

THE REPRESENTATION PROBLEM OF CAUSAL RELATIONSHIPS IN
COMPLEX SYSTEMS MODELING

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ABSTRACT

THE REPRESENTATION PROBLEM OF CAUSAL RELATIONSHIPS IN COMPLEX SYSTEMS MODELING

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An adequate representation for causal relations of a phenomenon offers (i) an explanatory architecture of the phenomenon; (ii) a basis for modeling the phenomenon; and thus, (iii) a way to make predictions about similar events. However, the criterion of the ‘right’ way to represent causation is highly disputed among econometrists and computer scientists as well as philosophers. Each representational framework may bear different ontological commitments concerning the nature of the causal connection. In this thesis, it is argued that the current representations embrace an ontology bound to linearity and will remain inadequate to represent complex systems as long as linearity is presumed. To characterize the relation between cause and effect in those systems it is needed that a representational framework for nonlinearly interacting complex phenomena. As a conclusion, the major obstacle in the way of representing nonlinear causation addressed as an ontological problem.

Keywords: Causality, Complex Systems, Modeling, Nonlinearity, Relatedness.

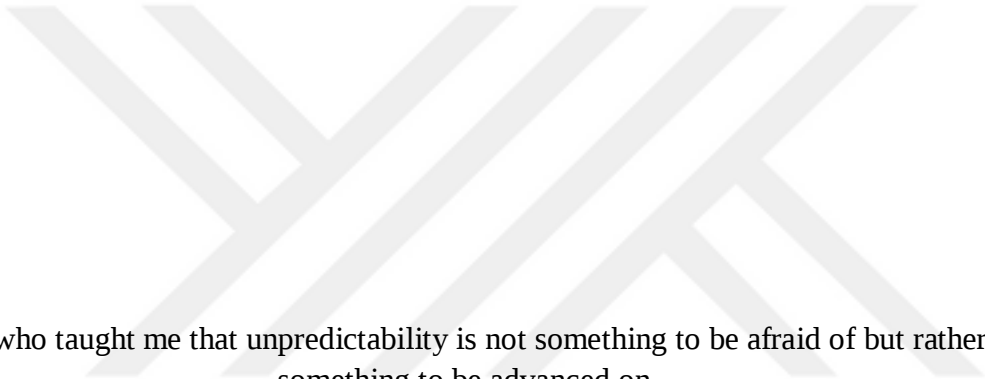
ÖZ

KOMPLEKS SİSTEMLERİN MODELLENMESİNDE NEDENSEL İLİŞKİLERİN TEMSİLİ PROBLEMİ

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Bir fenomenin temsil edilmiş nedensel ilişkileri fenomene dair (i) açıklayıcı yapısını (ii) modelleme için altyapısını, ve (iii) benzer durumlar hakkında öngöründe bulunmayı sunar. Ancak, nedensel ilişkileri ‘doğru’ temsil etme biçimleri filozoflar kadar ekonometristler ve bilgisayar bilimciler tarafından da çokça tartışmalı bir konu olmuştur. Mevcut temsil yapılarında nedensel bağıntının doğasına dair farklı ontolojik bağlanımlar bulunabilir. Bu temsil yapılarının lineerliğe dayalı bir ontolojisi olduğunu ve bu lineerlik varsayıldığı sürece söz konusu temsillerin kompleks sistemleri temsil etmede yetersiz kalmaya devam edeceklerini iddia edilmiştir. Kompleks sistemlerdeki neden-sonuç arasındaki ilişkinin betimlenmesi için, lineer olmayan yollarla etkileşen kompleks fenomenlerin temsilini veren bir “lineer-olmayan nedensel açıklama”ya ihtiyaç duyulmaktadır. Sonuç olarak, lineer-olmayan nedenselliği temsil etmedeki en büyük engelin ontolojik bir sorun olduğuna işaret edilmiştir.

Anahtar Kelimeler: Nedensellik, Kompleks Sistemler, Modelleme, Lineer-olmayan, İlişkisellik.



To who taught me that unpredictability is not something to be afraid of but rather
something to be advanced on.

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When I just started to write down this thesis I lost my mother who was my whole family. I kept studying harder because in deep down I believed that there are causal processes that lead to her death by cancer and I might slow down the process even if I could not stop it. The commitment that I have to this thesis' subject is mostly because of her. Yet, there were many others who believe in me and what it is intended to achieve in this study. Foremost my dearest friend Gamze Uzun was always there for me. My best friends, of course, Zeynep Yasak, Ece Senem Kondakcı, Doğan Kahraman, Tolgahan Toy who patiently listened my complaining about the thesis writing process, were there for me. I would like to thank Ece specifically for mocking me around in order to propel me to study more, although she did not study at all but still forced me to come with her to METU Library. Besides all, Sinem Odabaş who is my roommate should be acknowledged for being such a fun, a great critique of my dishes, and her tolerance of my study style at home. I would like to extend my sincere thanks to Şükrü Bezen for his support in many aspects. He spent hours for the latex code of this thesis that I somewhat distorted and could not fix it back. Indeed, this thesis embodies his efforts also.

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TABLE OF CONTENTS

PLAGIARISM.	iii
ABSTRACT.	iv
ÖZ.	v
DEDICATION.	vi
ACKNOWLEDGMENTS.	vii
TABLE OF CONTENTS.	viii
LIST OF FIGURES.	x
CHAPTER	
1. INTRODUCTION.	1
1.1 Causation as a Problem in Science and Philosophy.	2
1.2 Causation and Modeling.	5
2. THE REPRESENTATION PROBLEM IN COMPLEX SYSTEMS	
MODELING.	10
2.1 Systems.	12
2.2 Linear Systems.	13
2.2.1 Linearity and Linear Models.	13
2.2.2 Linear Assumptions in Data Generation Process. . .	15
2.3 Complex Systems.	19
2.3.1 Complexity.	19
2.3.2 Chaos.	21
2.3.3 Characteristics of Complex Systems.	22
2.3.4 Nonlinearity.	25
2.3.4.1 Order and Disorder.	26
2.3.4.2 Spatiotemporal Patterns.	28
2.4. The Insufficiency of Linear Methods to Represent Complex Systems. . .	29
2.4.1 Linear Methods.	30
2.4.2 Nonlinearity and Complexity.	32

3. CAUSAL REPRESENTATIONS AND THE CONCERNING PROBLEMS.	35
3.1 Causal Representations in Models.	36
3.1.1 Causation as Dependence.	37
3.1.2 Causation as Production.	49
3.2 Causal Models.	50
3.2.1 Structural Equation Models.	51
3.2.2 Causal Graph.	52
3.3 Linear Assumptions in Causal Models.	56
3.4 Linearity, Nonlinearity, and Causality.	59
4. IS THERE AN ALTERNATIVE?	62
4.1 On Possibility of a Nonlinear Causal Account.	63
4.2 On the Ontological Status of Relations in Complex Systems.	70
5. CONCLUSION.	76
REFERENCES.	78
APPENDICES	
A TURKISH SUMMARY/TÜRKÇE ÖZET.	89
B TEZ FOTOKOPİ İZİN FORMU.	100

LIST OF FIGURES

Figure 2.1 a. A model of system with its internal states, retrieved from Gorinevsky (2005), b. Another exemplar for modeling the systems, retrieved from Hover (2009).	13
Figure 2.2 A simple representation of a linear electrical circuit where R stands for resistor, v as battery and i as current.	15
Figure 2.3 An illustration for the state of interest and collected data.	16
Figure 2.4 Collected and organized data of first-grade children. Retrieved from Gravetter and Wallnau (2016).	17
Figure 2.5 Descriptive statistics for the samples A and B. Retrieved from Gravetter and Wallnau (2016).	18
Figure 2.6 An illustration for current state of interest and interpreted data.	19
Figure 2.7 (a) A simple representation of a feedback relation, (b) An example of a positive feedback loop: "a fluid particle hotter than its environment encounters ever colder fluid as it rises, which leads to the instability" (Manneville, 2006).	23
Figure 2.8 Snapshots of Rayleigh-Bénard cell convection from the experiment conducted by Pfander and Haupt (2015).	24
Figure 2.9 Knitting patterns.	28
Figure 2.10 A conceptual view of the spatial and temporal order in the behavior of neural ensembles (retrieved from Tognoli and Kelso, 2013). Complex systems are in between order and disorder in time and space.	29
Figure 2.11 A normal distribution graph (adapted from Gravetter and Wallnau, 2016).	31
Figure 3.1 A directed acyclic graph (DAG).	41
Figure 3.2 A directed acyclic graph (DAG) with the distributed probabilities. The probabilities to specify are $P(A)$, $P(B)$, $P(C A, B)$, $P(E C)$, $P(C D)$, $P(F E)$, and $P(G D, E, F)$. Retrieved from Glymour and Cooper (1999).	41

Figure 3.3 A causal Bayes net that is retrieved from Glymour and Cooper (1999).....	41
Figure 3.4 A causal DAG.....	42
Figure 3.5 An example for spurious regularity retrieved from Hitchcock (2018) .	42
Figure 3.6 The variable E cancels (or say, inhibits) the variable D	43
Figure 3.7 In this case, collider variable is C	43
Figure 3.8 A causal Bayes net with a representation for causal mixture from Glymour and Cooper (1999).	43
Figure 3.9 An oversimplified suggestion of representing causal mixture models. .	44
Figure 3.10 The causal structures for given equations of 3.2 and 3.3, 3.4. Retrieved from Hausman and Woodward (1999).....	46
Figure 3.11 Path diagram of $X = \beta M + U$ and $M = A$. It is interpreted as A causes M and M causes X under the conditions of U	52
Figure 4.1 Image is retrieved from Hordijk and Steel (2015).	67
Figure 4.2 A vector model and causal powers of a, b, c, d, e, f which give rise to R as a sum of the causal powers. Retrived from Mumford and Anjum (2011)	72
Figure 4.3 3D quality space by Mumford and Anjum (2011)	72
Figure 4.4 Image is retrieved from Mumford and Anjum (2011).	73
Figure 4.5 A case for two drugs introduced simultaneously react reverse effect. G space represents wellness of the patient and F space stands for illness, as put by Mumford and Anjum (2011). Notice that causal powers do not intersect even in the cases where causes are ‘acting together’	74

CHAPTER 1

INTRODUCTION

Modeling in sciences has an essential role of representing the systems in question to serve as an instrument of understanding and prediction. What makes models so important to sciences is that, especially for some systems, direct intervention to the systems may be inaccessible. Complex systems modeling considering the intricate nature of those systems, thus, constitutes a challenge for understanding and prediction. One of the main reasons of that being challenge is representing causal relationships.

The aim of this thesis is to expose a problem with the ontological commitments that are made by causal models. Models on offer, often, are not intended to be bound to any ontological claim on causal relations. Rather, causality is represented as a lawlike relation that is to say a fixed bond which is applicable to any system. However, even at this point, to be noticed, an ontological commitment is already made considering the identifying properties of a causal relation. To be more precise, it is assumed that the nature of causality is such a thing that there are fixed, lawlike relations that hold across entities to affect and be affected by. Thus, this reciprocity between models and entities to be modeled itself require a philosophical scrutiny, yet, in this thesis, it is restricted to causal models that are proposed to be the representations of causal relationships in complex systems. In this sense, this thesis will question the applicability of causal models to complex systems. By the term of complex systems, however, it is denoted that the ‘whole’s that are constituted by various kinds of interconnected elements through nonlinear relations. What makes a system complex is the web of interrelations of the system. It is because given the same components, systems may still differ. The difference is built up by rearrangements of the components.

Multifunctionality, degeneracy, self-organization, autopoiesis, emergence are all arise according to such interrelations that are dynamically rebuilt in each state of the systems. Besides such internal dynamicity of the complex systems that those systems are co-evolving with their environments. That is to say, complex systems actively affect and get affected by the media that they are in. So, complex systems are also coupled with their environments.

In modeling, there are fundamental problems considering the representations of these highly intricate relations. The most striking problem is that in order to represent a complex system, simplification of the system in question has to be made. If the systems are consisted of billions of heterogeneous components (such as neurons, ganglions, pyramidal cells in the human brain) and even more relations between those components what should be done to gain a simplified yet adequate picture of these systems? Or, is it possible to represent complex systems in a simplified way but, at the same time, to be inclusive enough to capture such a complex causal structure? These concerns seek further clarification.

1.1 Causation as a Problem in Science and Philosophy

The main problem with the causation is that: we know, or to say least, we assume that there are some events that cause some others, yet we do not know what causation exactly is. It seems that smoking causes lung cancer, fast-food consumption causes obesity, a lack of specific gene causes to a disease, carbon emission causes global warming, and so. Also, we can predict and/or control such events, for example the lacking gene might be grown in laboratories and when it is injected to the patient, it can cure the disease. Or, with a dietary routine obesity could be prevented. On the other hand, these are not always the cases. Even though a person, who does not consume any fast food may still suffer from obesity. For instance, obesity may be caused by excessive hormone secretion. In this case, should we disregard all the cases that we observed obesity as a concomitant of eating fast food? Or, if we should count on causality in both of the cases, then what is the criterion of identifying causal relations? There are different answers for that, but, today there is no consensus on the identifying conditions or on the definition of causality. In scientific practice the answer is often given in terms of statistical inferences. Philosophical approach, however, is twofold: metaphysical and

epistemic. The definition of causation which concerns the realm of causal bonds constitute the metaphysical research paradigm whereas epistemic research focuses on our knowledge on causal relations. In that sense, scientific answers are also included in epistemic studies. It should be noted that, philosophically, it is a problem on its own that where to draw a line between ontology and epistemology. At this point, it can be said that this thesis walks on that alleged line; while causal models which are epistemic ‘devices’ for representing causality are investigated, the discrepancy between these models and the nature of the modeled entities will be questioned. The main problem, it will be argued, stems from the definition of causality that is embedded in causal models on offer.

The systematic reasoning on definition of causality in philosophy can be traced back to Aristotle who has proposed a typology of causes (material, formal, efficient, final causes) to be found in the substances. During the medieval period the Aristotelian typology has dominated the studies on causal understanding of the nature, however, it was a theological interpretation of Aristotle’s works by the scholastic thinkers such as Saint Thomas Aquinas (Wallace, 1972). God, in that sense, is seen as the ultimate cause of all the ‘things’ replaced Aristotle’s unmoved mover. Besides Occasionalism which states that God is the only cause of all beings in the world, there are similar accounts of causation. The common ground in those accounts is that causation is taken to be granted for being necessary relation where such necessity could only ever be provided by God. On the other hand, there was a striking problem within Scholastic view on causation, if God is the cause of everything then what is left to bodies to do? With such legacy, thus, philosophers of early modern period had to deal with metaphysical problems of causation. In fact, early modern philosophers “with the exception of Hobbes, hold that some knowledge of God is critical to understanding nature and natural laws” (Clatterbaugh, 1999). Cartesian causation, for instance, accounts such laws of causal interactions where those laws are carried out by a law-giver, namely, God. In the works of Spinoza, however, God as *natura naturans* constitutes a non-scholastic depiction of God. But, similar to early modern philosophers, causal connection has seen as logical connection where according to rationalist school, is the way how the world is constructed. It should be also noted that Francis Bacon

whom lived at the very beginnings of modern era, suggests that a methodology for systematic causal inference by induction (Reiss, 2007). Bacon has not (seem to) interested in metaphysical side of causality but rather its importance in controlling the nature. In late modern philosophies, it can be seen that a similar approach to causation in order to “identify genuine causal connections” (Clatterbaugh, 1999) than the metaphysical explanations of how bodies interact.

David Hume, in that sense, challenged the common understanding of causality in terms of psychological experience of causal events. Consequently he found that whenever one refers causality all s/he has an idea of events that show (i) contiguity in space, (ii) temporal priority and, (iii) constant conjunction. It can be said that following attempts to describe causation, including contemporary efforts, rely on that analysis of causation put by Hume. Those accounts, thus, can be divided into two groups roughly: causal realists and reductionists. Causal realists are also recalled as anti-Humeans since it is thought that causal relation exists beyond the human experience. Reductionism in causality, contrarily, accounts causation in non-causal terms. Note that, one can be a causal realist while methodologically bound to reductive analysis of causality. In fact, causal models of today are mostly fell under this category.

Reductionist attempts to describe causation employed logical methods which are followed by mathematical analyses. The logical analysis of causation initially set by John Stuart Mill and advanced by John Leslie Mackie with INUS conditions where necessary and sufficient causes are discretely studied. Following those developments, logical analysis has gained its current form in terms of counterfactuality by David Lewis and his followers. The governing idea in here is that analyzing causal state-ments in conditional form. However, some philosophers like Curt John Ducasse are relied on the idea which is a stronger version of logical analysis: the ‘correct’ definition of the causal relation can only be given by conditionals, and the aspect of “constant conjunction is, therefore, no part of it” (Sosa and Tooley, 1993). A major problem within this framework is that in cases where there are more than one cause (‘overdetermination’), it becomes harder to represent causality in the form of conditional. The mathematical analysis, on the other hand, takes causation as a ‘functional dependency’. To represent causality in

functional dependencies was first made precise by Hans Reichenbach. He paved the way for causal inference from probabilistic dependencies since he put the idea that “simultaneous correlated events must have prior common causes” (Arntzenius, 2010). By this way, causality could be detected in correlations¹. Thus he proposed a principle which suggests that “two factors X and Y neither of which causes the other, if X and Y have a cause C in common (and C is the only factor in common in their causal past), then, $P(X.Y/C) = P(X/C)P(Y/C)$ ” (ibid). Today, it is proved that this principle is untenable in many cases, yet, it led the way of formalizing ‘conditional independencies’ among variables. Indeed, the principle of ‘causal Markov condition’ (Spirtes and Glymour, 1993) directly derived from the common cause principle. The causal Markov condition simply indicates that given its direct causes a variable is probabilistically independent of its non-effects. Thus it implies a version of common cause principle:

If coincidences of two events A and B occur more frequently than would correspond to their independent occurrence, that is, if the events satisfy relation $P(A.B) > P(A).P(B)$, then there exists a common cause C for these events such that the fork ACB is conjunctive, that is, satisfies relations $P(C, A.B) = P(C, A).P(C, B)$, $P(\bar{C}, A.B) = P(\bar{C}, A).P(\bar{C}, B)$, $P(C, A) > P(\bar{C}, A)$, $P(C, B) > P(\bar{C}, B)$. (Reichenbach, 1956)

Based on the concept of conditional independency, probabilistic analyses of causation are advanced by Patrick Suppes, Irving John Good, Wolfgang Spohn, John Williamson, Judea Pearl, Peter Spirtes, Clark Glymour, and Richard Scheines. The causal models that are investigated in this thesis, in that sense, embody both logical analysis (counterfactuals) and mathematical representations (probabilistic (in-) dependencies) of causation.

1.2 Causation and Modeling

The tools for modeling that are available to us today rely on the assumption that the systems behave linearly. However, there such systems that do not show linearity at all which are – reasonably, called nonlinear systems. Thus, when the linear methodology is applied to nonlinear data we face an incompatibility between state-of-affairs² and representations of them. Causal models that are hitherto put forward,

¹ See Reichenbach (1956).

² The way things are.

are also based on such linear methodology. In this thesis, it is argued that it is not plausible to apply those models to understand complex (thus, nonlinear) systems. This thesis, in this sense, suggests other routes to model causal relations in complex systems by exposing some handicaps concerning the current causal models. Thus, firstly, it will be introduced that the basics of the representations of systems. The discussions will start from the very beginnings of what is meant by systems and will extent to how to model them. Since the context is bound to modeling, the mathematical representation of the systems will be presented. Based on mathematics of the systems, models are distinguished as dynamical and stationary based on the behavior that is the way the output produced by the systems. Stationary systems are out of the context since they tend to stay in a balanced state which means that there is no activity and/or out- put production within those systems. Dynamical systems, on the other hand, may behave in linear or nonlinear way. Notice that throughout this thesis the deterministic systems are the ones that under investigation. If the systems are additive and show homogeny, consequently, the response (to say, the next state) is determined to be the linear combination of the previous (or initial) states. In this case systems are called linear. Linear combinations, mathematically, do hold analytical solutions. That is to say, each component are independent, thus adding or subtracting do not affect the nature of the components. Here, mathematical components stand in lieu of the decomposed parts of the represented entity.

Linear models, thus, are perfect for representing analyzable entities, for example the electronic devices or like, watches. In linear models, due to the properties of additivity and homogeneity, arrangement of the parts is nothing but a linear combination. Nonlinearity, as it is expected to be, lacks additivity and homogeneity yet it is representable as a function. The lacking of those properties, mathematically, hinders any analytical solutions for such functions. Yet, as a representation of an entity, it is not restrictive in the sense that any system that behaves nonlinearly is not de facto unanalyzable. We can analyze the biological organisms, for example. Rather, it has other restrictions/implications in terms of modeling such nonlinear behaviors. But to model the systems, at first, we need data.

