor organism itself (Smith et al., 2007). Relevant environmental characteristics include factors that affect the speciation and hence the bio-accessibility of the chemical, such as soil and sediment characteristics, surface water characteristics, and temperature (Plette et al., 1999; Sauvè et al., 2000; Newman and Unger, 2003). Species-specific characteristics of the organism play significant roles in the likelihood that exposure pathways, from source to receptor, will be completed (Smith et al., 2007). Life history traits of organisms directly influencing the likelihood as well as the magnitude of exposure include diet preferences, activity patterns (e.g., swimming, digging) and physiological factors such as respiratory rate, food ingestion rate, and absorption and excretion rate (Berglund et al., 2001). Further, there are numerous traits playing an indirect role, such as age, developmental stage, physical condition and gender, which affect exposure via for example activity patterns and contact rates.

The spatial distribution and temporal variation of environmental contaminant concentrations combined with the mobility of the receptor organism may result in considerable variation in exposure (Smith et al., 2007). Their mobility enables the organisms to shift between different micro-environments (Figure 1), i.e. well-defined surroundings that can be treated as homogeneous (or well-characterized) with regard to the concentrations of a hazardous agent (Duan, 1982). Micro-environments specific for humans include for example different rooms within diverse buildings, each with a specific air contaminant concentration; examples of wildlife micro-environments are specific habitat patches with a homogeneous contaminant concentration in the soil. The selection of a specific micro-environment can be assumed to partly reflect the organism’s response to ambient conditions in the different micro-environments (e.g., in response to weather conditions, humans might go indoor and wildlife organisms might change habitat patch in search for shelter), and partly result from species-specific characteristics that drive its activity.
patterns. Thus, total exposure will ultimately be a function of the ambient environmental concentrations within different micro-environments, combined with the time spent in each of these environments (Zidek et al., 2007). In order to provide realistic exposure estimates for the individuals or populations of concern, it is therefore felt that receptor-oriented exposure models should take into account all relevant locations, sources, and pathways of exposure (Berglund et al., 2001).

**Fig. 1.** Exposure of a mobile individual to different environmental media along a trajectory through different micro-environments.
3. Wildlife exposure models

Five receptor-oriented wildlife exposure models and five receptor-oriented human exposure models were selected for inclusion in this comparative study. The models were selected based on their receptor-oriented nature, their ability to model chemical exposure, and the availability of data in the open literature. The five state of the art wildlife exposure models selected (i.e., ALMaSS, RSEM, SE\textsuperscript{4}M, SpaCE, and WBM) are described in more detail below.

**ALMaSS**

ALMaSS (Animal, Landscape and Man Simulation System) is an agent-based modelling tool developed as a versatile decision-support tool for answering management questions regarding the effect of changing landscape use or agricultural practices (e.g. pesticide use) on several animal species. ALMaSS was developed at the National Environmental Research Institute (NERI) of Denmark. It was applied to assess the impact of a fictitious insecticide relative to changes in landscape structure on populations of skylarks in the UK. The backbone of the ALMaSS model framework is a temporally and spatially explicit dynamic landscape simulation model, which is linked to detailed behavioural individual-based wildlife species models. Individuals are modelled as objects that interact with their environment and with other individuals. They possess states (e.g. age, weight, internal contaminant concentration) and behaviours to change these states. The model calculates the exposure to pesticides via food ingestion as the daily estimated theoretical exposure for adults and young. The exposure is a function of the movement and feeding behaviour of the individual in combination with the contemporaneous local pesticide concentrations. Non-chemical stressors include land-use change and food availability.

**RSEM**

The Resource Selection Exposure Model (RSEM) is a GIS-based Monte Carlo simulation model developed to provide a general framework for predicting exposure of midsized wildlife species to soil contamination. The model was developed for the U.S. Department of Energy and was applied to assess exposure of Wild hogs and Raccoons to aluminium, nickel, uranium, and radiocesium in the Savannah River Site, i.e. a nuclear materials processing center in South Carolina. RSEM consists of a probabilistic resource selection model that predicts the spatial distribution of wildlife species, coupled to a generalized exposure model. Species' habitat characteristics and landscape indices, either known or derived from locations in which it occurs using logistic regression, are used to extrapolate areas of suitable habitat from the spatial data layers containing information on the environment. By iteratively sampling possible home ranges different individuals of a population are mimicked. For each home range an area weighted average use probability of all cells within the home range is estimated. The external and internal exposure estimates are calculated for oral exposure only. These estimates are modelled as a function of the ratio of
waste site area to home range area weighted by the probability of the animal inhabiting the waste site area.

**SE**^4**M**

SE^4M (Spatially and bioEnergetically Explicit terrestrial Ecological Exposure Model) is an individual-based, random walk model for exploring the influence of spatial and bioenergetic factors on a receptor’s acquisition of energy and contaminant tissue residues as it moves through space and time. The model was developed by Hope (2001; 2005) at the Oregon Department of Environmental Quality, Portland and has been applied to mercury exposure in hypothetical case studies only. Each Monte Carlo iteration of the entire program represents one individual foraging in an area for a specified number of days by moving over a rasterised landscape. Movement is governed by movement strategies and response to environmental variation of habitat quality defined in terms of the gross food energy available to a given receptor. SE^4M calculates chemical exposure via ingestion of soil and food as a function of the spatially variable chemical concentration and habitat-dependent daily ingestion rate. Tissue residue levels are calculated dynamically using toxicokinetics. Non-chemical stressors include food availability and lack of, or denial of access to, suitable habitat.

**SpaCE**

SpaCE (Spatially explicit Cumulative Exposure Model) is an individual-based model developed as a tool to assess dietary exposure of terrestrial vertebrates to cumulative chemical stressors, thereby explicitly addressing spatial heterogeneity. SpaCE was developed at the Department of Environmental Science at the Radboud University Nijmegen, the Netherlands. The model has been applied to predict exposure of several mammal and bird species to cadmium, nickel and zinc in a Dutch floodplain. Like SE^4M, an individual is simulated as a foraging receptor moving through a heterogeneously contaminated, rasterised landscape. Movement is directed by spatial heterogeneity in habitat quality. Contaminant exposure and accumulation are simulated following a food web-based approach, accounting for prey preference and availability. Exposure to heavy metals is calculated both as the average concentration in food and as a total concentration in the receptor’s body, based on metal-specific toxicokinetics.

