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Doctoral Dissertation

**Causation in ontology engineering:
A functional perspective**

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Abstract

Causation is shrouded in mystery. Notwithstanding much work on causation in a number of different domains, it remains a challenge to build an adequate theory of causation for ontology engineering. This dissertation aims to offer a functional perspective on causation, thereby enabling domain experts to have a fairly expressive representation of multifarious causal phenomena. As for its theoretical basis, causation is modeled upon the device ontology view of reality, assuming an intimate connection between causation and context. Combined in order with the device-ontological understanding of change, the systemic-functional notion of lawhood, and the systemic conception of function as goal achievement, this fundamental idea finally leads to the conceptual mapping of causation onto function and the **achieves** causal relation (as well as the **prevents** causal relation). Underpinned by the idea of ‘causally efficacious occurrents’, the **achieves** causal relation can hold between a specific pair of three subtypes of occurrents: events, processes, and states.

In addition, an expressive causal representation is practically developed based on the observation that an ontological modeling of causation would be particularly useful for expressing causal chains of various phenomena. This contributes to the precondition-for relations and the development of indirect causal relation (**allows** and **disallows**) that are based on direct causal relations and that are diagrammatically conceptualized as the Configuration of State-mediated Causation (CSC). These four kinds of causal relations are conceptually organized in the form of the functional square of causal relations. Those accomplishments are supported by the idea of a state-centered approach to causation. The explanatory force of the proposal is shown by its ability to accommodate a wide variety of examples extracted from the relevant literature. The dissertation also provides a preliminary formalization of these four causal relations and takes the first step towards a full-fledged functional theory of causation in ontology engineering.

To illustrate the application of the functional view of causation, the causal evolution of the River Flow Model (RFM) of diseases is attempted so that careful consideration of the relationship between disease and causation can be given. In biomedical ontology research, a disease ontology is built to meet a high demand for a common semantic framework in which an increasing amount of medical information and data are shareable among different information systems. An accurate conceptualization of disease is thus helpful for the robust construction of disease ontologies; but disease nevertheless remains an elusive notion from an ontological viewpoint. Against this background, the RFM was proposed around 2010 to explain the disease notion that has a close affinity with medical practitioners’ typical understanding of disease. The core idea of the RFM is that a disease is a dependent continuant constituted of causal chains of abnormal states. For instance, diabetes is a dependent continuant whose causal chains have as part the causal relation between the state of the deficiency of insulin and the state of the elevated level of glucose in the blood. The practical utility of the RFM is indicated by its active domain-level implementation for the last decade. One remaining theoretical problem with the RFM is its explicit reference to the notoriously difficult concept of causation. The application of the functional perspective on causation to the RFM leads to the improved RFM conception of disease: a disease is constituted of abnormal states to which events, processes, and states bear either the **achieves**, **prevents**, **allows**, or **disallows** causal relation.

With knowledge as its key concept, knowledge science aims to offer a systematic understanding and facilitation of the creation, exploitation, and the accumulation of knowledge involved in individuals as well as in society. The dissertation contributes to knowledge science by building a common ground for representing different causal phenomena in different domains and facilitating an integration of and an interdisciplinary collaboration across research fields dealing with the concept of causation. It also furthers the causal underpinning of the RFM, thereby not only enhancing the interoperability and flexibility of an increasing amount of disease-related data and information but also showing the practical potential for creating of medical knowledge in the long run.

Keywords: causation, function, the River Flow Model (RFM) of diseases, biomedical ontology, ontology engineering

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List of abbreviations

Abbreviation	Explanation
APA	American Psychiatric Association
BFO	Basic Formal Ontology
COM	the Computational Ontology of Mind
CSC	the Configuration of State-mediated Causation
DOLCE	the Descriptive Ontology for Linguistic and Cognitive Engineering
GFO	General Formal Ontology
ICD	the International Classification of Diseases
MF	the Mental Functioning Ontology
OBO	Open Biomedical Ontologies
OGMS	the Ontology for General Medical Science
RFM	the River Flow Model of diseases
RPOA	the Restricted Potential Outcomes Approach
SNOMED-CT	the Systematized Nomenclature of Medicine - Clinical Terms
YAMATO	Yet Another More Advanced Top-level Ontology

1. Introduction

1.1. Causation in everywhere

The notion of causation is part and parcel of our thinking and talk of causation is ubiquitous. An ontological analysis of causation would be therefore valuable for an adequate representation of various causal phenomena in information systems. Accordingly, there is a vast amount of literature on causation in a number of different domains, ranging from philosophy and jurisprudence to linguistics, cognitive science, and computer science. Consider for instance a scientific explanation: sky is blue because the molecules in the atmosphere of the earth will scatter more blue light towards the ground than other colors. This explanation is not merely linguistic or cognitive: it does not explicate (our understanding of) the meanings of the terms ‘sky’ and ‘blue’. It is not logical either: the blueness of sky is not a logical consequence of the working of the molecules in the atmosphere of the earth. Rather, the explanation at issue is the cause of the blueness of sky that is brought about by some ‘natural feature’ of the world. In this way, causation is indispensable for virtually all kinds of intellectual practice.

1.2. Causation in ontology engineering

The term ‘ontology’ is presently used mainly in two academic disciplines: philosophy and information/knowledge science. It originated as a philosophical word that is nowadays employed in two ways. First, it means the list of entities the existence of which a certain theory is committed to. For example, to say that God is part of deists’ and theists’ ontologies entails the existence of God with respect to their worldview. Second, ontology refers to the subfield of metaphysics (which is orthodoxly taken to be the study of the fundamental structure of reality) that investigates what exists or more generally what kinds of entities exist. Whether numbers exist is an ontological question, for instance.

The dissertation focuses on the other usage of the term ‘ontology’, namely the one that is prevalent in information and knowledge sciences, and ontologies in this sense of the word will be employed throughout this work unless otherwise specified (e.g., ‘philosophical ontology’). One of the most widespread definitions of ontology is Gruber’s (1993), which was further elucidated by Borst (1997) and later restated comprehensively by Studer, Benjamins and Fensel (1998) as follows: “An ontology is a formal, explicit specification of a shared conceptualization.” This definition of ontology was also logically analyzed so rigorously by Guarino, Oberle and Staab (2009) according to whom an ontology is “a logical theory designed to account for the intended meaning of the vocabulary used by a logical language” that it has been a popular understanding of ontology in the ontology community (but see Neuhaus, 2017 for criticism).

Another alternative conception of ontology is Smith, Kusnierczyk, Schober and Ceusters's (2006) definition, which was sophisticated by Arp, Smith and Spear (2015: Chapter 1) as follows: an ontology is "a representational artifact, comprising a taxonomy as proper part, whose representations are intended to designate some combination of universals, defined classes, and certain relations between them." The details of the technical terms therein (e.g., 'defined class') are omitted to circumvent unnecessary complications, but quite importantly, this interpretation is motivated by Smith's (2006) critique of the 'concept-oriented' view of ontologies as representations of agents', or precisely ontology users' conceptual schemata and his coupled endorsement of ontologies as representations of what exists in reality. It would require ideally all the ontology developers to embrace the realist philosophical view in order to ensure interoperability (Borgo and Hitzler, 2018).

Those two definitions of ontology, despite some non-trivial differences between them, share the conviction that an ontology is an explicit representation of the categories and the relations between them in a certain domain such that ontologies owe their explicitness largely to the usage of some logical representation language. This core idea of ontology is only needed for the purpose of this dissertation, which thus remains neutral on the conceptualist/realist confrontation sketched above. Examples of formal logics commonly used include first-order (modal) logic (Fitting and Mendelson, 1998) and Web Ontology Language (OWL) (Horrocks, Patel-Schneider, McGuinness and Welty, 2007), in spite of their problematicity (Borgo, Porello and Troquard, 2014; see also Guarino, 2009 for the relationship between representation languages and their ontological assumptions).

There are currently many kinds of ontologies, two of which will be focused on below: domain(-specific) ontologies and upper ontologies. Domain ontologies are ontologies that are intended to represent the categories and relations in a particular area of interest. The term 'ontology' will be hereafter used to mean domain ontology as convention, and biomedical ontologies are domain ontologies around which this dissertation pivots. Upper ontologies (aka foundational ontologies) are, by contrast, ontologies that strive to provide the most general categories (e.g., space and time) and relations (e.g., identity and parthood) to serve as a useful guideline for building domain ontologies of high interoperability.¹ They are in this respect comparable to philosophical ontologies (in their first sense). Upper ontologies are characterized by their ontological choices (Borgo and Masolo, 2010), i.e., choices as to whether a certain ontological category or relation is adopted or not; and their meta-ontological choices (de Cesare et al., 2016), i.e., choices (e.g., as to whether the realist or conceptualist approach to ontology is taken) that are fundamental enough to determine ontological choices.

There are some basic categories and relations that are relatively widespread in upper

¹ Top-level ontologies are sometimes used synonymously with upper ontologies, but Borgo and Hitzler (2018: 3) spell out the difference between them as follows: "(...) while a top-level ontology is a classification system that deals with general domain-independent categories only, a foundational ontology is a top-level (formal) ontology that has been built and motivated by the upfront and explicit choice of its core principles."

ontologies. Entities fall into two kinds: universals (aka types, classes) and particulars (aka tokens, instances). Particulars (e.g., Mary) bear the instance-of relation to universals (e.g., Human). Particulars fall into two categories: continuants (aka endurants) and occurrents (aka perdurants). Characteristically, continuants can persist, that is to say, they can exist at one time and also exist at another different time; whereas occurrents extend through time. Continuants (e.g., a stone) can participate in occurrents (e.g., a fall of the stone). Continuants can be further divided into independent continuants (including objects) and dependent continuants (including properties in the broad sense of the term). Independent continuants, or especially objects (e.g., stones) can be bearers of properties (e.g., hardness).

Ontology engineering (aka applied ontology, formal ontology) as understood in the dissertation is the field within information/knowledge science that aims at a deeper understanding of domain-specific knowledge by creating an ontology. It is well worth noting that ontologies can be distinguished by their dependence on philosophically inspired principles, or rather on upper ontologies, from other traditional classifications, viz. catalogues, glossaries, thesauri, and taxonomies; and connectedly, ontology engineering would be theoretically more well-founded than other preceding classificatory approaches.

In ontology engineering, causation remains far more unexplored than other topics that are closely related to it (e.g., agency, dispositions, and functions). This is due partly to its crucial role in an ontological modeling of reality. The notions that are commonly used in ontology engineering (as well as in other disciplines) may well be arranged in order of fundamentality or grounding (Correia and Schnieder, 2012). A notion is more difficult to analyze when it is more fundamental to reality. For instance, existence is generally believed to be so basic that virtually no further elucidation of the concept can be given, even if it is frequently discussed whether a given entity (e.g., numbers) exists or not. Examples also include identity, parthood, and causation.

Causation is particularly difficult to model from an ontological viewpoint. One of the arguably most sound reasons for this is that causation is in nature so intimately connected to grounding that grounding may be sometimes taken to be a type of causation (Wilson, 2017). Generally speaking, to say that a notion X is explicable in terms of another notion Y requires that X be grounded in Y (or in other words, Y grounds X). However, it is not only the case that there are few plausible candidates for a notion in which causation is grounded; but also that the very concept of grounding has a close affinity with causation. Those considerations may lead to primitivism about causation (Tooley, 1987).

There may be nevertheless another approach to a better knowledge of fundamental notions (including causation) which may be called ‘*conceptual mapping*’ and according to which a notion X is better understood through its conceptual mapping onto another notion Y such that (i) Y is more

approachable than X and (ii) there is a *sufficient conceptual overlap* between X and Y . It is not required in this method that X be grounded in Y . On the contrary, the conceptual mapping of X onto Y is effective especially when Y is grounded in X or when X appears to be too basic to be explainable. The notion Y , onto which X is conceptually mapped, is of great use as a close ‘conceptual approximation’ of X , thereby helping to have a clearer understanding of the nature of X .

1.3. Purpose and methodology

The goal of this dissertation is to elaborate upon a functional perspective on causation based on the idea that causation can be better understood through its conceptual mapping onto function. Certainly the notion of function is a highly controversial topic in ontology engineering. There would be nonetheless a broad consensus that function has been investigated carefully enough to be more lucid than causation. Seen in terms of the categories that are commonly accepted in upper ontologies, for instance, function falls into the category of specifically dependent entity (i.e., an entity that depends existentially on a particular, independent entity) and it also has a close relationship with dispositions and roles (see Röhl and Jansen, 2014).

To achieve this goal, a purely theoretical inquiry into causation is conducted by considering carefully the role of notion of *context* with respect to causation. In contrast to a conceptual and/or linguistic view of the relationship between causation and a context (Mackie, 1980: 34-35; Schaffer, 2005), what is assumed in this dissertation is that each causal relation *necessitates* its context *ontologically*. The causal world is in itself disordered owing to closely intertwined causal phenomena with one another. *Once it is articulated ontologically*, however, a causal phenomenon holds with respect to and only to the context that is specific to the causal phenomenon. It is because causation is ontologically linked with (but not epistemically relativized to) some context that we are able to understand causation. The causal phenomenon that is ontologically picked out is to be explored in this dissertation. Another assumption that is herein taken is that there is an intimate relationship between causation and *change*: causation typically involves some relevant change. Change is comprehensively modeled upon a *behavior* within the framework of the *device ontology* view of reality (Mizoguchi and Kitamura, 2009). The device ontology is a specific method for assigning roles to object that is elaborated in compliance with the YAMATO notions of processes, states, and roles.

Those preparatory works enable causation to be conceptually mapped onto *systemic function*: the function whose core idea is *goal achievement* and which is determined by a *systemic context* (Mizoguchi, Kitamura and Borgo, 2012; Mizoguchi, Kitamura and Borgo 2016; Borgo, Mizoguchi and Kitamura, 2016). Assuming a relevant connection between causation and *laws of nature* (roughly, general facts concerning lawhood that do not merely happen to be true), the conceptual mapping of causation onto function is countenanced by the observation that, for a given

causal relation, there is the systemic context that represent the laws of nature that are enough for explaining the causal relation.

All these ontological considerations crystallize into the **achieves** causal relation (as well as its sibling **prevents** causal relation) and its three kinds of relata: events, processes, and states. The **achieves** causal relation is further elucidated through the idea of *causally efficacious occurrents*, according to which events, processes, and states are *causally efficacious* (see e.g., Galton, 2012). One significant consequence of this is that, contrary to popular belief that a state is at most of secondary importance as a causal relatum, the causal relation between an event and a state is arguably the most paradigmatic causal pattern. This is in detail analyzed in terms of two device-ontological concepts: the ‘*how to achieve*’ and the ‘*what to achieve*’.

An ontological theory of causation needs to satisfy an actual demand for causal modeling in application domains, however. This is because the **achieves** causal relation is not expressive enough to represent causal chains of various phenomena, many of which arguably involve states. Elaborated in order to address this issue are *state-mediated causation* and the *precondition-for* relation (see e.g., Galton, 2012), which is used in turn to define *indirect causal relations*: **allows** and **disallows**. Those practical results are diagrammatically summarized in the table of the functional square of causal relations, a classification of **achieves**, **prevents**, **allows**, and **disallows**, and in the Configuration of State-mediated Causation (CSC), a representation of indirect causal relations.

In particular, CSC is well worth noting as the first step towards a full description of the causal history of the world. For one thing, CSC offers a new perspective on the so-called problem of ‘negative causation’ or ‘absence causation’: e.g., “The *absence* of vitamin C caused scurvy.” For another, the extended usage of CSC indicates a promising way of capturing more complex causal chains. Furthermore, a preliminary formalization of causal relations in the functional square is offered. Consequently, the functional perspective on causation of the dissertation is expected to develop into a full-fledged functional theory of causation that is adequately designed for ontology engineering. An attempt to build a robust ontology of causation made in the dissertation would be of great value for knowledge science because it helps to form a common ground for representing different causal phenomena in different domains and to facilitate an integration of and an interdisciplinary collaboration across research fields dealing with the concept of causation.

Furthermore, the application of the functional view of causation is illustrated with the River Flow Model (RFM) of diseases (Mizoguchi et al., 2011). There is a growing interest in a general ontological model of disease in biomedical ontology research because it would provide a solid foundation for disease ontologies in general. Among other things, the RFM consists in saying that a disease is a dependent continuant constituted of causal chains of abnormal states. One theoretical problem with the RFM is that the notion of causation on which the RFM is based remains obscure.

The dissertation proposes that the causal nature of the RFM be clarified from a functional perspective, hence the improved RFM definition of disease and a clearer comparison between the RFM and other major general models of disease.

1.4. Structure in content

Section 2 provides a brief overview of work on causation in other disciplines than ontology engineering. Section 3 specifies the scope of the current work and conducts a theoretical and ontological examination of causation. Section 4 considers a representation of causation that builds upon ontology of causation. Section 5 delineates the application of the functional view of causation to causal elucidation of the River Flow Model (RFM) of diseases. Section 6 is devoted to the general discussion and also comparison with on related work in ontology engineering. Section 7 summarizes the dissertation with some brief remarks on its possible ramifications for knowledge science and future possible directions of research.

2. Causation as an interdisciplinary concept

2.1. Linguistics

Linguistic research into causation generally centers on the syntactical and semantic analysis of linguistic constructions comprising a causative verb, namely a verb that expresses causation. Clear examples include overt causatives such as ‘causes’ appearing in the sentence: “John caused the robber to die.” What receives more attention is concealed causatives such as ‘killed’ appearing in the sentence “John killed the robber” (Bittner, 1999). This line of investigation is usually coupled with the underlying study of events, space, and time in natural language discourse.

2.2. Cognitive science

In discussing causation, cognitive scientists are typically interested in causal reasoning rather than causation as such. Although they traditionally take it for granted that causal reasoning is a special kind of more general forms of reasoning (including logical and probabilistic ones), there is a growing acknowledgement of the need for considering the causal feature that is specific to causal reasoning (Waldmann, 2017). For instance, a force theory of causal reasoning assumes that people represent singular causal events as generated by ‘hidden forces’ and says, for instance, that the impression of alleged causal asymmetry is reflected in judgments about force.

2.3. Artificial intelligence

The most well-known approach to formal causal representation would be causal models: mathematical models representing causal relationships within an individual system or population (Pearl, 2009). More specifically, causal modeling leverages a directed graph in graph theory, which is mathematically defined as a pair $G = (V, E)$ where V is a set (‘vertices’) and $E \subseteq V \times V$ (‘edges’); see e.g., Harris, Hirst and Mossinghoff (2000) for details. By facilitating inferences about causal relationships from statistical data, causal models are so useful for epistemology of causation that some contemporary theories of cognitive reasoning are based on them.

2.4. Philosophy

One of the most influential philosophical theories of causation is a counterfactual analysis; and its most basic version says that c was a cause of e if and only if e counterfactually depends on c : that is to say, if and only if c and e occurred, and if c had not occurred, then e would not have occurred (Lewis, 1973a, 2000). This view has been however severely criticized for many counterexamples such as preemption and overdetermination (Collins, Hall and Paul, 2004; Paul and Hall, 2013); but see Becker and Vennekens (2018) for a recent work.

3. Theory: Ontology of causation²

3.1. Scope

3.1.1. Ontological substantivity

Regarding causation in physics, for instance, Russell (1913) argues that the notion of causation is useless in physical theories and eliminable from physics. A lot of controversy still continues surrounding the role of causation in physics in general (Price and Corry, 2006). To keep things manageable, the investigation will be pursued within the context of classical (Newtonian) mechanics. Consequently, no further complication is added by other physical theories (such as general relativity and quantum mechanics). It is certainly well worth considering the non-trivial relationship between causation and classical mechanics (Lange, 2009). Following Barton, Rovetto and Mizoguchi (2014), however, it will be assumed that causation plays such a vital explanatory role in Newtonian mechanics that the ongoing exploration will cohere with contemporary (classical) physics. More generally, it is presupposed throughout in this dissertation that causation is ontologically substantive. Causation is part of reality and it is neither merely conceptual (dependent on our cognition) nor merely verbal (dependent on our language).

3.1.2. Simultaneous causation and backwards causation

The issue of simultaneous causation and backwards causation has been widely debated (Taylor, 1966; Dummett, 1954; Flew, 1954). Simultaneous causation and backwards causation are the kinds of causation in which the cause occurs *at the same time as* and *later than* its effect, respectively. Simultaneous causation is intuitively plausible: given Boyle's law with the constant temperature, for instance, the increasing pressure of a gas happens simultaneously with its decreasing volume, the former causing the latter. By comparison, backwards causation seems less probable but is not obviously absurd. It is at least conceivable, for example, that my prayer in 2017 is so powerful as to have saved somebody's life in the Second World War. To simplify the matter, simultaneous causation and backwards causation will be left aside. A standard, 'forwards' conception of causation will be assumed: the cause occurs *earlier than* its effect.

3.1.3. Mental causation and agent causation

It is perfectly legitimate to state ordinarily: "I ate a cake because I was hungry." This kind of causation (usually called 'mental causation') is particularly problematic from an ontological viewpoint. Mental causation means that mental functionings (e.g., beliefs and desires) cause further mental workings and physical events (e.g., actions). It is nonetheless fairly difficult to figure out what mental causation

² This section and the next hinge generally upon Toyoshima, Mizoguchi and Ikeda (2019).

is supposed to be. It is also argued, especially in the context of free will, that an agent can cause something not determined by anything prior (O'Connor, 2000). Requiring a serious engagement in philosophy of mind, action, and free will, a discussion on mental causation and agent causation is too heavy to fall within the range of the present investigation. Discussed herein is mainly physical causation: the kind of causation that holds only among (purely) physical entities.

3.1.4. Causation as a binary relation between token-level occurrents

It is safely assumed in conformity with a general consensus that causation is a binary relation between the cause and its effect. Thus the terms ‘causation’ and ‘causal relation’ will be henceforth used interchangeably. The relata of the causal relation are a fairly contentious subject (see Schaffer, 2016 for a general survey). First, there are two contrasting levels of causation: type-level causation and token-level causation. Type-level causation holds between universals (e.g., “smoking causes cancer”); whereas, token-level causation between particulars (e.g., “Mary’s smoking causes her to get cancer”). Token-level causation is the primary focus of the current research, type-level causation being left aside hereafter. Second, and more controversially, there are several candidates for causal relata (e.g., objects, facts, and tropes). As will be detailed below, arguably the most plausible of them is occurrents given the categories that are commonly accepted in upper ontologies.

3.1.5. Causation as an irreflexive and asymmetric relation

It will be assumed in compliance with a general recognition that the (token-level) causal relation is irreflexive and asymmetric. Causation is irreflexive: no occurrent causes itself. For instance, John’s throwing a stone did not cause itself. Causation is asymmetric: if an occurrent Oc_1 caused an occurrent Oc_2 , then Oc_2 did not cause Oc_1 . For instance, if John’s throwing a stone caused the breaking of a window, the breaking of the window did not cause John’s throwing the stone. More contentious is whether it is invariably the case that if an occurrent Oc_1 caused an occurrent Oc_2 and Oc_2 caused an occurrent Oc_3 , then Oc_1 caused Oc_3 (see e.g., Paul and Hall, 2013: Chapter 5).

3.2. Yet Another More Advanced Top-level Ontology (YAMATO)

In what follows discussions will be centered on the upper ontology Yet Another More Advanced Top-level Ontology (YAMATO) (Mizoguchi, 2011). Although it has been developed merely for the last two decades and it is still under active construction, YAMATO is based on close examination of existing upper ontologies and has as one of its defining characteristics a delicate balance between philosophical rigor and engineering utility.³

³ Borgo and Hitlzer (2018) report that YAMATO is vaguely realist in spirit, while it is only indirectly inspired by some philosophical views and prefers a more pragmatic/engineering approach to ontology.