Data as a first step to represent an entity is generated through a process. This process is usually twofold: data collection and data interpretation. There are statistical assumptions made during data generation process. Setting aside the assumptions like the absence of any practical errors, it is exposed that these assumptions promote linearity. It is noteworthy since even though we expect from some systems to behave nonlinearly, at the very beginning, the collected data from the systems become linearized (at least to some extent) with such statistical assumptions. Throughout this thesis it will be discussed that whether linearization (at least to some extent) is a must in order to represent the systems.

Data interpretation process consisted of a set of inference procedures that rely on the statistical assumptions. What to read from the collected data is up to such assumptions. For example if positive correlations assumed to be the indicators of a causal relation, then the variables of interest will be interpreted as causally linked. In this sense, the classical debate on whether correlation implies causation comes up at this level of data generation.

What about nonlinearity? To dig into that issue there are two distinctions have to be made. Nonlinearity may be observed in simple systems like the behavior of a pendulum as well as complex systems. In that sense, complexity as a technical term seeks clarification in the context of systems. That is what it is intended to achieve in section 2.3.1. Hence, computational complexity will be distinguished from the term of complex systems. The relation that they have, on the other hand, will be briefly mentioned. The other distinction is made between the chaos and the complex systems. The phenomenon of chaos, indeed, is nonlinear in its nature; however the complex systems which are intrinsically nonlinear, are not necessarily chaotic. The reasons will be mentioned in the section 2.3.2. After those distinctions, it will be discussed that the characteristics of the complex systems in general. It will be emphasized that we observe such characteristics within complex systems due to nonlinear relations. What it is meant by nonlinearity also needs to be discussed. Thus, the focus will be on the term of nonlinearity in the context of complex systems and the patterns of behavior. How order is rebuilt from instability of the systems will constitute the main inquiry of the section 2.3.4.1. Following that, the specific features of the relations (that are causal) built up in those systems will be

put forward. My objective for the section 2.3.4.2 is to underline the importance of the relatedness among the complex systems.

Chapter 2 will be concluded with a comparison between the linear assumptions and the basic features of the complex systems. It is intended to show the gap between linear methods and the complex systems which constitute the application domain of those methods. It will be explicated that the linear methods are insufficient to represent complex systems. Chapter 3 comprises a general analysis of causal theories that are referred in causal models on offer. The analysis aims to show the reciprocity between the causal theories and the causal models (and/or modeling techniques). Hence the representations of causal relations will be categorized according to their ontological commitments. Dependence and Production are the main categories for the representational frameworks of causation. This categorization is made in accordance with the recent literature, yet, in some details it differs from those available classifications. An example for that is a relatively new account of causal modeling is the causal emergence account which is put forward by Erik Hoel, Larissa Albantakis, and Giulio Tononi. This account is categorized under the production framework (in accordance with Russo and Illari's classification), and more specifically, under the information-theoretical accounts for causation. It seems that, in fact, there is no strict line between those two frameworks of causal representations. In the same line Hoel denotes that information-theoretical account might be broad enough to include dependence accounts. I found such discussions on the information theoretical account of causation are fascinating but also, too demanding to be fairly summarized in a master's thesis; thus in this thesis information-theoretic accounts will not be explained in detail. In that sense, to remain adherent to the philosophical literature, it will be referred that the structural equation models (SEMs) and the causal graphs that are advanced by Judea Pearl, when the term of causal models is recalled. In fact, in section 3.2, It is put that SEMs as the underlying idea behind the causal graphs as Pearl also admits in his writings. Besides its success in modeling linear systems, however, these causal models seem to fail in order to model the causal relations among the complex nonlinear systems. The proofs are listed in section 3.3 which simply aims to expose that causal models on offer are strictly tied to linearity assumptions. In other words,

the linear assumptions that are discussed in section 2.2, are also embedded in causal models. However, the linear methods are proved to be insufficient, then, how can we expect that causal models that assume linearity to be sufficient representations of causal relations in complex systems? A list of reasons, in accordance to the list in section 2.4, is made in order to explicitly compare the linear assumptions in causal models with the complex systems which, in this context, constitute the entities that are to be modeled. Hence in this thesis, it is intended to make discernable that the discrepancy between the ontological commitments that are made in causal models and the realm of the complex systems.

Chapter 4 is reserved for the discussions of the possible solutions for representing causal relations in complex nonlinearity. Is linearization process (concerning the models) a requirement for to be represented? Is it possible to account a nonlinear causal framework for causal relations in complex systems? If possible then what steps have to be taken? If not what are the implications of that? Such questions will be asked throughout that chapter. In section 4.2, I ponder about the alternative theoretical frameworks for causal models through a discussion on the ontological status of the relations in complex systems. A representational framework of causality which emphasize relations, rather than emphasizing the nodes as current causal models did, seems promising to me. The representation problem, however, still remains to be solved. Concluding remarks will be canvassed in Chapter 5.

CHAPTER 2

THE REPRESENTATION PROBLEM IN COMPLEX SYSTEMS MODELING

What is an entity that we are encountered in nature? How it appears to us? Does it appear as itself or otherwise? Such questions on the relation between an entity and its appearance have been occupied philosophy for thousands of years. The entities that are whether processes or objects, assumed to be constituents of the nature; yet, we still lack of a general agreement on what is the relation between an entity and its appearance, and how to approach to these entities in order to understand how nature works. The principal instrument that is adopted by modern science (Frigg and Hartmann, 2018) is modeling. Models in science are the representations of target system that is an entity in the world. Scientific approach towards the entities, however, may also show variety depending on the research questions. For instance, to model an engine the researchers account entities as static elements while in biological modeling entities are considered to be dynamical organizations.

In scientific models, thus, there are different ontological commitments towards the entities that are represented. My standpoint in these discussions is that we are surrounded by systems (entities should be considered as systems) and thereby to understand the nature, systems approach is needed. By systems approach it is not indicated that holistic view of nature that assumes "[t]here are some wholes whose natures are simply not determined by the nature of their parts" (Healey, 2016), but rather there are entities that are constituted by specific interrelations of their subunits within the environment. What differentiates the system from its environment is that some organization of subunits that produce response as a whole. Thus, systems are usually classified in regard to the behavior, namely the response that is produced under the specific conditions (a set of inputs).

In that sense, different types of responses may correspond to different systems. Yet, it is not trivial as it is put in here, especially for natural systems – including social ones, that are multifunctional¹. That is to say, the same response can be produced within different components and/or different systems.

Different systems, also, can be classified under the same category. Consider, for example, the organization of the nervous system (especially, of primates) which is consisted of specialized structures for specialized tasks that are carried out by a parallel and hierarchic system. Even the simplest task of seeing an object involves different levels of organization. Visual top-down processes (the ‘information’ flow of brain depending on our previous experiences, predispositions), for instance, required for external world perception while bottom-up processes (from the retina to visual cortex) are at play. This highly-intricate structure is considered as a complex system. One can argue that social constitutions also show similar patterns of organization. For example, the social policies have an influence on the behaviors of the individuals (top-down) whereas the policy makers make their decisions under the influence of the individuals (bottom-up). It is not argued that nervous systems and societies are alike, but rather that they show similar characteristics of the systems that today we call complex systems. The challenges of modeling such systems, it seems, consisted of insufficiency of the representational frameworks.

The main obstacle in the way of representing the complex systems is the discrepancy between the ultimate response of the system and the specific outcomes of each system component. Any reduction of the system behavior to the sole component(s) in such a context, consequently, constitutes a problem. Ironically, to represent these systems, it is thought that, complexity needs to be reduced. In that sense, ‘how to represent’ is itself requires a philosophical investigation on whether it is possible to provide a proper representation for a complex system without making any concession from its complex nature.

¹ When the same (or similar-enough) components that are able to produce different responses it is called multifunctionality whereas ‘degeneracy’ indicates that functionally equivalent actions can be executed by different components. See section 2.3.4.

2.1 System

A general definition for a system is that the set of constituents that are related with each other in specific manners. Systems may be classified as ‘open’ or ‘closed’ in regard to the relation between the systems and their media. For example, if there is no exchange of matter and/or energy or, if the system is somewhat isolated from its environment it is referred as closed system. Closed systems, usually, are the artificial systems or the systems that are put in an artificial medium. On the contrary, natural systems (e.g., biological entities) are considered to be open systems as long as they continue to be ‘open’ to the inputs from the environment. In mathematics, a system is represented as "a well-defined set of states" (Vasbinder and Gao, 2017) which may dynamical or stationary considering the changes in the system behavior. Thus, a system corresponds to a formalae of the rules that govern the state changes. If a system is stationary, the rule describes a state of balanced input-output that equals to no change whereas a dynamical system (DS) depicts change between the states. Thus, DS is "a rule for time evolution on a set of all possible states" (Meiss, 2007).

A dynamical system, simply, the systems that change over time. Time evolution of the DSs can be "described over either discrete time steps or a continuous time line" (Sayama, 2015). DS in continuous time is defined in the form of differential equations and therefore, it is useful to represent abrupt changes. DSs in discrete time, on the other hand, are more useful to represent smooth changes in the system behavior. Discrete time DSs are consisted of iterated maps or, difference equations (ibid). A discrete-time dynamical system is represented as:

$$x_t = f(x_{t-1}, t) \quad (2.1)$$

Whereas continuous-time dynamical systems are represented as follows:

$$\frac{dx}{dt} = f(x, t) \quad (2.2)$$

In the equation of discrete DS, each state takes the state that is before as an argument of the function. By this way, states are determined stepwise by the previous states at chosen time intervals. On the other hand, the equation of continuous DS represents the derivation of states over time. There are no time intervals but continuous time. The time evolution or ‘the temporal evolution rule’ delineates the way of derivation of next state from previous state (Fusco et al., 2014). DSs are grouped as either linear or nonlinear in regard to the course of temporal evolution. A system is linear if the state variables is only a linear combination (e.g., of their sum), and nonlinear if otherwise. If the next state, for example, is a trigonometric function of previous state variables (Sayama, 2015), the system is a nonlinear DS.

2.2 Linear Systems

2.2.1 Linearity and Linear Models

Systems, in general, are modeled as the boxes that take inputs and produce outputs. Linear systems are the systems of equations where the previous states constitute the next states according to defined linear rules. Since linear equations are separable into its elements, the elements of linear systems can be studied analytically. Discreted elements can be recombined as such in the initial state of the system since linear systems hold additivity. Similarly, multiplication of the elements would result in multiplication of the following states (and/or responses) due to homogeneity. Hence, proportionality is preserved through the states of the linear systems.

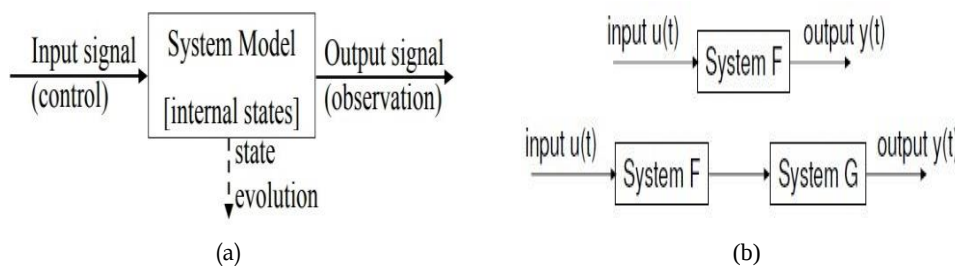


Figure 2.1: a. A model of system with its internal states, retrieved from Gorinevsky (2005), b. Another exemplar for modeling the systems, retrieved from Hover (2009).

Systems are called linear if for every time t_0 and any two state-input-output pairs (Chen, 1995):

$$\left. \begin{array}{l} x_i(t_0) \\ u_i(t), t \geq t_0 \end{array} \right\} y_i(t), t \geq t_0 \quad (2.3)$$

$i = 1, 2$, we have

$$\left. \begin{array}{l} x_1(t_0) + x_2(t_0) \\ u_1(t) + u_2(t), t \geq t_0 \end{array} \right\} y_1(t) + y_2(t), t \geq t_0 \quad (2.4)$$

and

$$\left. \begin{array}{l} \alpha x_1(t_0) \\ \alpha u_1(t), t \geq t_0 \end{array} \right\} \alpha y_1(t), t \geq t_0 \quad (2.5)$$

By additivity that is depicted in (2.4), we understand that the measured system-response is mere "the sum of its responses to each of the stimuli presented separately" (Heeger, 2000). The property of homogeneity that is depicted in (2.5), on the other hand, suggests that "as we increase the strength of a simple input to a linear system, say we double it, then we predict that the output function will also be doubled" (ibid). For example, when input is doubled α in equation (2.5) is substituted with 2, thus we would estimate that output as $2y_2(t)$ given $2x_2$ at time t_0 to a linear system under $2u_2$ (which stands for two times of noise and/or environmental constraints). These two properties, when combined, constitute a principle that is called 'superposition rule' which is a golden rule because it allows predicting the outcome given inputs. Thus, the behavior of the linear systems can be predicted. That is to say, given input x_1 at time t_0 to a linear system under u_1 , we observe y_1 as an output. Likewise, given an input, at time t_0 , $x_2(t_0)$ under u_2 it is yielded y_2 at time t ; then, if one introduces these two inputs $x_1(t_0)$ and $x_2(t_0)$ the output of y_1+y_2 can be perfectly predicted.

A real-world example for linear behavior would be the linear electrical circuits. Consider such a circuit, the current and voltage for any element in the circuit is the sum of the currents and voltages produced by each source acting independently

(Hayt et al., 1986). The manipulation of the batteries will proportionally result in an altered current. Linear systems, then, operate in such a way that same inputs always bring same outputs and thus, the response of the systems directly proportional to the inputs. The inputs that get into the system hold the role of causes while the outputs that are produced by the system considered as effects. The relation between cause and effect, then, is characterized as a linear relation.

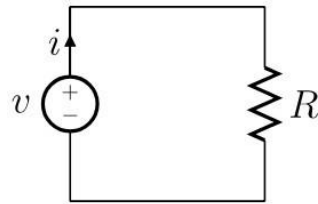


Figure 2.2: A simple representation of a linear electrical circuit where R stands for resistor, v as battery and i as current.

2.2.2 Linear Assumptions in Data Generation Process

To study the system behavior and/or the properties of the system, one needs to gather all the necessary data concerning the systems. The relevance of the data, mostly, depends on the focus of the research. For instance, if the researcher seeks to understand the relation between obesity and heart attacks in a population (the target system), then the data of IQ scores of that population would be redundant. If the data are obtained accordingly, then the target system is become represented in terms of obesity and heart attacks.

The conditions that change or has different values for different entities are represented with ‘variables’(Gravetter and Wallnau, 2016). In other words, an element that changes in each state is a variable. That change, mostly, attributed to a causal process where causes change the current state and/or property of the system. For example, x_i under u_i from the previous section, or the rates of obesity and heart attacks are the variables.

Besides the state variables, the other factors (e.g., parameters, omitted causes or error terms as u_i) are also measured to become quantitatively representable. Measurements are consisted of the collected data set which serves as a material for descriptive and/or inferential statistics (Daniel, 1991). The choice of statistical

means depends on the research question. For instance, if the question is that ‘what is the frequency of heart disease in Population-A?’ the descriptive means would be sufficient. However, if one seeks to understand the causes of the high rates of heart disease in Population-A, then inferential techniques (following the descriptive analysis of the current state of ‘high frequency of people with heart disease in Population-A’, for example) are used.

The data can be obtained through different methods in regard to the research question(s) and the hypothesis. The system properties of interest may already be in its quantitative form, such as in IQ test results. If not, then the measurement techniques (e.g., surveys) applied to the target system properties, in order to convert the qualitative data into the manageable forms. Once the collected data are quantifiable and organized, a representative data set from the data of whole population (target system) is selected.

The representative data set, namely ‘the sample’, is usually chosen via the method of simple random sampling. This sampling method, as like many others, relies on the idea that given data consisted of representatives of heterogeneous elements (if there is any). Hence the randomly selected set from data does not show any significant deviation from the overall representation of the data. In this sense, it is assumed that the sample can adequately or, is sufficient enough to represent the whole (the system) that of interest.

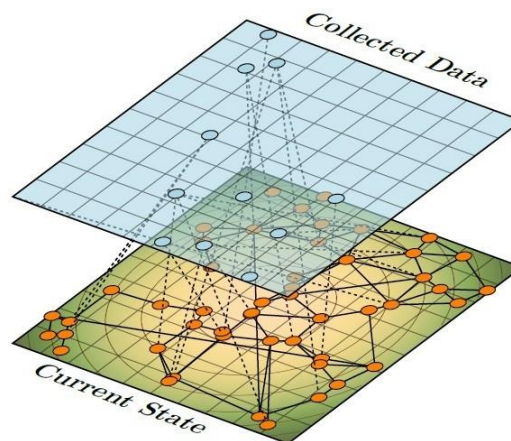


Figure 2.3: An illustration for the state of interest and collected data.

Data as measured properties, however, may show difference due to the way of quantification. For example, a survey (for example, Uncertainty Response Scale by Greco and Roger, 2001) that aims to classify decision-makers' attitude towards uncertainty, has a scale of points 1 to 5. The point 1 stands for unsentimental people whereas point 5 stands for very sensitive and impulsive persons. However, a person who is scored around the 3 points (quantifiable form of 'not-so-impulsive attitude' toward uncertainty) may get 15 points (quantifiable form of 'impulsive attitude' toward uncertainty) on a scale of 1 to 20 points. In that sense, the evaluation of the collected data depends on how we read the data as well as the ways of scaling and measurement.

The obtained data set is organized and simplified to get a 'neat' representation of the variables that are investigated. Especially, the techniques like functional magnetic resonance imaging (fMRI) to study the neural systems, demand 'cleansing' of the collected data from noises and artifacts. Artifacts in fMRI data would be, for instance, the fluctuations due to thoracic movements during breathing (Raj et al., 2001). After such removal processes, the specific changes in the variables can be easily observed. The evaluation of whether there is a relation between the variables requires inferential data analysis. Since the direct relations cannot be observed during an event, researchers trace the sequences of changes in the variables. Consider Figure 2.4 and Figure 2.5 that illustrate a study that questions the effects of the two teaching methods that are introduced to first-grade students:

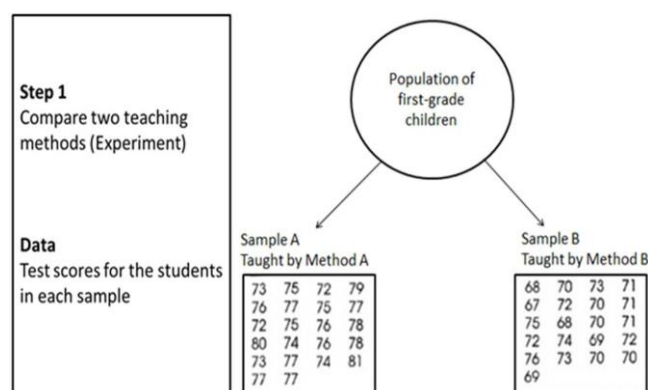


Figure 2.4: Collected and organized data of first-grade children. Retrieved from Gravetter and Wallnau (2016).

The system in question in here is a population of first-grade children. The study is conducted to determine the effects of two different teaching methods in this population. Thus the inputs are Method A and Method B whereas the outcomes are the test scores of children. Samples stand in lieu of all of the students (population) who hold similar features such as similar family backgrounds, taught by same teachers, or the stress levels. Thereby, it becomes more justifiable to argue that ‘the test scores are affected’ since the only difference is the test scores of the children. Notice that, it is an assumption of linearity that the components (individuals) of a system are analytically separable. Thereby, the arithmetic mean (average score) is become informative in showing the central tendency of the samples.

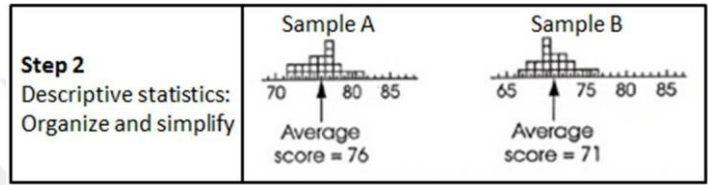


Figure 2.5: Descriptive statistics for the samples A and B. Retrieved from Gravetter and Wallnau (2016).

The central tendencies of the sample data show a difference (5 points) between two teaching methods. If there are no methodological errors such as the sampling errors, the data would be interpreted as ‘Method A is more efficient teaching method than Method B’. Think of an additional input; say a textbook that is previously proved to be a boosting factor for test scores. If this is the case indeed, then one expects that an additional input, the textbook, when introduced to the students who taught by Method A, would be a boosting effect for higher scores. As well, students who taught by Method B would be increased their scores with the help of the textbook. However, due to proportionality of the effects, it is expected that the students taught by Method A with the usage of the textbook would still have higher scores than the students who taught by Method B even with the textbook usage. Hence, the aspect of additivity is assumed in such cases.

