

Ontology of active and passive environmental exposure

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Abstract. Exposure is a central concept of the health and behavioural sciences needed to study the influence of the environment on the health and behaviour of people within a spatial context. While an increasing number of studies measure different forms of exposure, including the influence of air quality, noise, and crime, the influence of land cover on physical activity, or of the urban environment on food intake, we lack a *common conceptual model* of environmental exposure that captures its main structure across all this variety. Against the background of such a model, it becomes possible not only to systematically compare different methodological approaches but also to better link and align the content of the vast amount of scientific publications on this topic in a systematic way. For example, an important methodical distinction is between studies that model exposure as an exclusive outcome of some activity versus ones where the environment acts as a direct independent cause (*active vs. passive exposure*). Here, we propose an information ontology design pattern that can be used to define exposure and to model its variants. It is built around causal relations between concepts including persons, activities, concentrations, exposures, environments and health risks. We formally define environmental stressors and variants of exposure using Description Logic (DL), which allows automatic inference from the RDF-encoded content of a paper. Furthermore, concepts can be linked with data models and modelling methods used in a study. To test the pattern, we translated competency questions into SPARQL queries and ran them over RDF-encoded content. Results show how study characteristics can be classified and summarized in a manner that reflects important methodical differences.

Keywords: ontology, epidemiology, RDF, GIS, computer science

1. Introduction

The Exposome NL project studies the Exposome, i.e., the combination of the exposure to factors in the built, physico-chemical, food, and social environments over a person's life¹. Researchers across the Netherlands from several institutions are researching the effects of the exposome on the human body. There are many different disciplines that cover the exposome, including the built environment, early life, obesogenic environment, policy, geospa-

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¹Exposome-NL <https://exposome.nl/about-us/about-the-exposome/>.

tial modelling, and the microbiome, to name a few. Indeed, this project, along with other studies in the field of epidemiology, has an increasing amount of work measuring some form of exposure to the environment to study its effects on a person's behaviour and health [1]. Yet the increasing amount and variety of approaches make it very time-consuming for researchers to find and compare results across articles relevant to some analytic goal. For example, a health-related study on walking behaviour might target the effects of outdoor air pollution while walking, or measure the effects of green space on walking behaviour, or the effects of such behaviour on physical health. Which goal precisely was pursued is hard to tell from a distance. While some authors have emphasized the opportunities of a corresponding "spatial turn" in the health sciences [2, 3], others see the increasing need to synthesize such evidence and systematically structure underlying models with the help of information ontologies [4]. This may allow systematic comparisons of the effects of interventions on behaviour and health, and thus support evidence-based theory building [5].

Information ontologies provide a way to make the shared conceptualizations underlying a particular kind of information explicit [6]. Information ontologies can encode domain-specific knowledge and are then called domain ontologies. In this study, we will refer to the latter simply as ontologies. Since conceptualizations can differ greatly even between research on the same topic, understanding them is crucial for validating and comparing research results. Over the past couple of decades, ontologies have become increasingly useful across medical and epidemiological sciences [7, 8]. To make conceptualizations explicit, ontologies make use of formal logic, which not only helps unambiguously define ideas (contributing to theory development) but also makes definitions machine-readable and thus helps automatically classify results (contributing to comparison and information retrieval). Together with methods for extracting and annotating content in published texts, this methodology can be used to link various resources underlying exposure studies. However, an ontology for breaking down and organizing different exposure concepts is currently lacking (cf. Sect 2).

Systematically distinguishing and aligning exposure measurements involves two major challenges. For one, there is the matter of designing the ontology [9, 10] to capture the central differences in the way exposure is modelled and used in scientific studies, such that we can answer corresponding questions [11, 12]. One important kind of question is causal. It asks whether the health exposure studied is largely under the subjects' control or not. The former we call *active exposure* and applies, for instance, to the exposure to unhealthy food, whereas the latter is called *passive*, for example, when being exposed to air pollution [13]. In the former case, buying or eating food is an activity that causes an exposure that can potentially be controlled by the involved person, while in the latter case, such control is not possible (furthermore, there are different types of passive exposure, which will be further explained in section 4.1.3). This distinction² is relevant because it determines which model components are required. For passive exposure, tracking of people's (mobility) behaviour and environmental stressor concentrations need to be modelled in detail, while for active exposure, behavioural choices of humans move into focus [13]. The distinction also has ethical and intervention/policy implications, because it determines to what extent a health impact due to exposure can be attributed to a person's responsibility. However, it remains unclear how this distinction can be precisely defined and operationalized. In addition, to capture the type of exposure and the tools and data sets used, we also need to identify the involved types of activities and subjects, their involved risk and the underlying environments or "environmental stressors" and how they were modelled. The second challenge relates to *knowledge extraction*, namely how data for such an ontology can be extracted, and how this can be scaled up across many article documents. Manually annotating articles with ontology concepts is a time-consuming process which does not scale. Luckily, recent developments in Natural Language Processing (NLP), such as the development of pre-trained deep neural networks for language parsing [14] have vastly increased the chances of automating the detection of exposure concepts within article texts [15].

Since this latter challenge requires first addressing the former, we concentrate in this paper on the first step of ontology design: Which concepts are needed to define exposure in epidemiological and health geography studies, such that relevant methodical differences like the types of exposure, environmental stressors and activities can be distinguished, including the underlying tools and data sources used for modelling it? We develop an ontology pattern

²Sometimes also captured by the dichotomy *voluntary vs. involuntary* exposure. We prefer active/passive over voluntary/involuntary because the latter additionally implies an intention of the involved person, which we think is too restrictive.

to compare exposure methodologies across different domains of exposure in order to prove the ontology's generality and to highlight methodological differences in research papers. Our ultimate purpose is to help scientists compare, align, and understand research results from studies on health-related exposures that look similar on the surface but are actually not similar when delving deeper into the methods.

In Sect.2, we discuss related work and requirements for such an ontology. In Sect. 3, we explain our design method, and in Sect. 4, we introduce the conceptual model, its (Web Ontology Language) OWL axiomatization and our reuse of existing vocabularies. Finally, in Sect. 5, we test and evaluate our ontology pattern over sample articles for these requirements.

2. Ontologies of exposure and competency questions

In this section, we review related work on exposure-related ontologies and tools and formulate requirements for ontology design in terms of competency questions.

2.1. Approaches to modelling health-related exposure and the environment

Information ontologies can be used to structure information in epidemiology and related fields [8]. Facts can be organized in terms of a so-called *knowledge graph* [16], which can be used to query, link or embed knowledge in various AI systems, (e.g., for deep learning-based Natural Language Processing (NLP) and information retrieval [17]). However, conceptualizations, as well as terminology, can differ greatly not only between different fields but also within a single field, such as biomedicine [18]. Designing large general-purpose domain ontologies, as was often done in the past, has therefore turned out to be difficult [19]. More recently, researchers have therefore turned to model aspects of a knowledge domain in terms of small, reusable *design patterns* for particular purposes [20] (e.g., based on the types of questions they can answer [11]). Patterns can then be linked to form larger ontologies for specific purposes. Our ontology focuses on systematically comparing methodological approaches with the aim to better link and align the content of the vast amount of scientific publications on exposure epidemiology.

The concepts underlying *environmental exposure* may serve as a pattern to link domains such as epidemiology, environmental science, geography and behavioural sciences. Yet, researchers have modelled exposure from different angles in the past. In the following, we review ontologies and their limitations in the fields of biomedicine, healthy living, and epidemiology, as well as on particular exposure-related health factors, such as food, physical activities, as well as human behaviour. We also discuss related knowledge-based tools. Finally, we discuss the only existing ontology that specifically focuses on exposure.

The Ontology for Biomedical Investigations (OBI ontology) [21] is an example of a large general domain ontology. In a multidisciplinary field posing challenges to terminology agreement, OBI suffers from corresponding problems. External ontologies reused in OBI are often subject to change with independent release policies, which can impact the scalability of changes to OBI [21]. For our purpose, the ontology is too general to address the specific problem of modelling exposure.

Various ontologies focus on medical health services, such as the one by [22]. The authors explore the possibility of using ontology to counsel patients on adopting a healthier lifestyle. Since the ontology focuses on the cognitive requirements of human interactions, it is less suitable for exposure assessments. Another example is the medical ontology by [23], which was designed to aid in making rapid, crucial decisions in healthcare. This ontology does not capture exposure concepts. Similarly, the ontology by [24] concerns the treatment and diagnosis of diabetes but does not include exposure as a concept.

[7] noted that many epidemiology-related ontologies have described concepts of specific sub-disciplines such as the *Disease* ontology [25], *Vaccine* Ontology [26], and *Symptom* Ontology [27]. In these ontologies, important epidemiological concepts are not yet covered, such as exposure ratio and attack rate [7]. The authors, therefore, created a general domain ontology called *The Epidemiology Ontology (EPO)* which covers some of these gaps [7].

The ontology also models exposure, but not in terms of a general environmental concept. Rather, it regards exposure as a process of *transmission of infectious or other disease agents among persons*³.

The Environment Ontology (ENVO) represents biomes, environmental features, and materials pertinent to genomic and microbiome-related investigations [28]. While first described in 2013, it was expanded and enhanced in 2016 after there was steady growth and demand to adjust it to support increasingly diverse applications [28]. ENVO was also aligned with the Open Biological and Biomedical Ontologies (OBO). The fact that ENVO was later improved to bridge multiple domains illustrates how an exposure ontology could likewise be expanded and diversified depending on demand. ENVO itself could be used to classify environments for exposure measurements.

Several ontologies focus on modelling the food environment. *FoodOn* is an ontology that covers basic raw food source ingredients, and process terms for packaging, cooking, and preservation. It also includes an upper-level product type scheme under which food products can be categorized. This ontology helps describe and organise food in detail and has been successful in standardizing database content for food-related agencies and health organizations [29]. The NAct ontology by [30] focuses on connecting data about activities and nutrition. While many nutrition models already exist, NAct takes a holistic approach by combining and personalizing nutritional and physical activity recommendations to support healthy living. The authors adopt rules that connect each subject's implicit and explicit nutritional and well-being goals with the situational condition of the subject, as well as with standardized European nutritional and well-being directives [30]. Both ontologies may be useful to model aspects of a food environment but they lack notions of exposure.

ORBM+ [31] is an ontology that models human behaviour. The authors study how social relationships and personal factors contribute to macro-level behaviours, such as physical exercise. They developed the ontology using a knowledge-driven approach, followed by a data-driven validation and refinement approach. The key idea is that a representation of a concept will be learned by its own properties, the properties of its related concepts, and the representations of its sub-concepts [31]. This ontology is linked to a human behaviour deep learning prediction model to make the behaviour prediction explainable. By incorporating human behaviour determinants – self-motivation, implicit and explicit social influences, and environmental events, the model predicts the future activity levels of users more accurately than conventional methods [31]. However, the ontology does not model health-related exposures.