YAMATO builds fundamentally upon Galton and Mizoguchi’s (2009) view of reality according to which continuants and occurrents are existentially interdependent, neither of them being ontologically reducible to the other. Two prominent subcategories of occurrents are *processes* (e.g., walking) and *events* (e.g., a walk and a conference).⁴ The distinction between processes and events can be elucidated in several ways. Processes, like objects, can change; but events cannot. Processes, like objects, have no temporal part; but events do. Processes are intrinsically ‘ongoing’; but events are in nature ‘completed’. Unlike objects, both processes and events are nevertheless essentially temporal since they are occurrents. Another subcategory of occurrents is *states*: time-indexed qualitative occurrents. Examples include being hungry at time t_1 , sitting at t_2 , and speeding at t_3 . A number of kinds of relations hold among objects, processes, events, and states. For instance, an object *participates in* a process, an event, or a state; and any process *constitutes* a (unique) event: e.g., Mary participates in a walking process as well as the walk event that is constituted by that walking process.

Also unique to YAMATO is its understanding of objects (e.g., rivers) and roles (e.g., students). First, an object is conceptualized as a unity that *enacts* its *external process* or the ‘interface’ between its *internal process* and external process (Galton and Mizoguchi, 2009). As an object, for instance, a river has as its external process changing its course of water flowing (but not water flowing, which is its internal process). Second, YAMATO elaborates a ‘two-tiered’ theory of roles that divides the traditional conception of role into a *role* (an entity to be played) and a *role-holder* (the entity playing the role) (Mizoguchi, Sunagawa, Kozaki and Kitamura, 2007; Mizoguchi, Galton, Kitamura and Kozaki, 2015). Its basic schema is: “An entity *plays* a role in a *context*, thereby becoming a role-holder.” For instance, John plays a teacher role in the XYZ college (context), thereby becoming a particular teacher (role-holder). The notion of context is too complex to formulate, but a context for an entity e is roughly a complex entity that depends existentially on e and it identifies all the relevant objects (including e) and their states: e.g., an occurrent can be seen as a context in which continuants play participant roles, thereby becoming participants. Finally, YAMATO is partially axiomatized: e.g., its process/event-related module (Borgo and Mizoguchi, 2014) and its role-related module (ibid.; Mizoguchi, Galton, Kitamura and Kozaki, 2015).

3.3. From the device ontology via change towards causation

The device ontology, whose initial purpose was to analyze technical artifacts, is a specific way of assigning roles to objects in general (Mizoguchi and Kitamura, 2009). Its basic tenet is that an object plays an *agent role* as a *device* in a given *context*. A device processes something: receiving something to produce something. When received and produced by a device, an entity plays an *input role* as an *input* and an *output role* as an *output*, respectively. When processed by a device, an input plays an

⁴ The term ‘process’ will be hereinafter used in this YAMATO sense of the word unless otherwise specified.

operand role as an *operand* and so does an output. An entity carrying an operand plays a *medium role* as a *medium*. A behavior of a device is the ongoing progressive change from the value of an operand (which is usually a property of the medium) when input into the device to the value of the same operand when output from the device. In short, a behavior of a device is the process of change from the input to the output of the operand processed by the device; it is the transition between two states of the operand.

Note that any object (whether natural or artifact) can play an agent role, thereby becoming a ‘device’, once the harmonious way is identified in which its operand is input and output by the device, as is illustrated by the following examples:

- Example (i): Two magnets (say A and B) repel each other. Assume that A , as a permanent magnet, has a permanent magnet field, the magnetic force produced by which acts on B . B then plays an agent role in the context in which its input is the force received from the magnetic field and its output is the acceleration motion of B apart from the like magnetic pole (see Barton, Rovetto and Mizoguchi, 2014 for details on the ontological nature of forces). The same applies to the case in which B , as a permanent magnet, has a permanent magnet field, the magnetic force produced by which acts on A . Both phenomena simultaneously occur when A repels B and vice versa.
- Example (ii): A rock is falling, being accelerated. The rock plays an agent role in the context in which its input is the force received from the gravity field of the earth and its output is the acceleration with which the rock orients itself towards the gravity center of the earth.
- Example (iii): The human heart plays an agent role in the context in which its operand is the pressure of the blood as a medium. Its behavior is the process of pressuring blood: the process of change from the low pressure to the high pressure of the blood that has travelled through the human heart.
- Example (iv): A heat exchanger plays an agent role in the context in which its operand is heat carried by the fluid as a medium. Its behavior is the transition between two different temperatures (i.e., states of fluid) of the fluid.
- Example (v): There is a book on the desk, on the assumption that m and g refer respectively to the mass of the book and gravitational acceleration. The book plays an agent role in the context in which its operand is the force mg (operand). Its behavior is ‘pushing’ the desk: the change from the ‘gravity state’ to the state of exerting on the desk of the force mg . (When a book is put on the table, it begins to exert force on it. The book transfers the force exerted at the gravity center to the boundary between the book and the table. The table would be eventually broken or at least be damaged owing to the constant exertion of the force.)

There may appear to be no change in Example (v), but note that fatigue is brought about in the situation like this book-table case. Accumulation is the change in reality that is brought about by some integral effect. Imagine a water tank fed by an amount of water with a constant rate: the level of the tank increases as time goes. Similarly, the book on the desk continues pressing the table at a constant force mg . It must be emphasized that any given causal relation (e.g., “a table broke because the book was located on it for a thousand years”) can be modeled upon the device ontology view of reality. Therefore, far from offering a biased view of reality by accommodating the case of accumulation, the device ontology is, in fact, explanatory enough to capture the kind of change that is due to an integral effect as well as the sort of change that is ordinarily observed.

The device ontology takes a ‘black box’ approach to devices: devices can be nested. When an object plays an agent role as a device in a given context, what is happening inside the device is unknowable, insofar as the granularity level of reality corresponding to the context is concerned. To know the interior structure of the device requires another assignment of roles inside the device and this results in the emergence of more fine-grained devices as ‘black boxes’. To find out the inside of the human heart as a device, for instance, it is necessary to assign an agent role to the left ventricle of the human heart and see a behavior of the left ventricle. The same step is just needed to examine how a particular cell of the left ventricle works.

The device ontology is applicable not only to objects in different domains of reality but also to objects at different levels of granularity of a domain. The device ontology is insusceptible to domain knowledge, as illustrated by the examples of the human heart and a heat exchanger. Furthermore, the device ontology manages to describe consistently various layers of a portion of reality. For instance, the left ventricle of the human heart can also play an agent role as a device and so does a particular cell of the left ventricle.

Quite importantly, the notion of context in the identification of the device may be said to be ‘more ontological’ than is usually conceived, in that the context is uniquely determined by the causal relation and in turn uniquely determines the device. *If picked out according to some domain-specific knowledge*, therefore, the causal relation uniquely determines the device. It is in this sense that the insusceptibility of the device-setting to domain knowledge can be correctly understood: instead of being arbitrarily set up, the device accurately represents the objective, causal relation.

As was said above, it is fundamentally assumed that change and causation depend essentially on each other. In particular, whenever a change happens, there must be a *cause* of that change. Suppose that an object O plays an agent role in a context C_i (and with respect to its corresponding granular level G_i of reality), thereby becoming a device D_o . Suppose further that a behavior B of D_o is the change from one state S_1 to the other state S_2 of the operand OP that is processed by D_o . There

must be a causal connection that accounts for B , since it is by definition a change in OP .

What are the cause and effect of the causal linkage that explains B ? The effect is naturally the resulting state S_2 that OP participates in. Characteristic of the device view of change and causation is that the cause is what exists within D_o , or more specifically an occurrent that occurs within D_o with respect to G_{i-1} , i.e. a granular level of reality that is lower than G_i . This means that, for a particular effect E as the occurrent that occurs with respect to any granularity G_i , what is primarily responsible for E is the occurrent that occurs with respect to the finer granularity G_{i-1} .

Consider for instance that a cutting machine cuts a fish into two pieces. Given the device ontology, a cutting machine plays an agent role in a fish-cutting context, thereby becoming a cutting device, and a behavior of the cutting device is the change from the state of being one piece to the state of two pieces of the fish that is processed by the cutting machine. The effect and the cause of the causal connection that grounds the cutting behavior are the ‘two-piece state’ of the fish and what occurs within the cutting device (say, the cutting motion with a knife), respectively.

The occurrent that occurs within D_o with respect to the finer granularity G_{i-1} is of an inherently causal nature because it is plausibly taken to be primarily responsible for the causal relation that holds with respect to G_i . To see this, consider the notion of disposition that receives wide recognition in applied ontology, especially in its biomedical application. A disposition is a causal property in the sense of being causally linked to a specific type of performance (‘realization’) of its bearer under some appropriate circumstances (Röhl and Jansen, 2011). Given the device ontology view of causation, the occurrent under discussion that occurs within D_o can be called a ‘causally efficacious occurrent’ because it grounds the causal relation at the more coarse-grained level of reality (see Section 3.5 for details).

The causal relation is also modeled upon the notions that are correlated with the device ontology: *how to achieve* and *what to achieve* (Mizoguchi and Kitamura, 2000). Those notions are used in a meticulous analysis of functions according to which most ordinary functional concepts are a mixture of the goal to be achieved in virtue of a function and a way of achieving the goal. For instance, the cutting functional concept is a melange of the division of something (the ‘what to achieve’) and the use of a sharp tool such as a knife or scissors (the ‘how to achieve’).

The idea of the separation between the ‘what to achieve’ and the ‘how to achieve’ can be generalized to the nature of other entities. Consider for instance actions, or especially walking action. Someone’s walking can be analyzed as a conceptual mixture of the goal to change her position (the ‘what to achieve’) with putting one foot on the ground in front of the other sufficiently slowly (the ‘how to achieve’). For the sake of convenience, the term ‘ordinarily functional’ will be used to refer to the structure that is explicable in terms of the ‘what to achieve’ and the ‘how to achieve’.

The cause and its effect are also interpretable in terms of the ‘how to achieve’ and the ‘what

to achieve’, respectively. Causation is to be seen as a kind of goal achievement in such a way that something achieves a goal (effect) in a particular way (cause) in virtue of its ordinarily functional feature. According to the device ontology, it is the aforementioned occurrent that occurs within D_o with respect to G_{i-1} that is of an ordinarily functional nature, as shown by the fish-cutting example in which the more fine-grained occurrent of the cutting motion brings about the divided fish by means of a knife.

It might be worried that there seems to be no such ‘how to achieve’ entity as a cutting motion with a knife in many other causal phenomena than cutting. Indeed there may be no action that corresponds directly to the concept of cutting with a knife in the chemical domain. The device ontology view of reality can be nonetheless extended to chemical phenomena in such a way that the ‘cut’ of some chemical bond could be realized by different chemical reactions (‘how to achieve’) to bring about the equivalent separated chemical compounds (‘what to achieve’).

3.4. From the theory of systemic function to conceptual overlap between causation and function

The current debate on function assumes two major kinds of function: biological function (e.g., the function of the human heart to pump blood around the human body) and artifact function (e.g., the function of a heat exchanger to cool or heat by transferring heat from one liquid to another). The distinction between essential and accidental functions has been also actively discussed in the literature (e.g., Röhl and Jansen, 2014) and it is generally recognized that biological functions are essential. For instance, the function of a nose to let air enter into the body is essential and the function of the nose to hold glasses is accidental.

The theory of systemic function aims to build a unifying, ontological definition of function that encompasses both biological and artifact functions as well as both essential and accidental functions (Mizoguchi, Kitamura and Borgo, 2012, 2016; Borgo, Mizoguchi and Kitamura, 2016). Its main thesis is that any particular function is a specialization of a *systemic function*. It is based on the idea that both biological and artifact functions have three features in common.

First, a function of a certain object involves a system to which that object is connected: the human heart and the heat exchanger are connected to the blood circulatory system and to the dual fluid circulatory system, respectively. Second, the notion of goal matters to function: the goals of the human heart and the heat exchanger are to transfer oxygenated blood and to increase (or decrease) the temperature of one target liquid, respectively. Third, the goal to be achieved is determined in terms of a context but independently of human intention. The human heart has the goal to transfer oxygenated blood in the context of blood circulation. Contrary to popular belief, the human heart may well have the goal to make beating sound in the context of making sound. It is for some domain-

specific (especially biological) reason, but not for an ontological reason, that the function of the human heart is to pump blood, but not to maintain beating sound. This means that the essentiality of a function of an object is always dependent upon what kind of goal the object is supposed to have, or more specifically, the context in which the object is located.

To offer a strict definition of a systemic function requires four key notions.

(1) *Behavior*. The definition of a behavior has been already offered. Quite importantly, an object can have multiple behaviors and, if it plays an agent role in a certain context, the object displays the corresponding behavior. For instance, the human heart has as behavior not only the process of pressuring blood but also the process of making beating sound: the change from the low state to the high state of the beating sound (operand) of the blood (medium) that is processed by the human heart (device).

(2) *System*. Although it is too complex to be modeled readily, a system is roughly an entity that consists of some components and their relations and that, in virtue of their harmonization, plays an agent role, thereby becoming a device and displaying a behavior. For instance, the blood circulatory system has as component a number of objects such as the heart and blood vessels and works as a device because of the harmonious collaboration of its components with so many relations among them. A behavior of the blood circulatory system is the transition between two different states of oxygenation (operand) carried by blood (medium). Note that a system can have multiple behaviors and, if it plays an agent role in a certain context, the system displays the corresponding behavior.

(3) *Goal*. The intuitive notion of goal as a state to be achieved is here characterized in terms of roles: a state plays a goal role in a context, thereby becoming a goal. A goal role is thus an *occurrent role*, namely a role played by an occurrent (see Mizoguchi, Galton, Kitamura and Kozaki, 2015 for details). Goals fall into intentional goals and non-intentional goals. Examples of the former include the goal which is ascribed to a heat exchanger by its designer. It is important to note that the kind of goal that is primarily focused on in the dissertation, a systemic goal (elucidated below), is non-intentional (see Mizoguchi, Kitamura and Borgo, 2016 for details).

(4) *Systemic (function) context*. A systemic (function) context for an object O and a system S is, according to Mizoguchi, Kitamura and Borgo's (2012) simplified version of its definition, a context C in which (i) O plays an agent role, thereby becoming a device and displaying a behavior B_o and (ii) S plays an agent role, thereby becoming a device and displaying a behavior and (iii) O is a component of S and (iv) there is some systemic goal G such that (iv-a) G is specified by C and (iv-b) B_o contributes to the process of achieving G in such a way that a state of S will play a goal role, thereby becoming G .

The basic idea behind a systemic context is as follows. What is primarily responsible for the behavior that a system displays if it plays an agent role in the context C is the fact that the components

of the system interact so well with one another that those interactions realize the behavior of the system as a whole. This means that a behavior that an object (as a component of the system) displays if it is located in C contributes to the achievement of the state of the system that is specified by C .

The nub of the unifying theory of function is that function is explicable in terms of the interplay between behaviors and systemic contexts: an object has some function in virtue of some systemic context in which a behavior of the object is of a functional nature. More formally, an object O has some function if there is some systemic context C for O and a system S such that a behavior of O *plays a functional role*, and therefore, *O performs a systemic function*.

To illustrate a systemic context and a systemic function, take for example the function of the human heart to pump blood. Consider the blood circulation context in which the human heart and the blood circulatory system play an agent role as devices. A behavior of the human heart is the process of pressuring blood. A behavior of the blood circulatory system, which has as component the human heart, is the process of transferring oxygenated blood. Consider also a goal to transfer blood. The goal to transfer blood is specified by the blood circulation context. A blood-pressuring behavior of the human heart contributes to the process of achieving the goal to transfer blood in such a way that a state of the blood circulatory system plays a goal role as the goal to transfer blood.⁵ This means that the goal to transfer blood refers to a systemic goal. Thus, the blood circulation context is a systemic context for the human heart and the blood circulatory system. A pressuring blood behavior of the human heart plays the functional role to pump blood and thus the human heart performs the systemic function to pump blood in the blood circulation systemic context for the human heart and the blood circulatory system.

Given the theory of systemic function, the human heart also has the function to maintain beating sound. Consider next the sound-making context in which the human heart and the human body system play an agent role as devices. A behavior of the human heart is the process of making beating sound. A behavior of the human body system, which has as component the human heart, is the process of making body sound. Consider a goal to maintain body sound. The goal to maintain body sound is specified by the sound-making context. A sound-making behavior of the human heart contributes to the process of achieving the goal to maintain body sound in such a way that a state of

⁵ It might be argued that the goal here should be rather to maintain a constant circulation (process) unless further discussion (especially about granularity) is provided, based on the intuition that transferring blood is not like e.g., transferring a piece of furniture from one room to another, where there is a clear initial state and a clear final (goal) state. However, transferring blood could be plausibly taken to resemble conceptually transferring a piece of furniture from one room to another. For one thing, the former could look closer to the latter with a focus on the fact that the primary purpose of transferring blood is to bring each red blood cell from one bodily place to another, where one could observe well a pair of an initial state and a final state. For another, conversely, the latter could look closer to the former in the situation in which many pieces of furniture on the conveyor belt are transferred from one room to another consecutively, where one would have more difficulty in observing a pair of an initial state and a final state.

the human body system plays a goal role as the goal to maintain body sound. This entails that the goal to maintain body sound refers to a systemic goal. Hence, the sound-making context is a systemic context for the human heart and the human body system. A behavior of making beating sound by the human heart plays the functional role to maintain beating sound in the human body system and thus the human heart performs the systemic function to maintain beating sound in the sound-making systemic context for the human heart and the human body system.

As for the issue of essentiality/accidentality of functions, the theory of systemic function says that all the (specializations of) systemic functions are essential *with respect to some systemic context* on the grounds that essentiality of a systemic function should be judged by the existence of a contribution to the goal determined by the behavior that the system displays if it is located in the systemic context. This means that whether a function is essential or accidental to an object is determined by which systemic context the object lies in, which in turn depends on domain-specific knowledge and assumptions. For instance, the function to pump blood is essential to the human heart only in the blood circulation context (with which biologists are generally concerned); and the function to maintain beating sound may be accidental to the human heart in the blood circulation context, but it is indeed essential to the human heart in the sound-making context. In this way, both essential and accidental functions are explicable in terms of the context-dependent notion of essentiality of systemic functions.

One may think that the sound-making of the human heart is just an incidental side-effect that may happen to be of diagnosis value. Despite its possible diagnostic value, however, the sound-making (behavior) of the human heart should be treated as performing a *use function* to help the doctor's diagnosis, i.e., the function relativized to the *use context* in which the doctor uses the beating sound of the human heart to diagnose it, just as the human nose has the use function to support glasses: the function relativized to the use context in which the human uses her nose to wear glasses. Although the function of the human heart to make the sound may be taken to be essential with respect to the above-described use context, it should be understood as accidental like the glasses-use case because such 'use' is out of the scope of any biological organism's life plan.⁶

In addition, the theory of systemic function explains, in virtue of the separation between a functional role and a behavior to play the functional role, the notion of malfunction: an object can have a function, even though the object can fail to perform the function occasionally or even permanently. To say that an object malfunctions means within this framework that a behavior of an object does not play the given functional role: namely, it does not contribute to the realization of the systemic goal. Thus a malfunction of an object, which depends solely on its behavior, is sharply

⁶ The kind of use that is being discussed roughly means the interaction between the object under consideration and some (intentional) agent outside the organism.

distinguished from the case in which an object has no function, which is interpretable in terms of the non-existence of a systemic context and its corresponding functional role. As seen above, the theory of systemic function is such a full-fledged account of function that causation can be conceptually mapped onto a systemic function comprehensively. This point is to be discussed below together with its connection with lawhood and a systemic context.

It was argued above that causation can be seen as a kind of goal achievement, based on the device ontology view of reality. Since a systemic function is based on the idea of the achievement of a systemic goal by a behavior, causation may be regarded as a kind of goal achievement of a behavior within the theory of systemic function. As said above, however, talk of causation is always coupled with talk of laws of nature. Laws of nature are supposed to be a cluster of true general statements that are suitable for describing (and predicting) all the natural phenomena in the world. Examples include Newton's three laws of motion, Mendel's genetic laws, and the ideal gas law. It is reasonable to think that each causal phenomenon must comply with laws of nature. The conceptual mapping of causation onto function would require considering laws of nature from a functional viewpoint.

Interpreted from a causal perspective, systemic contexts are functional analogues of lawhood. This does not mean that laws of nature depend existentially on systemic contexts; rather, every law of nature holds independently of any systemic context. The claim is that the causal relation does not require all the laws of nature but a limited number of relevant laws among them and this corresponds closely to the fact that an object displays a particular behavior if it is located in a systemic context. A precise analysis of the causal relation in general needs an adequate delimitation of laws of nature, which would be convincingly taken to be brought about by some relevant systemic context.

To illustrate this, suppose that John ate a fish easily because he had cut it into bite-size pieces. Certainly this causal phenomenon must be naturally lawful. Not all the existing laws of nature nonetheless play a vital role for the causal relation under consideration. For instance, Newton's laws of motion would be explanatorily and/or inferentially important for a causal sequence of John's actions, but neither Mendel's genetic laws nor the ideal gas law, although those three laws of nature hold equally. On the contrary, it would be inappropriate to speak of Mendel's genetic laws or the ideal gas law in discussing the cause of John's easy eating of the fish.

The example being seen from a functional viewpoint, John displayed a variety of behaviors before eating the fish, including his cutting behavior, his behavior of cleaning a room, and his behavior of walking to the nearby station. Since John's eating the fish is currently focused on, John's cutting behavior is, according to the theory of systemic function, demarcated from other behaviors of his by the systemic context for John and e.g., a seafood cooking system. It can be thus said that, in

this ‘easy-to-eat systemic context’ for John and the seafood cooking system, John’s cutting behavior plays a functional role to (and John performs the systemic function to) make a fish easy to eat. It is not hard to see a conceptual affinity between ‘demarcated lawhood’ and a systemic context. Some relevant (but not all the) laws of nature are explanatory with respect to a particular causal relation; and similarly, some relevant (but not every) systemic context is with respect to a particular (specialization of a systemic) function. Which law of nature is pertinent can be reasonably observed by the nature of the causal relation; and likewise, which systemic context is suitable by the nature of the function. This meshes with the non-intentionality of a systemic context: *if* it is located in a systemic context, *then* an object displays the corresponding behavior (see for details Mizoguchi, Kitamura and Borgo, 2016: Section 6).

Viewed from another perspective, each systemic context essentially demarcates lawhood once it is set up. Consider a gene splicing context for John and a biological laboratory system. Unlike the easy-to-eat context discussed above, this gene splicing context involves some laws of nature concerning genes (whether they include Mendel’s or not) since gene splicing must obey them. Interpreted within the gene splicing context, John’s cutting behavior, together with his pasting process, would ground the causal relation between his use of a group of chemicals and the emergence of a new genetic combination.

In short, a proposed ontological picture of causation is as follows. The world is in nature causally so chaotic because of the highly complex interrelationship between causal phenomena. *Once it is picked out*, however, the causal relation *necessitates* the context (say *C*) in which it holds. Most importantly, it is *not* the case that the causal relation is relativized to and can change with a context that is externally set up. It is only when the causal relation goes together with *C* that it can be conceptually mapped onto the systemic function determined by the systemic context that is grounded in *C*. Finally, one can see a close conceptual affinity between the understanding of causation and that of (systemic) function.