The data interpretation, then, is an inference process that generalizes the results from sample data to overall system. The results, if show any ‘significant’² dif-

² The p-values are used to determine whether the independent variable has significant effect on the dependent variable.

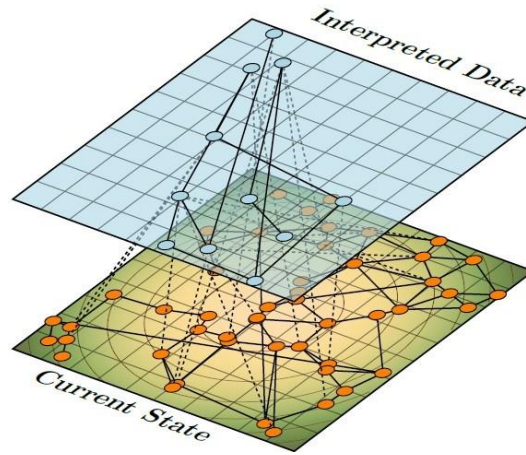


Figure 2.6: An illustration for current state of interest and interpreted data.

ference, indicate the possible causal relations between the variables. The way we read the data usually relies on the assumption that the relations between variables hold the properties of additivity and homogeneity, thus those relations are assumed to be linear.

2.3 Complex Systems

2.3.1 Complexity

Nervous systems, societies, stock markets, weather, etc. are all considered as complex systems whereas each of them has their own building blocks that completely differ compared to other systems. The scale, then, is not a criterion for complexity since even the microsystems (e.g., biological entities) may constitute a complex system. Yet, not all the systems that have interconnected parts (say, an electronic device) considered as complex systems. Then what is the criterion for distinguishing a complex system from any other system?

The efficiency of an algorithm when confronted with different sizes of input is measured according to the time that is taken to produce an output. The number of computational operations to execute an algorithm (that means to receive an output) is the criterion of complexity. Thus, computational complexity can be defined in polynomial time. The computational complexity is scaled with Big-O notation $O(n)$, which describes the upper bound of the algorithm's runtime with respect to the

amount of input (n). Each step of the operations may take different runtimes to produce an output, yet, each runtime is added in order to calculate the complexity of an algorithm. Because, the operations follow each other step by step, thus with an order. The steps never overlap. By this way, the input size (n) is directly proportional to the number of steps which follow orderly each other in time. By this way, $O(n)$ represents the time taken by an algorithm which is directly proportional to the input size.

An algorithm is considered to be solvable in polynomial time if the number of operations for a given input is $O(n^k)$ where k is a nonnegative integer and n is the size of the input. The algorithms that take polynomial time are consisted of tractable operations. However, some algorithms are not solvable in polynomial time. Such an algorithm is solvable only in non-deterministic Turing machine since, to produce an output, the number of steps to be taken are too much. On the other hand, systems' complexity is beyond algorithmic definitions since the systems' behavior is intricate in many respects (technically, high-dimensional), and such behavior cannot be assigned to merely the outputs of the components. Complex systems operate in a parallel and hierarchic manner. Thus the outputs can be produced through different ways rather than an orderly linear manner. In that sense, it is not trivial to provide a legitimate criterion of complexity in systems to apply all the complex systems. At this point, it is argued that nonlinearity may provide a criterion for deciding on whether a system is complex.

Linearity implies order, it means that to arrive a point there is a specific way to go through. Any interference in such processes would result in either failure to arrive or an altered outcome. However, in complex systems we observe such behavior like degeneracy that is the ability of systems to produce an output from different ways. Furthermore, it can be said that the number of possible ways to arrive a point would consequently increase the complexity in the systems. That is due to nonlinear dynamics which generate alternative ways to go through. Nonlinear dynamics seem to be ubiquitous in complex systems and constitute a steering factor of increasing instability, thus responsible for bringing the system into a state of the edge of chaos. At this stage, systems may result in more than one output, and such a result is specific to complex systems. However, every nonlinear behavior does not .

necessarily executed by a complex system. What needs to be done is that the characteristics of complex systems (e.g., self-organization) should be studied in respect to underlying nonlinear relations. Section 2.3.3 and 2.3.4 devoted to these discussions.

2.3.2 Chaos

As discussed in section 2.1, a dynamical system is a deterministic mathematical model, since the next states are determined by previous states. Chaos is a phenomenon that refers to sensitivity to initial conditions of nonlinear dynamical systems (Gleick, 1987). Even though the systems are deterministic, the long-term behavior of the systems is unpredictable due to nonlinearity³. In that regard, chaotic behavior can be defined as deterministic, nonlinear, and aperiodic behavior (Fuchs, 2013) that displays sensitivity to initial conditions. A linear dynamical system is considered to be predictable even in the long-term because, the next state of the system is the very linear combination of the previous. Thus the decomposition of that combination can reveal the previous state or else, the re-assembled components can reveal the states of the system. Likewise, if two systems hold the same time evolution rule with the initial conditions that are close enough (say, $x_1 = 1$ and $x_2 = 1.001$), these systems would follow similar trajectories. In this sense, they are not sensitive to initial (previous) conditions. What makes a chaotic system to be unpredictable is, then, inapplicability of the superposition rule. Nonlinearity is the main source of chaos since a small difference in “the system’s initial condition is quickly magnified under iteration”(Feldman, 2012). Such iterations bring aperiodicity, namely, non-repeatability of the previous conditions, in that sense only short-term prediction is feasible. However, within one (e.g. logistic growth) or two dimensional (e.g. pendulum) nonlinear systems the chaotic behavior cannot be observed because of the topological reasons⁴. At least three variables are required for chaotic behavior as such in three celestial bodies or ‘3-body problem’ (Poincaré,

³ Any imprecision in the calculations of the chaotic systems followed by amplified deviations from the original trajectory.

⁴ From the video recordings of MAE5790-Nonlinear Dynamics and Chaos Lectures at Cornell University taught by Steven Strogatz.

1913) that interact under simple (Newtonian) rules. Thus, chaotic behavior may be seen in very few interacting elements whereas complex systems are consisted of enormous amounts of interactions within dynamic components and the environmental constraints. Due to the dynamic interactions of large amounts of elements complex systems are able to arrange itself under the changing conditions. In that sense, complex systems are usually considered to be at ‘edge of chaos’ (Kauffman, 1996) rather than to be strictly chaotic. Then, although the fact that a complex system may be chaotic, it is not implied that chaos necessarily a property of complex systems.

2.3.3 Characteristics of Complex Systems

The term of complex system stands for the ‘whole’s that are constituted by various kinds of inter-connected elements and their nonlinear relations such that these wholes behave in an untraceable manner yet deterministic, de-centralized, self-organized and cannot be reconstructed via simply summing up the elements. The very reason for the difference between a lump of components and a complex system is the new characteristics that gained through interrelations among components. ‘Emergence,’ ‘autopoiesis’ and/or ‘self-organization,’ ‘edge of chaos’ are classified as such characteristics. However, today, we are unable to reach beyond the vague descriptions of these generic concepts.

The ambiguity in the generic concepts of emergence, self-organization, complexity, etc. stems from the fact that there is no consensus on the methodology to study these characteristics. The standard way to study the system behavior is analysis of the functions that are assigned to components. For example, to understand the role of a gene in producing a phenotype, researchers usually conduct knock-out experiments in which the interested gene is specifically deleted. If the phenotype does not appear after such a knock-out process, it is thought that that gene is responsible for the emergence of that phenotype. Similarly, it is expected that once the responsible element(s) that give(s) rise to self-organization (or any other characteristics) of a complex system, such systems will be explained. However, this

⁵To find more sophisticated examples of degeneracy, see Edelman and Gally (2001), Sporns et al. (2000).

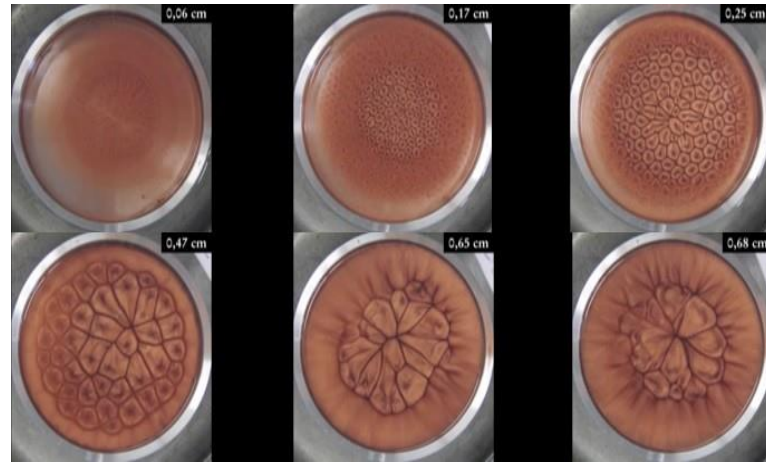


Figure 2.8: Snapshots of Rayleigh-Bénard cell convection from the experiment conducted by Pfander and Haupt (2015).

In the example of Rayleigh-Bénard convection, we see that "the bulk motions of fluids generated by temperature inhomogeneities" (Nicolis, 1995) as an instance of self-organizing behavior. Such a phenomenon – a new structuration, requires high degrees of incorporation of the local elements (Nicolis and Prigogine, 1971). Given the heat from below, a positive feedback loop is initiated: the lower side of the fluid layer becomes heated then it rises, the upper side which is cooler moves below while the effects of gravitational forces compete with heated molecules. The positive feedbacks reiterate the local elements in each turn that results in fluctuations, and thus the stability of the system breaks down. At a critical value⁶ the system responds to instability with the decentralized control which arises from local relations. Macroscopically – as it can be seen from the figures 2.7 (b) and 2.8, the fluid rearranges itself into a new type of organization through decentralized control. Such an example shows that "with self-organization, a new order of the system 'emerges,' an order of non-equilibrium, a non-static order" (Bertuglia and Vaio, 2005).

Emergence, on the other hand, is a characteristic that suggests a novel phenomenon that arises due to the interrelations of the system components. It is often aforementioned as the phenomenon of 'a whole is more than the sum of its parts'. In other words, it is thought that there are such higher level entities that cannot be one-to-

⁶ For details, see Nicolis and Prigogine (1971)

one mapped onto their constituent lower level entities. But, how could such a novelty appear? The answer, as it is argued for the other features of complex systems, is in the dynamical relations of the systems. However, it should be noted that kind of depiction of wholes and parts – which historically corresponds to Aristotelian metaphysics, is misleading if it is taken seriously. It is because ontological discrimination of the parts and the wholes leads to somewhat isolation of the parts while the relations are undermined. Philosophically it is called mereological ontology which is an unrealistic way to approach complex systems. In that sense, recent literature is more apt to Kantian description of wholes where "in an 'organized being' the parts exist for and by means of the whole, the whole exists for and by means of the parts" (Longo et al., 2012). Kauffman et al. offer many examples of Kantian wholes in biological context. Yet, to study such wholes is still a problem.

As complex phenomena the social systems, similar to biological organizations, hold systemic characteristics. We encounter with (i) self-organization and autopoiesis where⁷, for example, two persons have met and decided to establish a family that followed by generations, or in sects; (ii) emergence where the relations at the level of individuals result in novel assemblies that cannot be predicted from merely personal characteristics; and with (iii) complexity since even the data among a group of people (say, consisted of 3 individuals) would be enormous considering all of the aspects of the group's dynamics and personal traits. In this sense, to get a comprehensive understanding of such social phenomena the system-characteristics should be considered. What makes a social system different from other one depends on those characteristics. Think of, for example, in the same country within similar-enough genetic inheritance and similar-enough environmental factors, we may see (and as the world history shows, we mostly see) that the social movements do not show similar patterns compared to past generations. The difference, then, must lie in the relations of the components which, in this case, are the individuals and their constitutions. The relations, the causes and their effects, are in fact, nonlinear, and thus interactions seem to be ever-changing.

Nonlinear interactions may also give rise to the other aspects such as chaotic behavior. The trick with the nonlinearity is the disproportionality between the inputs (causes) and the

⁷ In the cases of when there is no external specific ordering influence.

outputs (effects) of a system. In fact, nonlinear relationships yield the system to become sensitive to external influences. Yet, as discussed above, it is not certain that whether there is a certain recipe for characteristics of complex systems. On the other hand, the nonlinear relations seem to precede all of the characteristics that appear in a complex system.

2.3.4 Nonlinearity

Nonlinearity as a shunned term of the mathematics has been used to describe the dynamics among complex systems which are, simply, not analytically solvable. The term encompasses all of the situations where linearity is not applicable. So, it is abstruse in the sense that there is no clarification about the set of not-being-linear entities. In fact, philosophically, it would be an intriguing research question that whether it is possible to classify different types of nonlinear relations. However, I stress on the definition of not-behaving-linearly in this paper. Nonlinear dynamics, then, consisted of the relations that do not follow a linear trend. It is expected that within that line of thought, all of the properties that constitute linearity can unravel what nonlinearity is not. Hence, to define nonlinearity, we can, roughly, exclude the properties of linearity. Indeed, within nonlinear systems, the tools for linearity can not applicable unless the data set is linearized. Recall the positive feedback loop that is discussed in section 2.3.3. Firstly, the input and the output are incommensurate since they feed each other at every turn. Also, an increase in the input may result in exceeding the critical threshold which can lead to self-organization as in the example of Rayleigh-Bénard cells; thus, the ultimate effect is not a superposition of the causes. Yet, in such nonlinear cases, during the evolution of the system, the link between cause and effect is not traceable, and this is the very reason that we cannot precisely predict the future outcome of the complex systems.

2.3.4.1 Order and Disorder

Do nonlinear dynamics encompass some order or, to say, follow some pattern? Such a question is put forward, because, we observe characteristics like self-organization, and/or the emergence of new forms due to cooperativity of the components through nonlinear relations of the systems. The components are re-combined

⁸ To see other related discussions Prigogine, 1978; Bak et al., 1987.

in each turn of positive feedback loops, by this way; the system is steered into an instable state. Surprisingly, the instabilities of the system yield to new formations of patterns. In that sense, new order seems to arise from the disorder⁸. Questioning whether the recipe of the new order is somehow given in nonlinear relationships, does not necessarily imply linearity within the term of order. However, there is no convenient way to think any order in terms of nonlinearity since nonlinearity appeals to instability and disorder in the systems. The clarification is indeed, needed considering the terms of order and disorder in the context of complex systems. The one possibility for defining order may be based on regularity. The events are regular, that is to say, they follow each other regularly and thus show a regular pattern. That sounds like causation, yet, insufficient to be. As another candidate to define order, one can consider the order as stability – a state of equilibrium, again, in the context of the systems. But how come an entity in a state of equilibrium "manifests itself as the collapse of a state, following internal instability or an action external to the system, and with the adjustment of the system to a new state" (Bertuglia and Vaio, 2005)? It is important to keep in mind that, especially while system organizes itself, the new pattern appears "with no specific ordering influence from the outside" (Kelso, 1997). If that is the case, then one should expect that some specific rules of interactions between the components, that is to say, an 'internal logic' (as Bertuglia & Vaio put) steers such behavior. Note that, there is no centralized control over the complex systems, thus that putative internal logic must lie in the relations between the components. But, how can we detect such an internal logic if there is? Can we extract "a deeper level of patterned order" (Capra, 1997)? Perturbing the system is one of the ways to detect that. In this sense, researchers (e.g. Scheffer et al., 2012; Carpenter and Kitchell, 1988; Dai et al., 2013) have been studying the factors that propel the system an abrupt transition toward an alternative state. A shift toward another state requires new ways of connections, thus a new ordering within the system. The point where systems transited to another state – namely the 'critical point', indicates that the interaction rules are about to change. Carpenter and Kitchell (1988) experimented with a whole-lake (in addition to another lake nearby as a reference in Michigan, USA) to specify the early warning

signals that indicate the affinity to the critical point for the change in food web. The lake that is manipulated is consisted of a low population of the predator fish (large-mouth bass) in contrast to large populations of the prey fishes (minnows). For several times, the researchers were intended to tweak the food web in the lake (Peter Lake) by supplying additional largemouth basses (in controlled amounts). The response of the lake consisted of some local repairs which carried out by feedback relations to preserve its state of prey-dominance. However, at some point, the system (food web in Peter Lake) has become slower in order to execute a response to those perturbations. Following that, the food web is completely traversed: Peter Lake became predator-dominant. The phenomenon of slowing down in producing a response is called ‘critical slowing down’ and it is observed in many complex systems. For example, in populations of budding yeast *Saccharomyces cerevisiae*, the indicators of critical slowing down are also observed based on spatiotemporal fluctuations in the system (Dai et al., 2013). The researchers put yeast cells in media consisted of sucrose which allow the yeast cells to grow cooperatively by sharing the hydrolysis products. By this way, positive feedback loops initiated between the cells which lead to bistability and a critical point (ibid). Since the response of the system is carried out by feedback relations, the critical slowing down may be implying a change in the relations. It would also be applicable to ‘flickering’ behavior (indecisiveness between alternate states, see Dakos et al. 2013) of the systems. Such experiments indicate a possibility to anticipate an upcoming pattern change once the rules of relations are comprehended.

2.3.4.2 Spatiotemporal Patterns

The transition process of stability to instability and then to (a new kind of) stability constitutes a spatiotemporal pattern of activity which we observe in nonlinear systems⁹. Spatiotemporal patterns imply both spatial and temporal regularities. In this sense, different regularities embody different patterns. Such regularities correspond to the different types of connections between the components and between the systems and its media. A commonsensical example for a spatial pattern would be a knitting pattern. These spatial patterns, simply, are formed by different arrange-

⁹ Although all complex systems are nonlinear, a nonlinear system is not necessarily a complex system as we have seen in Rayleigh-Bénard convection in section 2.3.3 (for more details see Walgraef, 2012).

ments of the yarn. The way of stitching the each inch of the yarn to another inch yields a specific pattern (if one knows to how to knit, for sure). Notice that, even the components re- main the same (in this case, the same yarn) patterns show variety. Hence we can say that, the relatedness of each inch of the yarn determines the upcoming pattern.



Figure 2.9: Knitting patterns.

Unlike the knitting patterns, in complex systems components are dynamically ‘coupled’ with each other and the environmental factors that affect (and may be affected by) the system without any centralized unit. The spatial couplings may be strong or weak (Kelso, 2012). The strength of coupling determines the system’s stability (whether it is stable, unstable, or metastable). If the coupling strength is considered to be weak, the system would be either metastable or unstable. In such states, the change in the interconnections of the system is highly possible.

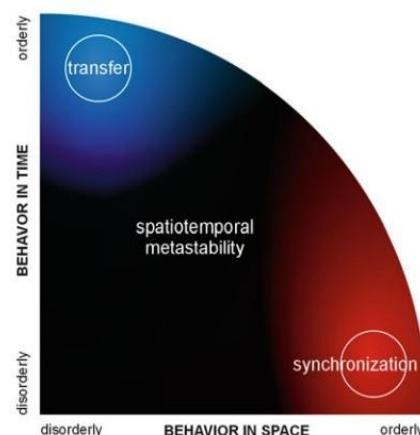


Figure 2.10: A conceptual view of the spatial and temporal order in the behavior of neural ensembles (retrieved from Tognoli and Kelso, 2013). Complex systems are in between order and disorder in time and space.

systems are In regards to couplings, some temporal patterns of activities (as such in developmental processes of an organism) arise due to the system dynamics. The pig embryo, for example, reaches 5 mm at approximately 17 – 18 days whereas the chick embryo of 4 - 4.5 days is approximately 5mm (Dye, 2011). Such spatiotemporal patterns can also show variety even in the same system. The very reason for that is the interactions between the components are changing, namely, dynamical. Since the relations between components are not fixed (Tognoli and Kelso, 2013), a complex system can be both stable enough to preserve itself and flexible enough to change itself.

2.4 The Insufficiency of Linear Methods to Represent Complex Systems

In section 2.2 and 2.3, it is distinguished that systems are either nonlinear (and complex) or linear depending on the behavior of the system. To study linear systems, there is a wide range of tools which extracts the relations between the variables (by the means of inferential statistics) and, provides a neat way of modeling and prediction (based on the superposition principle). However, when these tools are applied to complex systems which are intrinsically nonlinear, the overall result is nothing but lack of understanding.

2.4.1 Linear Methods

(a) Optimized representation: Since most of the time the system of interest is overwhelmingly detailed to examine, we seek for the representations that do not contain any ‘unnecessary’ detail. Whether or not a datum is necessary depends on the research question. For example, if the research is intended to investigate the causal relationship between sugar consumption and obesity, a datum of ‘95% of people who suffer from obesity wear dark-colored clothes’ would be an unnecessary detail. The data of sugar consumption and obesity rates are optimized whereas the other conditions count as equal and thus are not represented.