[32] addresses the general conceptual challenges of exposure science with the *ExO* ontology. The authors note that while exposure-related terms are widely used in exposure science, definitions and descriptions are often inconsistent. The ontology is used to translate findings in various environmental disciplines, including epidemiology, for exposure and risk assessment and decision making and for improving public health [32]. The authors base their ontology on the gene ontology project, an ontology that describes the functions of gene products from all organisms [33]. ExO is structured hierarchically to allow the representation of data and concepts at varying levels of detail [32]. [32] suggest that the essence of exposure science is the study of the co-occurrence of an *environmental stressor* and a *receptor* or a target. However, as we will explain later, reducing exposure to cases induced by stressors is too narrow, since not in all cases, stressors or targets that receive the impact of an environmental stressor are available. Also, ExO lacks formal definitions of exposure and related concepts that can be used to automate the classification of different types of exposures, such as passive and active exposure.

Several knowledge-based tools are also of relevance in this context. For example, MOMO, described as a microbiology analytics and clinical tool for analyzing and reporting pathogens and antimicrobial resistances [34], was designed in response to aiding in the assessment and surveillance of infection in hospitals. MOMO's QuickScan function provides an overview of the data of an individual patient, can accommodate different kinds of data items such as PCR and microscopy results, and is updated daily. MOMO presents an efficient and powerful way to support an increasing body of knowledge in health and medicine and patients [34]. This study shows how technology alternatives to ontologies could be used for the same functionality.

Another alternative tool to ontologies is methodologies like the one developed by [35]. The researchers in this study recognized that in-depth analysis and extraction of knowledge have become more challenging in the era of big data. The aim of KNARM (Knowledge Acquisition and Representation Methodology) is to handle big data in

³EPO defines exposure as a BFO span:ProcessualEntity with the informal description "Proximity and/or contact with a source of a disease agent in such a manner that effective transmission of the agent or harmful effects of the agent may occur."

the form of large amounts of textual information and translate it into axioms by using description logic [35]. The authors demonstrated the methodology's functionality by implementing the Drug Target Ontology (DTO). Results showed that the methodology is capable of building useful, comprehensive consistent ontologies, and helps with acquiring and representing knowledge in a systematic, semi-automated way [35]. This approach and the findings of this study are comparable to ours, so we assume that populating our ontology can be done in a similar way in the future.

While all ontologies and tools discussed above touch on some aspects of exposure, including the behavioural component, different kinds of environmental stressors, as well as more general medical terms or risks, it is still unclear how concepts fit into each other when determining and measuring exposure. Furthermore, it also seems that even existing exposure ontologies such as ExO are not general enough and thus fail to capture important variants of exposure (e.g., the difference between active and passive exposures or environments that are not environmental stressors but that beneficially affect people). The ontology that we propose in this paper exactly addresses this gap by taking the different components underlying exposure measurement into focus.

2.2. Competency questions about constituents and types of exposure measurement

As our discussion illustrates, ontologies relevant to exposure range from understanding human behaviour and classifying physical activities and chemical substances to the kinds of nutrition and their effect on people's health. At first look, these cases are hard to align with each other in one model. Secondly, there are significant differences between exposure measurements in terms of methods and data. Given this variety, the question is what an overarching model of exposure could look like, which can be reused across all these cases to answer fundamental questions about methodology.

To capture such requirements, we formulate competency questions [12]. Competency questions should include those types of questions that an ontological model of exposure should be able to answer across all applications. We focus on understanding the conceptual model used in an article, and how it serves to link the used methods and data sources. Below, the rationale for each question is explained.

Question 1. *What kinds of exposure are modelled in this article?*

The variety of health-related exposures needs to be distinguished systematically and automatically. Identifying which type of exposure (e.g. passive or active) is used in a paper helps the reader determine if the article is of relevance. It also determines which environmental and individual aspects are relevant for modelling.

Question 2. *Which activities are involved in the exposure and who is exposed?*

Activities cause exposure to the people involved in them. At the same time, different kinds of activities mediate exposure. For instance, walking to school may cause higher exposure to air pollution than driving to school for the same route. Children who need to walk 2km to school will have a higher exposure to physical activity than children who only need to walk 800 meters to school. Additionally, the health conditions of people involved in an activity can also influence how they react to exposure. For instance, children may be more susceptible to NO₂ than adults. Thus, the demographic characteristics of persons and their activities both modify their health risks.

Question 3. *What are subjects exposed to?*

Whenever we are exposed to an environment, we are exposed to some of its aspects in more direct or indirect ways. Accordingly, the exposure can be quantified in different ways, which determines the specific kind of exposure. Note, that what a person is exposed to is not necessarily that person's environment. For example, a person's exposure to unhealthy food is not directly caused by the environment but is rather a consequence of an eating activity that is influenced by some (eating or buying) decisions that take place in the environment.

Question 4. *What is the health risk of exposure?*

This question identifies the potential health risk(s) an exposure may have for a person. For instance, the risk of children who live near busy roads developing asthma. Note that exposure can either decrease or increase risk and thus may have positive or negative associations.

Question 5. Which environments influence the exposure and from which data sets were environmental measures derived?

Depending on whether the exposure promotes risk or not, the effect of the environment on health can be positive or negative. For instance, an environment with negative influence could be high-temperature fields, which are themselves caused by impervious surfaces, in what is called the *heat island effect* in a city [36]. An example of a positive environment would be a park in a city increasing a citizen's recreational activity, which in turn reduces the risk of obesity. Environmental measures are often derived (e.g., mean temperatures or object densities in a neighbourhood around the home). This means the analytic methods involve a workflow that derives *spatial and temporal measures* from other environmental measurements. Therefore, if available, we are also interested in the workflows used for measuring these environments, including the data sources.

Question 6. What are the environmental stressors?

An *environmental stressor* is an *environment* that negatively influences the *health risk* of a *person* via her *exposure*. For example, high temperatures and impervious surfaces can be *environmental stressors* for elderly people in a city.

3. Methodology

In this section, we explain our approach to developing and evaluating the ontology pattern. Fig. 1 shows an overview of the development process, where numbers in brackets point to corresponding sections of this article.

3.1. Overview

Our design is roughly based on the steps in [10] with a particular focus on pattern development based on competency questions [9, 11, 12]. As an *empirical basis* for developing and evaluating the pattern, we *selected six articles* (knowledge acquisition) that covered diverse kinds of exposure and risk (Fig. 1) (see next subsection).

Ontology design methods [10] usually start with *requirements and purposes*. Following the idea of pattern development [11], these requirements were captured by *competency questions* [12] (Fig. 1, *empirical basis*). Focusing ontology design patterns around questions helps address basic design principles, such as clarity (questions can be understood without technicalities), extendibility (integration with other patterns), and minimizing ontological commitments (only those concepts needed for answering questions are formalized) [6].

We formalized the pattern in OWL 2⁴. Based on the questions, we designed a preliminary *pattern* (Fig. 1, *TBox*) [9] including classes and relations (OWL object properties) that capture the distinctions needed for answering the questions. The pattern describes the exposure theory (*TBox*). As far as possible, we thereby inherited classes from existing ontologies. The ontology design was done iteratively in several rounds revising the ontology based on the content of the articles (see dotted feedback arrows in Fig. 1).

To test the pattern, we populated a *knowledge base* by adding facts extracted from the articles, see Fig. 1, *ABox*. We *encoded the article content* by filling the slots of the pattern with text snippets and examples manually extracted from exposure articles. If needed, the ontology was extended with new concepts. We fully encoded the content of each article into RDF⁵, using classes and relations from the pattern. Using a mature version of the ontology, we then automatically *enriched* (Fig. 1) the RDF-encoded article contents by running OWL-RL⁶ and RDFS⁷ inference over the data. This step adds automatic class instantiations to the article content based on the formal definitions specified in the ontology pattern, and in this way allows us to classify article content based on logical reasoning (e.g., the fact that a certain exposure is of a certain type).

⁴Web Ontology Language, <https://www.w3.org/OWL/>.

⁵<https://www.w3.org/RDF/>

⁶https://www.w3.org/TR/owl2-profiles/#Reasoning_in_OWL_2_RL_and_RDF_Graphs_using_Rules

⁷<https://www.w3.org/TR/rdf11-nt/#rdfs-interpretations>

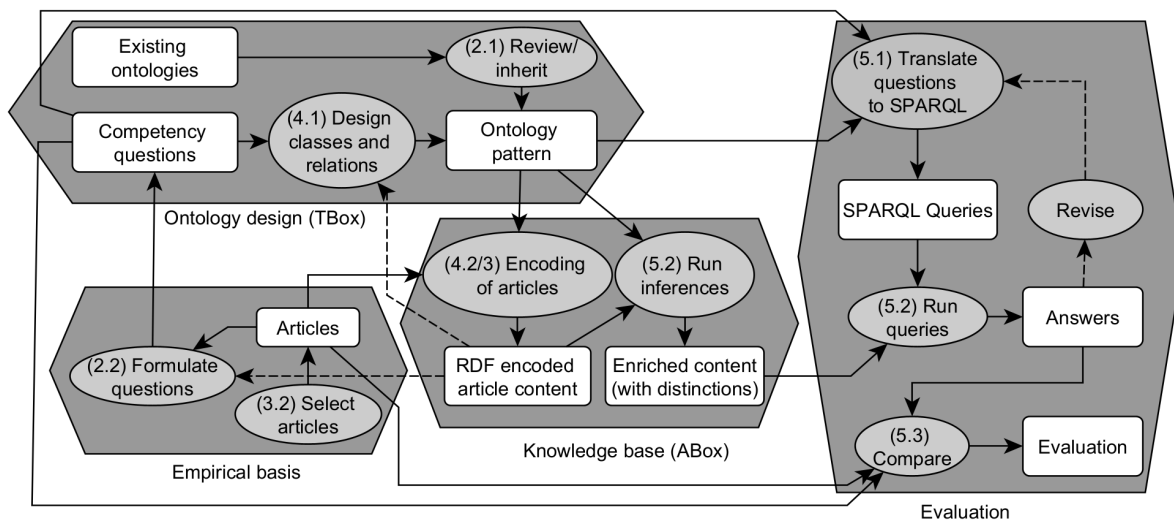


Fig. 1. Steps in building and evaluating the ontology pattern. Numbers refer to corresponding sections in this article. Ellipses denote processes, rectangles denote artefacts.

To *evaluate* the pattern, we (Fig. 1) *translated the competency questions into SPARQL*⁸ queries and finally *ran* all queries over the enriched article contents to analyse the content and to automatically classify and compare the articles against each other. We also compared the results against our expectations from reading the articles. This tests two things: first, whether the pattern is general enough to cover the diversity of exposure methods and specific enough to distinguish important methodical differences. And second, to what extent the pattern can be used for retrieval of methodological content. We discuss the results in Sect 5.

3.2. Selection of articles

Articles were selected from literature databases⁹ such that they should cover varying epidemiological risk factors and exposure types. We selected six articles on exposure to fast food outlets, neighbourhood social norms, air pollution (household and outdoors), crime, violence, urban green space, natural and built environment, and travel mode (see Table 1).