**WBM**

The Wading Bird Model (WBM) is an individual-based exposure model for wading birds, developed by Wolff (1994). Initially, it was constructed to assess the effects of different water level patterns on the reproductive success of wood storks. Later it was applied to assess dietary mercury exposure of wood storks in the Everglades, Florida, and to quantify effects of chronic dietary PCB exposure on the reproduction of small groups of interacting Great Blue Herons in Poplar Creek, Tennessee. The model uses the object-oriented programming technique and consists primarily of a spatially explicit landscape submodel, containing prey information and contamination data, behavioural
submodels for the individuals, and a bioaccumulation submodel for the uptake of the contaminant. An individual in this model is represented by a single object with characteristic state (age, fitness, size, etc.) and methods (forage, fly, search, etc.). Foraging behaviour depends on the availability of food. Exposure is calculated as the internal concentration in the bird using contaminant-specific toxicokinetics.

Table 1
Wildlife exposure model characteristics

<table>
<thead>
<tr>
<th>Short description</th>
<th>ALMaSS</th>
<th>RSEM</th>
<th>SE*M</th>
<th>SpaCE</th>
<th>WBM</th>
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<tr>
<td><strong>Species</strong></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Skylark, Field vole</td>
<td>An agent-based modelling tool for assessing the effect of land use change or agricultural practices (e.g. pesticide use) on several animal species.</td>
<td>A GIS-based Monte Carlo simulation model for predicting exposure of mid-sized wildlife species to soil contamination.</td>
<td>An individual-based, random walk model for exploring the influence of spatial and bioenergetic factors on a receptor’s acquisition of energy and contaminant tissue residues.</td>
<td>A spatially explicit, individual-based model for assessing dietary exposure of terrestrial vertebrates to cumulative chemical stressors.</td>
<td>An individual-based exposure model to assess dietary contaminant exposure of interacting individuals of a wading bird colony.</td>
</tr>
<tr>
<td><strong>Development stages</strong></td>
<td>For Skylark: Egg, nesting, young, adult; for Field vole: male, female.</td>
<td>For Raccoon only adult stage; for Wild hog 5 age classes.</td>
<td>None</td>
<td>None</td>
<td>Nestling, young, adult.</td>
</tr>
<tr>
<td><strong>Energy budget</strong></td>
<td>Yes, only for Skylark.</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Substances</strong></td>
<td>Fictitious pesticides.</td>
<td>Uranium, nickel, aluminium, radiocesium.</td>
<td>Not chemical specific; mercury used as an example.</td>
<td>Cadmium, nickel, zinc.</td>
<td>PCBs, mercury.</td>
</tr>
<tr>
<td><strong>Non-chemical stressors</strong></td>
<td>Food availability, starvation, human management (e.g. harvest, ploughing, mowing roadides etc.).</td>
<td>None</td>
<td>Food availability, habitat availability.</td>
<td>None</td>
<td>Food availability.</td>
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<td><strong>Exposure routes</strong></td>
<td>Ingestion.</td>
<td>Ingestion.</td>
<td>Ingestion.</td>
<td>Ingestion.</td>
<td>Ingestion.</td>
</tr>
<tr>
<td><strong>Exposure endpoint</strong></td>
<td>Daily dose (mg/kg/d); internal concentration (mg/kg).</td>
<td>Daily dose through ingestion (mg/kg/d); radiocesium uptake and burdens in muscle tissue (Bq/g) for the Raccoon.</td>
<td>Internal concentration (mg/kg).</td>
<td>Average exposure concentration in food (mg/kg); internal concentration (mg/kg).</td>
<td>Internal concentration (mg/kg).</td>
</tr>
<tr>
<td><strong>Effect level and endpoint</strong></td>
<td>Population level; abundance, growth rate, population persistence.</td>
<td>Individual level; comparison with LOAEL (for renal effects in rabbits).</td>
<td>Individual level; tissue residues and energy balances.</td>
<td>Individual level; risk indicator based on comparison with NOECs.</td>
<td>Population level; colony survival.</td>
</tr>
<tr>
<td><strong>Landscape-organism interaction</strong></td>
<td>Yes, foraging in suitable habitat only; behavioural responses (e.g. dispersal) to management (e.g. mowing).</td>
<td>Yes, foraging in suitable habitat only.</td>
<td>Yes, foraging only in suitable habitat; may or may not transit non-habitat areas.</td>
<td>Yes, foraging in suitable habitat only.</td>
<td>Yes, foraging in suitable habitat only.</td>
</tr>
<tr>
<td><strong>Interaction between organisms</strong></td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Space representation</td>
<td>Rectangular grid</td>
<td>Hexagonal grid</td>
<td>Rectangular grid</td>
<td>Rectangular grid</td>
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<tr>
<td><strong>Movement</strong></td>
<td>Based on assumptions and on observed behaviour</td>
<td>No movement</td>
<td>Modelled after several receptor behaviours presented in the literature</td>
<td>Based on assumptions and behaviours presented in the literature</td>
<td>Based on assumptions and on observed behaviour</td>
</tr>
<tr>
<td><strong>Activity patterns</strong></td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td><strong>Time step</strong></td>
<td>1 day</td>
<td>Not applicable</td>
<td>1 day</td>
<td>Species dependent (¼ - 10 days)</td>
<td>Bioaccumulation module: 1 day; foraging module: 24 time steps of 15 min, representing a ‘foraging day’</td>
</tr>
<tr>
<td><strong>Time window</strong></td>
<td>1 day to many years</td>
<td>Not applicable</td>
<td>1 to many days</td>
<td>1 lifetime</td>
<td>1 to many days</td>
</tr>
<tr>
<td><strong>Model platform</strong></td>
<td>C++, object-oriented</td>
<td>A dynamic linked library (DLL) in ArcMap (GIS platform) using Visual Basic, probabilistic resource selection model, Monte Carlo Simulation</td>
<td>Visual Basic Application for Excel with Monte Carlo (Crystal Ball)</td>
<td>Visual Basic Application for Excel</td>
<td>Object-oriented, language unknown</td>
</tr>
</tbody>
</table>

| References² | a,b,c,d | e,f,g,h,i | j,k | l,m,n,o | p,q,r,s |

**Wildlife-specific**

| Space (scale / resolution / extent) | 1 cell = 1m²; 1 hexagon = 7.8 ha; 10^4*10^4 cells | 1 cell = 0.1 ha; 100*100 cells | 1 cell = 25m²; 13*13 cells | 1 cell = e.g. 6.25ha; 913*247 cells | 1 cell = 1600km²; 160*160 cells |