(b) Analysis: Analysis means that examining the parts (components) of the system separately. In other words, system is decomposed into its components. The analyst focuses on the mere components in order to understand system behavior. In population statistics, for example, to understand the population (system), the specific properties of a selected group of individuals (parts), namely, of a sample are analyzed. In biological systems, an organism is divided into its sub-units (e.g.,

organelles like ribosomes). The analysis is performed with the extracted sub-units.

(c) Normally distributed data: The collected data represent the population of interest. It is assumed that the data show a normal distribution which means that the values in data are more or less close to each other. Then, the average (mean) is "the one number that best describes what the data is like" (Liebovitch and Shehedah, 2003). As the averages of the samples from the population get larger those means approach to a limiting value that is thought to be the real value of the population mean (ibid). Thus, the average value characterizes the data pretty well (ibid). As a graphical representation, normal distribution or, Gaussian distribution aims to show the distribution of population in regard to the central tendency which is the arithmetical average (mean) of the variables. It forms a 'bell curve' that is symmetrical "with the highest frequency in the middle and frequencies tapering off as you move toward the extreme" (Gravetter and Wallnau, 2016). The tails of the normal distribution graph indicate that extreme cases which are represented with low probabilities since the data are normally distributed the extremities hold low chance to be/or happen.

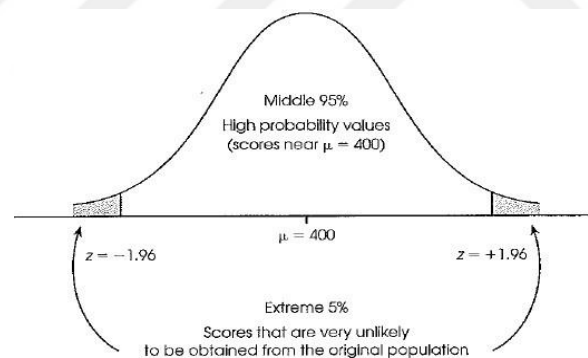


Figure 2.11: A normal distribution graph (adapted from Gravetter and Wallnau, 2016).

(d) Connecting dots: The analyzed data constitute a fragmented picture of the event of interest. Then to answer the research questions, inferential procedures are required. The relationships between the variables are inferred according to specific methods. These methods look at the analyzed data to obtain information such as

high correlation rates, or temporal precedence (as in Granger causality) between variables. With the help of inference methods the dots connected via specific relations (e.g., causality) when the specific conditions (temporal precedence and significant correlation) are met. For instance, (1) sugar consumption ‘caused’ obesity and (2) the teaching method A ‘caused’ an increase in the test scores. In such cases, the same conditions are met for causality. Notice that, it is assumed that causal relation is linear in regards to proportionality: if sugar consumption is increased even more, the obesity rates would have also increased. The dots, then, connected through fixed linear relations.

(e) Equation solving: A linear system can be mathematically represented as $S = \alpha x + b$ where S stands for the state of the system, x as a variable and, α and b as the parameters. S will change in regards to any change in x or b . This change is ‘proportional’, since a slight change in the value of x (or b) causes a slight change in the system behavior (to its new state) as a response. As discussed in section 2.2.1, due to additivity and homogeneity properties, if the system is introduced with additional variable y , the response will look like $S_2 = \alpha x + y + b$. If $x + b + y$ are the solutions as in this case, then $P = cx + dy + eb$ is also a solution. In this sense, such equations can be solved analytically.

2.4.2 Nonlinearity and Complexity

To apply linear methods to complex systems, at first, nonlinearity has to be linearized (usually by approximating) in order to fit the data in the models. Furthermore, systems’ complexity is reduced to a non-complex representation of the system. Consequently, complex systems lose their characteristics when the linear methods adopted.

(a*)¹⁰ To decide on which detail is unnecessary is not a trivial task in complex systems. It is also disputable that whether there is any unnecessary datum in those systems. As discussed in section 2.3, complex systems are constituted by many interacting parts which cooperate without a centralized unit. Involvement of each component under the environmental constraints is a must to be a complex system. In such a compact context, it is harder and possibly misleading to ‘pick’ a sample from

¹⁰ The enumeration of the articles involves a sign (*) to address that all articles are put orderly in accordance to previous list. In section 3.4 the same rule is followed considering the lists that are presented in here.

many interacting parts and get an optimized representation of the system. Moreover, isolating the system from its environment is not informative considering the undergoing feedback relations. Complex systems are mostly open systems and thus usually, co-evolving with their environments; it means that both environment and the system change the other accordingly.

(b*) In complex systems, parts are dynamically integrated such that systems can produce behavior as a whole without any command center. This composed structure can also yield novel phenomena (namely, emergence) that cannot be assigned to components solely. In that sense, decomposition of a complex system is not an efficient way to represent (and/or explain) such phenomena.

(c*) Statistical analysis requires that the variables are independent from each other (heights of the persons in a class) and characterizable by the mean value. However, since the behavior is nonlinear, the distribution of the data cannot be characterizable with 'one value'. Even if the averages of whole population were calculated, the calculated value would not get ever closer to a fixed value, that means there is no population mean at all (Liebovitch and Shehedah, 2003). Also, due to linearity, extreme events represented with very low frequencies as 'thin' tails. But extreme cases (for example, the earthquakes that scaled 6+ in Richter scale) occur more frequently than a normal distribution graph represents¹¹.

(d*) The major difference between linear systems and complex systems is that non-linear nature of the latter. The behavior of complex systems is governed by nonlinear relations. Thus, the proportionality (between the input and the out- come) does not hold within the complex systems. Positive feedback cycles as an exemplar of nonlinear relations (as discussed in section 2.3.3) show that relations are not fixed yet they are dynamical. In each turn of the feedback cycles, the relations are restored and thus there is a continuum change considering the relations. Also the couplings rest upon such nonlinear dynamics. The phenomena of generation of new configurations within systems depends on coupling strength which is determined by nonlinear interactions of both the components of the systems and the environment.

¹¹ For technical details please see the literature on heavy-tailed distributions and the power law.

(e*) Linear (differential) equations imply that the next state of the evolution of the system is the linear combination of its elements. Then it is possible to analytically solve the linear equations. It is also possible that linearize the state of the systems that are close to the conditions of the stable equilibrium (Bertuglia and Vaio, 2005) by approximation. However, such stable conditions are only available at laboratories or in any isolated environment. Nonlinear equations, on the other hand, are too hard to solve and any imprecision in the conditions lead to enormous differences in the outcomes. The best way to cope with nonlinearity today is provided by computer simulations. Yet, they are limited in the sense of computational power, and representing all of the variables and parameters of an event.



CHAPTER 3

CAUSAL REPRESENTATIONS AND THE CONCERNING PROBLEMS

Is it possible to prevent global warming? Is carbon emulsion reduction really an effective way? If customers buy more goods, would the markets improve? How do neurons give rise to cognitive functions? Why do placebo pills affect some people? Why do systems behave in such ways? To answer such questions we appeal to causal explanations¹. However, in contrast to growing body of scientific knowledge about those processes, there is no single, one-to-fit-all answer. Rather there are different answers² which can deliver limited insight on the ongoing processes since the systems in question are complex.

The difference, most of the time, stems from the opt for different representations of causality in the explanatory models. Then what is the reason for the employment of different causal representations? It is because we do not have a generic or universal definition of causality yet, and as a consequence, causal relationships may be depicted disparately. On the other hand, it is essential to adequately represent the causal relations since such a representation offers (i) an explanatory architecture of the phenomenon; (ii) a basis for modeling the phenomenon; and thus, (iii) a way to make predictions about similar events. The criterion of the adequacy of a causal representation is, however, problematic in itself. What is the ‘right’ way of representing causation? The one would be that best captures the state-of-affairs, or say, the reality itself.

All of the causal representations, even if it is not explicitly put forward, rely on a theory of causality. That is to say, each representational framework (may) make

¹ My concern in this work is limited to causal explanations. However, in the literature there is an ongoing debate on causal and non-causal explanations which is not discussed in this paper. For more information, please see the works of Skow (2014), and Chirimuuta (2017).

² The answers may be compatible or in contrast with the other.

(different) ontological commitments concerning the nature of the causal connection. Counterfactual accounts, for example, rely on the ontological assumption of "[. . .] if the first object had not been, the second never had existed" (Hume, 1748). The current attempts to represent the relation between cause and effect, either reductive or non-reductive in the Humean sense. Concessions have been made on at least one of the features of causality as such in the Bayesian approach: while the feature of difference-making is ensured; however, the feature of causal necessity is abandoned. Different representations, thus, may result in different causal inferences which can be contradictory if we hold the view that a particular set of causes always brings a particular set of effects. The situation gets even complicated when a complex phenomenon (e.g. neural system) brought into question since complex systems behave nonlinearly. My aim in this chapter is that to discuss the way we think of causal relations in 'structural' models. Models in general, of course, are not considered as complete descriptions of the state-of-affairs but rather, representations that can give an insight on what is going on in the real world. In this thesis it is concerned that the ontological commitments which are made within models in order to represent causality in complex systems.

3.1 Causal Representations in Models

In the literature, the research programs of "what are causal relationships?" and "how can one discover causal relationships?" constitute distinct territories (see Cartwright 2007, and Williamson 2007). The causal methods assume that there are some causal relations (often, deterministic) and do not question the nature of that relation. The focus is to detect causality. In that sense, the causal models that fit best to our understanding of the world are proposed as methodological devices to hunt causes. However the methods, in fact, determine what we are looking for. For instance, it would be arduous to investigate celestial events via microscopes. The methodology that we adopt in 'search of something' is attached to what we think of 'something is'. In philosophy, conventionally, we think of causation as a 'necessary' relation between events that display some features that are (i) contiguity in space-time, (ii) priority of causes, and (iii) constant conjunction (Hume, 1748). Following Hume, philosophical literature has divided into two camps: reductive accounts and causal realists. Former accounts approach to causal bond in terms of non-causal events such

as temporal priority of the events. Contrarily, causal realists are those who do not reduce causality to non-causal terms but rather seek a unique entity (can be whether relation or disposition) to ontologically admit that as causation. Since those non-causal features can be seen as symptoms, or say, footprints of the causal bond, they can be still used to detect causality.

In modeling it is vital to determine which assumptions are (going to be) regarded in order to provide the representation of a causal process. The reason for that is what it is seen as sufficient enough to claim that ‘something is a cause and/or causally related’ determines the interpretation of the model in terms of causal relation. In the literature, there has been an ongoing debate on whether the statistical relations are sufficient to claim causality. Considering the latest tools to be sufficient a set of extra-statistical assumptions are required. Still, however, extra-statistical assumptions of causality significantly constrain the phenomenon that is intended to be modeled.

3.1.1 Causation as Dependence

Based on the available theories of causation, causal relations are usually represented as sequences of events, i.e., a linear sequence of transitions from, or dependency of one state to the other. Yet, these two different representations differ in theories³ also. In that sense, contemporary philosophers (like Ned Hall) put an emphasis on that these representations indicate different "kinds of causation" (Hall, 2004). According to this classification, causal relation is regarded as either ‘dependence’(in other words, difference-making) relation or a process of the ‘production’ of the effect. There is no necessary mutual exclusion between two, yet, as Hall (ibid) put, some properties (e.g., transitivity) that are attributed to causal relations may contradict with other properties that have been seen as necessary counterparts for some causal theories. Dependence or difference-making accounts rely on the intuitive ideas of (1) ‘causes make differences in terms of effects’, and (2) ‘effects are dependent to their causes’. Most of the scientific experiments are designed in order to expose such

³ Since there is no available consensus on the classification of causal theories, that claim is also disputable. However, in this thesis the underlying idea is that each representation has its own ontological commitments whether or not it is intended to do so.

dependence relations. The basic design of an experiment consisted of (at least) two variables: dependent and independent variables. Independent variable is intentionally modified by researchers to observe whether any change is initiated on dependent variable. Even if there is a change it may not indicate causation. At this point one can substantiate only that there is a correlation between the variables. Thus, as it is discussed in section 2.2.2, the relations between the variables of interest are inferred according to observed changes in data. To causally interpret the data, however, additional assumptions must be introduced during data analysis. Those assumptions may vary due to the model that is intended to fit the data. Dependence/difference-making accounts provide a range of such assumptions that may⁴ reside in different causal theories. Causal theories that represent causality as a dependence relation and their extensions in causal models can be categorized as⁵:

i. Regularity: Consider a simple observation toward causation. Whenever *A* occurs, the occurrence of *B* is followed *A*, and these two events contiguous in space also. Then one might argue that "*A*'s cause *B*'s' iff *A*'s are regularly followed by *B*'s and contiguous" (Reiss, 2008). Note that, *A* and *B* stand for generalized cases of events (philosophically, 'types'). Such lawlike regular following of event (types) is the basis of regularity theory of causation. It is a reductive account in the sense that causality is reduced to non-causal terms (in this case, to regularity and contiguity). In other words, regularity theories intend to "analyze causation in terms of invariable patterns of succession" (Hitchcock, 2018). Invariability in patterns of succession implies a lawlike necessity which we seek to understand causation in terms of. However, necessity (in natural occurrences) is a philosophically challenging concept. Sufficiency, on the other hand, seems to be a convenient concept to reduce causal dependency to. In this sense, regularity theories had further improvements in terms of sufficient conditions by John Stuart Mill and John Leslie Mackie. With sufficient conditions, the focus is shifted to token-level events and singular causal claims from type-level events and general causal claims.

⁴ The theories are not necessarily mutually exclusive.

⁵ This categorization is based on the works of Hall (2004); Illari and Russo (2014), but differs from them in respect to some details.

According to Mill, the cause should be taken as the whole conjunction of the conditions that are sufficient for the effect:

The cause then, philosophically speaking, is the sum total of the conditions positive and negative taken together; the whole of the contingencies of every description, which being realised, the consequent invariably follows. (Mill, 1911)

In resemblance to Mill, Mackie claimed that there are such conditions that at least are Insufficient and Non-redundant parts of Unnecessary and Sufficient (INUS) conditions. INUS conditions are the least requirements for causality. However, taking causes as the sums of the sufficient conditions is problematic in many aspects. The problems will not be discussed in detail due to the limited space and the scope of this thesis; but they can be listed as:

- (a) Irrelevance: A causes B when A and I occur simultaneously. For example, salt that has been hexed by a sorcerer invariably dissolves when placed in water (Kyburg 1965 via Hitchcock 2018).
- (b) Imperfect regularities: A is a sufficient condition of a B such that the (differing) instances of A and B are spatiotemporally proximate, thus, clearly is not a necessary condition of A causing B (Baumgartner, 2008).
- (c) Asymmetry: Temporal precedence of cause(s). That is to say causes (C) cause effects (E) and effects (E) cannot cause causes (C).
- (d) Spurious regularities: Two parallel effects E1 and E2 of a common cause C.

In the philosophical literature there is an ongoing debate on whether regularity theories can handle with the problems enlisted. Today, at least there is a consensus on that even if regularity theories can handle the problems, the reassessed versions⁶ of the theory are warranted. The most important (and related to this thesis) aspect of regularity regards *ceteris paribus* conditions. *Ceteris paribus* suggests that ‘all other things being equal’ or, ‘other things held constant’. It is argued that the Humean proposal of "an object, followed by another, and where all the objects similar to the

⁶ In contemporary scene, Baumgartner (2008), Graßhoff and May (2001) and others attempt to provide an alternative account for counterfactual and probabilistic theories of causation.

first, are followed by objects similar to the second" (Hume, 1748) should be considered under *ceteris paribus* conditions. However, the application of *ceteris paribus* is itself problematic in respect to vague definition of it (Reutlinger et al., 2017). For example, which conditions should be considered as equal, fixed, or constant? *Ceteris paribus* conditions, indeed, constitute a fundamental problem in causal modeling ⁷.

ii. Probability: If A causes B , then in the presence of A , it is plausible to think that probability of the occurrence of B raises given the state of absence of A . In this regard, earlier accounts of probabilistic dependence (put forward by Hans Reichenbach, Irving J. Good, Patrick Suppes) grounded on that assumption: ‘causes raise the probability of their effects’. It is formalized as follows: A causes B iff $P(B|A) > P(B)$. However, there are some causes that seem to lower the effect’s probability. For example, a drug may inhibit releasing of a hormone and thereby regulate the function of an organ. In this case the drug (in fact, inhibition of the hormone) causes the regulation of the organ functioning. Or, similarly, omission of calcium-intake in human body may cause osteoporosis. To allow such chance-lowering cases, probabilistic accounts updated the central assumption as ‘causes change the probability of their effects’. Then, to detect causality one may look into statistical changes in data. As often put, correlation is not causation; but it can provide information about the underlying causal structure (Glymour and Cooper, 1999).

The idea of causal discovery from probabilistic dependencies is gained strength as Bayesian nets method is introduced. Clark Glymour, Gregory F. Cooper, Peter Spirtes, Richard Shenes et al. pioneered the use of Bayes-nets to detect causal relationships given observational data. In that framework, Bayesian net- works serve as a representation of causal relations.

A Bayesian network consists of a structural model and a set of probabilities. The structural model is a directed acyclic graph in which nodes represent variables and arcs represent probabilistic dependence. (ibid).

⁷ In the literature it is discussed as the ‘context-sensitivity’, or ‘background knowledge’ problem

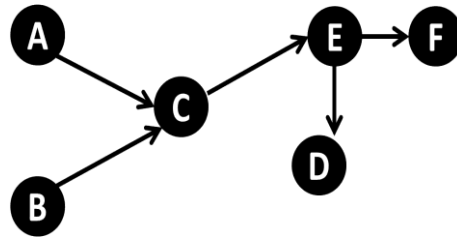


Figure 3.1: A directed acyclic graph (DAG).

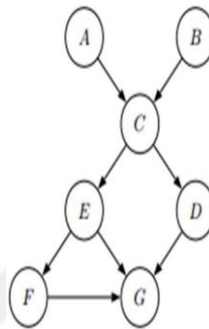


Figure 3.2: A directed acyclic graph (DAG) with the distributed probabilities. The probabilities to specify are $P(A)$, $P(B)$, $P(C|A, B)$, $P(E|C)$, $P(C|D)$, $P(F|E)$, and $P(G|D, E, F)$. Retrieved from Glymour and Cooper (1999).

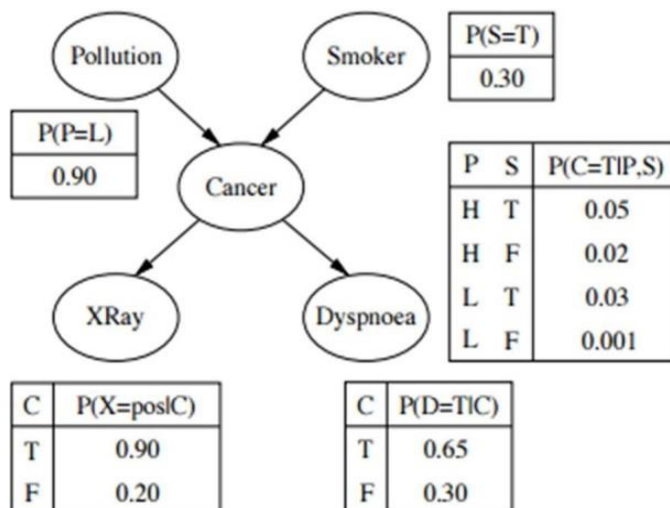


Figure 3.3: A causal Bayes net that is retrieved from Glymour and Cooper (1999).

In causal Bayes nets arcs are interpreted as causal influences. Such interpretation of Bayes nets, however, requires additional assumptions to be introduced. Causal Markov Condition (CMC) and Faithfulness are the essential ones. CMC stands for each variable to be probabilistically independent of its nondescendants given its parents (ibid). Thus, direct causation is implied. Faithfulness condition, on the other hand, suggests CMC: "In a causal graph, no probabilistic independencies hold other than those predicted by the CMC" (Reiss, 2007). Then all the interdependencies in data are not accidental but rather structural which means that resulting from the structure of the causal graph (Druzdzet, 2009). In the presence of (at least) those two assumptions manipulation is informative in terms of causation. In this regard, causal test undergoes with manipulations on selected variables. Consider figure 3.4:

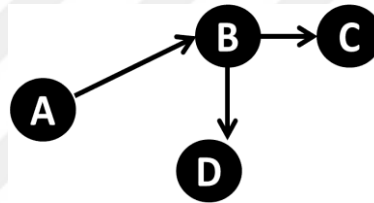


Figure 3.4: A causal DAG.

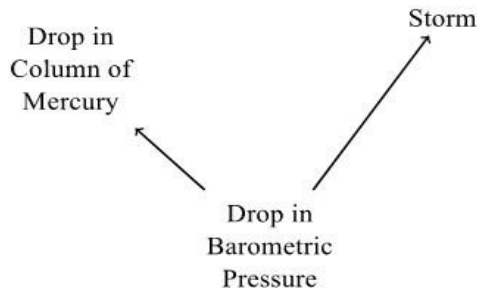


Figure 3.5: An example for spurious regularity retrieved from Hitchcock (2018).