3.5. Achieves as a direct and positive causal relation

3.5.1. From functional achievement to causal achieves

Causation is such a notoriously elusive notion that it is not uncommon to take it as primitive in some practically motivated situations, e.g., when it comes to the construction of knowledge representation systems. Indeed it is rather difficult to define explicitly the causal concept. One of the main objectives at present is nevertheless to *elucidate* the causal concept and help us understand it better than primitivism about it. To meet this goal, the authors focus on the traditionally underestimated role of the notion of context with respect to causation. Existing independently of our cognition and language, the causal world as such is chaotic owing to intimately connected, various causal

phenomena. Nonetheless, a certain causal relation, *once it is articulated*, involves the context in which it holds. It must be emphasized that this does *not* mean the epistemic relativization of the causal relation to some context. The kind of change to which the causal relation is intimately connected is modeled upon the device-ontological conception of change as behavior.

It is only when the causal relation expresses itself with its unique context that it can be conceptually mapped onto the systemic-theoretical function: the function that is determined by the systemic context that is grounded in the original context. As argued above, for a given causal relation, there is the systemic context that represent the laws of nature that are enough for explaining the causal relation. In this way, causation can be clarified from a functional point of view.

One significant result of those arguments is that the causal relation is well explicable in terms of the functional concept of *achievement*, by which is meant not the specificity of various kinds of functions but the broader view that the ‘cause’ *achieves* the ‘effect’ in the systemic context. This is, as said above, primarily because the cause and its effect are modeled upon the ‘how to achieve’ and ‘what to achieve’ in the device ontology, respectively; and the effect can be conceptually mapped onto some systemic goal in the systemic theory of function. In what follows the term ‘**achieves**’ is used to refer to the causal relation. Additionally the **achieves** causal relation holds between two actual occurrents because actualism is throughout assumed: the view that there are only entities (especially occurrents) in this actual world.

Another consequence is an explicit reference to a (systemic) context and its corresponding granularity in expressing the **achieves** causal relation: e.g., “An occurrent *X* achieves an occurrent *Y* with respect to a context *C* and its corresponding granularity *G*.” Although they are sometimes unmentioned for the sake of simplicity, a context and its corresponding granularity are naturally important for the causal relation, given the primacy of the notion of context over the causal relation. To illustrate this, consider the simple fact that Mary was unable to enter her house because its door was locked. It would make little sense to say so unless the context is assumed in which all the windows of her house were locked so that she could not go into the house through any of those windows. In this way, the causal relation essentially expresses itself together with some context and its corresponding granular level of reality, as is supported by the observation that one would fail to speak of the causal relation (as found in this example) without mentioning the scope in which it holds.

The **achieves** causal relation is described as *direct* and *positive* in the terminology of this dissertation. It is direct in the sense of ‘bringing about’ some occurrent and it is positive in the sense of holding only between two actual occurrents. On the other hand, the causal relation is *indirect* if it holds *via* some other occurrent (see below for details) and it is *negative* if it has as relatum at least one non-actual occurrent. Given actualism, negative causal relations are merely conceptual and/or linguistic, but not ontological. Finally, granted that causation is relational, the nature of the **achieves**

causal relation is fully clarified by the correct understanding of its causal relata, which will be provided below.

3.5.2. Causal relata

The **achieves** causal relata are primarily occurrents because so are the relata of goal achievement. Based on a detailed analysis of multifarious examples, three kinds of the **achieves** relata are proposed: events, processes, and states. Note that, to the best of the author's knowledge, all the possible pairs of the **achieves** relata are below presented.

A) *Event-state*

Example 1 (Cutting a loaf). John cut the loaf into thick slices.

Intuitively, the loaf was thickly sliced because John cut it. Interpreted in terms of the **achieves** relation, the occurrent of John's cutting the loaf **achieves** ('brings about') the occurrent of the loaf's being thickly sliced. The former occurrent is an event because its beginning and end are clearly demarcated; and the latter is a state because it is the time-indexed mode of existence of the loaf. Therefore: the *event* of John cutting the loaf achieves the *state* of the loaf being thickly sliced.

It might be said that, on closer examination, the event of John cutting the loaf **achieves** the *event* of the loaf *becoming* thickly sliced, which in turn **achieves** the state of the loaf's being thickly sliced. This reasoning is faulty, however. Given the notion of event of the dissertation, it is the case that the event of John cutting the loaf **achieves** the event of the loaf becoming thickly sliced only if the latter occurrent occurs after the former is 'completed'. What really occurred when John cut the loaf into thick slices is nonetheless that the loaf was in the state of being thickly sliced, because the loaf *had already become* thickly sliced.

It could be also suggested that the *process* of John cutting the loaf **achieves** the state of the loaf being thickly sliced. Certainly the loaf is being gradually sliced as John is *cutting* it. The loaf was separated into thick slices, however, when the process of John cutting the loaf became 'completed', i.e., when the event of John cutting the loaf occurred. Therefore, the event (but not process) of John cutting the loaf achieves the state of the loaf being thickly sliced.

B) *Event-process*

Example 2 (Billiard balls). On a billiard table, a red ball (called 'Red') moved at a constant velocity $v(> 0)$ towards a yellow ball (called 'Yellow') until the time t_l , when Red collided at v with Yellow, lost velocity, and stopped immediately. After t_l , Yellow instantly gained acceleration

and finally moved at a constant velocity $v(> 0)$.⁷

Pretheoretically, Yellow moved because it collided with Red. According to the event/state/process distinction, the event of Red's collision with Yellow **achieves** the process of Yellow's movement. It could be argued that the event of Red's collision with Yellow **achieves** the *event* of Yellow's movement. This is not the case, however; and furthermore, no event *directly achieves* any other event (see Section 3.5.3 for details).

C) *Process-process*

Example 3 (A crankshaft and a piston). In an industrial machinery, a crankshaft converts reciprocating motion of a piston into rotational motion of the crankshaft.

Seen from the engineer's point of view, a crankshaft is rotating because a piston is reciprocating, or technically speaking, the reciprocating motion occurrent of the piston **achieves** the rotational motion occurrent of the crankshaft. The reciprocating motion of the piston *constantly* transforms into the rotational motion of the crankshaft; that is, the rotational motion occurrent of the crankshaft occurs *at any time* at which the reciprocating motion occurrent of the piston occurs. Since those two occurents are intrinsically 'ongoing' and 'ongoing' occurents are classified as processes, the reciprocating motion *process* of the piston **achieves** the rotational motion *process* of the crankshaft.

In more detail, each of the processes in question consists of a continuous succession of positional states. The piston and crankshaft are so linked that the positional state of the former necessitates (and thus **achieves**) the corresponding positional state of the latter. This gives rise to a continuous series of **achieves** relations between the state of the piston and the state of the crankshaft, and this series cumulatively constitutes an **achieves** relation between the processes constituted by the two series of states. It should be emphasized that it is *not* the case that the reciprocating motion *event* of the piston **achieves** the rotational motion *event* of the crankshaft. As discussed above, one event **achieves** another event only if the latter event occurs after the former is 'completed'. Once the piston finishes reciprocating, however, the crankshaft ends up rotating. The reciprocating motion of the piston and the rotational motion of the crankshaft do not constitute a consecutive series of events; rather, they are concurrently occurring processes.

D) *Process-state and state-state*

Example 4 (Thrombosis). In a person with thrombosis, a growing blood clot in a blood vessel

⁷ It is safely assumed, given physical reality, that neither Red nor Yellow is a (completely) rigid body: a body that can rotate with all its parts locked together and without any change in its shape.

obstructs the flow of blood and the decreased flow rate of blood brings about the short supply of oxygen in the affected part of her body.

Example 4 appears to be closely analogous to Example 3 at first, which produces the following undesirable consequence: (i*) the growing *process* of a blood clot in a blood vessel **achieves** the *process* of getting smaller of the cross-sectional area of the blood vessel and (ii*) the *process* of getting smaller of the cross-sectional area of the blood vessel **achieves** the decreasing *process* of the flow rate of blood. For it is not the growth of a blood clot in a blood vessel but the clinically abnormally low flow rate of blood (brought about by the clinically abnormally small cross-sectional area of the blood vessel) that cause the lack of oxygen in the vessel; one would otherwise mistakenly believe that vital organs would be no longer seriously damaged owing to an oxygen supply shortage if the blood clot ceased to grow.

In other words, what is primarily responsible for thrombosis is being low *at a particular time* of the flow rate of blood (which is brought about by being small *at a particular time* of the cross-sectional area of the blood vessel). Since time-indexed (qualitative) occurrents are grouped under the heading of states, Example 4 is adequately analyzed as follows: (i) the growing *process* of a blood clot in a blood vessel **achieves** the *state* of being small of the cross-sectional area of the blood vessel; (ii) the *state* of being small of the cross-sectional area of the blood vessel **achieves** the *state* of being low of the flow rate of blood; and (iii) the *state* of being low of the flow rate of blood **achieves** the *state* of being in short supply of oxygen (see below for details on the state-state achieves causal relation).

To summarize, there are five types of causal pair: event-state, event-process, process-process, process-state, and state-state. Characteristic of this is that, with respect to causal relata, states are on an equal footing with events and processes. Most importantly, the event-state pair is a canonical form of causal relata. An interesting case is the process-process pair because it is based on the observation of a continuous succession of states. Further theoretical explanation is to be provided below.

3.5.3. 'Causally efficacious occurrents'

Broadly speaking, there are two types of approaches to causation. Although a large cluster of those approaches offer a *reductive* analysis of causation, others endorse a *non-reductive* view of causation. One version of the latter is a dispositional theory of causation. Given the ontology of dispositions as *causally efficacious properties* or *causal properties*, the dispositional theory of causation says that (the simplest case of) causation occurs when a disposition is realized/manifested (Mumford and Anjum, 2011). A glass broke because the fragility disposition of the glass was realized/manifested,

for instance.

Taking a cue from a non-reductive, dispositional view of causation, the functional perspective on causation of the dissertation explicates causation in terms of *causally efficacious occurrents*. This *very roughly* means that occurrents (events, processes, and states) as such have the ‘power’ or ‘capacity’ to bring about some occurrent. It is important to remember that this statement should not be taken at face value in such a way that there exists such ‘causal power’ or ‘causal capacity’ that occurrents are allowed to exemplify it; as with dispositions as causal properties, it is a *primitive* claim that occurrents are causally efficacious. Seen from another perspective, causation is claimed to be unanalyzably ‘grounded’ in the causal efficacy of occurrents.

The causal efficacy of occurrents could be nonetheless further elucidated. As said, the **achieves** causal relation can be modeled upon the device-ontological combination of the ‘what to achieve’ with the ‘how to achieve’ (which, if any, both exist in a single occurrent). It is hypothesized, based on this idea, that the causal efficacy of occurrents may be explainable in terms of the ‘what to achieve’ and the ‘how to achieve’. To illustrate this, consider first the causal efficacy of events. Recall that the event of John cutting the loaf **achieves** the state of the loaf’s being thickly sliced (from Example 1 “Cutting a loaf”). Interpreted in the current context, this **achieves** causal relation holds in virtue of that causal efficacy of the event of John cutting the loaf which brought about the state of the loaf’s being thickly sliced (the ‘what to achieve’) by way of John’s use of the knife (the ‘how to achieve’).

As this example shows, in virtue of its causal efficacy, an event leads primarily to a state and only secondarily to a process as a sequence of firmly glued states. Seen from this viewpoint, talk of the **achieves** causal relation between events means either that an event directly **achieves** another, specific type of event (e.g., an ‘initiation event’) so that no state could occur between those two events (see below the discussion on Example 2 “Billiard balls”); or that there exist a causal sequence of occurrents from the event as the cause to the event as the effect.

The causal efficacy of processes is significantly different from that of events, as is supported by the observation that there could be some cases in which a process bears the direct **achieves** to a process, as illustrated with the detailed discussion on Example 3 (“A crankshaft and a piston”). To clarify further the difference in the causal efficacy between events and processes, consider carefully Example 2 (Billiard balls) which is usually (but mistakenly) taken to mean that the event of Red’s collision with Yellow **achieves** the event of Yellow’s movement. Analyzed meticulously, what occurred during the collision between Red and Yellow is that the *process* of Red’s movement **achieves** the *process* of Yellow’s movement with respect to a systemic context C_I (whose ‘demarcated lawhood’ relates particularly to Newton’s second law of motion represented by the formula $F = ma$) and its corresponding granular level G_I of reality, since each state (position) of Red *ceaselessly achieves*

some state (position) of Yellow during that period of time.

Therefore, the event of Yellow's movement (which is constituted by the process of Yellow's movement) was primarily due to the causal efficacy of the process of Red's movement with respect to C_1 and G_1 . Note that the collision event between Red and Yellow **achieves** the process of Yellow's movement, but with respect to a systemic context C_2 and its corresponding granularity G_2 where one observes the collision phenomenon at such a coarse-grained level of reality that the collision counts as an instantaneous event and hence as a causal relatum (in particular, as the cause). The event to which the collision event bears the **achieves** causal relation is, if any, the 'initiation event' of Yellow *beginning* to move, which can be seen as the exceptional, 'event manifestation' of the causal efficacy of the collision event. See also Section 6 for a remark on Galton's (2012) notion of causal relation as the relation between events.

Finally, a state is causally efficacious in a quite distinct sense from both events and processes. A state can be causally efficacious and hence it can be a cause of the **achieves** causal relation when it is an 'internal state' of some object (device) that participates in the occurrent that is relevant to the occurrent as the effect. To see this, recall Example 4 (Thrombosis): the state of being small of the cross-sectional area of the blood vessel **achieves** the state of being low of the flow rate of blood. The clot and the cross-sectional area of the blood vessel are analogous with a (flow control) valve and the valve opening of the valve in the domain of fluid mechanics, respectively, and the blood clot participates in the flowing process of blood, which is crucial to the state (effect) of being low of the flow rate of blood.

To sum up, **achieves** causal relations generally have at least one state, because the causal efficacy of events consists in bringing about states. Exceptions include the process-process **achieves** causal relation found as in Example 3 (A crankshaft and a piston). Additionally, no event bears any direct **achieves** causal relation to another event unless an 'initiation event' is treated as an effect (which is also among **achieves** causal relations that is not mediated by any state). This is due to the fact that, being a 'completed' occurrent, no event as such *directly* bring about any following event.

3.5.4. Prevents as a negative counterpart of achieves

Although each causal relation is theoretically explicable in terms of the **achieves** causal relation, ordinary causal discourse is not limited to talk of the **achieves** causal relation. Consider the following example:

Example 5 (Election). In the United States presidential election in 2016, Donald Trump defeated Hilary Clinton, thereby becoming the 45th U.S. president.

Common sense tells us that Trump is the 45th U.S. president because he won the election. It would be equally normal to say that Trump's victory 'prevented' Clinton from being the 45th U.S. president. The latter statement including the term 'prevent' seems to refer to the state of affairs that is represented by the former: the fact that the event of Trump's victory **achieves** the state of the 45th U.S. president being Trump. This consideration leads to another way of speaking of the **achieves** phenomenon: the **prevents** causal relation. Interpreted in terms of the **prevents** causal relation, Example 5 implies that the event of Trump's victory **prevents** the state of the 45th U.S. president being Clinton.

Elucidation of the **prevents** causal relation requires careful consideration of the effect of the **achieves** causal relation. As said above, the event of Trump's victory **achieves** the state of the 45th U.S. president being Trump. Precisely put, this **achieves** causal relation holds with respect to a systemic context (say C_2) of the U.S. presidential election in 2016 and its corresponding, macroscopic granular level (say G_2) of reality at which, e.g., Trump and Clinton are observed, but neither subatomic particles nor the whole universe.

Quite importantly, this fact can be paraphrased as follows: the event of Trump's victory **achieves** the state of the 45th U.S. president being *not* Clinton with respect to C_2 and G_2 . The state of the 45th U.S. president being not Clinton appears to be non-actual and to lie outside the range of actualism because it includes the term 'not' and it can be loosely labelled as a negative occurrent. This linguistic worry is misguided, however. *Insofar as C_2 and G_2 are concerned*, the state of the 45th U.S. president being not Clinton must be the same as the state of the 45th U.S. president being Trump, since either Trump or Clinton (but not both) must be the 45th U.S. president with respect to C_2 and G_2 .

The **prevents** causal relation is a way of speaking of the **achieves** causal relation whose effect is a negative occurrent. It is the case that the event of Trump's victory **achieves** the (actual) state of the 45th U.S. president being not Clinton with respect to C_2 and G_2 . For the actualist, to say that the event of Trump's victory **prevents** the state of the 45th U.S. president being Clinton is merely a way of describing the same fact using the negative term 'prevent' instead of the term 'not'. It should be emphasized that, even given actualism, the negative **prevents** causal relation can be 'conceptually translated' into the positive **achieves** causal relation in virtue of the conceptual mapping of causation onto function, or rather its explication of the relationship between the causal relation and context.

The **prevents** causal relation is generally defined in terms of **achieves** as follows. Note that the variables presented in this definition (and all others) refer to token-level occurrents:

Definition 1 (Prevents). An occurrent X **prevents** an occurrent Y with respect to a systemic context C and its corresponding granularity G if and only if there exists an occurrent Z such that (i) Y and Z are incompatible with respect to C and G and (ii) X **achieves** Z with respect to C and

G.

As this definition shows, **prevents** is a direct and negative causal relation. **Prevents** is direct because, as is the case with the **achieves** causal relation, no occurrent (state) plays an intermediary role between the cause of **prevents** and its effect. Prevents is negative as well because it has as relatum a non-actual occurrent. Notice that, given actualism, **prevents** is a negative counterpart of **achieves** in the sense of offering a negative representation of the **achieves** causal relation holding in the actual world.

3.5.5. A state-centered approach

It was argued that all three major kinds of occurrents (namely events, processes, and states) can be relata of the **achieves** causal relation and that there are some different combinations of causal relata, including the event-state and the process-process pairs. This fairly generous view of causal relata is supported by the pivotal role of states in the functional perspective on causation. It is assumed throughout that causation bears an intimate relationship to change, which can be modeled roughly as the transition between states. This means that each causal relation (and its relata) must in some way involve a relevant transition between states.

The centrality of states to the causal relation generally explains how events, processes, and states are vindicated as causal relata. First and foremost, states can be causal relata. States can be causes, as illustrated by Example 4 (Thrombosis: state-state): the *state* of being small of the cross-sectional area of the blood vessel **achieves** the state of being low of the flow rate of blood, and the *state* of being low of the flow rate of blood **achieves** the state of being in short supply of oxygen. States can be also effects, as illustrated by Example 1 (Cutting a loaf: event-state) and Example 4 (Thrombosis: process-state).

Moreover, states are the primary kind of causal relata, since no **achieves** causal relation holds unless its corresponding change as the state transition occurs. This view sheds new light on the way events are causal relata. In most cases, events can be causal relata (especially causes) because they bring about a new state of some participant in the event. In Example 1 (Cutting a loaf), the event of John cutting the loaf is of great importance not because of the relevance of its occurrence as such but because it resulted in the state of the loaf being thickly sliced.

Three cautionary notes are made on the state-centered approach to causation. First, states are causally efficacious despite their ‘static’ appearance: loosely speaking, they have the ‘power’ or ‘capacity’ to cause something. Consider Example (v): there is a book on the desk, on the assumption that *m* and *g* refer respectively to the mass of the book and gravitational acceleration. Intuitively, the desk participates in the state of being located on the floor and there seems to be no substantial change in the scenario over a period of time. Seen from a different perspective, however, the desk is so

cumulatively pressed with and hence influenced by the force mg that relevant change in the desk can occur in the sense that the desk is physically damaged.

Second, a causal role of an event must be sharply distinguished from that of the process that constitutes the event in such a way that the process does not consist in bringing about a new state which the event will have produced when completed; a process in general can be seen as a sequence of firmly linked states. The causal role of processes was illustrated above with close examination of Example 3 (A crankshaft and a piston).

Third, it is the case that an event **achieves** another event but only in the sense that the latter event refers to a typically instantaneous, ‘initiation event’, illustrated by the above-given discussion on Example 2 (Billiard balls). What should be focused on is the process of Yellow’s movement *as a whole*, but not the initiation event of Yellow’s movement. The main lesson to be drawn from this is that, in principle, no event-event direct **achieves** causal relation holds.

All those arguments given above indicate the functional feature of causation given the device ontology and the theory of systemic function, thus enabling us to use the **achieves** causal relation as the consequence of the conceptual mapping of causation onto function. The **achieves** causal relation is further supported by the idea of ‘causally efficacious occurrents’ (especially the causal efficacy of events whose manifestation brings about states) and a state-centered approach to causation. It would be thus no exaggeration to say that the ontological (rather representational) inquiry for causation herein conducted ends with the explanation of the **achieves** causal relation (as well as the **prevents** causal relation).

Despite the essential directness of the **achieves** causal relation, the idea of causal chains (which involves *indirect* causal phenomena) is indispensable for representing a rich diversity of our actual understanding of causation. A fourfold distinction of causal relations will be therefore proposed later according to the direct/indirect and positive/negative dichotomies in order to offer a more expressive causal representation.

4. Practice: Representation of causation

4.1. State-mediated causation

4.1.1. Precondition-for relations

As said above, the direct/indirect distinction is introduced in order to accommodate a wide range of ordinary causal discourse. Consider a commonplace statement: “Mary went into a gallery because its door was unlocked.” Intuitively, Mary entering the gallery was not brought about by its unlocked door; rather, the unlocked door ‘enabled’ Mary to enter the gallery. In this respect, the unlocked door or the *state* of the door being unlocked is a *precondition* (see Galton, 2012) for the causal relation whose effect is Mary’s action. This kind of precondition may well be said to be *facilitative*: when a state Z is a facilitative precondition for an occurrent Y , the occurrence of Y always involves the occurrence of Z .

Then a negative counterpart of a facilitative precondition, a *preventive precondition*, may be elaborated: when a state Z is a preventive precondition for an occurrent Y , the occurrence of Y has the role to bring about the non-occurrence of Z . For instance, the locked state of the door of a gallery is a preventive precondition for the event of Mary entering the gallery.

It is important to note the following relationships between those two kinds of preconditions:

Proposition 1. If a state Z is incompatible with a state Z' with respect to a systemic context C and its corresponding granularity G and Z' is a facilitative precondition for an occurrent Y with respect to C and G , then Z is a preventive precondition for Y with respect to C and G .

Proposition 2. If a state Z is incompatible with a state Z' with respect to a systemic context C and its corresponding granularity G and Z' is a preventive precondition for an occurrent Y with respect to C and G , then Z is a facilitative precondition for Y with respect to C and G .

Given the context in which going through the door is the only way of entering the gallery, for instance, the locked state of the door of the gallery and the unlocked state of the door of the gallery are mutually incompatible. One is a facilitative (preventive) precondition for the event of Mary entering the gallery, then the other is a preventive (facilitative) precondition for Mary entering the gallery.

4.1.2. Configuration of State-mediated Causation (CSC)

The complex relationships among the cause, its effect, and the ‘mediating state’ that are commonly found in indirect causal relations are to be well conceptualized and visualized as a triangle-shaped configuration of nodes represented as a square or an oval and the arrows among them which will hereinafter be called the ‘Configuration of State-mediated Causation (CSC)’ (Figure 1).