¹ The mentioned space characteristics (scale, resolution, extent) correspond to case studies that have been carried out. For most models other spatial scales, resolutions, and extents are also possible

² a = Topping et al., 2003; b = Topping and Oddenskær, 2004; c = Roeflofs et al., 2005; d = Heneghan et al., 2003; e = Chow et al., 2005; f = Gaines et al., 2005a; g = Gaines et al., 2005b; h = Gaines et al., 2004; i = Sample and Suter, 1994; j = Hope, 2005; k = Hope, 2001; l = Loos et al., 2006a; m = Loos et al., 2006b; n = Schipper et al., 2008; o = Tramper et al., 2007; p = Wolff, 1994; q = Matsinos et al., 1994; r = Matsinos and Wolff, 2003; s = Hallam et al., 1996
4. Human exposure models

The five state of the art human exposure models selected for inclusion in this comparative study were CARES, LifeLine, NORMTOX, SHEDS and TRIM.Expoy/APEX. The models were selected based on the same criteria as for the wildlife exposure models. They are described in more detail below.

CARES

CARES (Cumulative and Aggregate Risk Evaluation System) is a non-spatial model for estimating aggregate risks from drinking water, residential and dietary exposure for a single pesticide, and cumulative risks from pesticides that have a similar mechanism of toxicity. Acute (1 minute to 1 day), short term (2-30 days), intermediate-term (1 to 3 months), and chronic (1 year) exposures and risks can be evaluated for a reference population of 100,000 individuals (or a user-specified subset thereof) which are parameterised using US Census data. Each individual is represented by a vector of personal characteristics. Individual daily consumption and activity patterns for an entire year are generated from a database based on the degree of similarity between the personal characteristics in the vector and the database. CARES was originally developed by Crop Life America (CLA) and is currently owned by the International Life Sciences Institute (ILSI) Research Foundation.

LifeLine

LifeLine is a non-spatial model for the assessment of aggregate and cumulative exposures to pesticides in support of the regulatory decision making under the US Food Quality Protection Act. The model addresses exposures that occur from the use of pesticides on agricultural crops, in residences (homes and yards), and pesticide residues that occur in water supplies. The model’s focus is the general US population and subgroups thereof. LifeLine™ models longitudinal variation of individuals over their lifetimes starting with the characteristics of the individuals at birth, and based on longitudinal models of growth that have been developed using data collected in the US Third National Health and Nutrition Examination Survey (NHANES III). Like CARES, individual daily consumption and activity patterns are generated from a database based on the degree of similarity in personal characteristics. LifeLine is owned by the LifeLine Group; a US non-profit organization.

NORMTOX

NORMTOX is a non-spatial, non-dynamic spreadsheet model for estimating human exposure to environmental contaminants from multiple exposure media, i.e. food, air, soil, drinking and surface water (Ragas and Huijbregts, 1998, Ragas et al., 2008, Oldenkamp, 2008). The model predicts the long-term averaged daily intake of contaminants for the Dutch population or specific age classes thereof. Individuals are simulated by generating random values from predefined variability distributions that describe relevant intake and activity patterns. Correlations between the intake rates of different food products are not taken...
into account. One-stage or two-stage Monte Carlo simulation is used to produce distributions of exposure that reflect the variability and/or uncertainty in the input parameters. NORMTOX is owned by the Department of Environmental Science of the Radboud University in the Netherlands.

**SHEDS**

The Stochastic Human Exposure and Dose Simulation (SHEDS) model estimates aggregate (single-chemical, multi-route/pathway) and cumulative (multi-chemical, multi-route/pathway) human exposure to environmental chemicals such as pesticides, metals, and persistent bioaccumulative toxins. SHEDS is a physically-based, probabilistic model that predicts, for user-specified population cohorts, exposures incurred via inhaling contaminated air, touching contaminated surface residues, and ingesting residues from hand-to-mouth or object-to-mouth activities. It combines information on chemical usage, human activity data (e.g., from time/activity diary surveys and videography studies), environmental residues and concentrations, and exposure factors to generate time series of exposure for simulated individuals. One-stage or two-stage Monte Carlo simulation is used to produce distributions of exposure for various population cohorts (e.g., age/gender groups) that reflect the variability and/or uncertainty in the input parameters. SHEDS is maintained by the National Exposure Research Laboratory of US EPA’s Office of Research and Development.

**TRIM.Expo/APEX**

APEX is part of US EPA’s TRIM model framework; a time series modelling system with multimedia capabilities for assessing human health and ecological risks. APEX estimates human exposure to air pollutants at local, urban, and regional scales in the USA using a stochastic, “micro-environmental” approach. The model randomly selects data for a sample of hypothetical individuals from an actual population database and simulates each individual’s activities over time, in different locations (e.g., at home, in vehicles) to estimate its exposure to (and, optionally, dose of) the modelled pollutants. Probabilistic algorithms are used to estimate the pollutant concentration and ventilation (respiration) rate associated with each exposure event. APEX optionally calculates hourly, daily, monthly, and annual average uptake dose values for each of the simulated individuals. This option currently is available only for the pollutant Carbon Monoxide. APEX is maintained by US EPA’s Office of Air Quality Planning and Standards.