Any intervention on A will result in change in the causal chain of $B \rightarrow C$, $B \rightarrow D$ then we can infer that A causes B. In this case, regularity theory would be insufficient to explain the independency between C and D. Notice that spurious regularity problem is solved within probabilistic theory. Also, the problems of imperfect regularity and irrelevance are ruled out within this account. Imperfect

regularities such as in the case of "smoking is a cause of lung cancer, even though some smokers do not develop lung cancer" do not constitute a problem in probabilistic accounts since causes change the probability of their effects and thus, "an effect may still occur in the absence of a cause or fail to occur in its presence"(Hitchcock, 2018). The problem of irrelevance is ruled out because if there is no difference in terms of effects one cannot claim the presence of causal influence. The problem of asymmetry, however, remains.

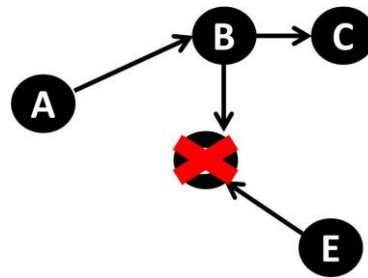


Figure 3.6: The variable E cancels (or say, inhibits) the variable D .

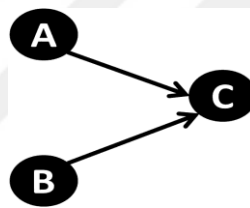


Figure 3.7: In this case, collider variable is C .

As a structural model, causal Bayes nets model the entity that is assumed to have a common set of causal relationships. If there are different sets of causal relationships then it becomes a causal mixture model where the mixture is represented by using a hidden binary variable (Glymour and Cooper, 1999).

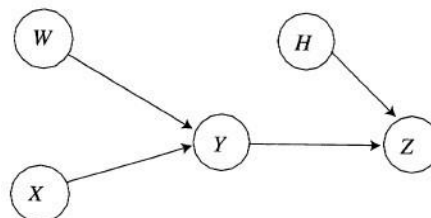


Figure 3.8: A causal Bayes net with a representation for causal mixture from Glymour and Cooper (1999).

Representing the different causal relations as a node (see H in the figure above), however, does not seem satisfactory to me contrary to Glymour and Cooper who claimed that is an adequate way to represent mixtures. The relation(s) between the mixture (H) and any other variable (Z) would be different (and probably more complicated) rather than the structural relation that is already assumed to be different from. In the next chapter, it is rather suggested that representing the different causal relationships appealing to the term of relation itself. In this specific case, it would correspond to such qualitative (and unfortunately loose) representation in figure 3.9.

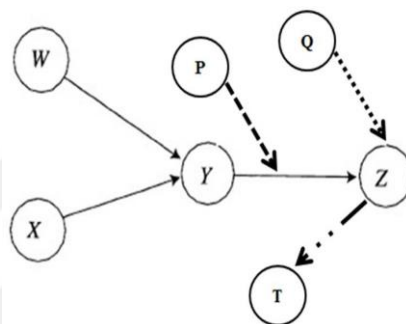


Figure 3.9: An oversimplified suggestion of representing causal mixture models.

Although the discussion is kept it short in this section, as Glymour and Cooper (ibid) put, the discovery (and to me, the representation) of mixtures of causal structures is a challenging and largely open problem. After all, probabilistic dependence is neither necessary nor sufficient for causation (Reiss, 2008). It is not necessary since there may be cancelling causes and not sufficient since there are cases of collider variables, and non-stationary time series (ibid) where the changes in probabilities can not be accounted for its direct causes (namely, its parents).

iii. Invariance: Invariance condition is met when "a relationship between two or more variables is invariant if it would remain continue to hold - would remain stable or unchanged- as various other conditions change" (Woodward, 1997). A safe definition for causality would be the one that holds property of invariance, thus a causal claim becomes generic and cleansed from any spurious or correlational relations. In that sense, invariance method is

developed as a test for causality in the early econometric models (suggested by Cowles Commission), and had further improvements by the works of James Woodward and Daniel Hausman with philosophical insights. Woodward and Hausman use the term of invariance in the sense that stability of the functional relation under some changes. Such that, there are equations that have a causal interpretation, satisfy certain requirements like invariance under intervention and independence of mechanisms (Hausman and Woodward, 1999). The equations that are studied by Woodward and Hausman mostly have the linear regression form as in equation 3.1.

$$Y = \alpha X + U \quad (3.1)$$

In the equation above Y represents the dependent variable and X represents the independent variable where U is the error term that stands for omitted causes. The parameter α represents the magnitude of X . If Y changes in the way described by the equation then this equation represents a causal relationship (between X and Y). As Woodward and Hausman put it, if Y doesn't change in that way as a result of intervention that changes the value of X , then the equation will not be a correct description of the causal relationship between X and Y . Thus, in this framework, invariance is a property of being causal whereas intervention is a way to test that invariance conditions. Woodward and Hausman also note that even some regression equations have the same mathematical solution, they might stand for different systems of causal relations. The structure of the equations bears a syntax for specific causal route.

$$Y = aX + U \quad (3.2)$$

$$Z = bX + cY + V \quad (3.3)$$

$$Z = dX + W \quad (3.4)$$

(where $d = b + ca$ and $W = cU + V$)

Equations 3.2 and 3.3, 3.4 represent two different systems of causal relationships:

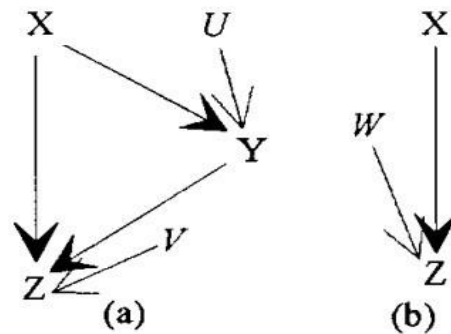


Figure 3.10: The causal structures for given equations of 3.2 and 3.3, 3.4. Retrieved from Hausman and Woodward (1999).

In the figure, (a) embodies the structure that is given by the equations of 3.2 and 3.3 whereas the structure of 3.4 is given as (b). In Woodward and Hausman's framework, the equations 3.2 and 3.3, 3.4 describe different (causal) mechanisms. Such "sets of simultaneous linear equations satisfying specific constraints" are, hence, thought to be causal representations in the framework of invariance accounts (Cartwright, 2007). David Hendry also advocates similar account in terms of social policies: "causes must [...] satisfy certain probabilistic conditions and they must continue to do so under the policy interventions envisaged" (ibid). As an additional assumption, Woodward (2003) puts forward 'modularity' condition which states that "the mechanism described by each individual equation be distinct from the mechanisms described by the others" (Hausman and Woodward, 1999). Furthermore, it is argued that "[...] as modularity fails, the asserted causal structure fails to mirror what will happen under hypothetical interventions and, [...] fails to represent correctly the causal structure of the system" (ibid). However, modularity seems to fail at many examples of causal relations⁸. In that sense, modularity is a highly disputed condition in the literature and it reaches beyond the scope of this thesis. Concerning the causal relation that Woodward and Hausman (1999) argue that within modularity there is no pre-supposition for linearity and/or additivity but rather the distinctiveness of the mechanisms is what modularity requires. In complex systems, as it will be discussed at the last section of this chapter, it is almost impossible to isolate such causal mechanisms. Moreover, Woodward and Hausman assert that their account is not

limited to linear equations, but rather such structural equations that are not linear can also be invariant under some changes and be modular:

$$Y = f(X) + U \quad (3.5)$$

$$Z = g(X, Y) + V \quad (3.6)$$

The structural representations for the equations 3.5 and 3.6 are not provided though, and as the authors admit such equations still assume that the error term, or say, the set of omitted causes, is additive (ibid). Hence, for nonlinear cases we have to assume that – at least the error term to be linear. To represent such cases like 3.5 and 3.6 in terms of causal structures, as it will be argued in section 3.3, linearity has to be assumed.

iv. Counterfactual: Hume had proposed a second definition (possibly he had not intended to do so) for causal dependence in a way that stands complementary to his views on regularity (Menzies, 2017): "where, if the first object had not been, the second never had existed." (Hume, 1748). Following Hume, Mill and Mackie are pondered about the logical forms of such conditional statements in order to analyze causal claims. The refined formalization of counterfactual account of causal dependence is found in the works of David Lewis. Counterfactual conditionals take the form of "if A had not occurred, C would not have occurred". The analysis of "had not - would not" clauses is at the center of Lewis' agenda of analyzing causality (Poellinger, 2012) in terms of possible world semantics. Lewis, indeed, takes counterfactual statements that are about possible alternatives to the actual situation (Lewis, 1973). Since "the semantics of conditionals exploits certain invariant relationships, certain dependencies" (Shulz, 2011), it is thought that causal claims can be reduced to counterfactual statements. Any alteration of the event changes the dependency relation between the events, thus expected to bring different consequences. Different versions of counterfactual dependence are available in the literature.

⁸ For details please see Cartwright 2007, Illari and Russo 2014, and others.

One of the versions⁹ of counterfactual account is "The Structural Equations Framework" that developed in the works of Hitchcock (2001, 2007); Woodward (2005); Woodward and Hitchcock (2003) in regard to Judea Pearl's works on causal inference besides the works of Peter Spirtes, Clark Glymour, and Richard Scheines on causal Bayes nets (Menzies, 2017). Pearl's agenda of causal inference, however, based on structural models rather than possible worlds semantics (Pearl, 2013). This account will be examined in section 3.2.

Despite the different versions of counterfactual account, the criticism toward counterfactuality mainly grounds on three basic assumptions of causation (Hall, 2004):

- (a) Transitivity: a is a cause of b, and b cause of c; then a is a cause of c.
- (b) Locality: Causal connection is provided via spatiotemporal contiguity of causal intermediates.
- (c) Intrinsicness: Causal relations hold by intrinsic, non-causal character.

Hall argues that counterfactual accounts are not compatible with those assumptions (where production accounts are) and in fact, require "an entirely different kind of analysis" (ibid). Similarly, Maria Carla Galavotti and Nancy Cartwright think that there might be a variety of different causal relations (Cartwright, 2007) which are suitable for different analyses. On the other hand, the issue of whether counterfactual accounts can handle the problems raised by the assumptions that are given above is still a disputable topic.

3.1.2 Causation as Production

Difference-making accounts, in general, put emphasis on the effects in terms of observed changes. It is thought that tracing the changes in the putative effects may unveil causation. Production accounts, on the other hand, focus on causal process itself. Since the idea behind the difference-making accounts is not in contrast with the idea of production accounts (at least in principle), there are also attempts to combine these accounts (for example, Handfield et al. 2008 attempt to integrate causal processes with causal Bayes nets). Yet, they still significantly differ in some respects.

⁹ Or, call it the extensions of counterfactual account.

Accounts that see causation as production (or say, process) depict causal link as "a continuous line in spacetime that transmits or propagates some kind of physical quantity or quantities" (Illari and Russo, 2014). In this sense, process accounts are more concerned to provide somewhat physical theory of causation. Earlier accounts of production are put forward under the influence of Bertnard Russell's later works on causality. Contrary to his earlier thoughts on causation, Russell thinks that physical occurrences unfold in causal lines. These causal lines picture causality in spacetime. In the same vein, Jerrold Aronson and David Fair appeal to physical depiction of causality based on the exchange of energy and/or momentum (Dowe, 2008). Causal relations, in these accounts, are represented as transference processes. Hence, Aronson and Fair advocated that causal relation to be "an objective feature of the world" (Dowe, 2000).

The problems with transference account of causation had steered the discussion into a 'processual' view of causality. Wesley Salmon defended a process theory of causation which can be generalized as "a token event *c* caused a token event *e* if and only if *c* and *e* are connected by a series of intersecting causal processes whose intersections constitute causal interactions" (Gallow, 2017). In the framework of Salmon's theory of causation, causal processes are regarded as a characteristic of some processes which transmit 'marks'. However, a series of new problems have arisen due to mark transmission. Phil Dowe developed a revised version of Salmon's account by pointing out those problems. Dowe advocated 'conserved quantities' instead of transmitted marks (Dowe, 2000). Similar problems are revealed mostly due to putative physical correspondents of causal links. As it can be seen, within production framework there is no established methodology and/or proposed models for causal processes. The application domain of production accounts is the physical models¹⁰ (e.g., the model for the electrical charge-exchanges) instead of a proposed causal model that comprises causal processes. Accordingly, the models that are investigated throughout this thesis rely on difference-making accounts of causation. However, it is noteworthy that information theoretic account of causation (that is classified under production

¹⁰ There is a debate on the application domain of process theories of causation. For detailed discussion please see Russo (2010), and Machamer et al. (2000).

theories in Illari and Russo 2014) may constitute an exception in terms of causal modeling. In information theoretical framework, causal link is somewhat quantified and thus it is possible to propose a specific model based on this account. A recent causal account that is based on the information theory is recalled ‘causal emergence’ by Hoel et al. But information-theoretical account is not issued in this thesis since it would require a sophisticated study on itself¹¹.

3.2 Causal Models

Even if there is no consensus on the definition of causation and the method for detecting it, at least, we can expect that a causal relation to be a stable relation across a given domain of constraints. Then the question is: Can we extract a schema of stable relations given dataset? Such a schema would constitute a structure that represents a (causal) mechanism. If the model is intended to uncover the structure underlying the data then it is classified as a ‘structural model’. Causal models on offer are all structural, in that sense What it is meant by structure, however, remains disputable. Earlier accounts that are adopted by Sewall Wright and Cowles Commission imply a mechanism that is already defined in theory. The recent accounts of Pearl and Mouchart et al., on the other hand, aim to ‘give structure’ within the background knowledge that is provided by either theory or analyses of data (Illari and Russo, 2014). Structural (causal) models encompass structural equation models (SEMs), causal graphs, causal Bayes nets (CBN) methods, and variational model-framework all together. However, the methodology for finding a causal structure may show variety. CBN adopts an inductive methodology (thus, it is an exploratory) whereas others use hypothetico-deductive inference method (Mouchart et al., 2010) which is a confirmatory method. Thus, each modeling technique has its own peculiarity in terms of interpretation of data but also some commonalities such as extra-statistical assumptions they made. In the following sections, the focus is that some of those assumptions which are claimed to be essential for causally interpret the data.

¹¹ For a comprehensive approach, please see the works of Collier (1999), Illari (2011), Floridi (2016), Hoel et al. (2013) and the related literature.

3.2.1 Structural Equation Models

SEMs are consisted of a set of equations that take regression form (such as the equation 3.7). The general framework of SEMs, initially, put forward by Wright (1921) with the form of a path diagram (for example, see figure 3.11) to represent causal relations. Beyond the mathematical description, SEM has a syntax for causal interpretation.

$$Y = \alpha X + U \quad (3.7)$$

The equation 3.7 forms a structural equation where Y denotes effect(s) (according to right-hand-side convention), X represents cause(s) and how much X affects (α), and U stands for omitted cause(s) or, technically speaking, error terms in our observation. This form of structural equation (which is in the regression form) allow analytical solution and thus, to the separability of the variables. It means that when the variables and their determinants are known (or simply presumed) it is possible to predict the outcome of a causal chain:

If we know the extent to which a variable X is determined by a certain cause M, which is independent of other causes, combines with them additively and acts on X in a linear manner, and if we know the extent to which M is determined by a more remote cause A, the degree of de-termination of X by A must be the product of the component degrees of determination. (Wright, 1921)

In the presence of latent variables (omitted causes) it is still feasible to make predictions once the structure is put in the regression form. The form implies proportionality of the effects in regard to causes under given conditions. If the structure is known a priori, the data of the effects would be sufficient to infer (direct) causal influence or likewise, the data among observed causes and effects can reveal latent variables.

To analyze causal chains Wright suggests the method of path analysis. This method "allows one to decompose the covariance [that is a measure of how much two random variables vary together] between two variables in a structural equation model into additive components, thus helping to understand how the inter-

relationships between many variables in a model predict the covariance between two selected variables" (Boker and McArdle, 2014). Performing path analysis would provide the prediction of system-states given the conditions by tracing the dependence relations.

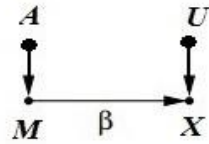


Figure 3.11: Path diagram of $X = \beta M + U$ and $M = A$. It is interpreted as A causes M and M causes X under the conditions of U .

Since the variables (the components) are decomposable, intervention (to specific variables) is possible. Remember the interventionist account which is based on structural equations. Due to the assumption of invariance, we expect that any change in the variables would steer a difference in the other as the way that is described by the equation. The equation propels a linear change in this form.

Notice that, even the analytical solutions for the equations are mathematically same, each set of equations stands for a unique solution (with right-hand convention) which turns out to be a unique structure. Thereby they might represent different causal routes which, philosophically, imply different (causal) mechanisms (Woodward, 1997).

3.2.2 Causal Graphs

Causal graphs are the DAGs that are causally interpreted (Scheines, 1997). As it is discussed in section 3.1.1, a DAG is a mathematical object that takes probability distributions among its nodes (variables). Thus, (in)dependency relations between the variables of interest are embodied in these graphs. Also, a DAG constitutes a structural model given the causal assumptions. Acyclicity (which means there are no feedback relations between variables), CMC, and Faithfulness are the basic assumptions that made up causal DAGs. However, to read the data in terms of causality — more specifically, the (in)dependency relations in the data, DAGs seek further assumptions.

Data generation process, as it is shortly discussed in section 2.2.2, is all about statistical assumptions that researcher appeals in regard to the observed (and/or experimented) phenomena. Linearity, normal distribution, additivity, homoscedasticity are among such statistical assumptions. Causal interpretation, on the other hand, brings extra-statistical assumptions on the table. Extra-statistical assumptions constitute the untested causal assumptions which include causal acyclicity, causal priority, causal mechanism (Russo, 2010). Accordingly, causal models, independent of whether the approach is exploratory or confirmatory, encode both statistical and causal assumptions.

SEMs in its traditional sense, do not hold specific causal assumptions but they impose a causal structure upon the system under investigation through the theories a priori given. Apart from traditional SEMs, Pearl in his seminal work on causal modeling (Pearl, 2009b) advanced the causal graphs with the encoded causal assumptions. Causal relations are represented in counterfactual forms. However, counterfactuality in Pearl's framework does not refer to possible world semantics that is put forward by Lewis. Pearl states:

In contrast with Lewis's theory, [structural] counterfactuals are not based on an abstract notion of similarity among hypothetical worlds; instead they rest directly on the mechanisms (or 'laws,' to be fancy) that govern those worlds and on the invariant properties of those mechanisms. Lewis's elusive 'miracles' are replaced by principled mini-surgeries, $do(X = x)$, which represent a minimal change (to a model) necessary for establishing the antecedent $X = x$ (for all u). (Pearl, 2013)

According to Pearl, to fit model to data a probabilistic analysis of counterfactuals is required, and that is provided through DAGs. Pearl delivers the link that serves as a bridge between causal models and observed data with the assumptions of d-separation and backdoor paths. d-separation is a criterion of determining whether a set of variables (say, X) is independent of another set Y , given a third set Z . With this criterion it is intended "to associate 'dependence' with 'connectedness' (i.e., the existence of a connecting path) and 'independence' with 'unconnected-ness' or 'separation'" (Pearl, 2009c). Backdoor and frontdoor conditions, similarly, rely on the separability of causal paths. Backdoor conditions are controlled by blocking the specific nodes.

Within such assumptions and counterfactuality, Pearl suggests a framework for causal calculus which allows to infer causal relations from a DAG under the "ideal manipulations and the changes in the probability distribution that follow such manipulations" (Scheines, 1997). Yet, the idea has roots in SEM:

[...]feature of invariance permits us to use structural equations as a basis for modeling causal effects and counterfactuals. This is done through a mathematical operator called $do(x)$ which simulates physical interventions by deleting certain functions from the model, replacing them by a constant $X = x$, while keeping the rest of the model unchanged. (Pearl, 2009a).

Causality is, thus, represented as (level) invariant, decomposable into the components that are assumed to be related causally (thus, additive), acyclic, and structural counterfactual relation. In the frameworks of Pearl and Spirtes et al. it is argued that linearity is not demanded and it is the most important departure point from traditional SEMs (Pearl 2009b; Morgan and Winship 2007). Then, structural equations are not necessarily consisted of linear functions. As an example, Pearl (2009b) presents the formalization below:

$$x_i = f_i(pa_i, u_i) \quad (3.8)$$

where $i = 1, 2, 3, \dots, n$

In equation (3.8), immediate causes of x_i , namely the connoting parents, are represented as pa_i , and omitted causes correspond to u_i . In this form, equation (3.8) constitutes a nonlinear, nonparametric generalization of the linear structural models (Pearl, 2009b). A similar argument is proposed by Woodward¹², where some nonlinear functions (like $Y = f(X) + U$ or $Z = g(X, Y) + V$) might represent invariant and modular structures. Yet, as it is discussed in previous sections, to argue that such equations to be structural, the functions should be linear in their omitted causes (e.g., U and V). In other words, to introduce invariance and modularity to nonlinear functions, at least the error terms (omitted causes) in the equation should be linear since it is not possible to decompose nonlinear equations analytically.