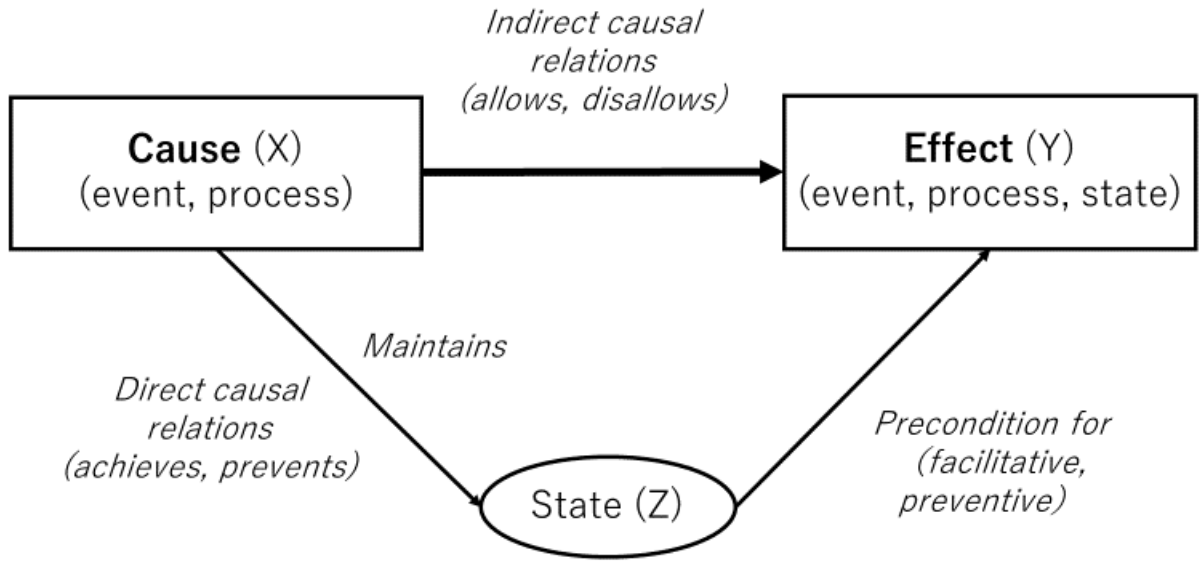


Fig. 1. The Configuration of State-mediated Causation (CSC)

CSC is one of the fruitful results of the functional perspective on causation. For one thing, CSC serves as a conceptual vehicle for a state-centered approach to causation, which underlies the proposed broad classification of causal relations and relations. For another, the full exploitation of CSC enables the functional perspective on causation (based on a solid theoretical foundation provided by the discussion on direct causal relations) to deal with the so-called problem of negative causation or absence causation in such a way that absence causation consists in bearing indirect causal relations to the effect in virtue of bearing a quasi-causal **maintains** relation (instead of direct causal ones) to a precondition for the effect (see Section 4.1.4).

One may wonder, taking into consideration the common claim that states are not inherently causally efficacious, whether the indirect (state-mediated) causal relations, **allows** and **disallows**, count reasonably as causal relations as well as the direct ones such as **achieves** and **prevents**. The incorporation of the relations that have as relatum states into the realm of causation is mainly motivated by the vital importance of those relations for an accurate description of what may be sometimes informally called ‘*causal chains* of various phenomena’ or the ‘*causal history* of the world’.

Causation is well worth investigating ontologically because it is fundamental to ordinary people’s (especially domain experts’) understanding of the course of states of affairs. Without indirect causal relations, one would face enormous difficulty in offering a full picture of (part of) the world. Spoken of within the CSC framework, this amounts to the underestimation of the causal role of the

precondition-for relation between a ‘mediating state’ (Z) and the effect (Y); and to the possession of a rather ‘low-resolution image’ of the world in which only discrete units of the **achieves** (and **prevents**) causal relations are observed, so to speak.

4.1.3. Functional square of causal relations

Interestingly enough, many causal examples discussed in the literature and extracted from our everyday life are explainable in terms of the sequence of direct causal relation (**achieves** and **prevents**) and *precondition-for* relations, to the best of the author’s knowledge. This motivates the conception of *indirect causal relations* as combinations of direct causal relations with precondition-for relations. In the terminology of this dissertation, positive and negative indirect causal relations are called ‘**allows**’ and ‘**disallows**’, respectively, and they are defined explicitly below. The direct/indirect and positive/negative features of the four kinds of causal relations are summarized in the table that may be called the ‘functional square of causal relations’ (see Table 1).

Table 1. The functional square of causal relations

	Positive	negative
Direct	Achieves	prevents
Indirect	Allows	disallows

I. *Allows as an indirect and positive causal relation*

The **allows** causal relation is defined as follows:

Definition 2 (Allows). An event or a process X **allows** an occurrent Y with respect to a systemic context C and its corresponding granularity G if and only if there exists a state Z such that either (i) X **achieves** or **maintains** Z with respect to C and G , and Z is a facilitative precondition for Y with respect to C and G ; or (ii) X **prevents** Z with respect to C and G , Z is a preventive precondition for Y with respect to C and G , and there exist no state W such that X **achieves** W with respect to C and G , and W is a preventive precondition for Y with respect to C and G .

As can be seen from the definition, **allows** is an indirect and positive causal relation. It is indirect because an occurrent can bear the **allows** causal relation to another occurrent only when some state acts as an intermediary between those two occurrents. It is also positive because its relata are actually occurring occurrents.

To illustrate this definition, consider the following examples. Note that the **maintains** case

will be explained in Section 6.4:

Example 6 (An unlocked door). Bill unlocked the door of his gallery and Mary went into the gallery.

Example 7 (Freezing ice cream). An ice cream froze because Williams put it into the refrigerator (extracted from Weber, 2008; with a relevant modification).

In Example 6, the event of Bill unlocking the door **allows** the event of Mary entering the gallery because it **achieves** the unlocked state of the door as a facilitative precondition for the event of Mary entering the gallery. In Example 7, the state of the ice cream being inside the refrigerator **allows** the event of the ice cream freezing because it **prevents** the state of the ice cream being above minus 18 degrees Celsius as a preventive condition for the ice cream freezing and there is no state to which the state of the ice cream being inside the refrigerator bears the **achieves** causal relation and which is a preventive precondition for the event of the ice cream freezing.

II. *Disallows as an indirect and negative causal relation*

The **disallows** causal relation is defined as follows:

Definition 3 (Disallows). An event or a process X **disallows** an occurrent Y with respect to a systemic context C and its corresponding granularity G if and only if there exists a state Z such that either (i) X **achieves** or **maintains** Z with respect to C and G , and Z is a preventive precondition for Y with respect to C and G ; or (ii) X **prevents** Z with respect to C and G , and Z is a facilitative precondition for Y with respect to C and G .

As the definition implies, **disallows** is an indirect and negative causal relation. It is indirect because an occurrent bears the **disallows** causal relation to another occurrent only when some state serves as a medium between those two occurrents. It is negative because its effect is a non-actual occurrent.

To illustrate this definition, consider the following examples. Note that the **maintains** case will be explained in Section 4.1.4:

Example 8 (Failed robbery). No robbery occurred because David locked the door of his house.

Example 9 (Non-melting ice cream). An ice cream did not melt because Williams put it into the refrigerator (extracted from Weber, 2008; with a relevant modification).

In Example 8, the event of David locking the door **disallows** the event of robbery because it **prevents**

the unlocked state of the door as a facilitative precondition for the event of robbery. In Example 9, the state of the ice cream being inside the refrigerator **disallows** the event of the ice cream melting because it **achieves** the state of the ice cream being below minus 18 degrees Celsius as a preventive condition for the ice cream melting. See Section 4.1.4 for details on absence causation.

4.1.4. Absence causation

Talk of absences as causes or effects is virtually omnipresent in scientific discourse. One ordinarily states, for instance, that the *absence* of vitamin C caused scurvy. The kind of causal cases that involve absences are loosely grouped under the heading ‘causation by disconnection’ (Schaffer, 2000), ‘negative causation’ (Schaffer, 2004), or ‘absence causation’ (Schaffer, 2005). Absence causation falls into two types. One type is *causation by absence* (aka causation by omission) where the absence is the cause; and the other is *causation of absence* (aka causation by prevention) where the absence is the effect.

It is a highly contentious subject whether absences serve as causal relata at all. It has been pointed out, for instance, that absence causation is rather troublesome for the kind of theory of causation (see e.g., Dowe, 2000, 2004) that posits a physical connection between causes and their effects (Schaffer, 2000, 2004, 2005). Absence causation poses a *prima facie* problem to the functional perspective on causation so far elaborated because the functional view takes causation to be based on a spatiotemporal relation between two occurrents. The state-centered approach to causation discussed above nonetheless helps us develop a strategy for dealing with absence causation, regardless of whether it is causation by absence or causation of absence.

I. *Causation of absence*

To examine causation of absence, consider first the less problematic case of ‘negative states’. Suppose that Bill unlocked the door of his gallery (recall Example 6 “An unlocked door”). The causal relation in the example is that the event of Bill unlocking the door of his gallery **achieves** the unlocked state of the door. It is nonetheless equally true that the same event **achieves** the not-locked state of the door. Strange as it may sound, the *not*-locked state of the door can be a causal relatum (especially the effect) because it is identical with the unlocked state of the door, but only given the systemic context and the granular level of reality behind the example.

Causation of absence is generally explicable in virtue of a state-centered approach to causation. In Example 5 (Election), for instance, it is intuitively acceptable to say that Trump’s victory brought about the absence of Clinton’s 45th U.S. presidency. To bring about the absence of Clinton’s 45th U.S. presidency means to **achieves** the state of the 45th U.S. president being not Clinton (and to **prevents** the state of the 45th U.S. president being Clinton, by the definition of the **prevents** causal

relation).

For a more paradigmatic case of causation of absence, it would be normal to say regarding Example 8 (Failed robbery) that David's action of locking the door of his house brought about the absence of robbery. Interpreted functionally, to bring about the absence of robbery is equivalent to the **achieves** causal relation to the locked state of the door as a preventive condition for the event of robbery (and to the **disallows** causal relation to the event of robbery, by its definition).

II. *Causation by absence*

Causation by absence is the more controversial case of absence causation. For it is rather unclear what it is supposed to mean to say that absences bring about something. There are various possible responses to causation by absence (see e.g., Mumford and Anjum, 2011: 144-146). Formulated in this dissertation is a proposal to interpret causation by absence in terms of the indirect causal relations (represented by CSC; see Figure 1) based on a quasi-causal **maintains** relation rather than direct causal ones. Moreover, virtually every case of causation by absence would be reducible to an instance of CSC.

To see this, consider the following examples:

Example 10 (Scurvy). Suzy contracted scurvy owing to her diet that is deficient in vitamin C (extracted from Schaffer, 2004; but with a relevant modification)

Example 11 (Examination). A student did not pass the examination because she did not study hard. (extracted from Weber, 2008; but with a relevant modification)

In Example 10, the process of Suzy's omission to take vitamin C from her diet **allows** the event of Suzy contracting scurvy because it **maintains** the state of the lack of vitamin C in Suzy's body as a facilitative precondition for the event of Suzy contracting scurvy. In Example 11, the event of the student's omission to study hard **disallows** the event of the student passing the examination because it **maintains** the state of the lack of knowledge in the student's head as a preventive precondition for the event of the student passing the examination.

Generally speaking, such typical cases of causation by absence as Examples 10 and 11 are interpretable in terms of the **maintains** version of CSC. Although it brings about nothing on its own, a non-actual event or process as omission (typically inaction) causally contributes to the effect in the sense that the omission performs the *function* to maintain a state which works as a precondition (whether facilitative or preventive) for the effect.⁸ Note that the combination of the context with a

⁸ See Kitamura, Sano, Namba and Mizoguchi (2002) for details on the function to maintain. It is interesting to note that the function to maintain is consistent with the device ontology view of reality. A device performs the

state-centered approach to causation enables the actualist to have a conceptual equivalence of those ‘omission occurrents’.

Consider two more examples:

Example 12 (A blind dog). Emily’s dog was bitten by an insect, contracting an eye disease as a result. Ignoring this, she did not take the dog to any animal hospital. Later the dog lost its sight. (extracted from Beebee, 2004; but with a relevant modification)

Example 13 (Plant). The fact that I did not give my houseplant any water caused its death. (Weber, 2008)

In Example 12, the event of Emily’s omission to take her dog to an animal hospital **allows** the event of the dog losing its sight because it **maintains** the state of the dog’s eyes not having been well treated as a facilitative precondition for the event of the dog losing his sight.

In Example 13, the state of the lack of water in my houseplant, which is incompatible with the state of my houseplant having enough water as a facilitative precondition for the growing process of my houseplant, so by Proposition 1, the state of the lack of water in my houseplant is a preventive precondition for the growing process of my houseplant.⁹ The event of my omission to water my houseplant **disallows** the growing process of the plant because it **maintains** the state of the lack of water in the plant as a preventive precondition for the growth of the plant.

Finally, it is interesting to note that some causal claims on causation by absence focus merely on precondition-for relations. Consider the following examples:

Example 14 (Scurvy: simpler case). An absence of vitamin C caused Suzy to contract scurvy (extracted from Schaffer, 2004; but with a slight modification)

Example 15 (Examination: simpler case). The lack of knowledge caused a student not to pass the examination. (extracted from Weber, 2008; but with a relevant modification)

In Example 14, the state of the lack of vitamin C in Suzy’s body is a facilitative precondition for the event of Suzy contracting scurvy. In Example 15, the state of the lack of knowledge in the student’s head is a preventive precondition for the event of the student passing the examination.

In the author’s view, most alleged examples of absence causation are to be interpreted in

function to maintain in the sense of keeping the state of the operand when output from the device is the same as (i.e., qualitatively identical to) the state of the operand when input into the device. In the case of Example 9 (Scurvy), Suzy’s body can be a device that performs the function to maintain: to retain the amount of vitamin C (operand) that is ‘processed’ by her body, irrespective of her diet.

⁹ It is important to remark that, *given the context that the causal relation of this example necessitates*, nobody else than I could water my houseplant.

terms of a combination of the functional square of causal relations (**allows** and **disallows**, especially in the case of causation by absence) with a state-centered approach to causation, which enables absences in causal talk to be well treated with (the extension of) CSC. It must be emphasized that (the extension of) indirect causal relations, **allows** and **disallows**, are explanatory forceful enough to deal with absence causation, primarily because of their dependence on the conceptually robust, precondition-for relations, which are in turn deeply rooted in the notion of incompatibility which is fundamentally based on the context that the causal relation *ontologically necessitates*.

4.1.5. False causal examples

This section further clarifies how the actualist's treatment of causation can be strengthened through the explication of the context and a state-centered approach to causation, by showing how the putative counterexamples to the counterfactual theory of causation (see Section 2.4) are to be interpreted from a functional perspective. All the examples herein presented are *pseudo-examples*: they are *assumed* to make false causal statements. Their presupposed falsity is explained in terms of indirect causal relations (CSC; Figure 1).

Example 16 (Terrorist: pseudo-example). I caused the terrorist attack in London by failing to be in a pub where I could have overheard the plot, and by failing to travel to the UK and blow up the terrorist's van. (Dowe, 2004)

Assuming that this is the case, "the event of my omission to be in the pub **allows** the event of the terrorist attack because it **maintains** the state of there being no preventive action of mine (e.g., to travel to the UK and blow up the terrorist's van) against the terrorist attack as a facilitative precondition for the terrorist attack. The state of there being no preventive action of mine against the terrorist attack is a facilitative precondition for the terrorist attack because of Proposition 2: the state of there being no preventive action of mine against the terrorist attack is incompatible with the state of there being some preventive action against the terrorist attack, which is in turn a preventive precondition for the terrorist attack."

Seen from a functional perspective, the falsity of the example stems from the falsity of the incompatibility under consideration, *given the context which the false causal relation necessitates*, because someone else (e.g., my friend) could take some preventive action against the terrorist attack.

Example 17 (Rock: pseudo-example). My not throwing a rock causes the window not to break. (Dowe, 2004)

Assuming that this is the case, "the event of my omission to throw a rock **disallows** the event of

the window breaking because it **maintains** the state of the window being not impacted by my throwing a rock as a preventive precondition for the window breaking. This is because of Proposition 1: the state of the window being not impacted by my throwing a rock is incompatible with the state of the window being impacted, which is in turn a facilitative precondition for the window breaking.”

Seen from a functional perspective, the falsity of the example stems from the falsity of the incompatibility under consideration, *given the context which the false causal relation necessitates*, because the window could be impacted by someone else (e.g., my brother) throwing a rock.

Example 18 (Plant: pseudo-example). The fact that you did not give my houseplant any water is the cause of its death. (Dowe, 2004)

Assuming that this is the case, “the event of your omission to water my houseplant **disallows** the growing process of my houseplant because it **maintains** the state of my houseplant not being watered by you as a preventive precondition for the growth of my houseplant. This is because of Proposition 1: the state of my houseplant not being watered by you is incompatible with the state of my houseplant having enough water, which is in turn a facilitative precondition for the growth of my houseplant.”

Seen from a functional perspective, the falsity of the example stems from the falsity of the incompatibility under consideration, *given the context which the false causal relation necessitates*, because someone else (e.g., my neighbor) than you and I could water my houseplant.

4.1.6. Extending CSC to more complex causal chains

As implied above, CSC (Figure 1) aims to be the first attempt to provide a full description of causal chains. The extensive usage of CSC would therefore take the further step towards the goal. The basic principle behind CSC is to characterize indirect causal relations in terms of the combination of *direct* causal relations and precondition-for relations. This idea can be extended to the combination of *indirect* causal relations and precondition-for relations in such a way that, e.g., the **allows** causal relation holds in virtue of either a coupling of the **allows** causal relation and the facilitative precondition or a coupling of the **disallows** relation and the preventive precondition.

The usage of CSC can be extended along this line of thought so that one instance (say CSC_m) of CSC can be ‘nested’ into another (say CSC_n) in such a way that positive (negative) indirect causal relation in CSC_m take the place of positive (negative) direct causal relations in CSC_n (see Figure 2 for a specific instance of this structure; see also Example 20). Therefore the extended usage of CSC has some kind of recursive feature: yet another instance of CSC, say CSC_l , can be ‘nested’ into CSC_m . In

this way, the extended, ‘nested’ usage of CSC serves as a conceptual tool for facilitative the reasoning about complex causal chains.

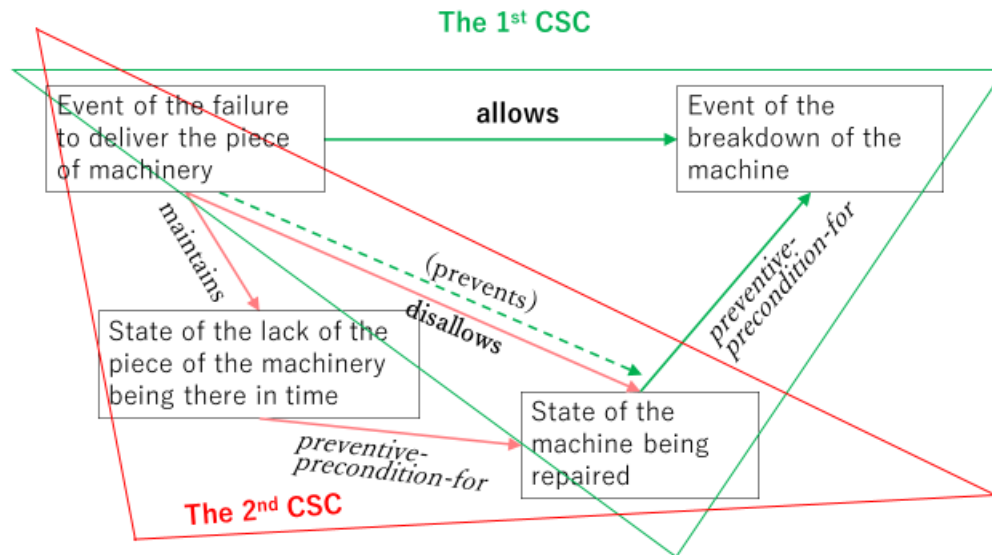


Fig. 2. An instance of the extended, ‘nested’ usage of CSC. The bracket refers to the direct causal relation that is replaced by the indirect one with respect to an extensive usage of CSC. (The same applies to all the other following figures.)

To illustrate this, consider the following example:

Example 19 (A child’s survival). A father pushing his child out of the way of a speeding car saved the child’s life (i.e. causes it not to die). (Weber, 2008)

One may want to think, with the standard usage of CSC, that the event of the father pushing his child out of the way of a speeding car **allows** the event of the child’s survival because it **prevents** the state of the child being hit by the car as a preventive precondition for the child’s survival. It is accurately found, however, that the event of the father pushing his child out of the way of a speeding car **disallows** (but not **prevents**) the state of the child being hit by the car because it **prevents** the state of the child being in the way of the speeding car as a facilitative precondition for the child being hit by the car.

The extended usage of CSC enables the second CSC to be ‘nested’ into the first one as follows: the event of the father pushing his child out of the way of a speeding car **allows** the event of the child’s survival because it **disallows** the state of the child being hit by the car as a preventive

precondition for the child's survival (see Figure 3). That is to say, the extended, 'nested' usage of CSC solves the problem of the failure of its standard usage to express the correct causal relation, by replacing coherently the **prevents** causal relation therein by the **disallows** causal relation, i.e. the indirect causal relation that has the same causal ground (causal negativeness) as the **prevents** causal relation does.

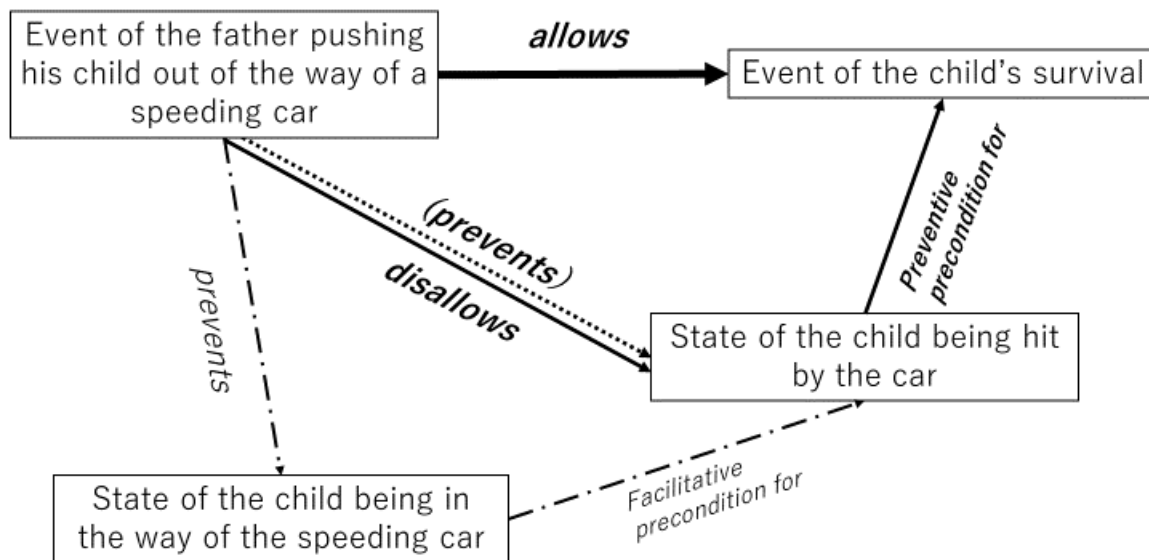


Fig. 3. The extended, 'nested' usage of CSC in Example 19

A few more examples are presented below for an illustrative purpose, together with their simple explanations and their extended, 'nested' usages of CSC:

Example 20 (Machine). The failure of delivering a piece of machinery in time causes a machine to break down. (Schaffer, 2004)

The **prevents** causal relation in the standard form of CSC is, in a 'nested' manner, replaced by the **disallows** causal relation between the event of the failure to deliver the piece of machinery (which the bears **maintains** quasi-causal relation) and the state of the machine being repaired. See Figure 2 for details.

Example 21 (A child's death). A father not pushing his child out of the way of a speeding a car is the cause of its death. (Weber, 2008)

The **achieves** causal relation in the standard form of CSC is, in a 'nested' manner, replaced by the **allows** causal relation between the event of the father not pushing his child (which bears the

maintains quasi-causal relation) and the state of the child being in the way of the speeding car. See Figure 4 for details.