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Human exposure model characteristics</th>
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<tr>
<td></td>
<td>CARES</td>
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<td>Version</td>
<td></td>
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<tr>
<td></td>
<td>3.0</td>
</tr>
<tr>
<td>Short description</td>
<td>An object-oriented windows application originally developed by Crop Life America (USA) that predicts aggregate and cumulative exposure to air</td>
</tr>
</tbody>
</table>
predicts the daily intake of pesticides from different exposure routes in a US reference population. Cumulative exposure to pesticides for the US population. Long-term average daily intake based on intake and contamination data for the Dutch population. Exposure after inhalation, ingestion (by hand or object mouthing), and dermal contact for the US population. Pollutants at local, urban, and regional scales in the USA using a stochastic, "micro-environmental" approach.

<table>
<thead>
<tr>
<th>Species</th>
<th>Energy budget</th>
<th>Substances</th>
<th>Non-chemical stressors</th>
<th>Exposure routes</th>
<th>Exposure media</th>
<th>Exposure endpoint(s)</th>
<th>Effect level and endpoint(s)</th>
<th>Landscape-human interaction</th>
<th>Interaction between humans</th>
<th>Space representation</th>
<th>Movement</th>
<th>Activity patterns</th>
<th>Time step</th>
<th>Time window</th>
<th>Model platform</th>
<th>References</th>
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<tr>
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<td>Pesticides</td>
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<td>Ingestion</td>
<td>Food</td>
<td>Average or Maximum Dose (mg/kg/d)</td>
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<td>1 minute (DMM) or 1 day</td>
<td>1 day to 1 year</td>
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<td>Average Daily Intake (µg/kg/d)</td>
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<td>Metals</td>
<td>Dermal Inhalation None</td>
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<td>Average daily exposure and Average Daily Dose (mg/kg/d)</td>
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<td>Time Series of Exposure Concentrations (µg/m³); CO in blood (%)</td>
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<td>None</td>
<td>Geo-referenced (home and work) and Non Geo-referenced (other) micro-environments (related to activity pattern)</td>
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<td>US EPA’s Consolidated Human Activity Database (CHADs)</td>
<td>None</td>
<td>1 minute to lifetime</td>
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<tr>
<td>Humans</td>
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<td>Individual level: Carboxy-hemoglobin in blood</td>
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<td>None</td>
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</tbody>
</table>

Species: Humans

Age classes: 7 age classes between <1 and 55* years (or user defined classes).

Energy budget: No

Substances: Pesticides

Non-chemical stressors: Ingestion

Exposure routes: Dermal Inhalation

Exposure media: Food

Exposure endpoint(s): Average or Maximum Daily pesticide Dose (mg/kg/d)

Effect level and endpoint(s): Individual level: Aggregate Margin of Exposure

Landscape-human interaction: None

Interaction between humans: None

Space representation: Non Geo-referenced micro-environments (related to activity pattern)

Movement: None

Activity patterns: Combination of demographic data and user provided data on use

Time step: 1 minute (DMM) or 1 day

Time window: 1 day to 1 year

Model platform: Visual Basic 6.0 and Notitia™

References: a, b, c, d, e, f, g, h, i, j, k, l, m
### Human-specific

<table>
<thead>
<tr>
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<th></th>
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<tbody>
<tr>
<td>Food consumption data</td>
<td>Continuing Survey of Food Intake by Individuals 1994-1996, 1998 (CSFII; n=21662; m=2) and US EPA’s Food Commodity Intake Database (FCID)</td>
<td>CSFII (see CARES)</td>
<td>Dutch Food Consumption Survey 1997-1998 (n=6250; m=2)</td>
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<td>Not Applicable</td>
</tr>
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<td>Impact of food processing included?</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Not Applicable</td>
<td>Not Applicable</td>
</tr>
</tbody>
</table>

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1 Case studies have been carried out for the mentioned (groups of) substances. Fundamentally, most models are also suitable for other (groups of) substances.

2 a = CropLife America, 2002; b = ILSI, 2008; c = The Lifeline Group, 2007; d = Price et al., 2003; e = Ragas and Huijbregts, 1998; f = Ragas et al., 2008a; g = Oldenkamp, 2008; h = Zartarian et al., 2005; i = Zartarian et al., 2006; j = Xue et al., 2006; k = Zartarian et al., 2007; l = US EPA, 2006a; m = US EPA, 2006b
5. Model comparison and discussion

When comparing the exposure models for humans with those for wildlife, similarities as well as differences are revealed (Table 3). One of the similarities concerns the selection of chemical stressors, as the majority of the contaminants investigated are metals and pesticides. Nevertheless, the focus is slightly different - most wildlife models focus on metals, whereas most human models concentrate on pesticides. In addition, similarities can be observed regarding the exposure endpoints. Exposure is generally expressed either as a dose (intake or uptake) or as an internal body concentration, though most of the human models focus on the former and most of the wildlife models on the latter. Further, there are no conceptual differences with respect to the simulation of the physiological characteristics and development of the receptor populations. Where most human models divide the receptor population into more or less homogeneous cohorts according to e.g. age and sex, some of the wildlife exposure models distinguish cohorts based on development stages such as weaning, juvenile and adult, which is conceptually similar. Finally, a time step of one day is applied in most human and wildlife exposure models that are temporally explicit.

Besides these similarities, some interesting differences can be noted between the two groups of exposure models. These differences have been divided into four categories: (1) effect endpoint, (2) exposure routes and media, (3) incorporation of other stressors, and (4) activity pattern and space. Per category, the differences will be discussed and, if possible, explained. Options where human models can learn from wildlife models and vice versa will be identified and discussed.