¹² See in section 3.1.1.

Mini-surgeries that are operated in Pearl's framework, CMC, Faithfulness and d-separation are, indeed, useful when the system in question is decomposable into some disjoint events (or parts, mechanisms) that can remain unchanged under specific interventions. However, in nonlinear cases (such as complex relations of biological systems mentioned in section 2.3.4) decomposing the system would result in loss of underlying (causal) relations. Think of, as an example, consciousness in humans. We (at least) know that specific brain regions are essential to be conscious; but even when we decompose all the neural system we are not able to explain how conscious processes are generated. Besides the 'big issues' like consciousness, at the abstract level of mathematics, nonlinear functions are not decomposable and thus treated with linearization methods. Likewise, in causal modeling we see that nonlinearity treated with the assumptions of additivity in noise (error terms) and/or additivity in parameters (see equation 3.9 from Mulaik 2009).

$$y = \alpha_1 + \alpha_2 x + \alpha_3 x^2 + \alpha_4 x^3 + \dots + \alpha_k x^{k-1} \quad (3.9)$$

Thus, linearity is assumed (at least to some extent) within the nonlinear representations of causal relations. Causal models on offer, in that sense, do not seem applicable to nonlinear cases unless the nonlinearity is being linearized. But why linearized modeling is not a satisfactory way to represent causality in nonlinearity is the question that it is intended to be answered in the following sections.

3.3 Linear Assumptions in Causal Models

The structure that is described by the equations, represents a causal relation. What is implied by a structural equation in terms of causality then, would suggest what kind of causal relation that we are looking for. Although Pearl (2012), and others (like Spirtes et al.) have argued that it is not limited to linear functions, "a structural equation suggests that the relation between [the given variables are] linear" (Illari and Russo, 2014). Indeed, in contrast to a wide range of examples of linear causal structures in the literature, nonlinear cases are often either neglected or linearized (with the assumption of additivity of the error terms or the parameters, or with the approximation). It is argued that causal models on offer depict linear causal

relations (even when nonlinearity is admitted) since the structure hold a linear(ized) form. The form of linear structural equation (as we have already seen many times) goes like this:

$$Y = \alpha X + U \quad (3.10)$$

In a causal model, this equation offers:

1. in which conditions X causes Y (α times X cause Y in the presence of U ¹³).
2. what remains unchanged in case of X causes Y .
3. X is proportional to Y (α times X causes Y).
4. what would happen when components are manipulated (if X is erased Y would not be occurred).
5. resolution of the structure by superposition:
 - (a) additivity of the components (each cause is additive, and U the error term is independent of X the cause).
 - (b) homogeneity of the system (as α increases the effect Y increases).
6. a causal mechanism that is not interbedded with any other. An intervention on X leave intact all other mechanisms besides the mechanism that previously determined the value of X (Woodward, 2016).
7. a description for graphical representation (two disjoint arrows that point Y and the strength of X to Y arrow is α).

The nonlinear functions in structural equations take the forms of:

$$Y_i = f_i(X_i, u_i) \quad (3.11)$$

$$Y = f(X) + U \quad (3.12)$$

¹³ Generally such a claim is supported with a set of structural equations in order to specify the conditions.

The given forms of equations in a causal model suggest the causal claims of:

1. Y is affected by X
2. in which conditions X causes Y (X causes Y in the presence of U).
3. (partial) additivity of the components (U the omitted cause is independent of X the cause).
4. the graphical representation is the same as the linear ones (two disjoint arrows that point Y : one is directed from U and the other defines a nonlinear function of $f(X)$).

The function $f(X)$ is not decomposable if it constitutes a nonlinear function. In terms of causality, thus, there is nothing much to infer about the underlying causal relation from this form. However if it is presumed (or known) that noise is additive¹⁴, and the nonlinear function is a priori given (in principle) the effects (or the future states) can be approximately estimated. Or else, if the nonlinear function is linear in its parameters (as α 's in equation 3.9), it provides proportionality which makes easier to trace the changes in the effect(s). With such assumptions prediction becomes possible - at least - to some extent.

Causation, indeed, seems to hold its predictive power in regard to proportionality. Think of, simply, if I consume fast food I will end up with increased body fat and weight; following the same reasoning if I consume too much fast food, the fat that I will gain would be commensurately increased. Under *ceteris paribus* conditions (e.g., same metabolic rate, same physical activity, etc.) it can be argued that fast food causes weight-gain. What if we observe that 90% of a fast-food-consumer population suffers from overweight? Does the same causal relation underlie in here?

In structural models, it is assumed that causal relation is implanted in the probability distributions. In fact, according to this framework, there are causal relations within probabilistic relations that realize in structural form. That is to say, each input related to an exact output (by this way, proportionality ensured) thus, each input is

¹⁴ For technical details please see Kun and Aapo (2016) and Peters et al. (2014).

actualized in a distinct mechanism. Causal processes, then, consisted of linear sequences of causes and effects that are not interbedded. Thereby, causation within the linear assumptions embodies such rules:

- i. The ultimate effect of the combined action of two (or more) different causes is merely the superposition of the effects of each cause taken individually (Nicolis, 1995). It does not matter whether causes are given separately or in a combined way.
- ii. Mathematically, causal processes can be modeled by (deterministic linear) differential equations since for any cause X , which is determined by Y with the unknown causes, $Y = X + U_Y$.
- iii. We can pilot the system's evolution by means of the causes of known consequences, which are always the same type and, above all, that are proportional to the intensity of the cause (Bertuglia and Vaio, 2005). It is simply because if X caused Y , we expect that two X 's will cause two Y 's.
- iv. Causality depends on the inputs merely; the combination of the inputs does not qualitatively change the output. The causal relation is fixed given the same causes.

3.4 Linearity, Nonlinearity, and Causality

In section 2.4 It is presented a list that comprises why linear methods are insufficient to represent the nonlinear phenomena. Likewise, throughout this chapter it is attempted to make explicit the linear assumptions made in causal models. Following that, here, it is intended to expand why causal models that are based on the assumptions of linearity are insufficient to represent the causal relations in complex (thus intrinsically nonlinear) systems.

(a**) *Ceteris paribus*: Causal models offer an optimized representation of a causal relation while others (independent factors) are held constant or fixed. That is to say, a causal event is modeled under *ceteris paribus* condition. Since each causal model stands for a causal mechanism that is not interbedded with another, the causality is represented under heavily restricted conditions. Consequently the causal model we have is an isolated representation of "a" causal relation. On the other hand, in complex systems all of the components are interrelated in such a way that we

cannot speak of isolated (causal) mechanisms. Moreover, complex relations that are realized during adaption and/or co-evolution would not be modeled with current causal models. The reason is that acyclic relations like feedback mechanisms are not allowed in causal graphs¹⁵.

(b**) Analysis: Causal models decompose the system that is to be modeled into probability distributions of the variables. Thus, the entity to be modeled is disintegrated. Separability of the components (which consist of all the variables, parameters, and omitted causes) makes what causal model is analyzable. Structure of the models is built on the assumptions that rely on the separability. Error terms (omitted factors) are separable since it is assumed that error terms are uncorrelated with each other and all the other variables in the model. The assumption of d-separation, especially, allows to determine the connectedness and separation of the variables under given conditions.

Causation, as a relation between the variables, is inferred from the probability distributions of the components. However, decomposing a complex system would not be a favorable method if one seeks to understand the behavior as a system, or any other systemic properties since analysis results in interruption of the relations. Complex systems, on the other hand, are highly-integrated entities and hold characteristics like emergence which cannot be foreseen by the mere information on the components.

(c**) Normal distribution: Causal models take probability distributions. Yet, to model dependency that is believed to indicate causality, a variety of statistical assumptions have to be made to get an interpretable dataset. One of the statistical assumptions is that the variables are normally distributed. But in nonlinear systems the distributions may (in fact, most of the time) show asymmetry. Given the causal model which is structural the parameters and the structure forms do affect the variables. Error terms, similarly, are not affected¹⁶ by intervention not since they stand for omitted factors (or say, noise). Since the relation between change under intervention. Intervention, if the model is adequate, would only

¹⁵There are studies on causal modeling with acyclic relations (e.g., Hoyer et al. 2012); however, the extent is limited to acyclic linear relations. In complex systems, acyclic relations are accompanied with positive feedback loops which result in nonlinearity as it is discussed in section 2.3.3.

¹⁶It is also an assumption as it is explicated in previous sections.

variables is nonlinear, in (complex) nonlinear cases, causal models assume that (at least) the omitted factors are additive which means that error terms are linear, and thus, normally distributed in the data. Consequently, I found that it is not a realistic treatment for nonlinear phenomena considering the open- systems where the omitted causes are more complicated to be classified under the term of noise or error terms. Variables and parameters are often found to be dependent on each other. This makes evaluating the system according to those average values of the data to be less reliable method.

(d**) Connecting dots and dependency: The main purpose of the causal models is to detect causal relations by exposing dependency relations. The dependency relation we look for is the one that remains invariant under specific changes. It is still an assumption; but also a good reason to believe that there is an underlying causal relation. However condition of invariance restricts causality to be fixed, proportional and thus, linear. The causal relation is fixed since it should remain unchanged under specific manipulations. Likewise, proportionality and linearity is encoded in the invariant (and modular) structural equations since the same proportion holds for same relation. On the other hand, complex systems are dynamical entities that undergo continuous changes. Also, historicity matters. That means a factor that is once a cause may no longer be a cause in the future states (one may consider the developmental processes, in this sense). Moreover, due to nonlinear relations proportionality does not hold within inter- relations of such systems.

(e**) Equation solving: Causal models offer a structural equation that holds an analytical solution, and a semantics to read the equation in terms of causality. Such structural equations hold the properties of additivity and homogeneity. Hence given superposition of the causes we can perfectly estimate the effects. The structural equation, thus, is a recipe for manipulation and prediction given the conditions. Even the system is manipulated, (it is assumed that) parameters do not change. It provides proportionality between the causes and their effects. However, today, complex systems can be modeled only in terms of nonlinear differential equations (because of the dynamicity). Nonlinear differential equations do not have analytical solutions. In this sense, they are not decomposable into its constituents.

Considering the problems that are listed above, the linear approach within causal models seems to be an untenable strategy to model causal relations of complex systems. To regard nonlinearity among causal relations, however, a new representational framework is needed since all of the modeling tools that we have today rely on linearity assumptions. Yet, at first, it should be provided that an ontological basis that embraces nonlinear relations. The last chapter, with that in mind, is reserved for alternative routes to take in order to find a causal representation that best fits to complex nonlinear phenomena.



CHAPTER 4

IS THERE AN ALTERNATIVE?

"How does causation work in nonlinear (and complex) systems?" is the big question that is mainly concerned in this paper. Since it cannot be answered easily, I attempted to approach the question through meditating on the nature of relationships in complex systems. In complex systems, the components are interconnected in such a way that the action of each component can produce more than one response. That is the point where linear causation becomes obsolete. On the other hand, the problem arises: How can we detect (and/or model) causality in such a mass of outputs?

The linear approach towards causation seeks agreement on the context, or to put in philosophical jargon, propels *ceteris paribus* conditions. Then, the components are represented as somewhat isolated from the environment and the complexity gets lost. Causality, due to linearity, bounded to the components solely, thus the relations that are assigned to the components do not hold dynamicity on their own. In other words, the components become overemphasized whereas the relational dynamics are ignored. On the contrary, in complex systems we see that the very same components may build connections in different compositions, thus can lead to different characteristics. A given representational framework that focuses on the causal relations rather than solely components, is it possible to capture the process of system's evolution?

I think it is possible and the very reason for that is any formalization that undermines the relations will not be sufficient to account complexity since it arises from the relations among the components (within their environments). As it is discussed in this paper, the relations are nonlinear as in the positive feedback loops. In the literature, positive feedback loops are recalled 'circular causation' or 'reciprocal causation' as well, and yet, we lack of a sound formalization for such loops in terms of causation. In fact, they are ignored since positive feedback cycles violate the assumptions of causal acyclicity.

Contrarily, the argument in here is that representations based on the dynamics of nonlinearity may provide causality in complex systems. To achieve that, however, we need "new tools of thought" (Prigogine and Stengers, 1984) rather than the linearized models of nonlinear relations.

4.1 On Possibility of a Nonlinear Causal Account

Modeling as a tool for thinking in sciences (and philosophy of sciences, of course) has its pros and cons. First of all, we develop models because models help us improve our understanding of entity of interest. Entity is being represented in models in a way that it becomes idealized in regard to manageable information load for humans. Due to the limited humane abilities, thus, models hold cognitive significance in sciences at the least. If not just for understanding the entity in question, models, when are adequately put, serve as prediction machines that can yield accurate results for circumstances given the input. In parallelism with the entity being represented, the trajectory of the behavior of the entity can be inferred through the related models. Yet, there are strict restrictions within the models. For example, models – by definition, can represent the entity only to some extent. They are restricted representations of the entities. In that sense, models are applicable to a class of circumstances rather than all of the possible scenarios. In linear systems, however, as it is discussed in section 2.2 that restriction may not constitute a problem at all. The reason for that is the behavior of the linear systems can be precisely estimated through a modeling principle which is called the rule of superposition. Contrarily, complex systems cannot be dispersed to the superposition of their parts. Besides such a drawback, a few other reasons that restrain to model complex systems are also presented in sections 2.3.1, 2.3.2, and 2.3.4. When the linear modeling tools are applied to such systems it does not seem that complex systems are represented fairly. Supporting that claim, it can be also considered that the everyday examples like inaccuracy in the predictions for the behaviors of stock markets, societies, or epidemics, etc. The insufficiency of linear modeling is already discussed in section 2.4. The lack of tools that regard nonlinearity and complexity is apparent. Then what can be done? First point that is in need of urgent clarification is that the procedures of representation and linearization in respect to modeling complex systems. It is said that models are kinds of idealized representations of the

entities of interest. As an example of representation consider Figure 2.4 in section 2.2.2 where the understanding of the subject that is taught to the school children is represented in the numbers of their test scores. An example of a model as a representation, on the other hand, would be the SEMs that stand for underlying causal mechanism of an event. In each case there are some assumptions – mostly statistical, and within such assumptions entities in quest are represented as in a linearized form. That is to say, the representations hold the properties of linearity such as separability or being normally distributed. The linearized form allows us to analyze the systems without disturbing the other factors which are not interested in our research question. In that sense entities cleansed from unnecessary details and thus representations can refer the entities in manageable forms. Otherwise, if all of the details were somewhat represented, the information load of those would be enormously high to comprehend. Moreover, as we have seen in the proposed causal models, nonlinearity does not allow decomposing the system mathematically and consequently, it is treated with either an additional compact term of some nonlinear function, such as $f(X)$, or linearization in terms of parameters. Even though such linearization processes are executed, the models are insufficient to provide understanding and to predict the behavior of the entity. Yet, due to the properties of nonlinearity and being complex any attempt to represent such complex entities seem obliged to be linearized at least to some extent. Then the question is that: do representing and modeling necessarily prompt to (at least to some extent) linearization of the systems of interest?

I think there is no such an obligation, however, we lack of tools to represent otherwise. Linear tools are handy and convenient whereas the models that are provided today by Nonlinear Dynamics Theory are mostly accessible to us via computer simulations since the data among complex systems are too hard to process. Eventually, it makes us to be disposed to use linear methods. The way of representing should be in regard to the nature of the system in question. Thus, how to achieve such a representational framework for complex systems is bound to our understanding of the nature of the complex systems. As it is discussed in previous chapters, dynamical relations rather than the mere components may provide an insight for understanding complex systems. At this point, then, I could only suggest to follow that line of thought: the representations should be based on relational

dynamics. But, how to capture relational dynamics? That is the exact point where we require new tools of thinking. The seduction of the linear thinking is so dense that, as it is discussed in section 3.3, even though we admit the nonlinearity we still attempt to reduce such nonlinearities into linearized forms. The reason for inability of linear thinking to capture relational dynamics is, first of all, that the linear approach assumes that the relations are fixed. That is to say, the relation between two (or more) components cannot vary. The only way to change a relation is that changing the components. In that sense, it is thought that relations are fixed by the bound components. Likewise, relations that hold efficacy are considered to be causal relations and such relations are also thought to be fixed, component-dependent, and invariant. Causation, indeed, seems to agree with such conditions intuitively. For example, if aspirin relieves headache we expect that every intake of aspirin will do that. Does that mean causation is necessarily a linear concept? It is the second issue that needs clarification.

In modeling the researcher deals with the states that stand in lieu of events, and their relations. The way of states relate may show linearity or may not. If the concomitant state is the linear combination of the previous one then it is called a linear relation between those states of interest. With additional assumptions of causation those states may be considered as causally linked also. But, notice that, causation as a relation type does not impose linearity at all. In SEMs and causal graphs that additional assumption toward causation is usually acyclicity of events¹. Cyclic relations, on the other hand, mostly yield disproportionality between the initial state and following states². In such cases, we expect that states are somehow related and there is causality in between, yet, the concomitant state (namely, the alleged effect) does not follow linearly. In linear systems causation may work as a linear relation, but not in nonlinear cases. In that context, I would suggest that there has to be a different causal account for nonlinear relations. Before making any suggestions for a new causal account, first, we need to know that under which circumstances a relation is called causal but not the otherwise. What is it expected from a causal

¹ If not, linearity of cyclic relations is usually what it is assumed. Please see section 3.3 and the related literature.

² See section 2.3.4.

relationship? This takes us back to the fundamental question: what is causation? The challenges to answer that are already made explicit in earlier debates on causation, as an instance, remember INUS conditions that are put by Mackie as the least requirements for being a cause. In the contemporary scene we have seen that identifiability of causes has gained more attention rather than understanding the nature of causal relation. It is believed that once the causes are identified, the effects following those will be unraveled or vice versa. The most striking drawback with all of those accounts of causation, I believe, is that we became so obsessed with hunting causes that the true nature of causality namely, the causal relation itself is undermined. Such an attitude towards causation may not constitute a problem within linear systems since the behavior of the system (the effects) can be inferred through mere inputs, or say, causes. However, when the case is nonlinear complex systems, mere input is not informative in terms of the future behavior of the systems. Relations are dynamical in complex systems. Because of such relational dynamicity, the very same causes may bring out different effects³. For a nonlinear causal account, then, the relational dynamics should be considered. I think that if causation is regarded to be a special relation rather than some influence power of the causes, such an account would enable to capture dynamicity of the complex relations. Considering the causal models that are available to us today, I would say that in those models causality is trapped in the nodes and that is the main reason for their inability to capture dynamicity. It is because in those models causation is represented as a fixed dependence of a node to another node. A change in the causal relation can be realized only if the nodes are changed. Different causal relations, likewise, are introduced as another node (remember the H in causal mixture models from section 3.1.1) in the causal structure. However, there is another reason that I found it should be reconsidered in context of complex systems. It constitutes the third point that in need of clarification: the assumption of causal acyclicity. The assumption of acyclicity simply states that if C is a cause of E then E cannot cause C. It is, indeed, very intuitive to think like that, for example, throwing a stone causes windows to be broken but a broken window would not cause the act of throwing a stone. Nonetheless, there are such cases that the causal acyclicity seems to be lost.

³That ability is technically called multifunctionality.

Consider, for example, the process of child birth. Once the contractions of labor has begun, the baby's body is being pushed towards the cervix and the cervix is stimulated. That stimulation leads to activation of neural signaling which causes oxytocin release. The released oxytocin causes more uterine contractions that make pressure on the cervix. In this case, oxytocin release – which is the effect, steers pressure on the cervix – that is the cause, thereby this event constitutes an example of a positive feedback loop. In section 2.3.4, a similar case (Rayleigh-Bénard cell convection) is already discussed in detail and it is advocated that characteristics of complex systems like self-organization arise due to such feedback relations. Yet, how systems feature such characteristics (e.g. self-organization) through such complex interactions is not a completely resolved issue even for today. The answer, however, may be given in terms of autocatalytic processes, which is in support of my claims on relational dynamics.

An autocatalytic process is when is the case that the end-product of a (chemical) reaction is a catalyst of its own production.

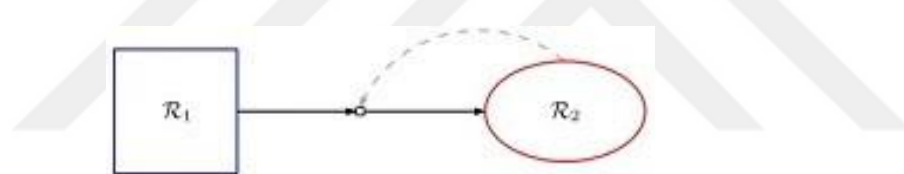


Figure 4.1: Image is retrieved from Hordijk and Steel (2015).