Exposure to fast food outlets includes places that sell unhealthy food [37]. Neighbourhood social norms are the perceived social norms that a person has in terms of what behaviours are acceptable with others in the neighbourhood (in this case, it specifically relates to how much fast food consumption is normally accepted in a person's neighbourhood) [37]. Both household and outdoor air pollution refer to the exposure of air pollution chemicals in a person's surroundings [38, 39]. Exposure to crime and violence refers to the exposure to such activities occurring in a person's immediate surroundings [40]. Urban green space refers to parks, gardens, trees, or other plants that a person may encounter in their immediate surroundings [41]. Travel mode in this case refers to whether a person travels by foot, bike, or motorized transport [42].

We chose these papers for two intentions. One is that they serve as empirical examples for exposure modelling in order to develop the ontology. The other reason is that they serve as a way to empirically evaluate the ontology by running queries over the statements in the papers and evaluating the answers. Papers should be as diverse as possible to make sure our ontology pattern can cover different types of health exposures. Furthermore, the chosen subject areas make our ontology compatible with the goals of the Exposome NL project. Because of the wide variety of

⁸<https://www.w3.org/TR/rdf-sparql-query/>

⁹<https://pubmed.ncbi.nlm.nih.gov>. However, note that a systematic review was beyond the scope of this article.

Title of article	Main Authors	Year Published	Health Exposure	Health Risk
The Associations of Area-Level Violent Crime Rates and Self-Reported Violent Crime Exposure with Adolescent Behavioral Health	Grinshteyn et al. [40]	2018	crime, violence	Adolescents, Behavioral Health, Mental health
Constituents of household air pollution and risk of lung cancer among never-smoking women in Xuanwei and Fuyuan, China	Vermeulen et al. [39]	2019	(household) air pollution	lung cancer
Long-term exposure to air pollution and cardiorespiratory disease in the California teachers study cohort	Lipsett et al. [38]	2011	air pollution	cardiorespiratory diseases
Neighbourhood fast food exposure and consumption: The mediating role of neighbourhood social norms	Rongen et al. [37]	2020	fast food outlets, neighbourhood social norms	fast food consumption
The relationship between access and quality of urban green space with population physical activity	Hillsdon et al. [41]	2006	urban green space	physical activity levels
Natural and built environmental exposures on children's active school travel: A Dutch global positioning system-based cross-sectional study	Helbich et al. [42]	2016	natural and built environment, travel mode	activity level of children

Table 1

The content of six articles was used for the development and evaluation of the ontology

topics that cover the exposome, the papers were also picked with the intention to represent this assortment of topics. The chosen papers represent scenarios studied by researchers associated with Exposome NL.¹⁰ [42] and [41] cover the topics of the obesogenic and built environments. [39] and [38] cover air pollution and lung or cardiovascular diseases. [37] explores the topics obesogenic and social environments, and [40] covers the social environment.

We made sure the papers that cover the same risk factor have different underlying exposure concepts, to show that even though a study may look similar at first glance, fundamental parts of it are structured differently, showing that it can be useful to use an ontology to more quickly discover these differences. We also made sure to cover a diversity of both active and passive exposure examples, including passive exposure examples of perceptual and physical nature. A short description of each article can be found in the Appendix.

4. Ontology design

This section describes our ontology design, motivating concepts, and the types of relations used to build it with the aid of *description logic axioms*¹¹. Description Logic (DL) is implemented in the W3C standards OWL and RDF. Many fragments of this logic are decidable and thus allow not only defining classes and relations between classes, but also the automatic inference of class subsumption (whether classes are subclasses of each other), and class instantiations (whether e.g., data samples can be classified accordingly). The ontology pattern was tested for consistency/coherency using the Hermit reasoner¹².

Ontologies are often divided into upper/top-level and domain ontologies, as well as lightweight and heavyweight ones. Lightweight ontologies are mere taxonomies [44]. Upper ontologies axiomatize general categories that can be reused across many knowledge domains [45]. An example of an upper-level ontology is the Descriptive Ontology for Linguistic and Cognitive Engineering (DOLCE) ontology [46]. DOLCE embraces a pluralist, cognitive perspective rather than targeting a unique universal ontology for knowledge representation [46, 47].

Ontologies may also be built off one another, similar to *design patterns* in software engineering [11]. Our ontology pattern can be used across the domains concerned with health-related exposure such as air pollution, food consumption and dieting, neighbourhood activities (crime, social activities), physical activity, built environment

¹⁰For more information on topics studied in Exposome NL, please refer to the list of published articles by Exposome NL researchers at <https://exposome.nl/research/publications/>

¹¹For an introduction to the DL syntax, see [43].

¹²<http://www.hermit-reasoner.com/>

(grey, blue, green space), noise, radiation, sleep, social economic status, and much more. It goes beyond a mere taxonomy because it defines exposure-related classes based on causal structures. We aligned our classes with the top-level ontology DOLCE+DnS Ultralite ontology (DUL)¹³, as it includes basic ontological distinctions relevant for modelling environmental agency (discussed below). We also reused a recently published ontology on quantities (AMMO¹⁴ and GeoAMMO¹⁵) [48] to describe quantifiable measures of exposure. Finally, we linked occurrences of these concepts to the articles in which they appear, as well as to corresponding data sources, by reusing standard vocabularies (DCAT¹⁶ and PROV¹⁷). Our pattern *exposureBasis* (*exp*) is available online¹⁸ as well as on github together with all resources¹⁹.

4.1. Basic model of active and passive exposure

We start with an informal motivation of the main concepts before introducing formal definitions. We first discuss the role of *causal relations* in exposure measurement, before we introduce concepts for the phenomena involved, and how they are related to each other. Afterwards, we introduce exposure types that can be defined as classes.

4.1.1. Causal relations and measure-able phenomena

From an analytical perspective, exposure is an important *cause* for health risks or health benefits. For example, exposure to an environment can cause a particular behaviour (e.g., when we are triggered by a nearby park to go running), which can be an indirect cause of exercising more and spending more time outdoors. Furthermore, it can also be a direct cause of health risks (e.g., when a person runs near a busy road). Finally, the environment can be modified by behaviour (e.g., when we decide to take a car instead of walking). Thus in environmental exposure, the environment can occur both as *cause* and *effect* in various causal chains [13].

In general, *causal relations* link measurable phenomena in a way that goes beyond spurious correlations. From causal theory [49, 50], we know that measurable phenomena might not correlate even though there is a causal link between them, and vice versa. This is especially relevant for the environmental and health sciences [51]. For example, whether the environment causes health risks might be hidden by confounding effects (causal forks), such as residential self-selection [52]. The distinction between causal relations and non-causal relations cannot be made without background assumptions [50]. Making such assumptions explicit results in a causal diagram, where causal relations appear as directed arrows between measurable variables. In essence, such a diagram is a conceptual model [53] which can be formalised in an ontology. For this reason, we use a *generalized causal relation* as a basic primitive DL role for connecting exposure phenomena.

Which measurable exposure phenomena should be linked by cause-effect relations? DOLCE and other top-level ontologies distinguish *events* (phenomena measured in time) from *objects* (phenomena measured in space), and *causal relations* typically exist only between consecutive events [46]. Other types of relations, e.g., *participation*, are used to link events and the objects participating in them [54]. Furthermore, from a philosophical as well as a practical viewpoint, it can be important to distinguish *causality*, which is a general vehicle for *explanation* in the sciences, from *actions* and *intentionality*, which focuses on purposes needed for *understanding* people's motives when deciding about their actions²⁰. However, the practice of causal analysis seems to be rather tolerant allowing causal links also for human behavioural decisions (e.g., an object like a park can cause an event like a run). We think this practice has also important theoretical implications because it highlights the role of particular causal chains for the conceptualization of exposure. To still account for ontological nuance, more specific ontological relations could be used to differentiate between types of causality, or to separate ontological relations expressing participation or

¹³http://ontologydesignpatterns.org/wiki/Ontology:DOLCE+DnS_Ultralite

¹⁴<http://geographicknowledge.de/vocab/Ammo.ttl>

¹⁵<http://geographicknowledge.de/vocab/GeoAmmo.ttl>

¹⁶<https://www.w3.org/TR/vocab-dcat/>

¹⁷<http://www.w3.org/TR/prov-overview/>

¹⁸<http://geographicknowledge.de/vocab/exposureBasis>

¹⁹<https://github.com/simonscheider/exposureStudy>

²⁰Causality as a vehicle for explanation in science is not really the same thing as when motives and decisions (teleologically) "cause" human actions, cf. [55].

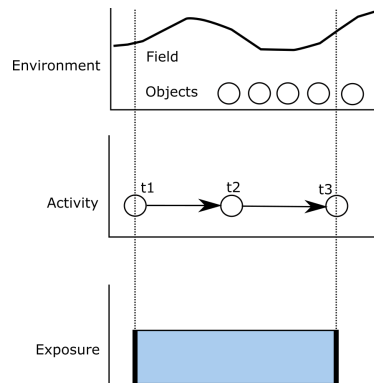


Fig. 2. Principle of exposure measurement: Geo-referenced environments (e.g. fields or objects) are summarized over the time and space of some activity in which the exposed person is involved. Exposure is a temporally extensive (accumulated) sum.

intentionality from causality. For example, we might specify that the *person* who decides to walk not only is a cause of the walking event but is also participating in this event and has the intention to walk. In the following, we reuse a *participation relation* in addition to a causal relation in order to express this, but we use a simplistic notion of causality that generalizes also over human intentions. In this way, we are able to distinguish intentional (caused) activities from unintentional participation. We leave more sophisticated types of specifications of effect relations open to sub-patterns of the ontology. For example, there is a causal relation between both food intake and health risk, and noise and health risk. However, they are based on very different physical processes that might be specified further in sub-ontologies.

4.1.2. Person, Exposure, Activity, Environment, Risk and Dose

A *person* is a human being who participates or initiates an *activity* that will cause an *exposure* impacting their health. The person is the main study subject of the observation being made about how exposure is impacting their health.

What exactly is *exposure*? In the following, we base our explanations on the notions of measurement control as introduced by [56], on a related amount theory [48], as well as on standard definitions in epidemiology. Without being too specific, we can say that exposure is a measurement of some amount that is controlled by (and accumulates) over time. In many cases, exposure refers to the amount of a particular environmental phenomenon that reaches a person, expressed in terms of physical state, concentration, duration, and frequency²¹. If you are exposed to some phenomenon for some time, and then again for another time, the total amount of exposure will increase by the amount of exposure in this additional time interval. Exposure therefore can be defined as a *temporally extensive (accumulative) amount*, i.e. an amount controlled by and adding up over an amount of time [48]. This amount of time is, in turn, controlled by some activity of the person who is exposed (see Fig. 2).²² For example, the amount of exposure to NO₂ and the amount of physical activity are both controlled by the time interval of a person biking along a road with traffic. The longer a person bikes, the more exposed the person is to both.