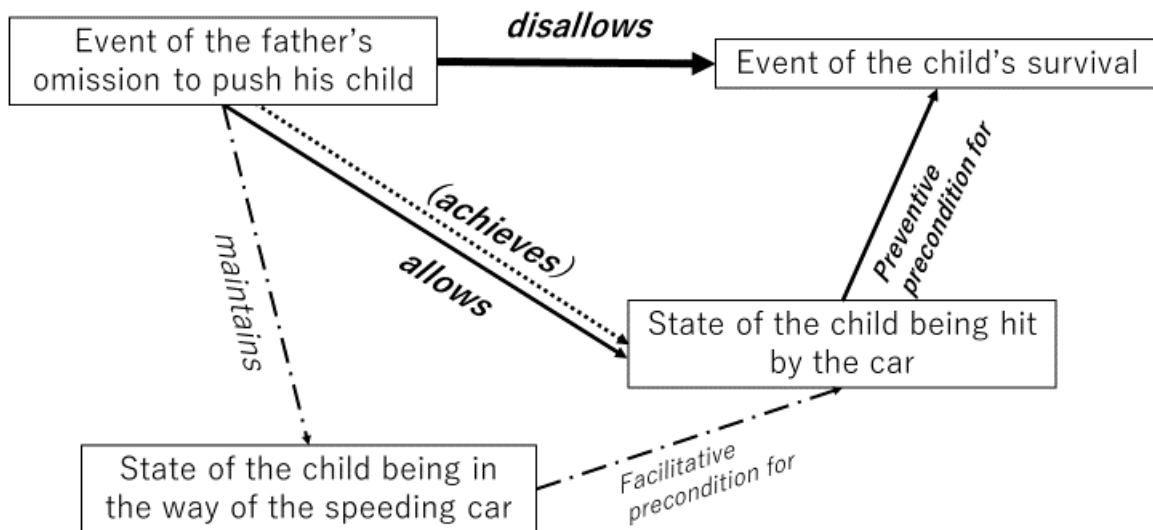


Fig. 4. The extended, 'nested' usage of CSC in Example 21

Example 22 (Terrorist). I caused the terrorist attack in London by failing to report information that I had about it. (Dowe, 2004)

The **prevents** causal relation in the standard form of CSC is, in a 'nested' manner, replaced by the **disallows** causal relation between the event of my omission to report relevant information about the terrorist attack (which bears the **maintains** quasi-causal relation) and the event of the police arresting the terrorist. See Figure 5 for details.

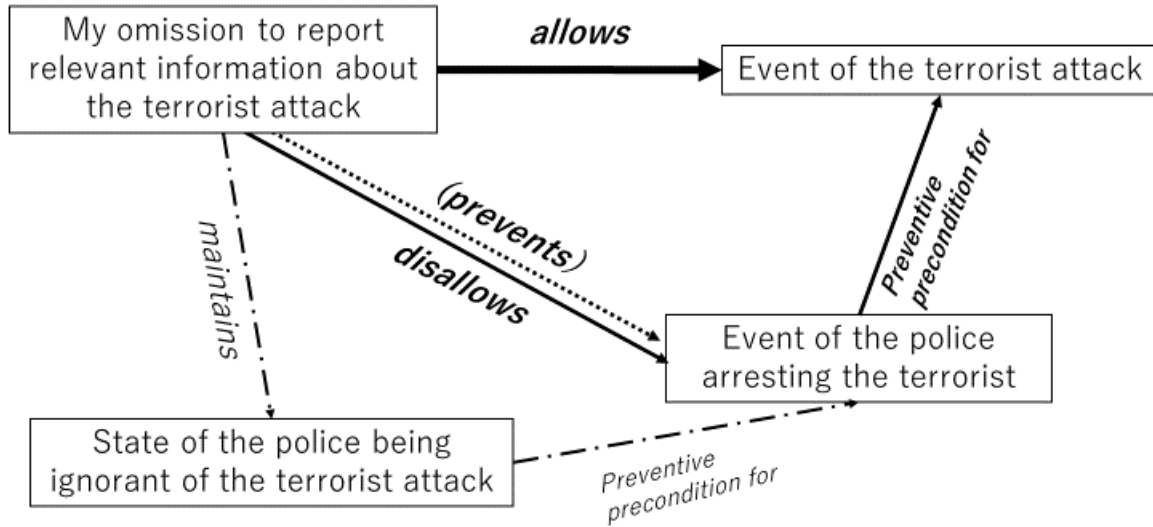


Fig. 5. The extended, 'nested' usage of CSC in Example 22

4.2. Preliminary formalization

This section presents a preliminary formalization of the four kinds of causal relations in the functional square (with the exception of the extensive usage of CSC). It should be emphasized that the core of the functional perspective on causation has been provisionally formalized as an initial step towards its available implementation in information systems. For instance, a rigorous formalization of the seemingly modal notion of incompatibility (found in the definition of a preventive precondition) might require some form of the modal possibility operator $\Diamond$. To avoid further logical complications, however, the formalization here has been developed in a first-order predicate language.

All the variables introduced here must be taken to represent particulars, but not universals, because token-level causation is the primary focus of the dissertation. In addition, small letter (' x ') and capital letters (' X ') are used to refer to actual and non-actual occurrents, respectively, so that it will be formally clear that the actualist's treatment of causation could be made as expressive as the non-actualist's by the conceptual tools of the functional perspective on causation of this dissertation: the context and a state-centered approach to causation (but see Section 7 for future development of the formalization). In particular, capital letters may seem to refer to particular non-actual occurrents, but given the actualist's usage of the incompatibility condition, they are merely *mentioned* rather than *used*; and the actualist can speak of them without any serious ontological commitment to them.

First off, the arguments of **achieves** causal relations are restricted as follows:

$$\begin{aligned} achieves(x, y) \rightarrow & (Event(x) \vee Process(x) \vee State(x)) \wedge \\ & (Process(y) \vee State(y)) \wedge \neg(Process(x) \wedge State(y)) \end{aligned}$$

$$prevents(x, Y) \triangleq \exists z (State(z) \wedge incompatible_with(z, Y) \wedge achieves(x, z))$$

$$\begin{aligned} prevents(x, Y) \rightarrow & (Event(x) \vee Process(x) \vee State(x)) \wedge \\ & (Process(Y) \vee State(Y)) \wedge \neg(Process(x) \wedge State(Y)) \end{aligned}$$

The arguments of both kinds of precondition-for relation are restricted as follows. For the sake of simplicity, the authors use the notation ‘ x/X ’ to mean an occurrent, regardless of whether it is actual or not:

$$\begin{aligned} facilitative_precondition_for(z, y/Y) \rightarrow & (State(z) \wedge \\ & (Event(y/Y) \vee Process(y/Y) \vee State(y/Y))) \end{aligned}$$

$$\begin{aligned} preventive_precondition_for(z, y/Y) \rightarrow & (State(z) \wedge \\ & (Event(y/Y) \vee Process(y/Y) \vee State(y/Y))) \end{aligned}$$

Although those two kinds of precondition-for relations are primitives, their fundamental features are clearly specified in terms of Propositions 1 and 2 in Section 4.1.1, resulting in the following:

$$\begin{aligned} incompatible_with(z, z') \wedge preventive_precondition_for(z', y/Y) \\ \rightarrow facilitative_precondition_for(z, y/Y) \end{aligned}$$

$$\begin{aligned} incompatible_with(z, z') \wedge facilitative_precondition_for(z', y/Y) \\ \rightarrow preventive_precondition_for(z, y/Y) \end{aligned}$$

The **allows** causal relation is defined and its arguments are restricted as follows. Note that **maintains** is a primitive predicate:

$$\begin{aligned} allows(x, y) \triangleq & \exists z/Z (achieves(x, z) \wedge facilitative_precondition_for(z, y) \\ & \vee (prevents(x, Z) \wedge preventive_precondition_for(Z, y) \\ & \wedge \neg \exists w (State(w) \wedge achieves(x, w) \wedge preventive_precondition_for(w, y))) \end{aligned}$$

$$allows(x, y) \rightarrow (Event(x) \vee Process(x)) \wedge (Event(y) \vee Process(y) \vee State(y))^{10}$$

$$allows(X, y) \rightarrow \triangleq \exists z((Event(X) \vee Process(X)) \wedge maintain(X, z) \\ \wedge facilitative_precondition_for(z, y))$$

Finally, the **disallows** causal relation is defined and its arguments are restricted as follows:

$$disallows(x, Y) \triangleq \exists z/Z(achieves(x, z) \wedge preventive_precondition_for(z, Y)) \\ \vee \exists Z(prevents(x, Z) \wedge facilitative_precondition_for(Z, Y))$$

$$disallows(x, Y) \rightarrow (Event(x) \vee Process(x)) \wedge (Event(Y) \vee Process(Y) \vee State(Y))$$

$$disallows(X, Y) \rightarrow \triangleq \exists z((Event(X) \vee Process(X)) \wedge maintain(X, z) \\ \wedge preventive_precondition_for(z, Y))^{11}$$

¹⁰ Note that *facilitative_precondition_for* is outside the scope of this formula.

¹¹ One may worry that the current formalization has to quantify over possible future events. It could be avoided if the idea of (non-)instantiated occurrent universals is introduced, although type-level occurrents fall outside the scope of the dissertation (see Section 3.1.4). The author thanks to Ludger Jansen for pointing out this problem and suggesting a possible solution.

5. Application: Causal evolution of the River Flow Model of diseases

5.1. Background and scope

Biomedicine is nowadays witnessing an unprecedented increasing amount of disease-related data and information, as is indicated by the emerging discipline called ‘biomedical informatics’ or ‘bioinformatics’. There is accordingly a growing demand for a common semantic framework in which many pieces of biomedical information are sharable among different information systems (e.g., databases) in order to maximize opportunities for medical practitioners to acquire medical knowledge and improve their clinical decisions. To surmount this difficulty of semantic interoperability has been nonetheless a considerable challenge, partly owing to the lack of broad consensus among biomedical experts on some key concepts: e.g., health, disease, and aging.

The long-term goal of health care and maintenance would not be achieved unless a thorough conceptual approach to those biomedically relevant notions is taken with the utmost seriousness. For instance, Stange (2009) reports that the global malfunctioning of the health care system is partly due to the problem of fragmentation: “focusing and acting on the parts without adequately appreciating their relation to the evolving whole.” Health improvement has been engaged in by a number of different people, ranging from health professionals to policy makers; and the disintegration of the healthcare system yields the failure to treat patients properly. The reduction of fragmentation would therefore constitute an initial step towards a solution to numerous healthcare problems. One of the most effective ways to address the problem of fragmentation is arguably to have a shared understanding of healthcare-related vital concepts in such a way that they are closely examined under the umbrella of the same theoretically well-founded notion; otherwise, for instance, health and disease would have undesirably no common semantic foundation, hence the remaining issue of fragmentation.

The concept of disease has been investigated with a central focus on causation because causation may well be seen as a common thread running through multifarious biomedical notions. A close relationship between disease and causation would be intuitively understood in actual clinical practice. To cure a certain disease properly, medical specialists must know why that disease emerged by examining the patient’s body and inquiring his/her medical history. This amounts to the exploration of the cause(s) of the disease in question; and causal inference plays a crucial role in bioinformatics (Kleinberg and Hripacsak, 2011). An operational conceptualization of disease thus needs to form the causal basis for medical practitioners’ causal reasoning about disease. Herein lies the problem of figuring out an inextricable connection between disease and causation.

There is limited scope for the dissertation to deal with disease and causation because they have been both too intensively researched to be discussed exhaustively in a single paper. In particular, three caveats will be given regarding disease. First, the main focus will be on a general notion of disease,

but not on any specific diseases, viz. cancer, pneumonia, and diabetes. Certainly discussions on those particular diseases would require fairly specialized knowledge and experience of them, which would lie outside the realm of the author's expertise. By contrast, the generic notion of disease can be investigated relatively independently of disease-related domain knowledge.

Second, the current approach to disease could have a close affinity with but nonetheless differs in some important respects from philosophy of medicine, which pivots around the debate about the concepts of health and disease between the naturalist, normativist, and hybrid accounts of them (but see Lemoine, 2013 for criticism of their conceptual analytic approach). Naturalism offers a value-free analysis of it by taking bodily dysfunction to be a sufficient condition for disease (Boorse, 1975, 1997). Normativism argues for the determination of the harmfulness of disease by social values (Reznek, 1987). Hybridism thinks of bodily dysfunction as a necessary, but not sufficient, condition for disease (Wakefield, 1992, 2014). Philosophers of medicine generally examine the nature of clinical abnormality of disease, or what kind of standard disease consists in deviating from. In contrast, the dissertation will proceed while taking the notion of clinical abnormality as primitive. This is mainly because different criteria for clinical abnormality depend so heavily on different medical fields and professionals that it would be impracticable to seek a single universal definition of clinical abnormality, although it may be well worth philosophical investigation (see Williams, 2007 for a similar line of research).

Third, discussions hinge upon the ontological notion of causation as such in biomedicine, but not the epistemic notion of causal inference or causal reasoning, although the former may have a downstream effect on the latter (see Section 5.5 for details). One may be indeed inclined to put more focus on causal inference in analyzing causation in biomedicine-related fields such as bioinformatics (Kleinberg and Hripcsak, 2011) and public health (Glass, Goodman, Hernán and Samet, 2013). For instance, Russo and Williamson (2007) point out two types of causal evidence in the health sciences: the probabilistic evidence (consisting mainly of observed dependencies in a range of similar studies) and the mechanical evidence (to be used to explain physical phenomena mechanically). They contend that the unification of both the mechanistic and probabilistic aspects of the health sciences can be achieved by the epistemic theory of causation: causal relationships are to be understood in terms of rational beliefs, or the causal beliefs of an (ideally) omniscient rational belief. It is equally true however that an ontological analysis of causation should not be underestimated in biomedicine; rather, prevailing epistemic approaches to causation must be supplemented with its deep ontological understanding to make a substantial contribution to evidence-based practice for healthcare (Kerry, Eriksen, Lie, Mumford and Anjum, 2012; Anjum, Kerry and Mumford, 2015).

5.2. The River Flow Model (RFM) of diseases

There is a growing amount of information and data about diseases, and it is highly desirable that they be used to enhance our scientific knowledge. However, such information is often collected and stored to meet local needs in the context of specific applications, so that potential benefits of data integration are lost. In biomedical ontology research, a disease ontology has to provide a common semantic framework which would facilitate the annotation of biological and biomedical datasets, and a proper understanding of disease is indispensable for the robust construction of disease ontologies (Bodenreider and Burgun, 2009). Disease nevertheless remains an elusive notion from an ontological viewpoint, and what is needed is an ontologically informed account of disease that can aid in the practical building of disease ontologies.

The River Flow Model (RFM) of diseases was proposed against the YAMATO ontological background as a clinician-friendly general account of disease (Mizoguchi et al., 2011). The basic tenet of the RFM is the analogy between a river and a disease. Just as a river enacts changing the course of the flow of water as its external process, a disease enacts as its external process a process of, e.g., spreading and disappearing. While a river is an independent continuant, however, a disease is a dependent continuant: it depends on an organism as its bearer. Moreover, just as a river has the flowing of water as its internal process (a process that occurs inside the river), a disease has as its internal process a number of chains of causal phenomena. A disease is in this respect *constituted of* causal chains of phenomena that are detrimental to the organism from a medical viewpoint. It is interesting to note that this constituted-of relation has a close affinity with the above-introduced constitution relation of a process to the event.

All these considerations lead to the RFM definition of disease as “a dependent continuant constituted of one or more causal chains of clinical disorders appearing in a human body and initiated by at least one disorder” (ibid.).¹² The term ‘clinical disorder’ initially shared its meaning with the OGMS conception of disorder. Since its active practical application, however, the RFM has regarded disease primarily as a dependent continuant constituted of causal chains of *abnormal states* (Yamagata, Kozaki, Imai, Ohe and Mizoguchi, 2014; Kozaki, Yamagata, Mizoguchi, Imai and Ohe, 2017). At first, a state therein was a time-indexed property (Yamagata et al., 2014; Kozaki et al., 2017), but later it is interpreted as an occurrent in the YAMATO fashion through theoretical sophistication of the RFM (Rovetto and Mizoguchi, 2015; Toyoshima, Mizoguchi and Ikeda, 2017). For instance, diabetes is a dependent continuant whose causal chains have as part the causal relation between the state of the deficiency of insulin and the state of the elevated level of glucose in the blood.

Another distinctive aspect of the RFM is its supplementation with the imbalance model

¹² The category of dependent continuant which is referred to in the dissertation corresponds to the YAMATO category of *specifically dependent entity*.

(Mizoguchi et al., 2011) according to which a clinically normal organism maintains homeostasis, which basically means a disposition of an organism to regulate its body in alignment with the OGMS (Scheuermann, Ceusters and Smith, 2009), when the ‘supply’ and the ‘demand’ are well-balanced as regards all the parameters that are relevant to the organism’s living condition. In a clinically abnormal organism, however, the supply and the demand for some (if not every) parameter that relates to the organism’s life are so different from each other that the difference between them lies outside the clinically permissible range for the maintenance of the organism’s homeostasis.

To illustrate the imbalance model, consider diabetes in general. In a patient with diabetes (whether Type I diabetes or steroid diabetes), the required amount of insulin (demand) exceeds to a clinically abnormal degree the amount available for working insulin (supply) and this imbalance state causes the state of being at an elevated level of glucose in the blood, which may result in the loss of sight of the patient over a long period of time. The difference between Type I diabetes and steroid diabetes resides partly in the fact that, in the case of Type I diabetes and steroid diabetes, causal chains that lead to the imbalance state described above include the state of having depleted pancreatic beta cells and the state of having large quantity of steroids, respectively. A clinical imbalance state is thus generally characterized as follows: a state of an organism such that, given a parameter p that is relevant to the organism, the mismatch between the supply and the demand that are specified with respect to p falls outside a clinically normal range for the organism’s homeostasis.

5.3. Restating the RFM definition of disease

Causation or causal relations has long been a focus of attention in the bioinformatical domain (Kleinberg and Hripsak, 2011; Agibetov et al., 2018), and the closest and most important related work is arguably Rovetto and Mizoguchi (2015) whose goal is to clarify the RFM with an emphasis on the concept of causation in disease ontology and knowledge representation. Their work has its limitations, although it would serve to highlight the relevance of causation to ontological accounts of disease. For one thing, they attempt to develop a formal representation of disease while taking the notion of causal chain to be primitive, and the causal character of the RFM notion of disease is still relatively unspecified.

For example, Rovetto and Mizoguchi (2015) provide two revised RFM definitions of disease, the first of which is: “An abnormal causal structure constituted of one or more causal chains of abnormal states located (or occurring) in an organism, and initiated by at least one abnormal state” (ibid.: 94, with some notational modifications for readability). An abnormal causal structure is a type of a *causal structure*, which is in turn a type of YAMATO-specifically dependent entity (dependent continuant). A causal structure is constituted of occurrents, specially types of *sequence of occurrents* such as *causally-linked occurrents* (ibid.: 92). This definition remains unspecified from a causal point

of view, as is witnessed by its dependence on the terms ‘causal structure’ and ‘causal chains’.¹³

The RFM description of disease can be arguably better ameliorated through the functional perspective on causation. For one thing, Rovetto and Mizoguchi (2015) endeavor to update the RFM definition in accordance with categories found in the YAMATO upper ontology, on which the functional view of causation can be well-founded. For another, as will be detailed below, the RFM central idea of disease as causal chains of abnormal states would receive further elucidation when the causal nature of abnormal states is looked from a functional standpoint. Recall a functional classification of causal relations (**achieves**, **prevents**, **allows**, and **disallows**) and causal relata (events, processes, and states). The RFM definition of disease can be now recast using the functional perspective on causation as follows: *a disease is a dependent continuant constituted of abnormal states (i) to which events, processes, and states bear either the **achieves**, **prevents**, **allows**, or **disallows** causal relation and (ii) initiated by at least one abnormal state.*

Some cautionary remarks are needed in order for this restatement to be properly understood. First, causal chains are, in this novel RFM characterization of disease, no longer taken to be as primitive as before notwithstanding its apparent usage of the term ‘causal relation’. Second, a disease must have at least one clinical imbalance state, although this is traditionally implicit in the RFM definition as such despite its great importance. Third, as was mentioned above, the RFM conception of clinical abnormality is virtually unanalyzable. This is vindicated through the sharp distinction between the domain-neutral notion of clinical abnormality and the domain-specific task of the identification of clinical abnormality. What counts as clinically abnormal would vary from clinician to clinician in a broad biomedical field. The RFM claims to be an ontological (domain-neutral) model of diseases and the RFM notion of clinical abnormality refers to the *existence* (rather than the content) of criteria for observing a state from a clinical perspective. For a practical application of the RFM understanding of abnormal states, see Yamagata et al. (2014) and Kozaki et al. (2017).

Moreover, the combination of the functional view of causation with the RFM would aid in clarifying the emergence of a disease. Since the event-state pair is a canonical pattern of causation (precisely: the **achieves** causal relation), it is reasonable to think that an initial abnormal state of a disease of an organism is typically caused by at least an event that is external to the organism’s body. One paradigmatic example would be that a person (say Mary) fell and fractured her skull. In this case, the event of Mary falling **achieves** an initial abnormal state of the fracture of her skull: e.g., an imbalance state characterized by the asymmetry between external pressure and resistance to it.

¹³ The same criticism applies to Rovetto and Mizoguchi’s (2015: 94) second revised RFM definition of disease, although it purports to match more closely the YAMATO categories and relations than the first: “An abnormal causal structure that inherits a causal structure, which is constituted of some causally-linked occurrents, and causal structure is located (or occurs) in an organism, and causally-linked occurrents has part (or is initiated by) at least one abnormal state.”

5.4. Illustrative examples

This section illustrates with several examples (some of which appeared above) the application of the functional perspective on causation to the RFM conceptualization of disease.

A) *Thrombosis.*

The causal mechanism involved in thrombosis was discussed through a meticulous analysis of Example 4 (Thrombosis). As said, (i) the growing process of a blood clot in a blood vessel **achieves** the state of being small of the cross-sectional area of the blood vessel; (ii) the state of being small of the cross-sectional area of the blood vessel **achieves** the state of being low of the flow rate of blood; and (iii) the state of being low of the flow rate of blood **achieves** the state of being in short supply of oxygen.

B) *Scurvy*

The causal character of scurvy was scrutinized by means of careful study on Example 10 (Scurvy) and Example 14 (Scurvy: simpler case). That is to say, assuming that Suzy contracted scurvy, the process of Suzy's omission to take vitamin C from her diet **allows** the event of Suzy contracting scurvy because it **maintains** the state of the lack of vitamin C in Suzy's body, which is a facilitative precondition for the event of Suzy contracting scurvy.

C) *Boerhaave syndrome*

People with Boerhaave syndrome tend to have their esophaguses (the tube through which food passes from the mouth to the stomach) get ruptured (suddenly, for instance).

Analysis. The event of the rupture of the patient's (say Nancy) esophagus **achieves** the state of Nancy's esophagus being torn. This represents a paradigmatic disease-related causal phenomenon since it has the event-state causal relata. Notice that one can speak of this case using the **prevents** causal relation: the event of the rupture of Nancy's esophagus **prevents** the (non-actual) state of Nancy's esophagus being unholed, which is assumed to be incompatible with the state of Nancy's esophagus being torn.