As it can be seen from the graphical representation, the catalyzer intervenes to the relation itself rather than the reactant(s). In that form of relations, it is possible to represent the disproportionality between causes and effects since effect can directly influence the relation that in turn affects itself. Due to the ability of direct interference to the relation itself, relations would be dynamical rather than static forms. I believe that causal relations in complex systems can be modeled in. such a form of representation where relations may bear dynamicity on their own That is to say, a causal relation can be changed without (heavily) disturbing the cause-component. Similarly, an effect can be produced via different causes. A simple illustration would be that a causal relation that ends up with an effect of raised arms. An activity of neural clusters in brain causes the act of raising the arms where the

arms could have been raised by an external force, say by ropes. Since there is no abnormality in neural paths that can block the act of raising the arm, cause-component in this case is not disturbed. It is already discussed in 3.3. This that mere dependency on inputs (the cause-components) disregards such cases of degeneracy. Counterfactual reading of the causal models – like, Y would not have happened if X is not present, fails to explain degenerate cases since effect is dependent on a cause which manifests a relation in a fixed structural form. If X is somewhat deleted then it would have resulted in absence of Y (since same causes lead same effects). By this way it is inferred that X causes Y . However, in complex systems there are many cases that deletion does not result in absence of effects. For example *Drosophila* neurons have cytoplasmic Abelson tyrosine kinase – which has a role in neural development of the animal, and when the researchers have deleted of the gene that produces that enzyme there is no observed abnormality in neural development of the animal. They have found that a protein fasciclin, a cell-adhesion protein that “has no obvious structural or functional similarity” to the enzyme, seems to taking the role of that enzyme (Elkins et al. 1990 via Edelman and Gally 2001). In this case the best tool that a causal modeler has is that absence of the kinase would be represented as a different condition U_2 which would be different state than ‘the kinase X causes some neural developmental products Y under condition U_1 ’. Yet, I found it is not appropriate because the conditions should be considered as equivalent due to the fact that there is no additional (external or internal) constraint in the developmental process of the animal but the mere intervention on the kinase X . I would argue that when the kinase is deleted the relational dynamics are changed in such a way that a new relation is built between the fasciclin and the products. Please note that it is not specifically advocated that causal pluralism (both kinase and fasciclin as the causes), but dynamicity of the causal relations is put forward as a game-changer. Dynamical causal relations which can be represented in ‘relation to relation’ form are promising in that sense. In this vein, as an alternative Stuart Kauffman’s work-constraint account of biological organisms (2000) and Montevil and Mossio’s work on organizational closure (2015) which is in the same line of Kauffman’s thoughts, may provide a framework of dynamical relations. Rather than emphasizing nonlinearity, however, they embrace relational dynamics of biological systems in their accounts.

Since the relations are not fixed, nonlinear causal interactions can be represented in such a form. On the other hand, a few drawbacks exist in that framework. A major disadvantage that I found is that there is no established mathematical framework of ‘relation to relation’ interactions. In causal models that are available to us today, since it is not allowed that kind of ‘arc to arc’ representation, nodes are responsible for the establishment of the (causal) relation. Each node as a variable is mapped to another, by this way, relations are expressed in terms of functions. Functions take arguments which can be a function on its own, yet, functions have to end up as variables. Relations are mathematically expressed as the form of equations and thus mapping a relation to another is not an acceptable way to represent in such a syntax. Another concern of a ‘relation to relation’ would be that the representation of the point of where the relations intervene to other relations. It can be clearly seen that point which is represented as a little black box in figure 4.1. Should that point be considered as a node? If it should, then what would be the difference of ‘arc to arc’ from ‘node to node’ ones? At this stance it seems problematic, indeed. Yet, I think the problem arises because of the ambiguity of our understanding of relations. In Montévil and Mossio’s account⁴ components are believed to be holding causal powers, or say dispositions to establish a relation. It makes components to be prior to relations which constitutes a claim that I found highly problematic. Besides the philosophical literature on such *potency* to cause which is disputable since back to Aristotle, I argue that causality should not be understood in terms of causal powers of the components. Because it will not be possible to postulate an account that allows to represent ‘relation to relation’ as long as causation is attributed to some causal powers of the components rather than the relation itself. Then, to suggest an alternative account, first, we need to tackle the question of where to localize causation in terms of ontology. I argue that causation should be localized on the relation that is not supervenient on properties but ontologically primary.

4.2 On the Ontological Status of Relations in Complex Systems

The basic tenet of current causal models is that the linearity of the causal relations linearity implies that causes (and given conditions) hold additivity and homogeneity

⁴ Even though it is not explicitly discussed, by the terms of ‘causal powers’ it is implied. Please see more in Mossio et al. (2009), Montévil and Mossio (2015), Mossio et al. (2013).

properties since each cause invokes a specific causal mechanism that is not interbedded with the others⁵. By this way, each causal mechanism can be modeled as a structure that is fixed between its relata⁶. That is the reason for the causal models are also called structural models. Here the structure serves as a template of causal relation that is to be work in the same principles (which are linearity, for proofs please see section 3.3) for all causal events. It is, indeed, appealing to intuitions about causation since we expect that a causal relation holds somewhat universality - same causes always bring same effects. Yet, it is discussed throughout this thesis that, nonlinear dynamical construction of complex systems contrasts with our intuitions of causality. Concerning causal models, on the other hand, the structure does not allow representing such nonlinear dynamicity. Besides the linearity assumption, causal assumptions like acyclicity constitute the ontological commitments toward the causal relations of the modeled system. I advocated in previous section that such assumptions disregard the dynamic-relational nature of complex systems. Here, I take my claim further: if ontological commitments of the models in regard to relations are not reworked, consequently, causal models will remain insufficient for complex cases. But, first, there is a philosophical challenge has to be canvassed: what is meant by the term of relation precisely?

Relation is such an entity that holds between its relata. In causal models relation is, as it is mentioned before, represented as a dependency between variables thus, relata are the variables. The dependency relation is embodied as the structure, and this structure is built by relata⁷. Any difference in the relata consequently changes the structure⁸ but not otherwise. Structure itself cannot change unless (at least one of) the relatum is changed. Ontological presupposition in here, then, is that causal relation is supervenient on its relata. To put in other words, causality is “no addition of being” (Armstrong, 1997). Thus ontological status of (causal) relations is a secondary

⁵ Separability principle.

⁶ Entities that get related. Since complex systems are made up many components, causality is already admitted as a polyadic relation.

⁷ Please consider variables in structural equations.

⁸ See figure 3.10 in chapter 3.

position. I found it is problematic since in complex systems, relational dynamics steer the system behavior such that these systems are decentralized yet operate as a whole, give rise to emergent properties, hold degeneracy and multifunctionality. Thus, rather than solely determined by its relata, causal relations seem to have dynamicity on their own. In that sense the ontological assumption of causal relation is determined only by its relata might be misleading considering causal models. Attributing dynamicity⁹ to causal relations, however, makes one a causal realist. That is to say, causation is a part of reality.

In causal models on offer, then, ontological commitments that are made in regard to causal relation itself rely on reductive analysis of causation. In fact, that should not be a surprise since it is already admitted that elusive nature of causal relation does not allow realistic approach and thus the adopted methodology is reductive in that sense. Yet, as I addressed throughout the thesis, to model dynamical causal relations an ontological commitment to causal realism would be more eligible in complex systems modeling. However, lacking of representational tools for causal realist modeling is a striking problem.

As a recent alternative, Mumford and Anjum (2011) proposed vector models based on (pan)dispositionalist ontology. Dispositionalism, roughly speaking, implies that there exist things which have properties and all these properties are (causal) powers, or say dispositions (ibid). Commitment to powers which are causal, it is argued, opposes with Humean reductive approach to causation. Thus vector models are supposed to embody causal realist attitude toward causal modeling. Authors explain this as follows¹⁰:

Neuron diagrams are conducive to a Humean ontology and, through a widespread and sometimes unquestioned use, they promote that ontology. If one were to be a realist about powers, however, one could opt for a better way of depicting a causal situation. Such a way will be offered – the vector model (...) (ibid).

⁹ Here, the ability to change per se interpreted as a sufficient condition for existing.

¹⁰ By the term of neuron diagrams they provide the examples of causal DAGs. Besides that, even they just mention SEMs that are advanced by Pearl in one sentence they do not claim any position for or against SEMs. However, the given descriptions for ‘neuron diagrams’ by their use of the term, it seems that they refer causal structural models in general.

Vectors, in that framework, represent “causal powers” which are “dispositions that are operating” upon a space called “quality space” (ibid). Direction (which state is to be disposed) and intensity (length of the vector) is also represented with vectors in 2D or in more complex cases 3D spaces as follows:

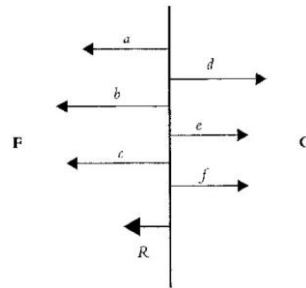


Figure 4.2: A vector model and causal powers of a, b, c, d, e, f which give rise to R as a sum of the causal powers. Retrived from Mumford and Anjum (2011)

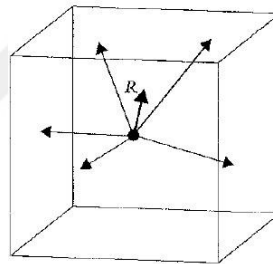


Figure 4.3: 3D quality space by Mumford and Anjum (2011)

As it can be seen from the figures, there are no relata represented but tendencies to be occurred. However, vectors are additive in resemblance to Mill’s sum of sufficient causes (ibid). Tendencies or their representations as vectors are drawn as in “flux”es while it is not shown that consequent (the effect) of exercising disposition. In that sense it is hard to imagine a relation between the cause and its effect but some ‘causing’s. In fact it is intended to model in that way since relations (according to dispositionalist ontology) are somewhat dispositional properties (Ellis, 2007). Thus dispositionalists rely on the idea that there exist non-relational monadic properties which are the real constituents of the world. Events, in that sense, are the

property instantiations where dispositions are manifested. Relations are not ontologically fundamental since causal work is executed via powers that are held by components. So, it is portrayed that “relatedness without relations” (Fisk, 1972). The ontological status of relations, in that sense, is again secondary in dispositionalist view. Yet, it is argued in Mumford and Anjum (2011) that vector causal models which are based on dispositionalist ontology can represent nonlinearity. In what follows I put my argument against it as an example of why undermining relations ontologically will not help us in our attempts to represent dynamicity of causal relations which most of the time lead to nonlinearity.

Vector models represent background conditions also as vectors, thus, a model comprises causes, conditions, and a side in quality space which stands for each of the causes and conditions is disposed to. Since there is no relation in terms of ontology, all there exist causal powers (as vectors). The effect which is to be disposed to, then, is determined via a calculation of the sum of vectors (ibid) and thus combination of powers does not matter. In this sense, vectors are (and obligated to be) additive. Although Mumford and Anjum acknowledge nonlinearity in complex cases, it is not provided a representation of non-additive vectors but a metaphoric representation given below. Yet, nonlinearity, to repeat what is put before, is the disproportionality between causes and their effects.

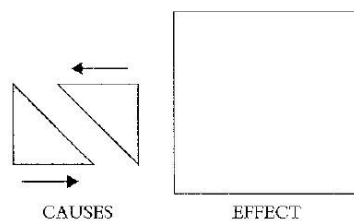


Figure 4.4: Image is retrieved from Mumford and Anjum (2011).

Butterfly effect which is the most known exemplar of nonlinearity of causation is, roughly, that small causes can lead big effects. Prediction horizon of such nonlinear cases is very narrow since dynamical relations – even within few numbers of interacting elements as in chaotic systems, make effects untraceable. Dynamicity of causal relations between the elements rather than the sum of causal potencies of elements is what makes up nonlinearity. Underrating the relations ontologically limits modeler to represent mere components (or powers that behold

by components) which result in, I recalled as, additivity fallacy¹¹. See that in vector models, causal powers do not intersect¹². Similarly, in structural models, causal relation that is the structure cannot intersect with any other and relata are (additively) placed in the regression form of equation. Dynamicity remains to be missing.

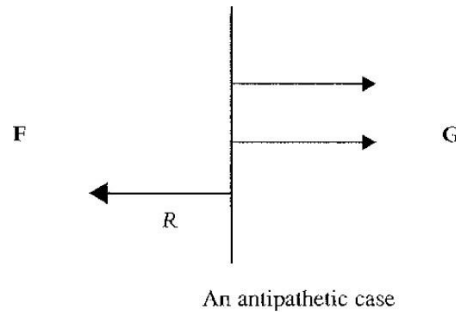


Figure 4.5: A case for two drugs introduced simultaneously react reverse effect. G space represents wellness of the patient and F space stands for illness, as put by Mumford and Anjum (2011). Notice that causal powers do not intersect even in the cases where causes are ‘acting together’.

All we can get by addition is that aggregates of elements (relata) where combination of the elements does not matter. But, in fact, combination is what really matters in complex systems. In its simplest terms think of two drugs which have different functions and lead to different effects by individually. These drugs when combined, however, may reveal wholly different function. The drugs cooperatively do work which means they are related and that relation is built in such a way that a new function is executed. However, if the ontological commitments that are made in causal models address that the relations as prior whereas properties or components are ontologically secondary, I argue, it might be possible to represent dynamical causal relations. Here, I mean that if the (causal) relations are taken to be real, then the representation of intersecting relations¹³ would not be disregarded. Metaphysical implications of such a stance, however, seem to recall

¹¹ Please do consider complex systems. If linear systems are issued, then it would not constitute a fallacy at all.

¹² In Nicholson and Dupré 2018 which is a recent book that is released just before this thesis being submitted, Mumford and Anjum put forward a revised version of their model to account overlapping causal processes at page 69. However, I found that it does not constitute a satisfying rework of their model since overlapping causality is represented as (distinct) causal powers exercising some effects in parallelism through a time interval rather than in cooperation.

¹³ I consider relations that are lacking in current models analyzed in previous sections.

ontic structural realism (OSR). OSR, simply, states that all there is that the relational structure rather than individuals (Ladyman et al. 2007 and Ladyman 2016). Thus relations are ontologically primary. Individuals as ontologically independent from relations cannot be existent. Some authors (such as Psillos 2001) stand against that since if all there is relation then there are relations without relata; however Ladyman emphasized that OSR does not require that but rather, relatum cannot be individual on its own. I found OSR as too radical considering their metaphysical attitude; on the other hand, for modeling purposes I believe that it might be helpful as an ontological basis to represent dynamical causal models since the relations are regarded. Yet, it is an open discussion in philosophy that how OSR accounts causal relations metaphysically¹⁴. Thus my claim is limited to modeling purposes: the ontological commitments in causal models for complex systems should regard relations as prior. In that framework, then, properties and/or components could be accounted secondary and it makes sense since in complex systems components do not exist individually yet their relations dynamically determined into their roles (or say, functions)¹⁵.

¹⁴ As a primary reference one can see chapter 5 ‘Causation in a Structural World’ in Ladyman et al. (2007) and for more Saatsi (2017).

¹⁵ One can think of plasticity and degeneracy like in case of *Drosophila* from previous section.

CHAPTER 5

CONCLUSION

Sciences make use of models in order to explain the phenomena at hand, and if possible, to predict the outcomes when manipulated. The scope of this thesis, thus, is consisted of causal models, since it is believed that explanation and prediction can be achieved through answering the ‘why-questions’ (Salmon, 1984) about the phenomenon. The model construction, on the other hand, requires some ontological commitments toward the nature of the phenomena. Notice that, during this process, it is not intended to question the nature of what-is out there but rather to assume what might be out there. Those assumptions constitute the frame of the models. All of the technical work is executed in that framework. Some models, however, may not be well-suited for a set of phenomena. In face of such inadequateness of models there are two main strategies to follow: either seek for advancements on the technical details (which sometimes lead to ad hoc) or rework on the framework itself. This thesis addressed a call for latter strategy within causal models concerning complex systems. I advocated that the ontological commitments should have been questioned and this would require a philosophical study on itself. Because it seems that building assumptions of the models – rather than the practical drawbacks like lack of full-knowledge of an event, in fact, impede adequately representing such systems. Those assumptions are, I argued, linearity and acyclicity of the causal connection.

Representing causality as linear and acyclic relation appears to be incompatible with nonlinear settlement of complex systems where, also, many cyclic events have been. In the literature, there are few technical interventions that have already pointed out that either additivity (which is a property of linearity) or acyclicity had to be revised in order to apply causal models to nonlinear phenomena. Yet, lacking of philosophical background is the reason of those revisions to be remained shallow. At that point, what does it mean for a system to be linear or nonlinear has to be distin-

guished. Such a comparison revealed that proposed nonlinear extensions of causal models, in fact, constitute the linearized versions of nonlinear cases.

Linearization would not be a problem in cases of nonlinear but close-to-equilibrium (which means near to being orderly) systems, however, it is a problem within complex systems since those systems are in between complete order and chaos. Such mediator state of complex systems is maintained through dynamical spatiotemporal relations. Linear framework, on the other hand, accounts (causal) relations to be fixed rather than dynamical. In that sense, I have indicated that a new framework is needed. However, since all of the available tools are developed to serve in linear framework, that alternative framework would have suffered from scarcity of tools. To overcome such challenges, it is implied that an ontology which prioritize relations rather than relata would be a way out.

REFERENCES

- Armstrong, D. M. (1997). *A world of states of affairs*. Cambridge: Cambridge University Press.
- Arntzenius, F. (2010). Reichenbach's common cause principle. In E. N. Zalta (Ed.), *The stanford encyclopedia of philosophy* (Fall 2010 ed.). Metaphysics Research Lab, Stanford University. <https://plato.stanford.edu/archives/fall2010/entries/physics-Rpcc/>.
- Bak, P., Tang, C., & Wiesenfeld, K. (1987, Jul). Self-organized-criticality: An explanation of the 1/f noise. *Phys. Rev. Lett.*, 59, 381-384.
- Baumgartner, M. (2008). Regularity theories reassessed. *Philosophia*, 36(3), 327– 354.
- Bertuglia, C. S., & Vaio, F. (2005). *Nonlinearity, chaos, and complexity: the dynamics of natural and social systems*. New York, NY: Oxford University Press.
- Boker, S. M., & McArdle, J. J. (2014). Path analysis and path diagrams. In *Wiley statsref: Statistics reference online*. American Cancer Society. Retrieved from <https://onlinelibrary.wiley.com/doi/abs/10.1002/9781118445112.stat06517>.
- Capra, F. (1997). *The web of life: A new scientific understanding of living systems*. New York, NY: Anchor Books.
- Carpenter, S. R., & Kitchell, J. F. (1988). Consumer control of lake productivity large-scale experimental manipulations reveal complex interactions among lake organisms. *BioScience*, 38(11), 764-769.
- Cartwright, N. (2007). *Hunting causes and using them: Approaches in philosophy and economics*. New York, NY: Cambridge University Press.
- Chen, C.-T. (1995). *Linear system theory and design* (2nd ed.). New York, NY: Oxford University Press.

- Chirimuuta, M.(2017). Explanation in computational neuroscience: Causal and non-causal . *The British Journal for the Philosophy of Science*.
- Clatterbaugh, K. C.(1999). *The causation debate in modern philosophy, 1637-1739*. New York, NY: Routledge.
- Collier, J. D. (1999). Causation is the transfer of information. In H. Sankey (Ed.), *Causation and laws of nature* (pp. 215–245). Dordrecht: Springer Netherlands.
- Dai, L., Korolev, K. S., & Gore, J. (2013). Slower recovery in space before collapse of connected populations. *Nature*, 496(7445), 355–358.
- Dakos, V., van Nes, E. H., & Scheffer, M. (2013). Flickering as an early warning signal. *Theoretical Ecology*, 6(3), 309–317.
- Daniel, W. W. (1991). *Biostatistics: a foundation for analysis in the health sciences*. U.S.A: Wiley.
- Dowe, P. (2000). *Physical causation*. Cambridge: Cambridge University Press.
- Dowe, P. (2008). Causal processes. In E. N. Zalta (Ed.), *The Stanford encyclopedia of philosophy* (Fall 2008 ed.). Metaphysics Research Lab, Stanford University, <https://plato.stanford.edu/archives/-fall2008/entries/causation-procesess>.
- Druzdzal, M. J. (2009, September). The role of assumptions in causal discovery. In *8th workshop on uncertainty processing (wupes-09)* (pp. 57 – 68). Retrieved from <http://d-scholarship.pitt.edu/6017/>.
- Dye, F. J. (2011). Embryology. In *Dictionary of developmental biology and embryology*. Hoboken, NJ: Wiley-Blackwell.
- Edelman, G. M. (1974). *Cellular selection and regulation in the immune response*. New York, NY: Raven Press.
- Edelman, G. M., & Gally, J. A. (2001). Degeneracy and complexity in biological systems. *Proceedings of the National Academy of Sciences*, 98(24), 13763–13768.