An *activity* happens in time and involves a person. We hold that exposure is always measured relative to some activity (e.g., it is always based on the duration of the activity and can be measured relative to the location of the person involved in the activity). Yet, how the activity influences exposure is different for different types of exposure: in the case of food intake, the amount and the quality of food are important. In the case of noise, the duration and the location of the person involved are relevant. As in the example above, activities are caused by persons. This could be anything from simply living in a certain place, to biking, or to buying food. Activities can be stationary or involve movement. If persons have control over an activity they can choose to perform it (for example, you choose to smoke

²¹Cf. <https://www.endocrinescience.org/glossary/exposure/>. We generalized "substance" in this standard definition to phenomenon since some exposure types are immaterial

²²Time as a control or measure is not explicitly modeled in this study because while it is an important component of exposure measurement, we tried to simplify the concepts as much as possible in the ontology.

or not). Sometimes there are many alternatives to choose from (for example, for your commute to work, you can choose to bike, take public transport, or walk). However, sometimes people do not have control over performing an activity. In fact, the environment constrains people's activity options, sometimes up to the degree that there is no choice and the activity becomes involuntary. In that case, the person does not cause the activity but the environment or biological need causes it (e.g., a person falling asleep because of exhaustion, or a person shivering because it is cold outside). In the following, we assume activities are not necessarily voluntary (i.e., caused by persons), even though they always involve some person.

An *environment* consists of characteristics within a neighbourhood of the location of a person. This could involve tangible phenomena of the landscape or of our home (road intersections, coal mines, fast food outlets, food in your fridge) or intangible ones (NO₂, farm odour), or even *fiat* phenomena like a culture or an administrative boundary [57]. What is considered an environment is therefore not only dependent on the *spatial location and scale* of a person, but it also depends on the person's activity. For example, the environment for shopping is constrained by the accessibility of shops. Thus activities become constitutive of environments. It is this connection between the activity, environment, and exposure that leads to health risks or health benefits. To model environments and their effects on activities and exposures, *spatial relations* (such as distance and topology) are thus needed to determine the *spatial context* [58, 59] of activities. Furthermore, different *conceptualizations of the environment*²³, as well as corresponding measurements [48], need to be taken into account (cf. Fig. 2). For example, one could distinguish between environments consisting of discrete, movable *objects* (such as cars), *events* (such as car crashes), or ones that consist of spatially continuous *fields* (such as air pollution), which all require different modeling approaches. In addition, one could distinguish between quantified measurements of such environments on a linear scale (*environmental factors or magnitudes*, such as car or air pollution density) and the underlying types of environmental phenomena themselves (*environments*) [48].²⁴ Note that in this article, however, we did not focus on ontological models of different kinds of environments and their quantification, which is considered future work. Instead, we use a generalized notion of environment which encompasses all of these options.

Dose is an amount of something accumulated in the person's body due to exposure. For example, it can be the amount of a passive environmental stressor (e.g., NO₂, noise) that enters a person's body dependent on the concentration or intensity in the environment and the physiological properties of the person.

Health Risk is a person's probability of participating in an event that negatively influences the person's health status within a specified period of time²⁵. For instance, a health risk could be a heart attack, disruptive behaviour, or obesity. The degree to which a health risk influences one's health or mortality varies. However, note that exposure might also decrease health risk. Health risks are often mediated by doses.

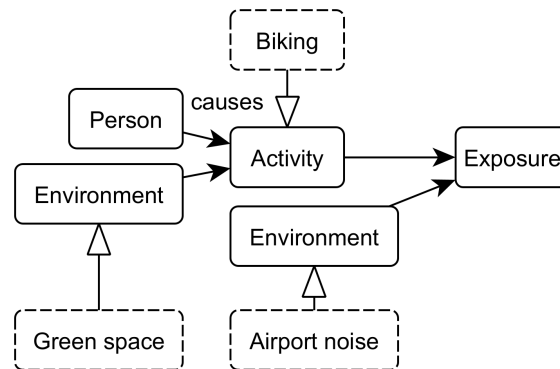
4.1.3. Active and passive exposure in a nutshell

While the same basic components, as outlined above, seem to constitute the parts of any exposure assessment, *their causal configuration differs from one case to another*. The argument we want to make in this article is that this causal structure precisely distinguishes the different cases of exposure from a methodological viewpoint. For example, for modelling the activity of food intake we have to consider the environment (e.g., fast food outlets), some activity (e.g., buying, eating), and the characteristics of a person involved (e.g., age), as well as the health risk involved (e.g., the risk for obesity). The activity of food intake can be caused by exposures to the environment as well as lead to exposures to food leading to a certain risk such as obesity. To model the exposure to noise, we likewise have to take into account an environment (noise level and noise sources), some activity (commuting to work) in which some person (school child) is involved, as well as some health risk (e.g., mental health). Yet, in the latter case, components are arranged in different order. Thus, to model the various cases of exposure, our pattern needs to allow the modelling of the *configuration of the causal relations* between these components.

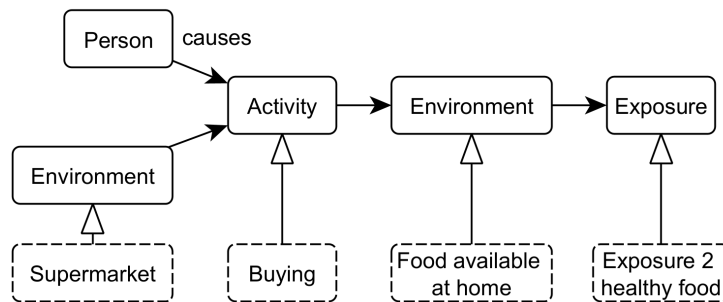
²³Including *core concepts of spatial information*, such as fields, objects, networks, and events [60].

²⁴Environmental factors include the aggregation/measurement of the environment on a linear scale for an exposed person, while an environment itself can be conceptualized as a layer of similar phenomena in space. The difference is reflected in the preprocessing of geodata layers. There is separate work on aggregation of environmental factors and recent ontological work on quantification of amounts in GIS [48]. However, such distinctions were not needed for the purpose of this paper.

²⁵Risk in epidemiology is commonly used more loosely to talk about probabilities of events more generally. We stick to a more restrictive interpretation which we think is more informative.



(a) Possible model of passive exposure. Though termed "passive", some activities are always implied. Examples are indicated by boxes with broken boundaries.



(b) Possible model of active exposure. Note that our definition does not exclude environmental causes, but restricts it to be at most an active cause of exposure (dotted box), or else a cause of an activity.

Fig. 3. Possible models of active and passive exposure.

Which causal patterns should be distinguished? In noise exposure, *the exposure is caused by a particular environment*. So there is a direct causal influence of the intensity of noise in the environment on the amount of exposure to noise, which then influences the amount of health risk. So we have a chain: *Environment* → *Exposure*. In addition, the activity likewise influences exposure, in the sense that it determines the spatial context of the person being exposed (cf. Fig.3a). This is different in the case of food exposure, which is an *exposure to (something caused by) an activity*. In the latter case, an environment (e.g., the density of fast food restaurants or the availability of food in your fridge) still plays an important role, but only as an activity triggering, indirect cause of the exposure. This means it is either itself caused by some activity (food in your fridge is caused by buying), or causes an activity that causes the exposure (number of fast food restaurants causes your eating there). This reflects the fact that no matter how many fast-food restaurants are around you, you are not forced to eat there. There is always an intermediary activity (and thus an implicit decision of eating or buying) involved between the environment and the exposure and the health risk. For this reason, it is not in itself risky to drive by a McDonald's restaurant, at least not in the same sense as driving by a polluted area. Thus, for the food case, we instead have a chain: *Environment* → *Activity* → *Exposure*.

We call the causal configuration in Fig.3b *active exposure*, where the exposure is controlled by a person, even if that person's activity might be influenced by the environment. Note that this distinction has important implications for (1) the modelling of exposure (which components need to be modelled, in which order), but also in terms of (2) ethics: while fast food restaurants can be avoided, no one can avoid noise around an airport when driving by. The causal configuration in Fig.3a is called *passive exposure*. Though an activity is always involved, there is also a component that is entirely independent of a person's activity (and thus beyond that person's control). Depending

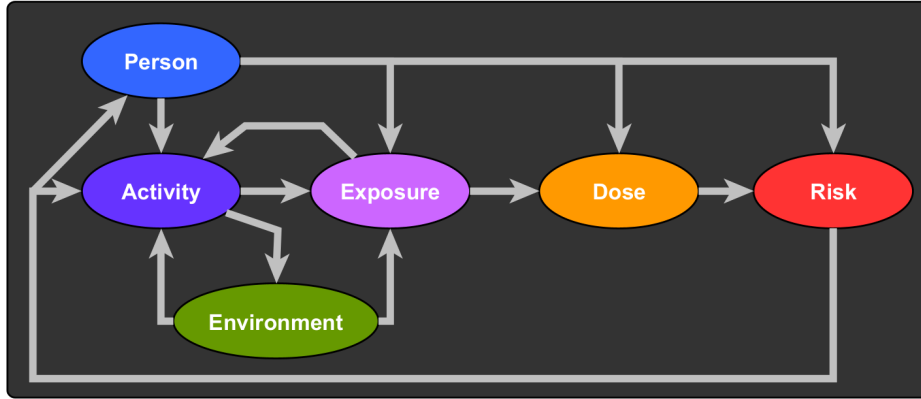


Fig. 4. Base classes of the ontology. Arrows show possible causal relations, ellipses are classes denoting concepts.

on how this component affects a person's body, exposure can be further distinguished into *physiological exposure*, and *perceptual exposure*. Physiological exposures include exposures that physically enter or affect the body (e.g., air pollution, sunlight). Perceptual exposures are exposures that involve perception (e.g., the perception of crimes and its effect on the feeling of safety).

An overview of the most important concepts (base classes) and their possible causal relations is given in Fig. 4. We believe that all base classes are relevant at least as background assumptions in a specific model, even if such assumptions may not explicitly be modelled with data. In the following, we will make these differences formally explicit in terms of our *basic exposure ontology* pattern.