Effect endpoints

When looking at the effect endpoints, there seems to be a slight difference in focus. In general, the human models aim at investigating adverse effects on individuals. Effects on the human population level are merely expressed as the number of individuals for which adverse effects might occur, determined by iterative model simulations and using risk indicators such as the ratio between the intake and Acceptable Daily Intake (ADI) or Reference Dose (RfD). Three of the wildlife model, i.e. RSEM, SEHM, and SpaCE, use a similar approach. However, two wildlife models, i.e. ALMaSS and WBM, include an extra level of biological organisation; they predict ‘true’ population-level endpoints, such as population growth rate or viability, derived from the consequences of reduced survival or reproductive success of individual organisms. This reflects a more fundamental difference between human health and wildlife assessments. Human health assessments generally aim at protection of the individual, whereas wildlife assessments generally aim at the protection of particular species or populations. A typical example of a human protection goal is the $1 \times 10^6$ risk limit for lifetime exposure to carcinogenic air pollutants used by the WHO (WHO, 2000). In contrast, wildlife risk assessments are typically aimed at protecting specific species, e.g., those defined in the US Endangered Species Act of 1973 and the EU Habitat Directive, or those playing a leading role in establishing and maintaining the structure and the functioning of the ecosystem (EC, 2003).
Exposure routes and media

In spite of the similarity in the selection of chemical stressors, the human models include a wider range of exposure routes and media than the wildlife models. The range of human models encompasses maternal transfer, ingestion, inhalation and dermal uptake and incorporates a variety of exposure media, whereas the wildlife models mainly simulate the ingestion of contaminated food. In some cases soil ingestion is also included, but inhalation, dermal uptake and maternal transfer are disregarded in all five wildlife models. The human exposure models thus have the ability to address a wider range of chemical substances than the wildlife models.

This can probably be explained by the fact that for humans multiple exposure routes are considered important, while for terrestrial wildlife ingestion is generally considered the predominant route of exposure, and all other routes negligible (Smith et al., 2007; Suter, 1993; Sample et al., 1996; Smith et al., 2007). For humans the relative contributions of the different exposure pathways can be as great as 40% for dermal intake (Khanal, 1999), 98% for ingestion (Khanal, 1999), and 70% for inhalation (Renner, 2002; Khanal, 1999; McKone and MacLeod, 2003), depending on the substance’s properties. As an example for wildlife exposure, Redfearn et al. (2004) showed that food chain exposures accounted for almost 100% of the total risks for all contaminants and all wildlife species considered in a risk screening study of polluted waters in the UK (Redfearn et al., 2004). A further reason for human exposure models being more sophisticated concerning the exposure routes included, might simply be that they have access to more detailed datasets than wildlife models. Estimates of dermal and inhalational exposure require intricate measurements that are largely absent for most wildlife species (Smith et al., 2007).

Nevertheless, in some cases exposure via other routes than food ingestion might significantly contribute to the exposure of wildlife and hence could be worth considering. For example, maternal transfer of contaminants could be an important exposure route for newborn animals (Zhang et al., 2008; Luebker et al., 2005). Inhalatory exposure to radiation might pose risks to burrowing wildlife in areas that are subject to radionuclic releases (MacDonald and Laverock, 1998). The assumption that routes other than ingestion are negligible may also be less appropriate when air emissions are the main route of contaminant entry into the environment, e.g., in the case of vehicular or incinerator emissions (Archbold, 2003). Contaminants such as volatile organic compounds (VOCs) may be more abundant in air than in other media. Moreover, for certain contaminants the inhalation pathway might yield a more severe adverse effect than ingestion as a result of differences in contaminant absorption, distribution, and metabolism (Driver et al., 1991; Bench et al., 2001). A comparative study on the relative contributions of inhalation and ingestion of, among others, VOCs and heavy metals (As, Ni, Cd) to the total exposure of Deer mice revealed that potential adverse effects were of comparable magnitude (Archbold et al., 2007). Effects of acute inhalation exposures have also been observed in birds at an oil industry flare stack (e.g., Bjorge, 1987). Inhalation risk to wildlife may also be important to explore in the case where the receptor’s home range is smaller than the compromised air shed, as may be the case with urban environments or large industrialised areas.
Incorporating other exposure routes and media in wildlife models might be relatively easy to achieve for external exposure, but will be much more challenging for internal doses. In the latter case knowledge about toxicokinetics of the substances (absorption via skin, lungs and gastro intestines) is needed. Data on internal concentrations in wildlife animals are hardly available (EC, 2003). Also data on toxicity remains a problem. The number of wildlife species for which toxicity data are available is limited (Munns Jr, 2006). And the data that is available is mostly expressed in term of concentrations of the food that the organisms consume (i.e. in mg·kg⁻¹ food) (EC, 2003). Because the responses of species of concern (often threatened and endangered species) rarely can be tested experimentally, expectations of how they might respond to stressor exposure must be extrapolated from the data of other, tested species. However, this might pose a problem, e.g. for inhalation exposure, where the use of allometric equations to account for differences in the inhalation rate of the receptor and the test species used to derive the toxicity reference values (usually rodents) is not well supported (US EPA, 1993). In the case of birds, the toxicological database remains too small to assess risks via inhalation (Archbold et al., 2007). Unfortunately, also little information about maternal transfer specific to terrestrial wildlife is currently available (Smith et al., 2007). A possible alternative method for ecological risk assessment for wildlife species in contaminated land might be the ecological vulnerability analysis, developed by Faber et al. (2004), and elaborated for wildlife by De Lange et al. (2006). In this method, ecological traits for individual species of wildlife are used to assess their exposure to soil contaminants, internal regulation and toxicological sensitivity to toxicants, and potential for population recovery from harmful effects at the level of the individual. However, due to unavailability of relevant literature data on the toxicity of the tested substances to the species the toxicological part of the vulnerability analysis remains problematic.

**Incorporation of other stressors**

When looking at the type of stressors addressed, all five human models were primarily designed to estimate exposure to chemical stressors, whereas two of the wildlife models, i.e. ALMaSS and WBM, were primarily designed to investigate impacts of other environmental factors, e.g. food availability, landscape structure and management. The latter approach is more general and allows the incorporation of multiple stressors and simulation of the receptors’ energy budgets, which is generally not accounted for in the models primarily designed for assessing chemical exposure.