D) *Ketoacidosis coma*

Ketoacidosis refers to acidosis (roughly, a decrease in pH) accompanied by the accumulation of ketone bodies in the body tissues and fluids. People with ketoacidosis are prone to go into a coma, in particular when they become dehydrated.

Analysis. The event of a person (say Bob) contracting ketoacidosis **allows** the event of Bob going into a coma because it **achieves** the state of being at an elevated level of glucose in Bob's blood,

which is a facilitative precondition for the effect.

Note that one can paraphrase this coupling of the **achieves** causal relation with a facilitative precondition in a ‘doubly negative’ way. That is, the event of Bob contracting ketoacidosis **prevents** the state of being at a normal level of glucose in Bob’s blood, which is a *preventive* precondition for the effect. Quite importantly, however, this is the case on the condition that there is no preventive precondition for the effect that is satisfied (**achieves**) by the event of Bob contracting ketoacidosis.

Lastly, since the RFM aims to promote therapeutic treatment of disease as well, medical prevention of a particular disease is illustrated as follows along with the RFM:

E) *Pellagra*

Pellagra is a disease due to a cellular deficiency of niacin (a vitamin of the B group that is found in foods such as milk and meat), and it is manifested by characteristic dermatitis (a skin condition in which the skin becomes red, swollen and sore) on areas of the skin that are exposed to the sun.¹⁴

Analysis. The process of a person’s (say John’s) dietary intake of abundant niacin **disallows** the event of John contracting dermatitis because it **prevents** the state of John having pellagra, which is a facilitative precondition for the effect.

5.5. Functional supports for causal inference in biomedicine

As was alluded to above, causal inference plays a vital role in biomedicine because clinicians and medical professionals are generally interested in how to extrapolate valid causal inferences from the observational data that they collect and analyze (Martin, 2014). Certainly the RFM has previously contributed to the effective deployment of biomedical experts’ causal knowledge, as evidenced by the development of Disease Compass: a navigation system for disease knowledge based on the RFM (Kozaki et al., 2017). It nonetheless remained unclear whether and how the RFM causal understanding of disease *per se* (rather than its application ontologies) is of benefit to causal reasoning in biomedical practice. The application of the functional view of causation to the RFM would help to bolster a sound argument for the practicality of the RFM with respect to causal reasoning.

For one thing, Kleinberg and Hripacsak (2011) hold that graphical models and Granger’s (1980) conception of causation provide useful frameworks for causal inference and explanation in bioinformatics. Being initially tailored for economics, Granger’s theory of causation consists in an

¹⁴ Tryptophan (a type of amino acid) is also nowadays referred to as a cause of pellagra, but it is presently omitted to be discussed for the sake of simplicity.

evaluation of the statistical significance of the relationship between two time series. Albeit a convenient hypothesis test, Granger causation is generally acknowledged to be severely defective with respect to bioinformaticians' more rigorous treatment of causation. To take one example, Granger causes may be suitable for prediction, but cannot be used for explanation or policy-making. The RFM presently fits well with a graphical representation of causal relationships, whose veracity would be higher in virtue of their functional (ontological) interpretation than Granger causation exhibits. Therefore, the RFM would provide a solid foundation for (instead of serving directly as) an efficient causal reasoning engine in biomedicine: e.g., an algorithm for determining under which bundle of pathological conditions a given disease begins to develop, irrespective of whether an effect is caused by a single factor or equally important multiple ones. For another, the functional approach to causation is designed to capture the counterfactual aspects of causation that are pertinent to causal inference in biomedicine (Martin, 2014; Strand and Parkkinen, 2014) because it comprises the notion of precondition, which is intimately linked with counterfactuality and which is used to define indirect causal relations, or especially the **allows** causal relation.

To be more concrete, consider causal reasoning in epidemiology. As with biomedicine in general, causation serves as one of the epidemiologists' most important conceptual tools in such a way that their causal inference is 'an exercise in measurement of an effect' (Rothman and Greenland, 2005) and a refined understanding of causation contributes to epidemiological practice (Maldonado, 2013; Glymour and Rudolph, 2016). There are many works on causal reasoning in epidemiology; and *inter alia*, Vandenbroucke, Broadbent and Pearce (2016) criticize an increasingly popular method for conceptualizing causation in epidemiology which they call the 'Restricted Potential Outcomes Approach' (RPOA). The RPOA falls, broadly speaking, into a loose group of interventionist approaches to causation (Woodward, 2003) according to which x is a cause of y if and only if there is a possible intervention on x that changes y . The RPOA faces some problems with regard to the assessment of causation in epidemiology, as they say. For one thing, the RPOA has trouble in dealing with the widespread concept of state in traditional epidemiological practice. For another, the RPOA provides no explicit way of overcoming the central epidemiological challenge of 'using different kinds of evidence to arrive at one overall verdict' because it fails to promote, e.g., 'interlocking of evidence': the convergence on a particular finding, of numerous pieces of evidence from a wide array of fields, including epidemiology. It is interesting to note that the RPOA may fare badly with respect to causal inference in public health as well (Glass, Goodman, Hernán and Samet, 2013).

The functional theory of causation may circumvent the difficulties with RPOA and thereby be utilized as the epidemiologist's toolbox, partly because it is capable of accommodating the notion of state in virtue of its state-centered approach to causation, partly because its causal identification can cut across various domains, as shown by the extension of CSC to more complex causal chains.

In contrast, Vandenbroucke et al. (2016) themselves suggest pragmatic pluralism: ‘a combination of quietism about the nature of causation, and pluralism about causal concepts’. The functional account would be more preferable to pragmatic pluralism because it furnishes functional elucidation of causation (the **achieves** causal relation) and elaborates upon an effective method (in particular: the **allows** causal relation) for representing epidemiologists’ common understanding of causation.

5.6. The Ontology for General Medical Science (OGMS)

One of the most influential general models of disease is arguably the one that is provided by the Ontology for General Medical Science (OGMS) (Scheuermann, Ceusters and Smith, 2009). The OGMS is designed to represent the entities that are involved in a clinical encounter in compliance with the Open Biomedical Ontologies (OBO) Foundry (Smith et al., 2007). The OBO foundry is a collaborative project to coordinate ontologies to support biomedical data integration and it adopts BFO as the standard upper ontology. One of the salient features of BFO is its approval of Smith and Ceusters’s (2010) methodology of ontological realism: “(...) the most effective way to ensure mutual consistency of ontologies over time and to ensure that ontologies are maintained in such a way as to keep pace with advances in empirical research is to view ontologies as representations of the reality that is described by science. This is the *fundamental principle* of ontological realism” (ibid.: 139; but see Merrill, 2010a, 2010b for criticism). As the realist methodology goes, for instance, ontologies can represent electrons and cells, but not unicorns.

The OGMS model of disease hinges upon the BFO category of dispositions. A disposition is a dependent continuant that exists because of certain features of the physical make-up (*material basis*) of the independent continuant (*bearer*) in which it inheres and whose instances can be realized in associated processes of specific correlated types in which the bearer participates (see for more thoughts Röhl and Jansen, 2011; Barton, Grenier, Jansen and Ethier, 2018). A process therein is the BFO category: an occurrent “that exists in time by occurring or happening, has temporal parts, and always depends on at least one independent continuant as participant” (Arp, Smith and Spear, 2015: 183). Classical examples of dispositions include fragility (the disposition to break when pressed with a certain force), solubility (the disposition to dissolve when put in a certain solvent), and flammability (the disposition to ignite when met with a certain heat source); and more specifically, fragility of a glass is the disposition of the glass (bearer) to break (realization) that depends on a particular physical molecule structure (material basis) of the glass. Characteristically, dispositions may exist even if they are not realized or even triggered. A glass is fragile even if it never breaks or even if it never undergoes any shock, for instance.

To introduce the OGMS dispositional account of disease, some core terms of the OGMS are presented: a disorder and a pathological process. A disorder basically refers to a material entity which

is clinically abnormal and part of an organism, although its precise definition has been repeatedly changed and seems to be under development (Scheuermann, Ceusters and Smith, 2009; Ceusters and Smith, 2010a, 2010b). A pathological process is a bodily process that is a manifestation of a disorder, where a bodily process is a BFO-process in which participate one or more material entities within or on the surface of an organism. Pathological processes are recognized through symptoms and signs.

For the OGMS, a disease is “a disposition (i) to undergo pathological processes that (ii) exists in an organism because of one or more disorders in that organism” (Scheuermann, Ceusters and Smith, 2009). As a disposition, a disease has some disorder as its material basis and a disease comes into existence when its corresponding disorder does, i.e., when the organism disposes towards its relevant pathological processes. A disease as a disposition may go unrealized, e.g., when it lies dormant over a long period of time. A related crucial term is a *disease course*: the totality of all BFO-processes through which a given disease instance is realized. A disease course of a disease ranges widely from potentially asymptomatic early stages of the disease to its recognizable, pathological processes. For instance, epilepsy as a disease is a disposition to undergo the occurrence of seizures (pathological processes) that exists owing to some clinically abnormal, neuronal circuitry of the brain (disorder); and the disease course of epilepsy would comprise the pathological processes of seizures and the BFO-processes of loss of consciousness.

5.7. Comparison with the OGMS dispositional model of disease¹⁵

The RFM and the OGMS account of disease have some common views on disease. First of all, an RFM-disease and an OGMS-disease both say that a disease is in nature a ‘causal pattern’ (see also Rovetto and Mizoguchi, 2015: Section 3.1). An RFM-disease is characterized by the regular way in which abnormal states are causally connected and the pattern nature of an RFM-disease may be represented in terms of a directed graph consisting of abnormal states as vertices and causal relations between them as edges. In a similar vein, an OGMS-disease reasonably qualifies as a causal pattern. For one thing, an OGMS-disease is causal, since a disposition is a causal property. For another, an OGMS-disease is patternized because a disposition which has a ‘specific’ material basis realizes its ‘corresponding’ BFO-processes when exercised by some ‘appropriate’ triggers.

It is important to emphasize that both models of disease share the opinion that a disease is a dependent continuant, but not an occurrent, although some existing ontologies classify a disease as a type of occurrent. A disease is an entity with which a patient is affected and which medical practitioners are able to identify, diagnose, and cure. A disease is something that comes into existence, grows, and finally disappears in the patient’s body. All these observations would mean that a disease is an entity that persists in time, i.e., a continuant. Additionally, a disease is a dependent continuant

¹⁵ This section derives largely from Toyoshima, Mizoguchi and Ikeda (2017).

that inheres in an organism.

Moreover, an RFM-disease and an OGMS-disease both involve what may be called a ‘clinical threshold’: the level at which a pathological state of affairs of a disease begins to develop. RFM specifies a clinical threshold employing the imbalance model. Pathological states of affairs of a disease show themselves when the clinical imbalance state that is relevant to the disease has occurred. Likewise, the OGMS model of disease explicates a clinical threshold in terms of the emergence of a disease as a disposition (and of its corresponding disorder as a material basis of the disease disposition). Having reached a clinical threshold, an organism disposes towards pathological processes, which are recognizable through (but may be not identical with) signs and symptoms.

It is nonetheless vital to clarify the difference between an RFM-disease and an OGMS-disease from the viewpoint of a clinical threshold. In the case of an RFM-disease, a clinical imbalance state of a disease is not always an initial state of the disease. As was indirectly shown above, the imbalance model abstracts from a disease its generality and eliminates its specificity. For instance, Type I diabetes and steroid diabetes fall into a group of diabetes, since they have the same kind of clinical imbalance state (i.e., the deficiency of insulin), but those two diseases still differ from each other because they have different causal chains of abnormal states (see Kozaki, Mizoguchi, Imai and Ohe, 2012 for more details on the identity of RFM-diseases).

By comparison, it is clear that the emergence of an OGMS-disease as a disposition is always at the beginning of the disease. To do justice to the specificity as well as the generality of disease, the OGMS covers a *predisposition to disease of type X*: a disposition in an organism that constitutes an increased risk of the organism’s subsequently developing the disease *X* (Scheuermann, Ceusters and Smith, 2009; Ceusters and Smith, 2010a). A predisposition is a disposition to acquire a further disposition and some diseases as dispositions (e.g., osteoporosis) are predispositions to further diseases as dispositions (e.g., fracture).

Roughly speaking, the generality and the specificity of an OGMS-disease are to be captured by a disease as a disposition and a predisposition to disease of type *X*, respectively. For instance, Type I diabetes and steroid diabetes belong to the same diabetes category because they are essentially the ‘diabetes disposition’. These two diseases are nevertheless different because a predisposition to have diabetes that is involved in Type I diabetes differs from that involved in steroid diabetes. A realization of the former predisposition, but not of the latter predisposition, may have as part the BFO-process of destruction of pancreatic beta cells; and conversely, a realization of the latter predisposition, but not of the former predisposition, may have as part the BFO-process of the increase of steroids (see Barton, Grenier and Ethier, 2018 for recent elucidation of the relationship between diseases as dispositions and predispositions in the OGMS).

Though conceived commonly as a causal pattern, an OGMS-disease and an RFM-disease

are significantly different in the sense that the basic ‘unit of thought’ of the OGMS is a disposition (dependent continuant) but that of the RFM is a state (occurrent). This fundamental difference between those two accounts of disease is largely, if not totally, explicable in terms of causation. For the OGMS, causation is dispositional: causation occurs when a disposition as a causal property realizes itself. The dispositional conception of causation would lead directly to an OGMS-disease as a disposition. For the RFM, causation is functional: causation occurs when the causal efficacy of occurrents (especially events) is manifested, which can be clarified when it is modeled upon the device-ontological concepts of the ‘what to achieve’ and the ‘how to achieve’. An RFM-disease obtains its causal features from causally efficacious occurrents that constitute the disease. Note that the functional view of causation does not *ipso facto* deny the existence of dispositions; but it would nonetheless imply that dispositions (if any) or their realizations serve at best as ‘contributors’ to causation, a complete picture of which is to be drawn only from a functional point of view.

Another indicator of the contrast between the OGMS and the RFM conceptions of causation is the difference in what is clinically abnormal between the OGMS and the RFM. Clinically abnormal are a disorder (continuant) in the OGMS and a state (occurrent) in the RFM. In the OGMS, a disease as a disposition inherits its clinical abnormality from a disorder as its material basis; and therefore, a disorder (a material entity) is defined as clinically abnormal. In contrast, the RFM notion of clinically abnormal state reflects well the idea of causally efficacious occurrents that is embraced by the functional view of causation. Consider for instance inflammation as a disease. The OGMS would say that inflammation as a disposition is clinically abnormal primarily because so is its material basis, e.g., the cells in the relevant part of the organism. The RFM contends however that the clinical abnormality of the cells therein is a contributor to that of states which inflammation has inside it. This marks a close analogy with the above-offered functional interpretation of (realizations of) dispositions as contributors to causation.

5.8. Towards an extension of the RFM to mental disease¹⁶

Mental functionings are highly relevant to disease ontologies, as is observed by the fact that mental and behavioral disorders constitute an acute problem for the public health all over the world (National Advisory Mental Health Council Workgroup, 2010). An ontology of mental disease would facilitate an interdisciplinary research on mental disease, thereby contributing to the improvement of psychiatric diagnostics and treatment (Ceusters and Smith, 2010b). In particular, it would help to fill a semantic gap between affective science and psychiatry, which have been historically separate in spite of their common goal to explore human mental phenomena (Larsen and Hastings, 2018).

This section discusses an extension of the RFM (which focuses traditionally on so-called

¹⁶ This section depends partially on Toyoshima (2018).

‘physical disease’ such as diabetes) to an intricate entity of mental disease, although a full-fledged advance of this project is outside the scope of this dissertation. First, it is proposed that causal functionalism (namely, a certain theory extracted from philosophy of mind) be used as a theoretical foundation for a general ontology of minds and mental states.¹⁷ Second, enlargement of the RFM to mental disease is sketched out against YAMATO ontological background on the assumption of a causal functional ontology of mind and the Belief-Desire-Intention (BDI) of agency.

5.8.1. A causal functional ontology of mind

The notions of minds and mental states are present in various disciplines ranging from philosophy and linguistics to cognitive science and artificial intelligence. Different theories of them are so far tailored to the demands of different domain experts, and several ontologies of mind have been built to serve as a ‘point of reference’ for evaluating those varying mental models. Examples include the Computational Ontology of Mind (COM) and the Mental Functioning Ontology (MF). Being motivated by the need in agent technology for an ontological modeling of a cognitive agent’s vision of the world, the COM provides a preliminary characterization of mentality that is integrable into the DOLCE upper ontology (Ferrario and Oltramari, 2004). By contrast, the MF is built in alignment with the BFO upper ontology in order to be employed especially in the context of bioinformatics and biomedical ontologies (Hastings, Ceusters, Jensen, Mulligan and Smith, 2012).

It is here suggested that a solid foundation for a general ontology of mentality be furnished by the kind of theory that comes loosely under the heading of ‘common-sense (or analytical) functionalism’ (Braddon-Mitchell and Jackson, 2007: Chapter 3) or ‘causal-theoretical functionalism’ (Kim, 2011: Chapter 10) in philosophy of mind. To refer to it, the term ‘causal functionalism’ will be hereafter used. Causal functionalism roughly says that each mental kind is characterized in terms of its own distinctive *causal role* with respect to its inputs (including sensory stimuli) and its outputs (including bodily behaviors) in the entire network of the causal relations involving all the psychological states.¹⁸ For instance, for an agent x to be in pain is for x to be in an internal state with a causal intermediary, in x ’s whole psychological system or simply in x ’s ‘mind as a causal system’ (ibid.), between tissue damage and some mental states (e.g., being normally alert) as causes; and groans, winces, and other mental states (e.g., distress) as effects.

First and foremost, causal functionalism is compatible with materialism. Philosophy of mind traditionally pivots on the debate between dualism and materialism (aka physicalism). Roughly

¹⁷ The term ‘physical state’ (resp. ‘mental state’ or ‘psychological state’) will be employed because of its widespread usage in everyday life as well as in academic disciplines; but ontologically speaking, it refers to a physical (resp. mental) *occurrent*, instead of being limited to a physical (resp. mental) state in the YAMATO sense of the term.

¹⁸ More precisely, causal functionalism herein means *role functionalism* rather than *realizer functionalism*, borrowing Ross and Spurrett’s (2004) terminology.

speaking, dualism says that minds and mental states are in nature something immaterial or non-physical; whereas, materialism says that anything that exists in the world (including minds and mental states) are bits of matter or aggregates composed of them. Since most modern scientific research favors materialism over dualism and ontologies in general are employed mainly in scientific fields, it is reasonable to anchor an ontology of mind to the materialist conception of mind.

Second, causal functionalism is consistent with folk psychology or common-sense psychology. Rather, causal functionalism is elaborated so that causal roles of mental states can be given by commonsensical claims about mental states (Lewis, 1972; Braddon-Mitchell and Jackson, 2007). Examples of folk psychology include the principle (sometimes called the ‘belief-desire principle’) according to which an agent’s action is a causal consequence of her various beliefs, desires and other mental states. In this respect, causal functionalism contrasts sharply with eliminative materialism (Churchland, 1981): the view that mentality as ordinarily conceived (e.g., pain and beliefs) should be eliminated by the latest scientific (e.g., neuroscientific) findings because it is a completely misguided conception of the nature of mental activities and the causes of bodily behaviors.

Third, and most importantly, causal functionalism (or functionalism in general) captures well the *multiple realizability* of mental states: there are generally two or more physical states that can ‘realize’ a certain mental state (Putnam, 1973). For instance, there must be indefinitely many physical states that can ‘realize’ pain in all sorts of pain-capable organisms and systems. Taking the defining feature of a mental state to be its causal role, the causal functionalist admits the possibility, e.g., that a pain is ‘realized’ not only by a neurological state of a person but also by an electromagnetic state of a highly developed robot. Causal functionalism is in this sense opposed to the identity theory (Smart, 1959): the view that identifies types of mental states with types of physical (neural) states (for instance, a type of pain is identical to a type of C-fiber stimulation). The causal functionalist ontology of mind would be therefore applicable not only to biomedicine but also to the domains (e.g., robotics) to which central are agents with no brain, or more precisely, with no biological basis for their action planning.

5.8.2. First steps towards mental disease in the RFM

For the present purpose of the RFM modeling of mental disease, it is further proposed that causal functionalism be interpreted in compliance with the YAMATO upper ontology, or more specifically that the paramount notion of causal role in this theory of mind be construed in terms of the YAMATO conception of role and the functional perspective on causation. One possible statement to be deployed along this line is that a physical occurrent (event/process/state) plays a *mental role* in the mind, thereby becoming a mental occurrent (event/process/state). Given the neuroscientific finding that the cerebral cortex is vital for various cognitive activities, for instance, an active state (player) of Mary’s cerebral cortex plays a belief role in her mind (context) when she looks outside from the

window, thereby becoming her belief state (role-holder) about snow. The multiple realizability of mental states is thus explicable in terms of the playing of the mental role of multiple physical states. Notice that, on this interpretation, mentality is an occurrent role.

To render this proposal more concrete, the Belief-Desire-Intention (BDI) model of agency is assumed, which would enable later the illustration of causal interrelationships between mental states from a functional viewpoint. Inspired by Bratman's (1987) philosophical work, the BDI model recognizes the primacy of beliefs, desires, and intentions in practical reasoning and rational actions (Wooldridge, 2010). Despite controversy as to the reducibility of intention to desire-belief pairs (Sinhababu, 2013), the BDI approach is widely used in applied ontology as well as in artificial intelligence in virtue of its implementational and logical benefits (see e.g., Trypuz, 2007). It is thus instructive the way the BDI model of agency works within the present framework for mentality.

Suppose for the sake of argument that, on the way to the station, Sofia realized that the window of her room was left open, so she came back home. A rough analysis of this case would proceed as follows. The event of Sofia coming back home occurred and it was caused (**allows**) by Sofia's (*new*) *intention event* of her intending (making a plan) to come back home, which a physical event (of part of her body, or especially her brain) plays an *intention role* in her mind, thereby becoming.¹⁹ Sofia's intention event was caused (**allows**) by her (*new*) *desire process* of her desiring to be at home. Sofia's desire process was caused (**allows**) by her (*new*) *belief process* of her believing that Sofia is outside home. Note the high relevance of state-mediated causation (underpinned conceptually by a state-centered approach to causation) to careful consideration of this simple scenario.

Not surprisingly, Sofia's desire to be at home is intimately linked with her 'deeper' desires. Sofia's desire process discussed above is caused (**allows**) by her *deeper desire process* of her desiring to close the window of her room. Moreover, Sofia's deeper desire process is caused (**allows**) by her *yet deeper desire process* of her desiring to prevent a robbery.²⁰ Sofia's mind change is also representable in terms of the functional square of causal relations. Sofia's desire process **disallows** her *old intention event* of her intending to go to the station. This is because Sofia's desire process **prevents** her *old desire state* of her desiring to arrive at the station and the latter is a facilitative precondition for Sofia's old intention event. Finally, the process of Sofia going to the station occurred until her realization and it had been caused (**allows**) by Sofia's old intention event.

Now that a causal functional ontology of mentality is well-specified given the YAMATO construal of causal role, one of arguably the most straightforward extensions of the RFM to mental

¹⁹ A reference to the playing of 'BDI roles' will be omitted below for the sake of simplicity.

²⁰ Needless to say, other numerous and multifarious mental occurrents that must be causally involved in this scenario are left aside to simplify the matter: e.g., Sofia's *knowledge state* of her knowing that Sofia can open the window of her room only when she is inside the room.

disease can be provided as follows: a mental disease is a dependent continuant constituted of abnormal mental events, processes, or states (i) to which mental events, processes, and states bear either the **achieves**, **prevents**, **allows**, or **disallows** causal relation and (ii) initiated by at least one abnormal mental event, process, or state. As compared with its original version, the extended RFM definition of mental disease takes a liberal view that mental disease can be constituted not only of abnormal mental states but also of abnormal mental events and processes. For one thing, it is assured theoretically and practically that one has only to examine abnormal states (paradigmatically brought about by events) in identifying physical diseases; whereas, the RFM definition of mental disease should not be currently as strict until mental causation (Heil and Mele, 1993; Walter and Heckmann, 2003) is fully understood in accordance with the functional perspective on causation.