4.1.4. DL-Axiomatization of exposure concepts

We first introduce base classes for the different open slots in our causal model of exposure (standing for the ellipses of Fig. 4), including *exposure*, *environment*, *activity*, *person*, *dose* and *health risk*. While it would be beneficial for an exposure ontology to also model the variety of *environments*, this is out of scope in this article. This could be added in a sub-ontology by inheriting from the environment class (see Sect. 6). The six classes mentioned above are all mutually exclusive, meaning that something cannot be of more than one of these classes at the same time (e.g., not a *person* and an *activity*):

Axiom 1. *Base classes are mutually disjoint*

$$\begin{aligned}
 & (Exposure \sqcap Environment) \sqcup (Exposure \sqcap Activity) \sqcup (Exposure \sqcap Person) \sqcup (Exposure \sqcap Dose) \sqcup \\
 & (Exposure \sqcap Risk) \sqcup (Environment \sqcap Activity) \sqcup (Environment \sqcap Person) \sqcup \\
 & (Environment \sqcap Risk) \sqcup (Environment \sqcap Dose) \sqcup (Activity \sqcap Person) \sqcup \\
 & (Activity \sqcap Risk) \sqcup (Activity \sqcap Dose) \sqcup (Person \sqcap Dose) \sqcup (Person \sqcap Risk) \sqcup (Dose \sqcap Risk) \sqsubseteq \perp
 \end{aligned}$$

Note that the phenomena that fall under these classes have measurable qualities that are not identical to the phenomena themselves. We distinguish different kinds of phenomena and their qualities using DOLCE+DnS Ultralite (DUL). *Objects* are phenomena whose qualities are controlled by time moments (*dul:Object*). For example, *persons* (*dul:Person*) as well as *environments* can change their qualities in time. We model *environments* as a *dul:PhysicalPlace*, i.e., phenomena where persons can be located. *Activities* are a form of an (*dul:Event*), i.e., entities whose qualities are not controlled by time moments, but which have some fixed temporal extent. More specifically, activities are a subclass of *dul>Action*, in which persons can participate and which they can cause. The following axioms specify *causal relations* (arrows) between the measured qualities of *exposure* concepts:

Axiom 2. *Causal roles*

$$\begin{aligned}
causes &\equiv causedBy^- & Disjoint(hinders, promotes) \\
hinders &\equiv isHinderedBy^- & hinders \sqsubseteq causes \\
promotes &\equiv isPromotedBy^- & promotes \sqsubseteq causes
\end{aligned}$$

We consider a single causal relation *causes* which is the inverse of *causedBy*, denoting whether some quality of some phenomenon is causally influenced by some quality of another phenomenon. For example, both the environmental concentration of NO₂ (a quality of some environment) and the duration of the cycling activity of a person (a quality of some activity) cause an exposure to NO₂ (an accumulation amount). This, in turn, *causes* a dose of NO₂ in this person's body. We only distinguish two sub-relations: *hinders*, which means a measured quality influences the other in a negative direction (the more, the less) or not (*promotes*).

Axiom 3. *Non causal roles*

$$\begin{aligned}
of &\equiv has^- & participatesIn &\equiv involves^- \\
\top &\sqsubseteq \forall has.Exposure & \top &\sqsubseteq \forall participatesIn.Activity \\
\top &\sqsubseteq \forall of.Person & \top &\sqsubseteq \forall involves.Person
\end{aligned}$$

We also consider some non-causal relations. *of* relates an exposure to the person exposed, and *has* denotes the inverse relation. We use standard ontological notions (inherited from DUL) for saying that a person *participatesIn* some activity, and that, vice versa, the latter *involves* some person. Note that a person's participation in an activity does not imply that the person is also causing (i.e., intending) it, though this fact can be expressed simply by using both roles *participatesIn* and *causes* to describe the relation between a person and an activity.

The classes *exposure* and *dose* correspond to a particular kind of amount (*AMMO:Amount*), namely an amount accumulated over (and thus controlled by) some time interval (*GeoAMMO:AccumulationAmount*)²⁶. More specifically, an *exposure* corresponds to a person's accumulated amount of exposure to something over some time interval during an activity in which the person is involved. The time interval can be the extent of the activity or any part of it. For example, residents are exposed to local air quality at any time during which they reside in the same place. In this case, the air quality at the place is measured by concentration, the persons are residents and the activity is living somewhere. A *dose* is an amount of substance left in a person's body as a consequence of its exposure. For example, this could be the amount of PM₁₀ in your lungs. An exposure magnitude might be measured as a temporal integral of intensities, e.g. as a sum of NO₂ concentration values over some time interval. Strictly speaking, the measured magnitudes (e.g. in grams) are not identical with the amount (e.g. the amount of NO₂ in the body) [48]. Yet, note that we do not further model the temporal aspect of these concepts in our design pattern, nor extract this information from the papers. This is because it is not required for answering the competency questions, and to keep the pattern as simple as possible.

We formalize the causal structure of these concepts by requiring that *exposures* always depend on particular *persons* via some *activity* in which they are involved during the exposure²⁷. For example, a person's exposure to NO₂ is caused by that person's biking. This requires *exposure* to be of *exactly one person* who is involved in some causing *activity* :

²⁶ An accumulation amount is measured by an accumulation measurement function. The latter is controlled by amounts of time [48].

²⁷ A more complete formalization of our exposure definition above would require modelling amount domains explicitly in the pattern. We have refrained from doing so to keep the pattern simple.

Axiom 4. *Exposures, persons and the activities in which they are involved*

$$\text{Exposure} \sqsubseteq ((\exists \text{causedBy.Activity}) \sqcap (\exists \text{of.Person}) \sqcap (\leq 1) \text{of.Person}))$$

$$\text{Activity} \sqsubseteq (\exists \text{involves.Person})$$

$$(\text{Person} \sqcap \exists \text{causes.Activity}) \sqsubseteq \exists \text{participatesIn.Activity}$$

$$(\text{Person} \sqcap \exists \text{has.Exposure}) \sqsubseteq \exists \text{participatesIn.}(\text{Activity} \sqcap \exists \text{causes.Exposure})$$

Furthermore, activities always involve some person, and if persons (intentionally) cause an activity, then they also participate in it. Finally, if a person is exposed to something, then this person also participates in the activity that causes this exposure²⁸. Note that though every exposure belongs to only a single person, still several persons might be involved, intentionally or not, in a given exposure activity. For example, though a family collectively might burn coal in a household, every member of the family has their own exposure to the emissions as a consequence of their participation in this activity.

Next, we specify the effects of exposure on this person in terms of its *dose* and *health risk*. We call an exposure or dose *health-relevant* if it causes some health risk for this person. For example, exposure to fast food may increase the health risk of obesity. Note some exposures are not health-relevant because no health risk is involved. For example, a traffic sign may have caused me to stop at a road intersection. Furthermore, we call the activities causing these exposures also health-relevant. We define this in terms of DL role restrictions.

Definition 1. *Health impacts*

$$\text{HealthRelevantDose} \equiv (\text{Dose} \sqcap \exists \text{causes.Risk})$$

$$\text{HealthRelevantExposure} \equiv ((\text{Exposure} \sqcap \exists \text{causes.Risk}) \sqcup (\text{Exposure} \sqcap \exists \text{causes.HealthRelevantDose}))$$

$$\text{HealthRelevantActivity} \equiv (\text{Activity} \sqcap \exists \text{causes.HealthRelevantExposure})$$

$$\text{RiskPromotingDose} \equiv (\text{Dose} \sqcap \exists \text{promotes.Risk})$$

$$\text{RiskPromotingExposure} \equiv ((\text{Exposure} \sqcap \exists \text{promotes.Risk}) \sqcup ((\text{Exposure} \sqcap \exists \text{promotes.RiskPromotingDose}))$$

$$\text{RiskPreventingExposure} \equiv ((\text{Exposure} \sqcap \exists \text{hinders.Risk}) \sqcup (\text{Exposure} \sqcap \exists \text{hinders.RiskPromotingDose}))$$

If we know such exposures (or doses) *promote* health risk rather than *hinder* it, meaning there is a promoting chain of causes from activity to health risk, then we speak of a *health risk promoting exposure (dose)*.

The term *environmental stressor* has been defined in various ways by different researchers. Most of these definitions involve both an *environment* and some (negative) response for the exposed *person*. For example, [61] describes an environmental stressor as "any intrinsic or extrinsic factor that challenges individuals and obliges them to adjust behaviour". [62] defines environmental stress as "the emotional, cognitive and behavioural responses to an environmental stimulus (or *environmental stressor*)". Thus "whether stress occurs is dependent on individual and contextual factors." [63] shows how environmental stressors can be further categorized according to the degree of actionability (directly or indirectly), its predictability, and how salient or identifiable it is.

We define *environmental stressors* simply based on the causal relation between *environments* and *health risks* of the *person* exposed. Environmental stressors are environments which *promote* some exposure that *promotes* some health risk. Note that environmental stressors therefore are not necessarily part of an exposure: For example, in the case of exposure to fast food, there is no environmental stressor involved, because the environment does not directly cause the risky exposure. Furthermore, there are also environments that cause exposures which *hinder* health risk and thus *promote* health, e.g., exposure to green space. Finally, note that our definition leaves room for all the environmental stressor-related concepts cited above, including controllability, cognitive, and physiological responses. These can be accounted for by distinguishing corresponding relations between actions and the kinds of exposure involved (see the distinctions defined below).

²⁸The latter statements cannot be fully formalized in DL because of its restrictions regarding triangular relations.

Definition 2. Environmental stressors

$$\text{EnvironmentalStressor} \equiv (\text{Environment} \sqcap \exists \text{promotes.RiskPromotingExposure})$$

Finally, we can define the difference between *active* and *passive exposures* based on distinguishing their causes in terms of involved activities, and thus in terms of personal responsibility. We first introduce a class *Active*, which is defined as something that is either itself an activity or caused by some activity:

Definition 3. Active

$$\text{Active} \equiv (\text{Activity} \sqcup \exists \text{causedBy.Activity})$$

Note that this class includes, besides activities, also environments whenever the latter are caused by some activity. For example, when we burn coal in an oven without a vent, we cause air pollution in our homes. Note that this definition serves only as a way to generalize over both options. Now, we call an exposure *active* if has only *active* causes, i.e., it is either directly caused by an activity or by something that is itself caused by one. We call an exposure *passive* if it is directly caused by some environment:

Definition 4. Active and passive exposures

$$\text{ActiveExposure} \equiv (\text{Exposure} \sqcap \forall \text{causedBy.Active})$$

$$\text{PassiveExposure} \equiv (\text{Exposure} \sqcap \exists \text{causedBy.Environment})$$

This definition builds on the following logical reasons: If the exposure is caused only by some activity (Def. 4), and thus, indirectly, by the causes of that activity (e.g., a person's decision to act) (by Axiom 4), then we know there is no independent influence of the environment on the exposure, and thus the responsibility of exposure lies entirely within the hands of the person who controls this activity. Our DL definitions above partially distinguish between these different models as illustrated in Figure 3.