A possible explanation might be the difference in legislation between human and wildlife protection. Regulations addressing human health risks are generally focussed on specific chemical substances (or mixtures) or on specific compartments. For example, widely used substances such as benzene and phthalates are evaluated within the context of substance-specific regulations such as the Toxic Substances Control Act in the USA and the new REACH programme in the EU. Substances encountered at polluted sites are evaluated within the context of compartment-specific regulations such as the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA; commonly known as Superfund) in the USA and the Water and Soil Framework Directives in the EU. Many human
exposure models are explicitly developed to support the implementation of these substance or compartment-specific regulations. Examples are CARES and LifeLine, which have been developed within the context of the US Food Quality Protection Act. As a result, these models focus on (cumulative and aggregate) exposure to pesticides, but fail to capture other stressors that may impact human health. Indeed, it is interesting to note that comprehensive regulations to protect human health against the potential effects of sequential or concurrent exposure to multiple stressors is currently lacking. To put it differently: who will protect the farmer who works with pesticides and lives on a metal polluted soil in a house with high radon exposure levels?

In contrast to human health policy, legislation concerning the protection of species is much more integrative and includes all kinds of stressors. Mandated by acts like the Endangered Species Act (ESA) and the Marine Mammal Protection Act (MMPA), governmental agencies such as the US Environmental Protection Agency are required to assess risks to wildlife populations, in its broadest definition, that result from exposure to anthropogenic stressors (e.g. agricultural and urban land use, introduced invasive and exotic species, nutrient enrichment, direct human disturbance, and toxic chemicals) (Munns Jr, 2006). The ESA specifically mandates the protection of the endangered species and its critical habitat, where the critical habitat extends to both the quality (suitability) and the extent (US EPA, 2003b). So risk assessment should determine whether the species’ critical habitat will be destroyed (reduced area) or adversely modified (reduced quality). The MMPA protects marine mammals from, among others, harassment, where the term ‘harassment’ means any act of pursuit, torment, or annoyance which has the potential to disturb a marine mammal or marine mammal stock in the wild by causing disruption of behavioural patterns, including, but not limited to, migration, breathing, nursing, breeding, feeding, or sheltering (US Code, §1362) (US EPA, 2003b). In other words, these harassments may be caused by chemical exposure, but also habitat loss (both quantitatively and qualitatively). Therefore, such regulatory laws promote exposure assessment models to address both chemical and other (physical, biological) stressors. ALMaSS is an example of a wildlife model that is integrative in its exposure assessment. This model was designed to answer policy questions regarding the effect of changing landscape structure or management on the population dynamics of key animal species (Topping et al., 2003; Topping and Oddenskær, 2004). These landscape structure and management changes include both physical stressors (e.g. mowing and crop rotation) and chemical stressors (i.e. pesticide use). The wildlife exposure models are generally better equipped to address cumulative exposure from a combination of chemical and non-chemical stressors.

Other stressors such as noise, heat, infection, etc., may play a significant role in the overall environmental health response (Robinson and MacDonell, 2004). Estimates of the adverse effects of chemical stressors can be expected to improve if they are considered in relation to the effects of other types of stress. So, human models might benefit from the inclusion of stressors that might interact with chemical stressors. Furthermore, human health policy starts to focus more and more on multiple and cumulative exposure assessments. EPA has begun to explore cumulative approaches to risk assessment (US EPA, 2003a). The EU-funded projects such as NOMIRACLE (NOvel Methods for Integrated Risk Assessment of Cumulative stressors in Europe, http://nomiracle.jrc.it/default.aspx) and more recent
European human health legislation also tries to address flooding, electromagnetic field, noise, nutrition flooding, heat, and mental health (http://ec.europa.eu/health/index_en.htm). This policy change could be a good incentive to develop more integrative human exposure models.

However, developing such models is challenging. Good human models for cumulative exposure assessment are not available and estimating effects of cumulative exposure is complex; many interactions between stressors may occur. Human exposure modelling, but also wildlife exposure modelling, might benefit from contributions made in the ecological field. The dynamic energy budgets (DEB) theory (Kooijman, 2000), tries to combine to effects of different types of stress using a dynamic energy budget, quantifying stress into energetic costs. This is a very interesting approach that might have potentials for being employed in the wildlife and human exposure and risk assessment.

**Activity patterns and space**

The most remarkable and fundamental difference between the two groups of exposure models concerns the simulation of the receptors’ activity patterns and food habits. While an individual’s activity pattern and food habits are mostly linked for wildlife, this is by no means valid for humans. The activities and food habits of humans tend to be determined entirely by predefined input parameters, derived from databases containing information on actual activity patterns and food habits of existing individuals. Contrastingly, parts of the activity patterns and food habits of wildlife receptors emerge during the model simulations, usually as a result of interactions between the receptors and the surrounding landscape. For example, in the SpaCE model the food habits depend on where the receptor forages and which preys are available at that location. Some of the wildlife models, i.e. ALMaSS and WBM, also include interactions between organisms, e.g. competition and predation, as a driving force for activity patterns.

The conceptual difference concerning the modelling of activity patterns is accompanied by a different treatment of the environment and the movement of the receptors therein. In wildlife models, the model landscape tends to have a clear geographic component. The different patches that make up the landscape are usually geographically embedded and reflect their actual positions with respect to each other and to the earth’s surface. Movements of receptors between the different landscape patches are explicitly simulated in the majority of the wildlife models, with the exception of RSEM. Contrastingly, the ‘landscape’ of the human exposure models generally consists of different micro-environments (e.g., home, workplace) that lack a geographical reference. Transfers of a receptor from one micro-environment to the next are never simulated in a spatially explicit manner. Only one of the human models included in this study (i.e. the APEX model) features a spatially explicit model landscape which consists of geographically embedded districts. APEX also predicts exposure during movement (commuting), but not in a spatially explicit manner, i.e., the exposure predictions are not geo-referenced.

An important reason for this difference might be the availability of data. Human activities impact the timing, location, and degree of pollutant exposure and play a key role in explaining exposure variation (Klepeis et al., 2001). Further, oral exposure is primarily determined by people’s food habits. This has
motivated the collection of activity and consumption data for specific use in exposure assessments (Klepeis et al., 2001). For human activity patterns and feeding behaviours, several extended databases are available, e.g., the Consolidated Human Activity Database (CHAD; US EPA 2003c) from the US EPA, and the Multinational Time Use Study (MTUS; MTUS, 2005) from the Centre for Time Use Research. Information on wildlife activity patterns is less detailed and more diffusely spread over different studies and literature resources. However, in 1993 the United States Environmental Protection Agency made an effort to put together scattered toxicity data on common wildlife species (US EPA, 1993).