To illustrate this preliminary picture of mental disease, consider the case of *persecutory delusions*, since they are the most common kind of delusions regarding schizophrenia spectrum and other psychotic disorders (American Psychiatric Association, 2013: 87). Imagine that a deluded person, Michael, forced all the other members in his company not to harass him physically. The event of Michael forcing all the other members in his company not to harass him physically occurred and it was caused (**achieves**) by Michael's *intention event* of him intending to force so. Michael's intention event was caused (**allows**) by his *desire process* of him desiring not to be physically harassed by anybody. In addition, Michael's desire process is caused (**allows**) by his *deeper desire process* of him desiring to have a pleasant working environment.

Most importantly, Michael's desire process was caused (**allows**) by his *abnormal belief process* of him believing *falsely* that all the other members in Michael's company are going to harass him physically. Michael's delusion is interpretable in terms of clinical abnormality of this belief process, which can be thus called a 'delusional belief process'. In general, it depends on the psychiatrist's judgment whether a certain mental occurrent is clinically abnormal or not. A further analysis of Michael's mentality could be added given some auxiliary assumptions. Michael's delusional belief process was caused (**achieves**) by an assemblage of various events that had occurred around him, including the event in which one of his colleagues accidentally trod on his toe. It could be said more technically that Michael's delusional belief process was due to the 'mutual manifestation' or 'co-manifestation' (a precise meaning of which is left for future work) of the causal efficacy of those incidental events.

6. Discussion and related work in ontology engineering

6.1. Discussion

6.1.1. The transitivity of causal relations

The transitivity of the causal relation is a highly controversial topic (see e.g., Hall, 2000). Suppose that a bomber placed a bomb on a road, the police managed to defuse it while they closed the road, and the road became safely open again. It is intuitively false to think that the road became safe because the bomber places the bomb there, although a naive conception of the transitivity of the causal relation might imply it. As illustrated with this example, a full-fledged specification of the (non-)transitive feature of the causal relations in the functional square is to be left for future work owing to its considerable difficulty.

It is nonetheless important to note that the idea of causal chains, to which the transitivity of the causal relation is closely related, has been partially covered in terms of indirect causal relations (represented by CSC) and the extensive usage of CSC. It is expected that a proper transformation of and/or a repetitive usage of CSC will illuminate the intricate issue of the transitivity of the causal relation based on the present functional view of causation.

6.1.2. Integration with different upper ontologies

As for the relationship with the functional perspective on causation and foundational ontological frameworks, three upper ontologies are briefly discussed. First, the current proposal can be fully exploited in compliance with the YAMATO upper ontology. This is because virtually all the conceptual vehicles employed in the dissertation are available there, ranging from the device ontology and roles to the event/process/state distinction and systemic function.

Second, the present functional view of causation would be adaptable to the Descriptive Ontology for Linguistic and Cognitive Engineering (DOLCE) (Borgo and Masolo, 2010), partly because this upper ontology has the potential to accommodate the notion of systemic function. One apparent problem is that DOLCE as such does not have as category the conception of processes as ‘ongoing occurrents’, but it could be addressed using the DOLCE-based analysis of the ongoing-ness of occurrence (see e.g., Guarino, 2017).

Third, it may be less clear how the functional approach to causation can be adjusted to Basic Formal Ontology (BFO) (Arp, Smith and Spear, 2015; Smith et al., 2015). For one thing, BFO classifies a state as a continuant rather than an occurrent (cf. Jansen, 2015) and does not elaborate the notion of ‘ongoing occurrent’. There are nonetheless some promising ways to harmonize the author’s main ideas and BFO, one of which is to use a recently proposed way of further classifying processes in the BFO sense of the term (Jarrar and Ceusters, 2017). Since BFO develops a rich theory of

dispositions, it would be also constructive to investigate the relationship between dispositions and functions (see e.g., Röhl and Jansen, 2014; Spear, Ceusters and Smith, 2016; Jansen, 2018).

6.2. Related work in ontology engineering

Lehman, Borgo, Masolo and Gangemi (2004) present a general conceptualization and a comparatively robust formalization of causal relations that pivot on the distinction between *causality*, a law-like relation between types of perdurants (occurents, in the terminology of this dissertation) and *causation*, the actual causal relations that hold between individual perdurants. Characteristic of their work is that the notion of causality and causation depends on a specific choice of constraints (structural, causal, and circumstantial) which are in turn to be explicable in terms of dependencies between (types of) *quality changes* against the background of the DOLCE upper ontology.

Their work is of great importance because it develops a basic framework for later discussions on causality and causation in applied ontology. Examples include the causality/causation distinction, the conception of causal relation as the relation between (types of) occurents, and the argument that there is no unique characterization of the notion of causality and causation in everyday practice. At the same time, however, this early work has some limitations partly because it revolves around one simple example (The Broken Window). For instance, ‘static events’ (e.g., the pen on the table does not fall ‘because’ of the presence of the table) are mentioned as the intuitively (but not clearly to them) causal case to which their proposal is not applicable. Being analogous to Example (v) “Book on the table” in Section 3.3, the case of ‘static events’ falls under the broader scope of our functional perspective on causation.

Michalek (2009) develops a formal ontological theory of causality that is conceptually based on the intuitions of regularity and counterfactual dependency within the framework of the upper ontology General Formal Ontology (GFO) (Herre, 2010).²¹ Given the GFO basic distinction between *presentials* (which is roughly something that defines objects) and *processes* (roughly, occurents), the basic causal relation between presentials is then extended to cover different kinds of causal relations between processes. The epistemic adequacy of the theory is also evaluated through a discussion on its application to scientific procedures, including experiments and clinical trials.

The author’s functional perspective on causation is fundamentally so different from Michalek’s GFO theory of causality that it would be rather difficult to compare them with each other. First and foremost, the notion of regularity and counterfactual dependency is at the nub of his approach, whereas the functional perspective is rooted in an intimate connection between change and causation. Second, he takes presentials and processes to be primary and secondary causal relata, respectively; and in contrast, the author justifies occurents as basic causal relata on the grounds of

²¹ The author follows Michalek’s (2009) own usage of the term ‘causality’ in referring to his theory.

the idea of ‘causally efficacious occurrents’. It could be nonetheless argued that the functional perspective on causation is (or at least aims to be) more comprehensive than Michalek’s theory of causality. For instance, the regularity and counterfactual aspect of causation is to be captured in terms of the **maintains** usage of indirect causal relations and CSC (see Section 4.1.4 for a related point). Furthermore, the functional perspective examines more closely the relation between lawhood and causation, on which he does not explicitly focus, and offers a detailed classification of causal relations based on the event/process/state ontology.

Arguably the closest and most important related work to the functional perspective on causation developed in this dissertation is Galton’s (2012) investigation of a (token-level) causal role of events, processes, and states as well as a range of different causal and causal-like relations among those occurrents. Despite the shared assumption of the event/process/state ontology, the authors’ view differs considerably from Galton’s in some crucial respects, most notably regarding the causal role of states and events. In Galton’s view, events and processes are in themselves causally efficacious, whereas states are not. The primary causal role of states is thus to enable (‘allow’) the causal relation (‘cause’) between events as well as the causal relation (‘perpetuate’) between processes and a state can be a cause only when it ‘maintains’ another state. For instance, the state of Ryan holding the ball ‘allows’ not only the event of Ryan starting moving his hand to ‘cause’ the event of the ball starting moving his hand to ‘cause’ the event of the ball starting moving but also the process of Ryan moving his hand to ‘perpetuate’ the process of the ball moving; and the state of the continuing presence of the cable ‘maintains’ the state of the continuing presence of the lamp in its position 2.2 meters above the floor.

It is however proposed in this dissertation that the causal role of states be more highly evaluated based on the state-centered approach to causation. States are causally more efficacious than Galton argues, as illustrated by Example 4 (Thrombosis). Furthermore, the vital causal role of states is clearly found in the context of absence causation, although Galton himself does not fully discuss it. In particular, causation by absence is supposed to be explicable in terms of CSC which involves some state (e.g., the state of the absence of vitamin C). The author nonetheless agrees with him on the more problematic identification of states than that of events (see Section 7 for details).

An intimately connected issue is the causal role of events. Following the functional perspective of the dissertation, an event can cause (**achieves**) a state in virtue of the functional structure of its causal efficacy and there is in principle no **achieves** causal relation between events; whereas, Galton says that an event bears a causal-like relation (‘initiate’, ‘terminate’) to a state and he takes the event-event causal relation to be most typical, as is shown by his usage of the term ‘cause’ to refer to the event-event causal relation. This broad difference seems to arise from the fact that causation is for Galton primarily the bringing about of the *beginning* of something; in contrast, the

author considers events *in their entirety* as causal relata. Since an event (but neither a process nor a state) has a definite beginning, the event-event causal relation is a paradigmatic case of his conception of causal relation. To take his example, the event of Ryan starting moving his hand ‘causes’ the event of the ball *starting* moving. While conceding the possibility of the causal relation between an event and an ‘initiation event’, the author denies the **achieves** causal relation between an event and an event *as a whole*. It is, rather, either a state (typically brought about by an event) or a process that **achieves** a whole event.

7. Conclusion

7.1. Summary

The functional perspective on causation is built on twin pillars. One is a theoretical exploration of the ontological nature of causation. Of paramount importance is the claim that the causal relation necessitates ontologically the context in which it holds. Combined in order with the device-ontological understanding of change, the systemic-functional notion of lawhood, and the systemic conception of function as goal achievement, this fundamental idea finally leads to the conceptual mapping of causation onto function and the **achieves** causal relation (as well as the **prevents** causal relation). The **achieves** causal relation is underpinned by the idea of ‘causally efficacious occurrents’. In particular, events have the causal efficacy to bring about states.

The other is a practical development of an expressive causal representation. A vital clue as to this is the observation that an ontological modeling of causation would be particularly useful for expressing causal chains of various phenomena. This contributes to the precondition-for relations and the development of indirect causal relation (**allows** and **disallows**) that are based on direct causal relations and that are diagrammatically conceptualized as the Configuration of State-mediated Causation (CSC). Those four kinds of causal relations are conceptually organized in the form of the functional square of causal relations. Those accomplishments are backed by the idea of a state-centered approach to causation. The explanatory force of the proposal is shown by its ability to accommodate a wide variety of examples extracted from the relevant literature.

It would be helpful to glance at the philosophical significance of the functional perspective on causation for the sake of its clarification. The aforementioned dispositional theory of causation explicates causation in terms of a constellation of a disposition and its related notions: e.g., a disposition as a ‘causal property’, its bearer, its base, its realization/manifestation, and its trigger. Similarly, the functional perspective on causation aims to model causation upon an arrangement of various concepts and conceptual tools, including events/processes/states as ‘causally efficacious occurrents’, a systemic context (and its corresponding granularity), the functional square of causal relations, and CSC (or the notion of precondition therein). Furthermore, it offers a unique treatment of the counterfactuality of causation in terms of the quasi-causal **maintains** relation. In this respect, the functional perspective on causation developed in this dissertation would have the potential to take an interesting middle course between the dispositional and counterfactual theories of causation.

Moreover, the usage of the functional view of causation was illustrated with its application to the River Flow Model (RFM) of diseases, which has remained obscure from a causal point of view. The RFM definition of disease was then updated from a functional standpoint, and it turned out to be more sophisticated than prior revised RFM definitions. This also served to provide a clearer comparison between the RFM and an influential dispositional account of disease offered by the

Ontology for General Medical Science (OGMS). An extension of the RFM to mental disease was also briefly discussed together with some conceptual considerations of mentality.

This work will yield wide implications for knowledge science: the discipline that aims to offer a systematic understanding and facilitation of the creation, exploitation, and the accumulation of knowledge involved in individuals as well as in society. The dissertation contributes to knowledge science by building a common ground for representing different causal phenomena in different domains and thus facilitating an integration of and an interdisciplinary collaboration across research fields dealing with the concept of causation. It also constitutes an initial step towards a solution to the problem of fragmentation in biomedicine because it not only enhances the interoperability and flexibility of an increasing amount of disease-related data and information but also shows the practical potential for creating novel medical knowledge in the long run.

7.2. Future work

There are a number of directions of research in which the work developed in this dissertation can proceed; and some future works on ontology of causation and ontology of disease will be listed below individually. First, for the sake of further development of the functional perspective on causation, a detailed investigation into each key element of the functionally interpreted notion of causation is evidently warranted. Examples include:

- *How can one identify states occurring in reality?* A state-centered approach to causation is one of the central tenets of the functional perspective on causation. Questions nonetheless remain about the nature of states. As Galton (2012) says, the identification of states (state tokens) is more problematic than that of events (event tokens), partly because of the difficulty of distinguishing sharply a state in the world from its description, typically with the usage of the term ‘state’. It is even debatable whether a state is a continuant or an occurrent (Jansen, 2015). Further elucidation of states (or even of the event/process/state ontology) is required.
- *What is a context?* The functional perspective on causation consists in saying that the causal relation necessitates the context in which it holds. There has been however a heated debate on the nature of a context in the discussion over causation (see Schaffer, 2005), let alone in respect of a general inquiry into it: see e.g., Stalnaker (2014) in philosophy and Mizoguchi, Kitamura and Borgo (2016) and Baclawski (2018) in applied ontology. Further clarification of the notion of context is needed.
- *What is granularity?* In the discussion above, granularity was mentioned explicitly and repeatedly because it is one of the most important elements of the notion of context. The ontological nature of granularity has been nevertheless obscure, in spite of some preceding work

(Bittner and Smith, 2003; Keet, 2008; Masolo, 2010). Granular levels of reality are informally said to be the kind of ‘divisions’ or ‘partitions’ that reflect the hierarchical structure of reality, but it is rather unclear what those phrases are supposed to mean. An in-depth investigation of the notion of granularity is required, although it may be one of the most challenging tasks regarding the epistemological foundation of ontologies.

- *What is (a causal role of) a precondition (whether facilitative or preventive)?* The notion of (facilitative or preventive) precondition in general (simply ‘precondition’ hereafter) is indispensable for the definitions of the indirect causal relations as well as the construction of CSC. Despite Galton’s (2012) preceding reference to a precondition, it may remain unsettled what a causal role of a precondition is like. For one thing, a precondition seems to have its own causal role, as is supported by the observation that some causal claims (e.g., Example 14 “Scurvy: simpler case” and Example 15 “Examination: simpler case”) refer only to some state bearing the precondition-for relation to the effect. An extensive exploration of a precondition (especially its causal role) is strictly necessary. It is interesting to note that the causal role of a precondition may be elucidated in terms of counterfactuality; for it is reasonable to think that the truth of the claim “A state *Z* is a facilitative precondition for an occurrent *Y*” is closely related to counterfactual dependence of *Z* on *Y*, or specifically, to the truth of a simple counterfactual conditional (“If *Z* did not occur, then *Y* would not occur”), i.e., a special case of the subjunctive conditional, which uses what is known in grammar as the ‘subjunctive mood’. It is therefore well worth investigation to construct a counterfactual logic (Stalnaker, 1968; Lewis, 1973b) for the preconditional conceptualization of causation.
- *How can the causal relations in the functional square be more rigorously formalized?* Section 4.2 aimed to clarify logically the actualist’s ‘functionally strengthened’ account of causation as compared to the non-actualist’s standard view of causation. There is nonetheless some room for improvement in the formal specification of the relationship between them. One promising formal candidate for this task might be the logic of interpretability (see e.g., Visser 1998), since it is necessary to formalize well how the non-actualist’s theory of causation is ‘interpretable’ in terms of or ‘translatable’ into the actualist’s theory enhanced by the functional perspective on causation, or especially by the coupling of the context with a state-centered approach to causation. Other possible formal techniques include intuitionistic logic (van Dalen, 1984) and relevance logic (Read, 1988) because those weaker logical languages are expected to specify the conditional or implicational nature of indirect causal relations better than classical logic (Fitting and Mendelson, 1998). This longitudinal line of research will comprise the building a full-fledged logical system (syntax and semantics) for the functional square and close examination of its meta-logical properties (e.g., soundness, completeness, and decidability), both of which would enable the full

computational implementation of the RFM.

Despite those remaining issues, however, the functional perspective on causation is currently theoretically and practically valuable enough to be expected to be closely investigated and extensively utilized in the context of ontology engineering. For one thing, the functional view has paved the way for the conceptual conversion of an extremely complex notion of causation into several simpler, functional ones. For another, the view is so well-designed to encompass a richly diverse range of examples not only from the philosophical literature but also from the domains where ontologies have been extensively employed.

Second, there are at least two promising further expansions of an ontological analysis of disease offered in the dissertation. One is the development of an ontological module for generic disease representation that would reconcile the RFM with the OGMS dispositional account of disease (Toyoshima, Mizoguchi and Ikeda, 2017; see also Barton, Rosier, Burgun and Ethier, 2014). An important clue as to this task would be the finding that disease can be well ontologized as a causal pattern with a clinical threshold. The other is an extension of a causal understanding of disease to other relevant biomedical entities such as health and aging (see e.g., Fuellen et al., 2018). When accomplished, both a general disease module and a comprehensive ontology of biomedicine will have crucial ramifications for the satisfaction of medical specialists' practical needs, e.g., for clinical terminologies such as the Systematized Nomenclature of Medicine - Clinical Terms (SNOMED-CT) and disease classification systems such as the International Classification of Diseases (ICD) (World Health Organization, 2018).²²

²² For details on SNOMED-CT, visit: <https://www.snomed.org/> (Last accessed on June 23, 2019).

Bibliography

- Agibetov, A., Jiménez-Ruiz, E., Ondrésik, M., Solimando, A., Banerjee, I., Guerrini, G., Catalano, C. E., Oliveira, J. M., Patanè, G., Reis, R. L. and Spagnuolo, M. (2018). Supporting shared hypothesis testing in the biomedical domain. *Journal of Biomedical Semantics* 9: 9.
- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorder - Fifth Edition*. Washington, DC.
- Anjum, R. L., Kerry R. and Mumford, S. (2016). Evidence Based On What? *Journal of Evaluation in Clinical Practice* 21(6): E11-E12.
- Arp, R., Smith, B. and Spear, A. D. (2015). *Building Ontologies with Basic Formal Ontology*. Cambridge, MA: MIT Press.
- Baclawski, K. et al. (2018). Ontology Summit 2018 Communiqué: Contexts in context. *Applied Ontology* 13(3): 181-200.
- Barton, A., Grenier, O. and Ethier, J.-F. (2018). The Identity and Mereology of Pathological Dispositions. In L. Cooper and P. Jaiswal (eds.), *Proceedings of the 9th International Conference on Biological Ontology (ICBO 2018)*, Corvallis, Oregon, USA, August 7-10, 2018. CEUR Workshop Proceedings, vol. 2285.
- Barton, A., Grenier, O., Jansen, L. and Ethier, J.-F. (2018). The Identity of Dispositions. In S. Borgo, P. Hitzler and O. Kutz (eds.), *Proceedings of the 10th International Conference of Formal Ontology in Information Systems (FOIS 2018)*, Cape Town, South Africa, September 17–21, 2018. Amsterdam: IOS Press, pp. 113-126.
- Barton, A., Rosier, A., Burgun, A. and Ethier, J. F. (2014). The Cardiovascular Disease Ontology. In P. Garbacz and O. Kutz (eds.), *Proceedings of the 8th International Conference of Formal Ontology in Information Systems (FOIS 2014)*, Rio de Janeiro, Brazil, September 22-25, 2014. Amsterdam: IOS Press, pp. 409-414.
- Barton, A., Rovetto, R. and Mizoguchi, R. (2014). Newtonian Forces and Causation: A Dispositional Account. In P. Garbacz and O. Kutz (eds.), *Proceedings of the 8th International Conference of Formal Ontology in Information Systems (FOIS 2014)*, Rio de Janeiro, Brazil, September 22–25, 2014. Amsterdam: IOS Press, pp. 157-170.
- Beckers, S. and Vennekens, J. (2018). A principled approach to defining actual causation. *Synthese* 195(2): 835-862.
- Beebe, H. (2004). Causing and Nothingness. In J. Collins, N. Hall and L. A. Paul (eds), *Causation and Counterfactuals*. Cambridge, MA: The MIT Press, pp. 291-308.
- Bittner, M. (1999). Concealed causatives. *Natural Language Semantics* 7(1): 1-78.
- Bittner, T. and Smith, B. (2008). A Theory of Granular Partitions. In K. Munn and B. Smith (eds.),

Applied Ontology: An Introduction. Frankfurt: Ontos Verlag, pp. 125-158.

- Bodenreider, O. and Burgun, A. (2009). Towards Desiderata for an Ontology of Diseases for the Annotation of Biological Datasets. In B. Smith (ed.), *Proceedings of the 1st International Conference on Biomedical Ontology (ICBO 2009)*, Buffalo, New York, USA, July 24-26, 2009, pp. 39-42.
- Boorse, C. (1975). On the Distinction between Disease and Illness. *Philosophy and Public Affairs*, 5: 49-68.
- Boorse, C. (1997). A Rebuttal on Health. In J. M. Humber and R. F. Almeder (eds.), *What is Disease?* Totowa, NJ: Humana Press, pp. 3-143.
- Borgo S. and Hitzler P. (2018). Some Open Issues After Twenty Years of Formal Ontology. In S. Borgo, P., Hitzler and O. Kutz. (eds.), *Proceedings of the 10th International Conference on Formal Ontology in Information Systems (FOIS 2018)*, Cape Town, South Africa, September 17-21, 2018. Amsterdam: IOS Press, pp. 1-9.
- Borgo, S. and Masolo, C. (2010). Ontological Foundations of DOLCE. In R. Poli, M. Healy and A. D. Kameas (eds.), *Theory and Applications of Ontology: Computer Applications*. Netherlands: Springer, pp. 279-295.
- Borgo, S. and Mizoguchi, R. (2014). A first-order formalization of event, object, process, and role in YAMATO. In P. Garbacz and O. Kutz (eds.), *Proceedings of the 8th International Conference of Formal Ontology in Information Systems (FOIS 2014)*, Rio de Janeiro, Brazil, September 22–25, 2014. Amsterdam: IOS Press, pp. 79-92.
- Borgo, S., Mizoguchi, R. and Kitamura, Y. (2016). Formalizing and Adapting a General Function Module for Foundational Ontologies. In R. Ferrario and W. Kuhn (eds.), *Proceedings of the 9th International Conference on Formal Ontology in Information Systems (FOIS 2016)*, Annecy, France, July 6–9, 2016. Amsterdam: IOS Press, pp. 241-254.
- Borgo, S., Porello, D. and Troquard, N. (2014). Logical Operators for Ontological Modeling. In P. Garbacz and O. Kutz (eds.), *Proceedings of the 8th International Conference of Formal Ontology in Information Systems (FOIS 2014)*, Rio de Janeiro, Brazil, September 22–25, 2014. Amsterdam: IOS Press, pp. 23-36.
- Borst, W. N. (1997). Construction of Engineering Ontologies for Knowledge Sharing and Reuse (PhD Thesis). Institute for Telematica and Information Technology, University of Twente, Enschede, The Netherlands.
- Braddon-Mitchell, D. and Jackson, F. (2007). *Philosophy of Mind and Cognition* (Second Edition). Malden, MA: Blackwell Publishing.
- Bratman, M. E. (1987). *Intention, Plans, and Practical Reason*. Cambridge, MA: Harvard University Press.