However, to implement this idea of *active exposures* in our model, we need to ensure that the exposure is not caused by something that is *not active*. This requires knowing whether something is not the case (logical negation \neg), which requires the *logical closure* of our knowledge base (cf. [64]). Since DL has an open-world assumption (what we do not know is not automatically false), this reasoning goes beyond standard DL reasoning. To account for this, we *locally closed our world* of causes to be able to make this inference within DL:

Inference rule 1. Local closure of causedBy Activity. *If something is caused only by activities in the graph g, then we add an all-constraint:*

```
def locallyCloseWorld(g, property=exp:causedBy, all = exp:Active):
    for s in g.subjects(property, None):
        allconstraint = False
        objects = g.objects(s, property)
        for o in objects:
            if (o, rdf.type, all) in g:
                allconstraint = True
            else:
                allconstraint = False
                break
        if allconstraint:
            g.add(s, rdf.type, \forall \text{forall } property.all)
```

Note that our two definitions for *passive* and *active exposure* are not mutually exclusive, namely, in case the environment is caused by some activity (e.g. burning coal). To make them mutually exclusive, we would need to request that passive exposure causes are never active, which requires another local closure of a similar kind. In

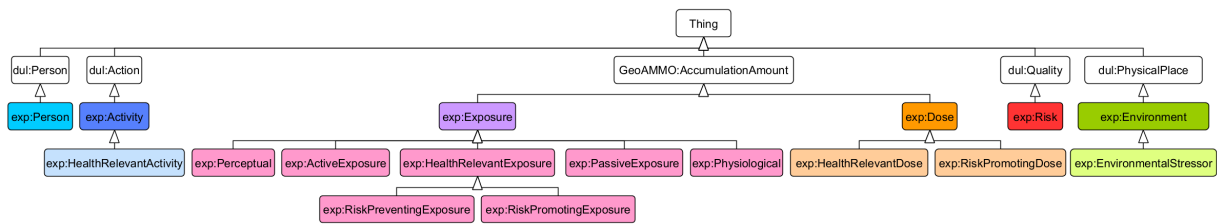


Fig. 5. Overview of class hierarchy of the Exposure Basis pattern including defined classes. The class *Active* is left out as it is used only for axiomatization purposes.

this paper, we decided to leave this stricter definition out, because the more loose definition also illustrates that sometimes exposure can be considered both *active* and *passive*. In addition, we added subclasses for *perceptual* and *physiological exposure* which capture differences in the way exposure is caused. The *exposure* class can be seen as a reified n-ary relation between the causes constituting the exposure. Thus, these *exposure* subclasses also capture specific ways in which an environment and a person are related, e.g., via perception or via physiological contact. Note that we do not restrict this to *passive exposures*, since in principle, *active exposures* could be caused by perceptual or physiological causes which are themselves controlled by activities (such as burning coal). These exposure subclasses are likewise not mutually exclusive:

Axiom 5. *Kinds of exposures*

$$\textit{Perceptual} \sqsubseteq \textit{Exposure}$$

$$\textit{Physiological} \sqsubseteq \textit{Exposure}$$

An overview of the entire class hierarchy focused around the base concepts can be seen in Fig. 5.

4.2. Modelling data generation

The previous section introduced an ontology that can be used to reason over the different concepts that are needed to understand how exposure is modelled in an article. An important aspect of this question concerns how concepts are represented in terms of data.

In general, concepts may either stay implicit in the actual analysis or else may explicitly be represented by data. Certain factors involved in the exposure process are often part of the background assumptions without any explicit modelling. For example, many studies neither model the persons involved in exposure explicitly nor the actual exposure event, while others leave the environments implicit. Yet, still, these concepts are important to understand the author's intentions and methodological approach. To investigate the extent of explicit/implicit modelling within an article, we indicate whether a concept has a data representation or not and if yes, from which data sources they might have been derived if this is known.

For this purpose, we use the *Data Catalog Vocabulary (DCAT)*²⁹ - Version 2, which is used to describe *datasets* and their *distributions* (via different URLs). To keep things simple, we label something as both an instance of a concept and of a data set (*dcat:Dataset*), meaning that the respective data set is instantiating the concept. For example, there might be a data set of temperature measurements which is at the same time an environment. Using the property *dcat:distribution*, we link the dataset to a particular distribution source (e.g., some URI from a public data catalogue).

Second, since data sources are often not used directly but need to be *transformed* to capture information about the intended concepts, we model such transformations by linking dataset nodes using the *provenance ontology (PROV)*³⁰, using the property *prov:wasDerivedFrom* (in case the derivation method is unknown), or else by triples of the following pattern (Fig. 6):

²⁹<https://www.w3.org/TR/vocab-dcat-2/>

³⁰<http://www.w3.org/ns/prov#>

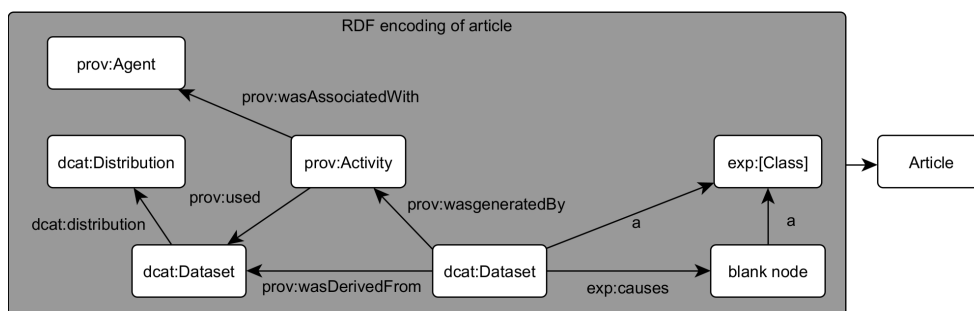


Fig. 6. Class diagram illustrating the encoding of article content. Occurrences of exposure concepts (`exp:[Class]`) can be causally linked to each other and can be either a `dcat:Dataset` (in case they are represented by a dataset), or otherwise just a blank node. Datasets may have been derived from other datasets (encoded using PROV relations). Datasets can have distributions and derivations can have tools (encoded as `prov:Agent`).

```
_:dataOutput prov:wasGeneratedBy _:a1 .
_:a1 prov:wasAssociatedWith <https://cran.r-project.org/web/packages/kdensity/>.
_:a1 prov:used _:dataInput.
```

, where `_:a1` denotes the application of some R tool to derive `_:dataOutput` from `_:dataInput`.

4.3. Encoding of example articles

To test the ontology, the content of all six articles was encoded in RDF. For this purpose, we first identified all article content/text snippets that denote instances of some class in our ontology. In this study, we did this thoroughly by reading the articles and manually identifying the text phrases that corresponded to classes in *exp* or the dataset/provenance ontologies. We then generated a blank node for each detected text phrase that stands for an instance of a class (e.g., *exp:Environment*) and saved the corresponding phrases into RDFS comments describing this blank node (Fig. 6):

```
_:proportionofcyclingpathlengths a exp:Environmental, dcat:Dataset;
rdfs:comment "proportion of cycling path lengths".
_:proportionofcyclingpathlengths prov:wasDerivedFrom _:cyclingstreets.
```

Text occurrences and *data artefacts* should in principle be distinguished from the *concepts* they represent (e.g. environments). Note that in our encoding, these can coincide. The reason is that for our purpose, it was not required to compare different datasets or text snippets representing the same concept. For this reason, we used a simplified encoding that does not force us to separate these items. If needed, this distinction can be drawn using existing ontology design patterns³¹, which distinguish information artefacts from what they represent.

In the future, this work may be automatized using a larger annotated corpus of articles and state-of-the-art deep learning-based NLP methods, similar to [65]. A particular challenge is that the concepts that play a role in the exposure assessment are sometimes left implicit by the authors. Furthermore, we added causal and other links between extracted instances whenever the authors either gave support for such a link (e.g., if they found a correlation) or when they mentioned or assumed such links in their overall approach. Both practices require implicit knowledge and therefore currently still pose a challenge for state-of-the-art NLP methods [65]. Since our article focuses on the modelling aspect, we did not use state-of-the-art text annotation techniques for finding text snippets [66].

5. Evaluation: comparing conceptualizations and methods for measuring exposure

To evaluate the pattern, we tested to what extent the competency questions can be answered automatically in a way that corresponds to our understanding of each article's method.

³¹<http://ontologydesignpatterns.org/wiki/Submissions:InformationObjectsAndRepresentationLanguages>

5.1. Translating competency questions into SPARQL queries

SPARQL³², the query language for RDF, is used here to automatically retrieve answers for competency questions. In the following, we go through each question and discuss its translation to SPARQL:

Query 1. ‘What kind of exposures are modelled in this paper?’

```
SELECT DISTINCT ?c ?y
WHERE {
  ?x a exp:Exposure.
  ?x rdfs:comment ?c
  OPTIONAL{?x a ?y.
  FILTER(?y not in (exp:Exposure, dcat:Dataset)).
  FILTER(!isBlank(?y))
  }
}
```

Here we query for exposures (?x) and retrieve the other classes (?y) they are instances of (other than *exp:Exposure* and not *dcat:Dataset*, constrained by *FILTER*), in case they exist (*OPTIONAL* statement).

Query 2. ‘Which activities are involved in the exposure and who is exposed?’

```
SELECT DISTINCT ?yc ?zc
WHERE {
  ?x a exp:Exposure.
  ?x exp:causedBy ?y. ?y a exp:Activity.
  ?y rdfs:comment ?yc.
  OPTIONAL{?x exp:of ?z. ?z a exp:Person.
  ?z rdfs:comment ?zc.}
}
```

In this query, we search for *activities* that cause some *exposure*, and optionally for exposed *persons*. Since the ontology does not involve any *activity/person* types, we just retrieve the text descriptions (*rdfs:comment*) about these *activities* or *persons*.

Query 3. ‘What are subjects exposed to?’

```
SELECT DISTINCT ?yc
WHERE
{
  ?x a exp:Exposure. ?x exp:causedBy ?y. ?y rdfs:comment ?yc.
  FILTER NOT EXISTS{?x a exp:ActiveExposure. ?y a exp:Environment. }
  FILTER NOT EXISTS{?x a exp:PassiveExposure. ?y a exp:Activity. }
}
```

In this query, we search for all phenomena that cause *exposure*. Yet, the focus on what we are exposed to changes with the type of *exposure*. In the case of *passive exposure*, we focus on *environments*. This is because if someone is *passively exposed* to air pollution (e.g., we are not interested in his or her *activity* performed when being *exposed*). Conversely, for *active exposure*, we are mainly interested in the *activity* that is performed, such as running. This focus is encoded in *FILTER NOT EXISTS* statements, and of course, it could be removed if needed.

Query 4. ‘What is their health risk of exposure?’

```
SELECT DISTINCT ?yc
WHERE {
  ?x a exp:Exposure.
  ?x rdfs:comment ?c.
  ?x exp:causes+ ?y. ?y a exp:Risk. ?y rdfs:comment ?yc.
}
```

³²<https://www.w3.org/TR/sparql11-query/>

In this query, we retrieve *health risks* caused by *exposures*, potentially via some causal chain (+). This is because the *exposure* may cause *health risks* directly or indirectly via *doses* first. We want to keep this possibility open.