Another potential explanation is related to the previously mentioned difference in protection targets. The focus on the individual implies that human health assessments must provide detailed and accurate estimates of individual exposure. Such accurate estimates can be obtained by imposing realistic activity patterns and food habits on the individuals modelled. The fact that wildlife protection focuses on the species or population level means that individual estimates are less important as long as the population endpoints are correctly predicted. If population endpoints such as the population viability (addressed by ALMaSS and WBM) are to be predicted, then factors that influence the viability of a population must be addressed. Such factors include habitat characteristics (i.e. contamination, availability of food and shelter) and coexisting individuals (predation, mating possibilities). Considering this and the fact that detailed data on individual activity patterns and food habits of wildlife are largely lacking, analysing the viability of populations can then be achieved by modelling the behaviour of the individuals as emerging from interaction with the surrounding environment and see how a population responds to a certain scenario.

The use of human activity pattern databases to simulate exposure is relatively simple and will generally result in relatively realistic exposure estimates. Wildlife exposure models generate behaviour by simulating underlying processes that are not always fully understood or that are simplified to reduce the computational costs. This approach, therefore, introduces some uncertainties. Consequently, their exposure predictions are less certain than predictions made with the human exposure models. Current human exposure models, on the other hand, only produce truly realistic exposure estimates as long as human activity patterns do not change. The imposed human behaviour assumes predefined activity plans and does not account for spontaneous behaviour potentially relevant for exposure. For example, if a person is shopping and meets a friend, this can influence its activity pattern (e.g., they might decide to drink something in a pub), and hence its exposure (e.g., to smoke or alcohol).

The mechanistic approach used in wildlife exposure models has the power to investigate the effect of manipulation of individual and environmental characteristics on activity patterns and exposure. The alternative approach, imposing behaviour derived from empirical data from human activity pattern databases, can be a simple, easy way to obtain the desired outcomes (Grimm and Railsback, 2005). Although modelling behaviour as emerging from behavioural rules has the disadvantage of being more complex, the benefit is a larger explanatory power (Grimm and Railsback, 2005). Human models could benefit from including interactions with the environment and/or other individuals, in order to account for those parts of human behaviour that are not entirely determined by planning and predefined intentions,
such as ad hoc decisions based on the weather. This can improve the understanding of underlying processes. Such models already exist for transport modelling. Originally, databases with predefined transport patterns were employed within the transport modelling. Nowadays, several object-oriented mechanistic transport models have been developed, taking into account individual behaviour as to enable modelling the effect of behavioural change on transport movements. Examples of such models are the mobility model for humans in urban areas (Meier et al., 2006), the Agent Based Meta-Model for Urban Mobility Modelling (Marilleau, 2005), and two agent-based models of pedestrian movement, i.e. STREET (Hacklay et al., 2001) and PEDFLOW (Kerridge et al., 2001; Kukla et al., 2001). Such models combined with urban exposure models might hold the future. One such exposure model is the interesting system combining two models to predict the noise and air pollution in urban areas (Tang and Wang, 2007). Another interesting exposure model, regarding noise, was recently developed by De Coensel et al. (2007). They followed an agent-based modelling approach to mimic the human response to environmental stressors and applied it to gain insight in the underlying mechanisms that lead to the emergence of noise annoyance.

For ecological risk assessors, it could be meaningful to investigate whether adequate exposure prediction of wildlife can be acquired with the use of activity pattern databases. Developing wildlife exposure models that make use of databases containing activity pattern information is conceptually quite simple. Therefore, the feasibility mainly depends on development of such databases for animal activity patterns. The development of human exposure models that incorporate emergent behaviour would be more complicated. First of all, the processes underlying behavioural choices of individuals are not fully understood to model behavioural (and exposure) patterns. Secondly, simulation of multiple interactions that characterize human behaviour might bring along severe computational challenges. Nevertheless, the examples of noise and transport models as mentioned above show that advances are being made.

Table 3
Comparison between five human and five wildlife exposure models

<table>
<thead>
<tr>
<th>Origin/purpose</th>
<th>human exposure models</th>
<th>wildlife exposure models</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- designed primarily to estimate chemical exposure (5)</td>
<td>- designed primarily to estimate chemical exposure (3) (SpaCE, SE^4M, RSEM)</td>
</tr>
<tr>
<td></td>
<td>- designed primarily for other purposes (2) (ALMaSS, WBM)</td>
<td>- designed primarily to estimate chemical exposure (3) (SpaCE, SE^4M, RSEM)</td>
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</table>

<table>
<thead>
<tr>
<th>Chemical stressors</th>
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<th>wildlife exposure models</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- metals (2) (NORMTOX, SHEDS)</td>
<td>- metals (4) (RSEM, SE^4M SpaCE, WBM)</td>
</tr>
<tr>
<td></td>
<td>- pesticides (4) (CARES, LifeLine, NORMTOX, SHEDS)</td>
<td>- pesticides (1) (ALMaSS)</td>
</tr>
<tr>
<td></td>
<td>- air pollutants (2) (APEX, SHEDS)</td>
<td>- PCBs (1) (WBM)</td>
</tr>
<tr>
<td></td>
<td>- others (Wood preservatives, Nitrate/Benzene) (2) (SHEDS, NORMTOX)</td>
<td>- others (uranium, radiocesium) (1) (RSEM)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Exposure routes</th>
<th>human exposure models</th>
<th>wildlife exposure models</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- ingestion (4) (CARES, LifeLine, NORMTOX, SHEDS)</td>
<td>- ingestion (5)</td>
</tr>
<tr>
<td></td>
<td>- inhalation (5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- dermal uptake (3) (CARES, LifeLine, SHEDS)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- maternal transfer (1) (LifeLine)</td>
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</tr>
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</table>