- de Cesare, S., Gailly, F., Guizzardi, G., Lycett, M., Partridge, C. and Pastor, O. (2016). 4th International Workshop on Ontologies and Conceptual Modeling (Onto.Com). In O. Kutz and S. de Cesare (eds.), *Proceedings of the 2nd Joint Ontology Workshops (JOWO 2016)*, Annecy, France, July 6-9, 2016. CEUR Workshop proceedings, vol. 1660.
- Ceusters, W. and Smith, B. (2010a). Malaria Diagnosis and the Plasmodium Life Cycle: The BFO Perspective. In *Proceedings of the Third Interdisciplinary Ontology Meeting*, Tokyo, Japan, February 27-28, 2010. Tokyo: Keio University Press, pp. 25-34.
- Ceusters, W. and Smith, B. (2010b). Foundations for a realist ontology of mental disease. *Journal of Biomedical Semantics* 1(1), 10.
- Churchland, P. (1981). Eliminative Materialism and Propositional Attitudes. *Journal of Philosophy* 78(2): 67-90.
- Collins, J., Hall, N. and Paul, L. A. (eds.). (2004). *Causation and Counterfactuals*. Cambridge, MA: MIT Press.
- Correia, F. and Schnieder, B. (eds.) (2012). *Metaphysical Grounding: Understanding the Structure of Reality*, Cambridge: Cambridge University Press.
- Dowe, P. (2000). *Physical Causation*. Cambridge: Cambridge University Press.
- Dowe, P. (2004). Causes are Physically Connected to their Effects: Why Preventers and Omissions are not Causes. In C. Hitchcock (ed.), *Contemporary Debates in Philosophy of Science*. Basil Blackwell, pp. 189-196.
- Dummett, M. (1954). Can an Effect Precede its Cause. *Proceedings of the Aristotelian Society* 28(Supplement): 27-44.
- Ferrario, R. and Oltramari, A. (2004). Towards a Computational Ontology of Mind. In A. Varzi and L. Vieu (eds.), *Proceedings of the 3rd International Conference of Formal Ontology in Information Systems (FOIS 2004)*, Torino, Italy, November 4-6, 2004. Amsterdam: IOS Press, pp. 287-297.
- Fitting, M. and Mendelson, R. L. (1998). *First-Order Modal Logic*. Netherlands: Springer.
- Flew, A. (1954). Can an Effect Precede its Cause. *Proceedings of the Aristotelian Society* 28(Supplement): 45-62.
- Fuellen, G., Jansen, L., Cohen, A. A., Luyten, W., Gogol, M., Simm, A., Saul, N., Cirulli, F., Berry, A., Antal, P., Köhling, R., Wouters, B. and Möller, S. (2018). Health and Aging: Unifying Concepts, Scores, Biomarkers and Pathways. *Aging and Disease* 10(4), 10.14336/AD.2018.1030.
- Galton, A. (2012). States, Processes and Events, and the Ontology of Causal Relations. In M. Donnelly and G. Guizzardi (eds.), *Proceedings of the 7th International Conference of Formal Ontology in Information Systems (FOIS 2012)*, Graz, Austria, July 24-27, 2012. Amsterdam: IOS Press, pp. 279-292.

- Galton, A. and Mizoguchi, R. (2009). The Water Falls but the Waterfall does not Fall: New perspectives on Objects, Processes and Events. *Applied Ontology* 4(2): 71-107.
- Glass, T. A., Goodman, S. N., Hernán, M. A. and Samet, J. M. (2013). Causal Inference in Public Health. *Annual Review of Public Health* 34: 61-75.
- Glymour, M. M. and Rudolph, K. E. (2016). Causal inference challenges in social epidemiology: Bias, specificity, and imagination. *Social Sciences & Medicine* 166: 258-265.
- Granger, C. W. J. (1980). Testing for causality: A personal viewpoint. *Journal of Economic Dynamics and Control* 2: 329-352
- Gruber, T. R. (1993). A Translation Approach to Portable Ontologies. *Knowledge Acquisition* 5(2): 199-220.
- Guarino, N. (2009). The Ontological Level: Revisiting 30 Years of Knowledge Representation. In A. Borgida, V. Chaudhri, P. Giorgini and E. Yu (eds.), *Conceptual Modelling: Foundations and Applications. Essays in Honor of John Mylopoulos*. Berlin, Heidelberg: Springer-Verlag, pp. 52-67.
- Guarino, N. (2017). On the Semantics of Ongoing and Future Occurrence Identifiers. In H. Mayr, G. Guizzardi, H. Ma and O. Pastor. (eds.), *Conceptual Modeling. ER 2017. Lecture Notes in Computer Science*, vol. 10650. Springer, Cham.
- Guarino, N., Oberle, D. and Staab, S. (2009). What Is an *Ontology*? In S. Staab and R. Studer (eds.), *Handbook on Ontologies* (Second Edition). Berlin, Heidelberg: Springer-Verlag, pp. 1-17.
- Hall, N. (2000). Causation and the Price of Transitivity. *Journal of Philosophy* 97: 198-222.
- Harris, J. M., Hirst, J. L. and Mossinghoff, M. J. (2000). *Combinatorics and Graph Theory*. New York, USA: Springer-Verlag New York.
- Hastings, J., Ceusters, W., Jensen, M., Mulligan, K. and Smith, B. (2012). Representing Mental Functioning: Ontologies for Mental Health and Disease. Towards an Ontology of Mental Functioning (ICBO Workshop), *The 3rd International Conference on Biomedical Ontology (ICBO 2012)*, Graz, Austria, July 22, 2012.
- Heil, J. and Mele, A. (eds.). (1993). *Mental Causation*. Oxford: Clarendon Press.
- Herre, H. (2010). General Formal Ontology (GFO): A Foundational Ontology for Conceptual Modelling. In R. Poli, M. Healy and A. Kameas (eds.), *Theory and Applications of Ontology: Computer Applications*. Netherlands: Springer, pp. 297-345.
- Horrocks, I., Patel-Schneider, P.-F., McGuinness, D. L. and Welty, C. A. (2007). OWL: a Description-Logic-Based Ontology Language for the Semantic Web. In F. Baader, D. Calvanese, D. L. McGuinness, D. Nardi and P. F. Patel-Schneider (eds.), *The Description Logic Handbook: Theory, Implementation and Applications* (Second Edition). Cambridge: Cambridge University Press, pp. 458-486.

- Jansen, L. (2015). Zur Ontologie sozialer Prozesse. In R. Schützeichel and S. Jordan (eds.), *Prozesse*. Springer VS, Wiesbaden, pp. 17-43.
- Jansen, L. (2018) Functions, Malfunctioning, and Negative Causation. In A. Christian, D. Hommen, N. Retzlaff and G. Schurz (eds.), *Philosophy of Science. European Studies in Philosophy of Science*, vol. 9. Springer, Cham, pp. 117-135.
- Jarrar, M. and Ceusters, W. (2017). Classifying Processes and Basic Formal Ontology. In M. Horridge, P. Lord and J. D. Warrender (eds.), *Proceedings of the 8th International Conference on Biomedical Ontology (ICBO 2017)*, Newcastle upon Tyne, UK, September 13-15, 2017. CEUR Workshop Proceedings, vol. 2137.
- Keet, C. M. (2008). A Formal Theory of Granularity (PhD Thesis). KRDB Research Centre, Faculty of Computer Science, Free University of Bozen-Bolzano, Italy. Retrieved from: <http://www.meteck.org/PhDthesis.html> (Last accessed on June 23, 2019).
- Kerry, R., Eriksen, T. E., Lie, S. A. N., Mumford, S. and Anjum, R. L. (2012). Causation and Evidence-based practice --- an ontological review. *Journal of Evaluation in Clinical Practice* 18(5): 1006-1012.
- Kim, J. (2011). *Philosophy of Mind* (Third Edition). Boulder: Westview Press.
- Kitamura, Y., Sano, T., Namba, K., and Mizoguchi, R. (2002). A Functional Concept Ontology and Its Application to Automatic Identification of Functional Structures. *Advanced Engineering Informatics* 16(2): 145-163.
- Kleinberg, S. and Hripcsak, G. (2011). A review of causal inference for biomedical informatics. *Journal of Biomedical Informatics* 44(6): 1102-1112.
- Kozaki, K., Mizoguchi, R., Imai, T. and Ohe, K. (2012). Identity Tracking of a Disease as a Causal Chain. In R. Cornet and R. Stevens (eds.), *Proceedings of the 3rd International Conference on Biomedical Ontology (ICBO 2012)*, Graz, Austria, July 21-25, 2012. CEUR Workshop Proceedings, vol. 897.
- Kozaki, K., Yamagata, Y., Mizoguchi, R., Imai, T. and Ohe, K. (2017). Disease Compass: a navigation system for disease knowledge based on ontology and linked data techniques. *Journal of Biomedical Informatics* 8: 22.
- Lange, M. (2009). Causation in Classical Mechanics. In H. Beebe, C. Hitchcock and P. Menzies (eds.), *The Oxford Handbook of Causation*. Oxford: Oxford University Press, pp. 649-660.
- Larsen, R. and Hastings, J. (2018). From Affective Science to Psychiatric Disorder: Ontology as a Semantic Bridge. *Frontiers in Psychiatry* 9: 487. doi: 10.3389/fpsy.2018.00487.
- Lehmann, J., Borgo, S., Masolo, C. and Gangemi, A. (2004). Causality and Causation in DOLCE. In A. Varzi and L. Vieu (eds.), *Proceedings of the 3th International Conference of Formal Ontology in Information Systems (FOIS 2004)*, Torino, Italy, November 4–6, 2004. Amsterdam:

IOS Press, pp. 273-284.

- Lemoine, M. (2013). Defining Disease Beyond Conceptual Analysis: An Analysis of Conceptual Analysis in Philosophy of Medicine. *Theoretical Medicine and Bioethics* 34: 309-325.
- Lewis, D. (1972). Psychophysical and Theoretical Identifications. *Australasian Journal of Philosophy* 50: 249-258.
- Lewis, D. (1973a). Causation. *Journal of Philosophy* 70: 556–567. Reprinted in D. Lewis. *Philosophy Papers: Volume 2*. Oxford: Oxford University Press, 1986, pp. 159-171.
- Lewis, D. (1973b). *Counterfactuals*. Oxford: Blackwell.
- Lewis, D. (2000). Causation as influence. *Journal of Philosophy* 97(4): 182-197. Reprinted in Collins, Hall and Paul (2004), pp. 75-106.
- Mackie, J. L. (1980). *The Cement of the Universe: A Study of Causation*. Oxford: Clarendon Press.
- Maldonado, G. (2013). Toward a clearer understanding of causal concepts in epidemiology. *Annals of Epidemiology* 23: 743-749.
- Martin, W. (2014). Making valid causal inferences from observational data. *Preventive Veterinary Medicine* 113: 281-297.
- Masolo, C. (2010). Understanding Ontological Levels. In F. Lin, U. Sattler and M. Truszczyński (eds.), *Proceedings of the Twelfth International Conference on the Principles of Knowledge Representation and Reasoning (KR 2010)*, Toronto, Ontario, Canada, May 9-13, 2010. Menlo Park, California: AAAI Press, pp. 258-268.
- Merrill, G. H. (2010a). Ontological realism: Methodology or misdirection? *Applied Ontology* 5(2): 79-108.
- Merrill, G. H. (2010b). Realism and reference ontologies: Considerations, reflections and problems. *Applied Ontology* 5(3-4): 189-221.
- Michalek, H. (2009). A Formal Ontological Approach to Causality embedded in the Top-Level Ontology of GFO (General Formal Ontology) (PhD Thesis). Retrieved from: www.onto-med.de/publications/2009/A_formal_ontological_approach_to_causality.pdf (Last accessed on June 23, 2019).
- Mizoguchi, R. (2011). YAMATO: Yet Another More Advanced Top-level Ontology. In K. Taylor, T. Meyer and M. Orgun (eds.), *Proceedings of the Sixth Australasian Ontology Workshop (Advanced in Ontologies)*, Adelaide, Australia, December 7, 2010. University of Adelaide, pp. 1-17.
- Mizoguchi, R., Galton, A., Kitamura, Y. and Kozaki, K. (2015). Families of roles: A new theory of occurrent-dependent roles. *Applied Ontology* 10(3-4): 367-399.
- Mizoguchi, R. and Kitamura, Y. (2000). Foundation of Knowledge Systematization: Role of Ontological Engineering. In R. Roy (ed.), *Industrial Knowledge Management --- A Micro Level*

Approach. Springer-Verlag, pp. 17-36.

- Mizoguchi, R. and Kitamura, Y. (2009). A Functional Ontology of Artifacts. *The Monist* 92(3): 387-402.
- Mizoguchi, R., Kitamura, Y. and Borgo, S. (2012). Towards a Unified Definition of Function. In M. Donnelly and G. Guizzardi (eds.), *Proceedings of the 7th International Conference of Formal Ontology in Information Systems (FOIS 2012)*, Graz, Austria, July 24-27, 2012. Amsterdam: IOS Press, pp. 103-116.
- Mizoguchi, R., Kitamura, Y. and Borgo, S. (2016). A unifying definition for artifact and biological functions. *Applied Ontology* 11(2): 129-154.
- Mizoguchi, R., Kozaki, K., Kou, H., Yamagata, Y., Imai, T., Waki, K., and Ohe, K. (2011). River Flow Model of Diseases. In O. Bodenreider, M. E. Martone and A. Ruttenberg (eds.), *Proceedings of the 2nd International Conference on Biomedical Ontology (ICBO 2011)*, Buffalo, New York, USA, July 26-30, 2011. CEUR Workshop Proceedings, vol. 833, pp. 63-70.
- Mizoguchi, R., Sunagawa, E., Kozaki, K. and Kitamura, Y. (2007). The model of roles within an ontology development tool: Hozo. *Applied Ontology* 2(2): 159-179.
- Mumford, S. and Anjum, R. L. (2011). *Getting Causes From Powers*. Oxford: Oxford University Press.
- National Advisory Mental Health Council Workgroup. (2010) From Discovery to Cure: Accelerating the Development of New and Personalized Interventions for Mental Illness 2010. Available online at: <http://www.nimh.nih.gov/about/advisory-boards-and-groups/namhc/reports/fromdiscoverytocure.pdf> (Last accessed on June 23, 2019)
- Neuhaus, F. (2017). On the Definition of ‘Ontology’. In A. Galton and F. Neuhaus (eds.), *Proceedings of the 3rd Joint Ontology Workshops (JOWO 2017)*, Bozen-Bolzano, Italy, September 21-23, 2017. CEUR Workshop proceedings, vol. 2050.
- O’Connor, T. (2000). *Persons and Causes: The Metaphysics of Free Will*. New York: Oxford University Press.
- Paul, L. A. and Hall, N. (2013). *Causation: A Use’s Guide*. Oxford: Oxford University Press.
- Pearl, J. (2009). *Causality: Models, Reasoning, and Inference* (Second Edition). Cambridge: Cambridge University Press.
- Price, H. and Corry, R. (eds.) (2006). *Causation, Physics, and the Constitution of Reality: Russell’s Republic Revisited*. Oxford: Oxford University Press.
- Putnam, H. (1967). Psychological Predicates. In W.H. Capitan and D.D. Merrill (eds.), *Art, Mind, and Religion*. Pittsburgh: University of Pittsburgh Press, pp. 37-48.
- Read, S. (1988). *Relevant Logic*. Oxford: Blackwell.
- Reznek, L. (1987). *The Nature of Disease*. New York: Routledge.

- Ross, D. and Spurrett, D. (2004). What to say to a skeptical metaphysician: A defense manual for cognitive and behavioral scientists. *Behavioral and Brain Sciences* 27(5): 603-627.
- Röhl, J. and Jansen, L. (2011). Representing dispositions. *Journal of Biomedical Semantics* 2(Supplement 4), S4.
- Röhl, J. and Jansen, L. (2014). Why functions are not special dispositions: an improved classification of realizables for top-level ontologies. *Journal of Biomedical Semantics* 5, 27.
- Rothman, K. J. and Greenland, S. (2005). Causation and Causal Inference in Epidemiology. *American Journal of Public Health* 95 (Suppl 1): S144-S150.
- Rovetto, R. J. and Mizoguchi, R. (2015). Causality and the ontology of disease. *Applied Ontology* 10(2): 79-105.
- Russell, B. (1913). On the Notion of Cause. *Proceedings of the Aristotelian Society* 13: 1-26.
- Russo, F. and Williamson, J. (2007). Interpreting Causality in the Health Sciences. *International Studies in the Philosophy of Studies* 21(2): 157-170.
- Schaffer, J. (2000). Causation by Disconnection. *Philosophy of Science* 67(2): 285-300.
- Schaffer, J. (2004). Causes Need Not Be Physically Connected to Their Effects: The Case for Negative Causation. In C. Hitchcock (ed.), *Contemporary Debates in Philosophy of Science*. Malden, MA: Blackwell Publishing, pp. 197-216.
- Schaffer, J. (2005). Contrastive Causation. *Philosophical Review* 114: 327-358.
- Schaffer, J. (2016). The Metaphysics of Causation. In E. N. Zalta (ed.), *The Stanford Encyclopedia of Philosophy* (Fall 2016 Edition), Available online at: <https://plato.stanford.edu/archives/fall2016/entries/causation-metaphysics/> (Last accessed on June 23, 2019).
- Scheuermann, R. H., Ceusters, W. and Smith, B. (2009). Toward an ontological treatment of disease and diagnosis. *Summit on Translational Bioinformatics*: 116-120.
- Sinhababu, N. (2013). The Desire-Belief Account of Intention Explains Everything. *Noûs* 47(4): 680-696.
- Smart, J. J. C. (1959). Sensations and Brain Processes. *Philosophical Review* 68: 141-156.
- Smith, B. (2004). Beyond Concepts: Ontology as Reality Representation. In A. C. Varzi and L. Vieu (eds.), *Proceedings of the 4th International Conference on Formal Ontology in Information Systems (FOIS 2004)*, Torino, Italy, November 4-6, 2004. Amsterdam: IOS Press, pp. 31-42.
- Smith, S. and Ceusters, W. (2010). Ontological realism: A methodology for coordinated evolution of scientific ontologies. *Applied Ontology* 5(3-4): 139-188.
- Smith B., Kusnierczyk, W., Schober, D. and Ceusters, W. (2006). Towards a Reference Terminology for Ontology Research and Development in the Biomedical Domain. In O. Bodenreider (ed.), *Proceedings of the 2nd International Workshop on Formal Biomedical*

Knowledge Representation (KR-MED 2006), Maryland, USA, November 8, 2006. CEUR Workshop Proceedings, vol. 222, pp. 57-66.

- Smith, B. et al. (2007). The OBO Foundry: coordinated evolution of ontologies to support biomedical data integration. *Nature Biotechnology* 25: 1251-1255.
- Smith, B. et al. (2015). Basic Formal Ontology 2.0: Specification and User's Guide. Available online at: <https://github.com/BFO-ontology/BFO> (Last accessed on June 23, 2019).
- Spear, A. D., Ceusters, W. and Smith, B. (2016). Functions in Basic Formal Ontology. *Applied Ontology* 11(2): 103-128.
- Stalnaker, R. (1968). A Theory of Conditionals. In N. Rescher (ed.), *Studies in Logical Theory: American Philosophical Quarterly Monograph Series, No. 2.*, Oxford: Blackwell, pp. 98-112.
- Stalnaker, R. (2014). *Context*. Oxford: Oxford University Press.
- Stange, K. C. (2009). The Problem of Fragmentation and the Need for Integrative Solutions. *The Annals of Family Medicine* 7: 100-103.
- Strand, A. and Parkkinen, V. P. (2014). Causal knowledge in evidence-based medicine. In reply to Kerry et al.'s causation and evidence-based practice: an ontological review. *Journal of Evaluation in Clinical Practice* 20(6): 981-984.
- Studer, R., Benjamins, R. and Fensel, D. (1998). Knowledge Engineering: Principles and Methods. *Data & Knowledge Engineering* 25(1-2): 161-198.
- Taylor, R. (1966). *Action and Purpose*. Upper Saddle River: Prentice Hall.
- Tooley, M. (1987). *Causation: A Realist Approach*. Oxford: Clarendon Press.
- Trypuz, R. (2007). Formal Ontology of Action: a unifying approach, (PhD Dissertation), 2007. Retrieved from https://www.researchgate.net/publication/233990262_Formal_Ontology_of_Action_a_unifying_approach (Last accessed on June 23, 2019).
- van Dalen, D. (1984). Intuitionistic logic. In D. Gabby and F. Guenther (eds.), *Handbook of Philosophical Logic III*, pp. 225-239.
- Vandenbroucke, J. P., Broadbent, A. and Pearce, N. (2016). Causality and causal inference in epidemiology: the need for a pluralistic approach. *International Journal of Epidemiology* 45(6): 1776-1786.
- Visser, A. (1998). An Overview of Interpretability Logic. In M. Kracht et al. (eds.), *Advances in Modal Logic* (Volume 1). Stanford: CSLI Publications, pp. 307-359.
- Wakefield, J. C. (1992). The Concept of Mental Disorder: On the Boundary Between Biological Facts and Social Values. *American Psychologist* 47: 373-388.
- Wakefield, J. C. (2014). The Biostatistical Theory Versus the Harmful Dysfunction Analysis, Part 1: Is Part-Dysfunction a Sufficient Condition for Medical Disorder? *Journal of Medicine and Philosophy* 39(6): 648-682.

- Waldmann, M. R. (ed.). (2017). *The Oxford Handbook of Causal Reasoning*. Oxford: Oxford University Press.
- Walter, S. and Heckmann H. (eds.). (2003). *Physicalism and Mental Causation: The Metaphysics of Mind and Action*. Exeter: Imprint Academic.
- Weber, E. (2008). The Debate between Causal Realism and Causal Constructivism: Metaphilosophical Reflections. *Philosophia* 81: 59-71.
- Williams, N. (2007). The Factory Model of Disease. *The Monist* 90(4): 555-584.
- Wilson, A. (2018). Metaphysical Causation. *Noûs* 52(4): 723-751.
- Woodward, J. (2003). *Making Things Happen: A Theory of Causal Explanation*. Oxford: Oxford University Press.
- Wooldridge, M. J. (2000). *Reasoning about Rational Agents*. Cambridge, MA: MIT Press.
- World Health Organization (2018). *International Statistical Classification of Diseases and Related Health Problems - 11th Revision - Version for 2018*. Geneva: World Health Organization.
- Yamagata, Y., Kozaki, K., Imai, T., Ohe, K. and Mizoguchi, R. (2014). An ontological modeling approach for abnormal states and its application in the medical domain. *Journal of Biomedical Semantics* 5(1): 23.

List of publications

Journal paper

Toyoshima, F., Mizoguchi, R. and Ikeda, M. (2019). Causation: A functional perspective. *Applied Ontology* 14(1): 43-78.

Conference proceedings paper

Toyoshima, F., Mizoguchi, R. and Ikeda, M. (2017). Causation and the River Flow Model of Diseases. In M. Horridge, P. Lord, J. D. Warrender (eds.), *Proceedings of the 8th International Conference on Biomedical Ontology (ICBO 2017)*, Newcastle-upon-Tyne, United Kingdom, September 13-15, 2017. CEUR Workshop Proceedings, vol. 2137, 6 pages.

Poster presentation

Toyoshima, F. (2018). Towards an Ontological Module for Mental Disease. *Early Career Symposium, the 10th International Conference on Formal Ontology in Information Systems (FOIS 2018)*, Cape Town, South Africa, September 17-21, 2018.