Query 5. ‘Which environments influence the exposure and from which datasets were they derived?’

```
SELECT DISTINCT ?yc ?zc ?d
WHERE {
  ?x a exp:Exposure.
  ?x rdfs:comment ?xc.
  ?x exp:causedBy+ ?y. ?y a exp:Environment. ?y rdfs:comment ?yc.
  ?y prov:wasDerivedFrom* ?z. ?z a dcat:Dataset; rdfs:comment ?zc.
  FILTER NOT EXISTS {?z prov:wasDerivedFrom ?u}
  OPTIONAL{?z dcat:distribution ?d}
}
```

In this query, we search for *environments* that (directly or indirectly) cause *exposure*. The causal chain (+) is needed since, in the case of active *exposures*, the *environment* is a direct cause of the *activity*, but only an indirect cause of the *exposure*, via the *activity*. Furthermore, we are also interested in the data sources of these *environments*, which could have been generated by zero or more (*) steps of derivation via the provenance ontology *prov:wasDerivedFrom*. We want to focus on the sources of data, not intermediary datasets (FILTER NOT EXISTS), and possibly (OPTIONAL) retrieve a web link to where the data is available (*dcat:distribution*).

Query 6. ‘What are the environmental stressors?’

```
SELECT DISTINCT ?xc
WHERE {
  ?x a exp:Environment; rdfs:comment ?xc.
  ?y a exp:RiskPromotingExposure; exp:causedBy ?x .
}
```

In this query, we are looking for *environments* that cause some risk-promoting *exposure* (see Definition 1), i.e., an *exposure* that causes a *health risk* level to increase with the amount of *exposure*. This is what we call an *environmental stressor*.

5.2. Running inferences and queries

We loaded RDF files for each paper together with our ontology into separate RDF graphs in RDFLib³³. We then used a brute force implementation³⁴ of the *OWL 2 RL*³⁵ and RDFS³⁶ inference schemes to expand each graph with all possible triples that logically follow from our ontology and the linked data encoding of a paper’s content. After this inference step, we applied locally closed world inferences to all unique *causedBy.Active* triples (as explained in Inference rule 1) using our script. Since the latter adds new OWL facts which serve as a start for further inferences, we needed to run the former inference steps again. Since the standard inference is conservative regarding *causedBy* triples, no further inference is possible. Afterwards, we fired all SPARQL queries over all graphs and summarized the answers.

5.3. Results

In this section, we discuss the potential of our model for filtering and classifying exposure-related concepts, data, and methods across studies. For this reason, we compare results across the six studies for each query individually. Retrieved answers to queries are shown in Tables 2 and 3.

³³<https://github.com/RDFLib>

³⁴<https://github.com/RDFLib/OWL-RL>

³⁵https://www.w3.org/TR/owl2-profiles/#Reasoning_in_OWL_2_RL_and_RDF_Graphs_using_Rules

³⁶<https://www.w3.org/TR/rdf11-mt/>

Query 1 As you can see in Table 2, the amount of answers in each study for this query already tells us something about the focus of a study. For example [41], [37], and [42] only study a single *exposure*, whereas [40], [39], and [38] study multiple *exposures*. For example, [38] focus on types of air quality *exposures* and [40] on different variants of crime *exposures*. All *exposures* are *health-relevant*. Furthermore, we can see differences in how these *exposures* are automatically classified using inference. According to our model, [39], [41], [37], and [42] all study some form of *active exposure*. According to Def. 4, this means that *exposures* have exclusively *active* causes (so either are activities or are caused by *activities*). [41] and [42] focus on *exposure to physical activity* (walking or biking or motorized transport), while [37] focuses on an individual's *exposure to poor diet and fast food*. Note that while these studies also take *exposure to environments* into focus, the latter are not direct causes of *exposure*. Furthermore, the poor diet *exposure* in the study of [37] is correctly classified as a risk-promoting *exposure*, whereas the other two kinds of *exposures* are correctly recognized as *risk preventing* instead. [39] is an interesting case of *active risk-promoting exposure*. Though air quality plays an important role in this process, the *exposure* is still classified as *active*, simply because burning coal is an *activity* causing air quality, and so the causal chain of *exposure* is entirely rooted in the underlying household decisions of the women. [40] is another interesting border case, because *exposure to crime* may be seen as an *active exposure* due to crime being an activity, yet it is classified as *passive* by our model. The reason is that [40] does not take into account crime as an *activity*, including the people committing the crime, but rather models crime as a (static) aspect of the *environment*. This way of modelling crime resembles the way any other *environment* is modelled.

Query 2 This query asks about the specific *activity* that causes health-relevant *exposure* and who is involved in that *activity* (see Table 2). In the case of [40], this *activity* is not committing a crime, but *living in a neighbourhood* with crime, as experienced by children. Living is also the prime *activity* considered in the study of [38] about air pollution, yet in this case, focusing on female teachers. Children's transport to school is the focus for [42], whereas [41] focuses on the physical activity of adults in Norwich. Interestingly, the *activity* causing the *exposure* in [37] is not the food buying behaviour (though this could be done when studying exposure to poor diet), but it is instead eating at fast food outlets. Note that this distinction is crucial to understanding whether studies about food are comparable to not. In [39], our model makes clear that the cause of the smokey coal exposure is *indoor fuel use* by *never smoking women*. This shows the study intends to measure a health effect that can be exclusively attributed to the household environment, instead of smoking behaviour.

As shown in Table 2, most studies only take a single kind of *activity* into account, except for [42], where transport to school is distinguished into 3 different modes: walking, biking, and motorized transport. Note that all studies define a certain study group, though some have tighter restrictions on their subjects. [39], [38], [41], and [37] examine adults, whereas [39] and [38] place additional requirements on these adults. The remaining two studies, [42] and [40], both study children of different age groups: 6 – 11 years and 11 – 18 years, respectively.

Query 3 The third query (Table 2) focuses on what a *person* is exposed to, dependent on whether the *exposure* is *active* (activity) or *passive* (environment). In all *active exposure* cases, a *person* is exposed to exactly the *activities* that are causing their *exposure*. For example, in [42], school children are exposed to walking, biking, or motorized transport. In [39] people are exposed to indoor fuel use (though indirectly via indoor air pollution), [37] subjects are exposed to eating at fast food outlets, and [41]'s subjects are exposed to physical activity. In the passive exposure studies, subjects are exposed to air pollution concentrations particulate matter 10, particulate matter 2.5, ozone, nitrogen dioxide, nitrogen oxides, carbon, and sulfur dioxide (PM₁₀, PM_{2.5}, O₃, NO₂, NO_x, CO, and SO₂) [38], and violent and non-violent crime [40].

Query 4 This query asks for the *healthrisk* of *exposure* (Table 3). All studies identified some health-related risks as a consequence of the exposure. [40] focuses on mental health rather than physical health (risk of adverse behaviour). [42], [37], and [41] focus on obesity (though using different methods and considering different groups of people). [39] and [38]'s look at different risks of air pollution in their study, namely lung cancer, myocardial infarction, and stroke.

Query 5 The first part of query 5 filters for *environments* that influence *exposure* (Table 3). In the study for [38], concentrations for PM₁₀, PM_{2.5}, NO₂, NO_x, CO, and SO₂ are identified as *environments*. While many different types of chemicals are considered, the study lacks other *environments* such as distance to highways, that could also

influence one's exposure to air pollution. In [42], a wide range of *environments* influence *exposure*: homes, schools, availability of major roads, distance to major roads, accident density, the proportion of cul-de-sacs, wind speed, temperature, global radiation, hourly precipitation, and proportion of green land use. Note that since [42] studies a form of *active exposure*, these factors directly influence physical activities, and only indirectly the *exposure* to those *activities*. [37] only considers two *environments*, fast food outlets and neighbourhoods, while [41] focuses on green space and the built environment: large urban green space, quality of the urban green space, distance to green space, and distance to the city boundary. [40] takes into account only the social environment, including violent crime, non-violent crime, crime rates, and neighbourhood. [39]'s study focuses on coal deposits or mines and homes located in Chinese counties Xuanwei or Fuyuan are from the built environment, and socio-economic status is from the social environment. Note that in all *passive exposure* studies [38, 40], the involved *persons* are directly *exposed* to these *environments*.

The second part of query 5 asks about the data sets from which *environments* were derived. In this query, there are many missing (labelled as "None" in the table) answers because most studies provided only incomplete information on where data sets were obtained, and this was not enough to provide a reliable answer to this query. As shown in Table 3, [42] provided links to data sources for wind speed, temperature, global radiation, hourly precipitation, and proportion of green land use. Our query also reveals that data on the availability of major roads, distance to major roads, and proportion of cul-de-sacs was *derived* from the same road data set. Similarly, accident density was derived from an accident data set. However, the data links to the road and accident data set are not available. Location data and other qualitative data on homes and schools were also not available (may be due to privacy reasons). [39] and [37] provided access to data about coal deposits or mines and fast food outlets, respectively. [40] and [41] did not provide any data sets for any of their *environments*. Only [38] provided data links for all their *environments* (monitoring stations).

Query 6 This query is about *environmental stressors* (Table 2). Answers for this query are lacking (labelled as "None" in the table) for all *active exposures* because they can never be caused by *environmental stressors* by definition (cf. Def 2 and 4). For example, large urban green space [41] is an *environment* but is not a *environmental stressor*. The only *environmental stressors*, therefore, are air quality [38] and crime [40].

6. Discussion and future work

Ontologies are a way to organize knowledge in a field according to well-defined concepts. In combination with automatic annotation and information extraction methods, it can be used to handle large amounts of evidence on the influence of exposure in the health and behavioural sciences. In this study, we have focused on the design of an ontology that captures and makes comparable the conceptualizations of exposure and the underlying methods and data across different studies. The ontology categorizes parts of an epidemiological study in terms of the following related classes: *person*, *activity*, *environment*, *exposure*, *dose*, and *health risk*. Using these classes as well as a universal causal relation, we defined different exposure concepts using OWL definitions. Based on OWL-RL/RDFS reasoning, we were able to categorize whether a given study in question focused on *active* and or *passive exposure*, which *environmental stressors* are involved, who is *exposed* etc.

Our model illustrates the potential for an ontology to organize and extract information from exposure-related studies and classify them. It shows the variability of *exposure* conceptualizations including *environmental* causes and *activities*, but also basic commonalities, which allows us to compare articles against each other regarding their content. An article's focus can be revealed by result frequencies (e.g., many *environments* causing one *activity*, vs different *activities* in the same *environment*). Also, *passive exposures* tend to neglect *activities* and *persons*, whereas *active exposures* tend to model both. Many articles tend to lack information on data sets that were used to measure exposure. The diversity of article topics and exposure cases encoded in our ontology shows that the ontology is general enough to cover various approaches to measuring exposure. This can be easily missed by keyword-based comparisons. For example, it is very easy to confuse the study by [39] about lung cancer and the study by [38] about air pollution if we remain unaware that the former focuses on indoor fuel use (an *activity*), not on the quality of air. Yet both studies involve the keyword "air pollution". Thus our model is effective in adding semantic depth to

meta-studies, which can now differentiate the underlying exposure model. Epidemiological researchers could use this to systematically compare approaches.