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<thead>
<tr>
<th>Exposure media</th>
<th>human exposure models</th>
<th>wildlife exposure models</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- food (3) (CARES, LifeLine, NORMTOX)</td>
<td>- food (5) (RSEM, SE^4M)</td>
</tr>
<tr>
<td></td>
<td>- soil/dust (4) (CARES, LifeLine, NORMTOX)</td>
<td>- soil (2) (RSEM, SE^4M)</td>
</tr>
</tbody>
</table>
| SHEDS) | - surface water (swimming) (1) (NORMTOX)  
- drinking water (3) (CARES, LifeLine, NORMTOX)  
- air (5)  
- residential media (e.g. coatings, building products, cleaners, play sets) (3) (CARES, LifeLine, SHEDS)  
- drinking water (1) (RSEM) |
|---|---|
| Exposure Endpoints | - intake dose (mg/kg·d) (3) (CARES, LifeLine, NORMTOX)  
- uptake dose (mg/kg/d) (1) (SHEDS)  
- internal concentration (mg/kg) (1) (APEX)  
- concentration in food (mg/kg) (1) (SpaCE)  
- intake dose (mg/kg/d) (2) (RSEM, ALMaSS)  
- muscle concentration (Bq/g) (1) (RSEM)  
- internal concentration (mg/kg) (4) (ALMaSS, SE\textsuperscript{4}M, SpaCE, WBM) |
| Effect endpoints | - parameters of individuals (expressed as risk indicators, e.g. intake/ADI-ratio, intake/RfD-ratio) (5)  
- population level parameters (e.g. population growth rate or viability) (2) (ALMaSS, WBM)  
- parameters of individuals (e.g. growth, survival, renal effects) (3) (RSEM, SE\textsuperscript{4}M, SpaCE) |
| Activity patterns | predefined, i.e. based on data from activity surveys (5)  
- emerging from interactions with landscape (5) and other organisms (2) (ALMaSS, WBM) |
| Space | - with geographical reference (1) (APEX)  
- without geographical reference (4) (CARES, LifeLine, NORMTOX, SHEDS)  
- with geographical reference (5) |
| Spatially explicit movement | not included (5)  
- included (4) (ALMaSS, SE\textsuperscript{4}M, SpaCE, WBM)  
- not included (1) (RSEM) |
| Receptor development | - categorised into cohorts (5)  
- simulated from day to day (1) (LifeLine)  
- categorised into cohorts (3) (ALMaSS, RSEM, WBM)  
- unspecified (2) (SE\textsuperscript{4}M, SpaCE) |
| Time step | - 1 day (2) (CARES, LifeLine)  
- 1 minute to 1 hour (2) (SHEDS, APEX)  
- not applicable/explicit (1) (NORMTOX)  
- 1 day (2) (ALMaSS, RSEM)  
- 15 minutes (foraging) / 1 day (exposure) (1) (WBM)  
- not applicable/explicit (2) (RSEM, SpaCE) |
| Time window | - 1 day to 1 year (2) (CARES, SHEDS)  
- 1 minute to lifetime (1) (APEX)  
- 1 day to lifetime (1) (LifeLine)  
- 1 year to lifetime (1) (NORMTOX)  
- 1 day to 120 days (1) (WBM)  
- 1 day to lifetime (2) (SE\textsuperscript{4}M, SpaCE)  
- 1 day to 66 year (1) (ALMaSS)  
- not applicable (1) (RSEM) |
| Other stressors | - not included (5)  
- included (e.g. lack of food or habitat, predation, weather) (3) (ALMaSS, SE\textsuperscript{4}M, WBM)  
- not included (2) (SpaCE, RSEM) |
| Energy budget | - not included (5)  
- included (3) (ALMaSS, SE\textsuperscript{4}M, WBM)  
- not included (2) (SpaCE, RSEM) |

* The number of models to which the characteristics apply is indicated between brackets.
6. Conclusions

The present paper compared five human and five wildlife receptor-oriented exposure models to gain a better insight in the modelling concepts employed and to make recommendations for improvement. Integration of human and wildlife exposure assessment is considered desirable for reasons of efficiency and quality of exposure assessment. This study can help risk assessors and modellers from both fields to improve the quality of their exposure assessment by adopting successful approaches employed by the other field. Based on the comparison, it is concluded that the human and wildlife models mostly address similar chemical stressors (i.e. pesticides and metals) and use comparable exposure endpoints (external and internal), time steps (i.e., one day) and concepts to simulate individual characteristics and development. However, there are also important differences. Human models tend to focus on the prediction of individual exposure and effect endpoints influenced by chemical stressors from multiple exposure routes and media, whereas wildlife models tend to focus on chemical exposure through food only, but include population effect endpoints (e.g. viability) influenced by non-chemical stressors (e.g. land use and food availability). The most outstanding difference is the way activity patterns are simulated. Where human models obtain activity patterns from databases, these are an emerging property in wildlife models, resulting from interactions between the organisms and the surrounding environment. This difference gives the wildlife models a better ability to address non-chemical stressors, whereas the human exposure models are generally more suitable to address a wider range of chemical stressors from different exposure routes. The differences can largely be explained by differences in protection targets (i.e., protection of human individuals versus wildlife populations or species), dominant exposure routes (multiple for humans versus ingestion for terrestrial wildlife) and data availability (activity data for humans are much more abundant and detailed than for wildlife).

Environmental policy in the USA and EU is slowly moving from a substance- and compartment-oriented approach towards a more comprehensive approach of integral health protection (US EPA, 2003a; CEC, 2003). This requires new tools to assess the effects from sequential and concurrent exposures to multiple stressors; chemical as well as non-chemical. The receptor-oriented approach seems particularly suitable for this purpose, and it is within this context that the human and wildlife models can learn from each other. Wildlife models can serve as a source of inspiration for human models when it comes to the simulation of activity patterns as emergent and the inclusion of non-chemical stressors (such as noise, heat, infection, etc.). Particularly, modelling human behaviour as emergent from individual traits and interacting with the environment might increase the explanatory power of human exposure models and improve exposure estimation by addressing those parts of human behaviour that are not entirely determined by planning and predefined intentions and lead to spontaneous exposure. Human models can serve as a source of inspiration for wildlife models when it comes to modelling exposure from multiple exposure routes and the compilation and use of activity data. This might eventually lead up to exposure
models employing a more holistic approach in line with the environmental policy needs that advance towards a more comprehensive and integral health approach.
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