However, our work remains preliminary in the following respects: One limiting factor of this study is that its empirical basis for testing is rather narrow, as only six articles were used to test the ontology. Such a small number was needed because each article needed to be read thoroughly and then encoded into the ontology and RDF manually, which is a very time-consuming, iterative process. How could our study be scaled up to analyse larger amounts of articles? It is possible to integrate natural language processing (NLP) and supervised classification into the framework to scale up the analysis of articles with our ontology. Such an approach has been proposed by [4, 67]. However, while it can be assumed that e.g., BERT based pre-trained deep learning models [14] can classify text snippets as *persons*, *activities*, *environments*, etc., with high-quality using named entity recognition, to date, it remains unclear to what extent such methods are also able to extract the rather implicit relations between the categories investigated here, which therefore remains a challenge [15, 65]. The latter would be needed to populate our ontology and to automatically infer different exposure categories. Furthermore, in the future, we should investigate to what extent the interpretation of ontology classes and relations are reproducible across different annotators based on measuring *inter-annotator agreement* [66]. In this way, we could find out to what extent our ontology classes allow for incompatible interpretations.

In addition, the ontology pattern that we proposed here can be improved in several ways. For one, the difference between passive and active exposure was modelled as a binary decision. However, it might be more adequate to allow for passiveness in degrees. For example, one could define an exposure as *semi-active* if all its causes are caused by some activity, depending on the length of a chain of such causes that are routed in activities. This would allow us to recognize [39]’s air quality study as an active case, even in case we conceptualized the indoor environment as an independent cause. The causal chain would still reveal that such causes are all routed in the activity of burning coal, which can be controlled by the involved person. More generally, future work should investigate to what extent the used ontology of quantities [48] could be extended to capture the various ways how exposure measures are generated computationally. This would allow us to reason about the validity of method applications for certain measurement goals and, at the same time, to investigate the influence of systematic method variations on the quality of exposure-based models in the health sciences.

Appendix (selection of articles)

Grinshteyn et al The paper by [40] studies how children’s mental health is impacted by witnessing, being a victim, or knowing a victim of violent or non-violent crime, specifically when the children show delinquent or aggressive behaviour. Data was collected from seven cohorts in which children had been asked about their crime exposure. This data was then linked to uniform crime reporting data of the Federal Bureau of Investigations (FBI). Based on this data, three sensitivity analyses were performed, with results showing that self-reported crime exposure was associated with increased scores [40].

Vermeulen et al The paper by [39] investigates the relationship between lifelong exposure to the constituents of smoky coal and other fuel types, and lung cancer in females who do not smoke in two provinces in China. The researchers collected lung cancer cases among non-smoking women from six hospitals and also used a control population. Both cases and controls were interviewed using a questionnaire that collected information on residential history, fuel use, and established or suspected risk factors for lung cancer [39]. Statistical analysis revealed that the strongest association with lung cancer was for a cluster of 25 polycyclic aromatic hydrocarbons (PAHS) and for NO₂ [39]. This finding is in line with other studies but this was the first study known to examine the role of specific household air pollution constituents exposure of the entire life and lung cancer risk [39].

Lipsett et al Lipsett et al [38] also look at air pollution, however from outdoors. The researchers’ goal was to examine associations of individualized long-term exposures to particulate and gas usage air pollution with myocardial infarction and stroke in female teachers in California. This was done by linking geocoded addresses with inverse distance-weighting monthly pollutant surfaces for two measures of particulate matter and for several gaseous pollutants [38]. They examined associations between these pollutants and risks of myocardial infarction and stroke using

Cox proportional hazard models [38]. Results showed long-term exposure to PM_{2.5}, PM₁₀ and NO_x were associated with elevated risks for ischemic heart disease mortality [38].

Rongen et al The study by [37] investigates Dutch neighbourhood social norms with respect to fast food consumption as a potential mediating pathway between fast food outlet exposure and residents' fast food consumption [37]. A sample of respondents living across the Netherlands completed a survey, where they reported on their fast food consumption and related perceived norms in their neighbourhood [37]. The exposure to fast food was measured by the average count of fast food outlets within a 400m walking distance buffer around the zip codes of respondents. Regression models were used to assess the association between residential fast food outlet exposure, fast food consumption, and social norm perceptions [37]. Results found that there was no overall direct association between residential fast food outlet exposure and residents' fast food consumption [37]. However, the researchers found that fast food outlet exposure was positively associated with neighbourhood social norms regarding fast food consumption, which was positively associated with the odds of consuming fast food [37].

Hillsdon et al The study by [41] examined the association between access to quality urban green space and levels of physical activity among adults living in Norwich, United Kingdom. This was done by performing three measures of access to open green space based on distance only, distance and size of green space, and distance, size, and quality of green space [41]. These measurements were done using GIS, and multiple regression models were used to determine relationships between the three factors and the level of recreational physical activity. Results showed that there were no clear relationships. The authors concluded that access to urban green spaces does not appear to be associated with recreational physical activity for their sample group [41]. This article is interesting because it is the only article included in this study where no relationship was found between environments with an action, exposure, or health risk.

Helbich et al Lastly, the study by [42] is about measuring how the natural and built environment impacts Dutch children's mode of transport to school, which may influence their exposure to physical activity, which in turn prevents obesity. This was done by giving children GPSs for several days, and by analysing the association between the environment on the school path and children's active/passive transportation behaviour using mixed models. Results showed that distance to school, green space, and weather are not significant, but well-connected streets and cycling lanes are [42].

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Paper	Which exposures are modelled in this paper? (Query 1)	What types of exposures are these? (Query 1)	Which activities are involved in the exposure? (Query 2)	Who is exposed? (Query 2)	What are subjects exposed to? (Query 3)
Helbich_2016 [42]	exposure to physical activity	exp:RiskPreventingExposure, exp:ActiveExposure	walking or biking or motorized transport	school children (GPS tracks)	walking or biking or motorized transport
Lipsett_2011 [38]	PM 10 exposure	exp:PassiveExposure, exp:RiskPromotingExposure	Living in California	female teacher	PM 10 concentration raster
	PM 2.5 exposure	exp:PassiveExposure, exp:RiskPromotingExposure	Living in California	female teacher	PM 25 concentration raster
	O ₃ exposure	exp:PassiveExposure, exp:RiskPromotingExposure	Living in California	female teacher	O ₃ concentration raster
	NO ₂ exposure	exp:PassiveExposure, exp:RiskPromotingExposure	Living in California	female teacher	NO ₂ concentration raster
	NO _x exposure	exp:PassiveExposure, exp:RiskPromotingExposure	Living in California	female teacher	NO _x concentration raster
	CO exposure	exp:PassiveExposure, exp:RiskPromotingExposure	Living in California	female teacher	CO concentration raster
Vermeulen_2019 [39]	exposure to smokey coal	exp:RiskPromotingExposure, exp:ActiveExposure	indoor fuel use, data collected on a questionnaire	never smoking women in Xuanwei and Fuyuan	indoor fuel use, data collected on a questionnaire
	exposure to smokeless coal	exp:RiskPromotingExposure, exp:ActiveExposure	indoor fuel use, data collected on a questionnaire	never smoking women in Xuanwei and Fuyuan	indoor fuel use, data collected on a questionnaire
Rongen_2020 [37]	poor diet	exp:RiskPromotingExposure, exp:ActiveExposure	eating at fast food outlets	adults in the Netherlands	eating at fast food outlets
Grinshteyn_2018 [40]	witnessed violent crime exposure	exp:PassiveExposure, exp:RiskPromotingExposure	living in crime neighborhoods	children aged 11 to 18 years old	violent crime
	hearsay violent crime exposure	exp:PassiveExposure, exp:RiskPromotingExposure	living in crime neighborhoods	children aged 11 to 18 years old	violent crime
	victim of violent crime exposure	exp:PassiveExposure, exp:RiskPromotingExposure	living in crime neighborhoods	children aged 11 to 18 years old	violent crime
	witnessed non-violent crime exposure	exp:PassiveExposure, exp:RiskPromotingExposure	living in crime neighborhoods	children aged 11 to 18 years old	non-violent crime
	hearsay non-violent crime exposure	exp:PassiveExposure, exp:RiskPromotingExposure	living in crime neighborhoods	children aged 11 to 18 years old	non-violent crime
Hillsdon_2006 [41]	exposure to physical activity	exp:RiskPreventingExposure, exp:ActiveExposure	physical activity	adults in Norwich, England	physical activity

Table 2

Answers to queries 1-3 retrieved from the knowledge base via inference

Paper	What is the risk of exposure? (Query 4)	Which environments influence the exposure? (Query 5)	From which datasets were they derived? (Query 5)	What are the environmental stressors? (Query 6)
Helbich_2016 [42]	obesity	homes	None	None
	obesity	schools	None	None
	obesity	availability of major roads	roads, None	None
	obesity	distance 2 major roads	roads, None	None
	obesity	accident density	accidents, None	None
	obesity	proportion of cul-de-sac	roads, None	None
	obesity	wind speed	https://www.knmi.nl/nederland-nu/klimatologie/uurgegevens	None
	obesity	temperature	https://www.knmi.nl/nederland-nu/klimatologie/uurgegevens	None
	obesity	global radiation	https://www.knmi.nl/nederland-nu/klimatologie/uurgegevens	None
Lipsett_2011 [38]	Myocardial stroke	Infarction, PM 10 concentration raster	PM 10 monitoring stations, https://www.arb.ca.gov/adam	PM 10 concentration raster
	Myocardial stroke	Infarction, PM 25 concentration raster	PM 2.5 monitoring stations, https://www.arb.ca.gov/adam	PM 25 concentration raster
	Myocardial stroke	Infarction, O ₃ concentration raster	O ₃ monitoring stations, https://www.arb.ca.gov/adam	O ₃ concentration raster
	Myocardial stroke	Infarction, NO ₂ concentration raster	NO ₂ monitoring stations, https://www.arb.ca.gov/adam	NO ₂ concentration raster
	Myocardial stroke	Infarction, NO _x concentration raster	NO _x monitoring stations, https://www.arb.ca.gov/adam	NO _x concentration raster
	Myocardial stroke	Infarction, CO concentration raster	CO monitoring stations, https://www.arb.ca.gov/adam	CO concentration raster
	Myocardial stroke	Infarction, SO ₂ concentration raster	SO monitoring stations, https://www.arb.ca.gov/adam	SO ₂ concentration raster
Vermeulen_2019 [39]	lung cancer	coal deposits or mines	https://doi.org/10.1002/ijc.32034	None
	lung cancer	homes located in Chinese Counties Xuanwei or Fuyuan	None	None
	lung cancer	socio-economic status	None	None
Rongen_2020 [37]	obesity	fast food outlets	https://locatus.com/applicatie/retail-facts/	None
	obesity	neighbourhood	None	None
Grinshteyn_2018 [40]	adverse health characteristics	behavioural violent crime	None	violent crime
	adverse health characteristics	behavioural non-violent crime	None	non-violent crime
	adverse health characteristics	behavioural neighbourhood crime rates	None	None
Hillsdon_2006 [41]	obesity, health issues	large urban green space	None	None
	obesity, health issues	quality urban green space	None	None
	obesity, health issues	distance to green space	None	None
	obesity, health issues	distance to city boundary	None	None

Table 3

Answers to queries 4-6 retrieved from the knowledge base via inference