- Elkins, T., Zinn, K., McAllister, L., Hoffmann, F. M., & Goodman, C. S. (1990). Genetic analysis of a drosophila neural cell adhesion molecule: interaction of fasciclin and abelson tyrosine kinase mutations. *Cell*, 60(4), 565–575.
- Ellis, B. (2007). Powers and dispositions. In *Revitalizing causality: Realism about causality in philosophy and social science*. New York, NY: Routledge.
- Feldman, D. P. (2012). *Chaos and fractals: an elementary introduction*. Oxford: Oxford University Press.
- Fisk, M. (1972). Relatedness without relations. *Nous*, 6(2), 139–151.
- Floridi, L. (2016). *The routledge handbook of philosophy of information*. New York, NY: Routledge, Taylor & Francis Group.
- Frigg, R., & Hartmann, S. (2018). Models in science. In E. N. Zalta (Ed.), *The stanford encyclopedia of philosophy* (Summer 2018 ed.). Metaphysics Research Lab, Stanford University. <https://plato.stanford.edu/archives/sum2018/entries/models-science/>.
- Fuchs, A. (2013). *Nonlinear dynamics in complex systems: Theory and applications for the life-, neuro- and natural sciences*. Heidelberg: Springer- Verlag.
- Fusco, G., Carrer, R., & Serrelli, E. (2014). The landscape metaphor in development. In A. Minelli & T. Pradeu (Eds.), *Towards a theory of development*. Oxford: Oxford University Press.
- Gallow, J. D. (2017). *Pittsburgh university seminar notes for causality*. Retrieved from <http://pitt.edu/jdg83/teaching/pdfs/Causality%20Seminar-%20Notes.pdf>.
- Gleick, J. (1987). *Chaos: Making a new science*. USA: New York, NY: Penguin Books.
- Glymour, C. N., & Cooper, G. F. (1999). *Computation, causation, and discovery* (C. N. Glymour & G. F. Cooper, Eds.). Cambridge, MA: AAAI and MIT Press.

- Gorinevsky, D.(2005). *Linear systems in control engineering industry in ee392m class archive*. Retrieved from <https://web.stanford-edu/class/archive/ee/ee392m/ee392m.1056/>. Stanford University.
- Graßhoff, G.,& May, M.(2001). Causal regularities. In W. Spohn, M. Ledwig, & M.Esfeld (Eds.), *Current issues in causation* (pp. 85–114). Mentis Verlag.
- Gravetter, F. J.,& Wallnau, L. B.(2016). *Statistics for the behavioral sciences* (9thed.). Belmont, CA: Wadsworth/Cengage Learning.
- Greco, V.,& Roger, D.(2001). Coping with uncertainty: The construction and validation of a new measure. *Personality and individual differences*, 31(4), 519–534.
- Hall, N.(2004). Two concepts of causation. In J. Collins, N. Hall,& L. Paul(Eds.), *Causation and counterfactuals*. Cambridge, MA: MIT Press.
- Handfield, T., Twardy, C. R., Korb, K. B.,& Oppy, G.(2008). The metaphysics of causal models. *Erkenntnis*, 68(2), 149–168.
- Hausman, D. M., & Woodward, J. (1999). Independence, invariance and the causal markov condition. *The British journal for the philosophy of science*, 50(4), 521–583.
- Hayt, W. H., Kemmerly, J. E., & Durbin, S. M. (1986). *Engineering circuit analysis* (4th ed.). New York, NY: McGraw-Hill.
- Healey, R. (2016). Holism and nonseparability in physics. In E. N. Zalta (Ed.), *The stanford encyclopedia of philosophy* (Spring 2016 ed.).Metaphysics Research Lab, Stanford University. <https://plato.-stanford.edu/archives/spr2016/entries/physics-holism/>.
- Heeger, D.(2000). Signals, linear systems, and convolution. Retrieved from <http://www.cns.nyu.edu/~david/handouts/convolution.pdf>. New York University.
- Hitchcock, C.(2001). The intransitivity of causation revealed in equations and graphs. *The Journal of Philosophy*, 98(6), 273–299.

- Hitchcock, C. (2007). Prevention, preemption, and the principle of sufficient reason. *The Philosophical Review*, 116(4), 495–532.
- Hitchcock, C. (2018). Probabilistic causation. In E. N. Zalta (Ed.), *The stanford encyclopedia of philosophy* (Spring 2018 ed.). Metaphysics Research Lab, Stanford University. https://plato.stanford.edu/archives/spr2018/entries/causation_probabilistic/.
- Hoel, E. P., Albantakis, L., & Tononi, G. (2013). Quantifying causal emergence shows that macro can beat micro. *Proceedings of the National Academy of Sciences*, 110(49), 19790–19795.
- Hordijk, W., & Steel, M. (2015). Autocatalytic sets and boundaries. *Journal of systems chemistry*, 6(1), 1.
- Hover, F. (2009). *Linear systems, class notes for design of electro-mechanical robotic systems*. Retrieved from <https://ocw.mit.edu/courses/mechanical-engineering/2-017j-design-of-electromechanical-robotic-systems-fall-2009>. MIT Open CourseWare.
- Hoyer, P. O., Hyvarinen, A., Scheines, R., Spirtes, P. L., Ramsey, J., Lacerda, G., & Shimizu, S. (2012). Causal discovery of linear acyclic models with arbitrary distributions. *arXiv:1206.3260*.
- Hume, D. (1748). *Philosophical essays concerning human understanding*. London: Retrieved from <http://www.davidhume.-org/texts/thn.html>.
- Illari, P., & Russo, F. (2014). *Causality: Philosophical theory meets scientific practice*. Oxford: Oxford University Press.
- Illari, P. M. (2011). Why theories of causality need production: An information transmission account. *Philosophy & Technology*, 24(2), 95–114.
- Kauffman, S. (1996). *At home in the universe: The search for the laws of self-organization and complexity*. U.S.A: Oxford University Press.

- Kauffman, S. A. (2000). *Investigations*. U.S.A: Oxford University Press.
- Kelso, J. S. (1997). *Dynamic patterns: The self-organization of brain and behavior*. Cambridge, MA: MIT Press.
- Kelso, J. S. (2012). Multistability and metastability: understanding dynamic coordination in the brain. *Phil. Trans. R. Soc. B*, 367(1591), 906–918.
- Kun, Z., & Aapo, H. (2016). Nonlinear functional causal models for distinguishing cause from effect. In *Statistics and causality* (chap. 8). Wiley-Blackwell.
- Kyburg, H. (1965). Probability, rationality, and a rule of detachment. In *Proceedings of the international congress philosophy and history of science*, North Holland, Amsterdam (pp. 301–310).
- Ladyman, J. (2016). Structural realism. In E. N. Zalta (Ed.), *The stanford encyclopedia of philosophy* (Winter 2016 ed.). Metaphysics Research Lab, Stanford University. <https://plato.stanford.edu/archives/win2016/entries/structural-realism/>.
- Ladyman, J., Ross, D., Spurrett, D., & Collier, J. G. (2007). *Everything must go: Metaphysics naturalized*. New York, NY: Oxford University Press.
- Lewis, D. (1973). Causation. *The Journal of Philosophy*, 70(17), 556–567. Retrieved from <http://www.jstor.org/stable/2025310>.
- Liebovitch, L. S., & Shehedah, L. A. (2003). Lecture 32: Statistics: Bell curve and fractal pdfs. In *The mathematics and science of fractals and chaos*. Electronic Source: Florida Atlantic University.
- Longo, G., Montévil, M., & Kauffman, S. (2012). No entailing laws, but enablement in the evolution of the biosphere. In *Proceedings of the 14th annual conference companion on genetic and evolutionary computation* (pp. 1379–1392).
- Luenberger, D. G. (1979). *Introduction to dynamic systems: theory, models, and applications* (Vol. 1). New York, NY: Wiley.

- Machamer, P., Darden, L., & Craver, C. F. (2000). Thinking about mechanisms. *Philosophy of Science*, 67(1), 1-25.
- Manneville, P. (2006). Rayleigh-bénard convection: Thirty years of experimental, theoretical, and modeling work. In I. Mutabazi, J. E. Wesfreid, & E. Guyon (Eds.), *Dynamics of spatio-temporal cellular structures: Henri Bénard centenary review* (pp. 41–65). New York, NY: New York, NY: Springer.
- Meiss, J. D. (2007). Dynamical systems. In *Differential dynamical systems, revised edition*. U.S.A: SIAM Press. Retrieved from <https://epubs.siam.org/doi/abs/10.1137/1.9781611974645.ch4>.
- Menzies, P. (2017). Counterfactual theories of causation. In E. N. Zalta (Ed.), *The stanford encyclopedia of philosophy* (Winter 2017 ed.). Metaphysics Research Lab, Stanford University. <https://plato.stanford.edu/archives/win2017/entries/causation-counterfactual/>.
- Mill, J. (1911). *A system of logic: Ratiocinative and inductive*. London: Longmans, Green Co. and Library of Alexandria.
- Montevil, M., & Mossio, M. (2015). Biological organisation as closure of constraints. *Journal of Theoretical Biology*, 372, 179–191.
- Morgan, S. L., & Winship, C. (2007). *Counterfactuals and causal inference: Methods and principles for social research*. Cambridge: Cambridge University Press.
- Mossio, M., Bich, L., & Moreno, A. (2013). Emergence, closure and inter-level causation in biological systems. *Erkenntnis*, 78(2), 153–178.
- Mossio, M., Saborido, C., & Moreno, A. (2009). An organizational account of biological functions. *The British Journal for the Philosophy of Science*, 60(4), 813–841.
- Mouchart, M., Russo, F., & Wunsch, G. (2010). Inferring causal relations by modelling structures. *Statistica*, 70(4), 411.

- Mulaik, S. A. (2009). *Linear causal modeling with structural equations*. Boca Raton, FL: CRC Press, Taylor&Francis Group.
- Mumford, S., & Anjum, R. L. (2011). *Getting causes from powers*. Oxford: Oxford University Press.
- Nicholson, D. J., & Dupre, J. (2018). *Everything flows: towards a processual philosophy of biology*. Oxford: Oxford University Press.
- Nicolis, G. (1995). *Introduction to nonlinear science*. Cambridge: Cambridge University Press.
- Nicolis, G., & Prigogine, I. (1971). Fluctuations in nonequilibrium systems. *Proceedings of the National Academy of Sciences*, 68(9), 2102–2107.
- Pearl, J. (2009a). Causal inference in statistics: An overview. *Statistics surveys*, 3, 96–146.
- Pearl, J. (2009b). *Causality: Models, reasoning and inference* (2nd ed.). Cambridge University Press.
- Pearl, J. (2009c). *d-separation without tears*. Retrieved from <http://bayescs.ucla.edu/BOOK-2K/d-sep.html>. Online resource.
- Pearl, J. (2012). *The causal foundations of structural equation modeling* (Tech. Rep.). University of California Los Angeles Department of Computer Science.
- Pearl, J. (2013). Structural counterfactuals: A brief introduction. *Cognitive science*, 37: 977–985.
- Peters, J., Mooij, J. M., Janzing, D., & Scholkopf, B. (2014). Causal discovery with continuous additive noise models. *The Journal of Machine Learning Research*, 15(1), 2009–2053.
- Pfander, P., & Haupt, J. (2015). *Rayleigh Benard cell convection experiment*. Retrieved from http://didaktik.physik.fuberlin.de/projekte/niliphex/material/-12%20Zusatz%20Rayleigh-Benard-Konvektion_Schichtdicken.mp4.

Poellinger, R. (2012, February). Concrete causation: About the structures of causal knowledge. *Inaugural-Dissertation zur Erlangung des Doktorgrades der Philosophie an der Ludwig-Maximilians-Universität München*.

Poincaré, H. (1913). *The foundations of science: Science and hypothesis; the value of science; science and method*. Lancaster, PA: The Science Press.

Prigogine, I. (1978). Time, structure, and fluctuations. *Science*, 201 (4358), 777–785.

Prigogine, I., & Stengers, I. (1984). *Order out of chaos: Man's new dialogue with nature* (Vol. 13). Boulder, CO: New Science Library.

Psillos, S. (2001). Is structural realism possible? *Philosophy of Science*, 68(3), 13–24.
Raj, D., Anderson, A. W., & Gore, J. C. (2001). Respiratory effects in human functional magnetic resonance imaging due to bulk susceptibility changes. *Physics in Medicine & Biology*, 46(12), 3331.

Reichenbach, H. (1956). *The direction of time* (M. Reichenbach, Ed.). New York, NY: Dover Publications.

Reiss, J. (2007). *Causation: An opinionated introduction*. Retrieved from <http://j-reiss.org/Causality%20Manuscript.pdf>.

Reiss, J. (2008, September). *The philosophy of causation lecture 1*. <https://blogs.kent.ac.uk/jonw/files/2015/04/Reiss12008.pdf>. Erasmus University Rotterdam.

Reutlinger, A., Schurz, G., & Httemann, A. (2017). Ceteris paribus laws. In E. N. Zalta (Ed.), *The stanford encyclopedia of philosophy* (Spring 2017 ed.). Metaphysics Research Lab, Stanford University. <https://plato.stanford.edu/archives/spr2017/entries/ceteris-paribus/>.

Russo, F. (2010). *Causality and causal modelling in the social sciences*. Dordrecht: Springer.

Saatsi, J. (2017). Structuralism with and without causation. *Synthese*, 194(7), 2255–2271.

- Salmon, W. C. (1984). *Scientific explanation and the causal structure of the world*. Princeton: Princeton University Press.
- Sayama, H. (2015). *Introduction to the modeling and analysis of complex systems*. Binghamton, NY: Open SUNY Textbooks.
- Scheffer, M., Carpenter, S. R., Lenton, T. M., Bascompte, J., Brock, W., Dakos, V., and others (2012). Anticipating critical transitions. *Science*, 338(6105), 344–348.
- Scheines, R. (1997). Causality in crisis? statistical methods and the search for causal knowledge in the social sciences. In V. R. M. . S. P. Turner (Ed.), (chap. An introduction to causal inference). Notre Dame, IN: University of Notre Dame Press. Retrieved from <http://repository.cmu.edu/cgi/viewcontent.cgi?article=-1428&context=philosophy>.
- Shulz, K. (2011). if youd wiggled a, then b wouldve changed: Causality and counterfactual conditionals.. *Synthese*, 179, 239–251.
- Skow, B. (2014). Are there non-causal explanations (of particular events)? *British Journal for the Philosophy of Science*, 65(3).
- Sosa, E., & Tooley, M. (Eds.). (1993). *Causation*. Oxford: Oxford University Press.
- Sporns, O., Tononi, G., & Edelman, G. M. (2000). Connectivity and complexity: the relationship between neuroanatomy and brain dynamics. *Neural networks*, 13(8-9), 909–922.
- Tognoli, E., & Kelso, J. S. (2013). On the brains dynamical complexity: coupling and causal influences across spatiotemporal scales. In *Advances in cognitive neurodynamics (iii)* (pp. 259–265). Springer.
- Tononi, G., Sporns, O., & Edelman, G. M. (1999). Measures of degeneracy and redundancy in biological networks. *Proceedings of the National Academy of Sciences*, 96(6), 3257–3262.

- Vasbinder, J. W., & Gao, H. H. (2017). *Selected papers of john h holland: A pioneer in complexity science* (Vol. 4). Singapore: World Scientific Publishing Company.
- Walgraef, D. (2012). *Spatio-temporal pattern formation: with examples from physics, chemistry, and materials science*. New York, NY: Springer Science & Business Media.
- Wallace, W. A. (1972). *Causality and scientific explanation. vol. 1: Medieval and early classical science*. Ann Arbor, MI: University of Michigan Press.
- Williamson, J. (2007). Handbook of philosophical logic. In D. Gabbay & F. Guentner (Eds.), (chap. Causality). Dordrecht: Springer Netherlands.
- Woodward, J. (1997). Explanation, invariance, and intervention. *Philosophy of Science*, 64, 26–41.
- Woodward, J. (2005). *Making things happen: A theory of causal explanation*. Oxford: Oxford University Press.
- Woodward, J. (2016). Causation and manipulability. In E. N. Zalta (Ed.), *The stanford encyclopedia of philosophy* (Winter 2016 ed.). Metaphysics Research Lab, Stanford University. <https://plato.stanford.edu/archives/win2016/entries/causation-mani/>.
- Woodward, J., & Hitchcock, C. (2003). Explanatory generalizations, part i: A counterfactual account. *Nous*, 37(1), 1–24.
- Wright, S. (1921). Correlation and causation. *Journal of agricultural research*, 20(7), 557–585.

APPENDICIES

A. TURKISH SUMMARY/TÜRKÇE ÖZET

Kompleks Sistemlerin Modellenmesinde Nedensel İlişkilerin Temsili Problemi

Bilimde modelleme, sistemlerin temsili olarak söz konusu sistemleri anlama ve öngöründe bulunma aracı olarak çok önemli bir role sahiptir. Modelleri bu denli önemli yapan şeyse, özellikle bazı sistemler için, sistemlere doğrudan müdahalenin mümkün olmayışıdır. Kompleks sistemlerin modellenmesi bu sistemlerin karmaşık doğaları göz önünde bulundurulduğunda, anlama ve öngöründe bulunma adına zorluk oluşturmaktadır. Bu sistemlerin modellenmesinin zor olmasının ana unsurlarından biri de nedensel ilişkilerin temsil edilmesi problemidir. Bu tezde amaç, nedensel modellerde yapılan ontolojik bağlanımlarla ilgili bir problemi ortaya koymaktır. Elimizdeki modeller, genel olarak, nedensel ilişkilere dair herhangi bir ontolojik iddiada bulunmama amacındadırlar. Modellerde, daha çok nedenselliğin kanun-benzeri bir ilişki olarak temsil edildiği görülmektedir. Kanun-benzeri bir ilişkiyle kastedilen şey ise her sisteme uyarlanabilen sabit bir bağıdır. Daha açık olmak gerekirse, nedenselliğin doğası gereği sabitlemiş, kanun-benzeri bir zorunluluk taşıyan, şeyleri etkileyen ve şeylerin etkilenmesini sağlayan ilişkilerden ibaret olduğu varsayılmaktadır. Buradaki modeller ve modellenen şeyler arasındaki karşılıklı ilişki üzerine felsefi bir inceleme gerektirmektedir. Ancak bu tez, kompleks sistemlerdeki nedensellik ilişkilerinin temsili olarak öne sürülen nedensel modellerle sınırlıdır. Bu anlamda, çalışmada, nedensel modellerin kompleks sistemlere uygulanabilirliği tartışılmıştır. Kompleks sistem terimiyle anlatılmak istenen şey ise lineer olmayan yollarla birbirine bağlanmış elemanlardan oluşan ‘bütün’lerdir. Eğer bir sistem kompleks ise sistemin karşılıklı ilişkiler ağının karmaşıklığı yüksektir. Çünkü sistemler aynı bileşenlere sahip olsalar dahi farklı sistemler oluşturabilmektedir. Farklılık, bileşenlerin farklı şekillerde biraraya gelmelerinden kaynaklanmaktadır. Bu biraraya gelişlerin oluşturduğu karşılıklı ilişkililik durumlarının dinamik olarak her evrede yeniden kurulmasıyla çok-fonksiyonellik, dejeneresi, kendini-örgütleme,

kendini-yaratım, belirlimlilik gibi karakteristikler ortaya çıkmaktadır. Böylesi bir içsel dinamikliğin yanı sıra, kompleks sistemler çevreleriyle birlikte evrilmektedirler. Bu demektir ki, kompleks sistemler aktif olarak içinde bulundukları çevreyi etkilemekte ve aynı şekilde çevrelerinden etkilenmektedirler. Yani, kompleks sistemler ayrıca çevreleriyle bağlıdır.

Modellemelerde, böylesi içiçe geçmiş karmaşık ilişkilerin temsil edilmesi açısından birtakım temel problemler yer almaktadır. En göze çarpan problem ise kompleks bir sistemin temsil edilmesi için basitleştirme yapılması gerekliliğidir. Eğer sistemler milyonlarca heterojen bileşenden oluşuyorsa (örneğin insan beynindeki nöronlar, ganglion hücreleri, piramitsel hücreler gibi) ve bu milyonlarca bileşenlerin daha da fazla sayıdaki ilişkileri göz önünde bulundurulduğunda, bu (kompleks) sistemlerin uygun ama basitleştirilmiş bir şekilde betimlenmesi için ne yapılmalıdır? Ya da, kompleks sistemlerin temsilini basit ama aynı zamanda onların karmaşık nedensel yapılanmalarını kapsayacak kadar geniş bir şekilde sağlamak mümkün müdür? Tüm bu meselelerin açıklığa kavuşturulması gerekmektedir.

Bilim ve Felsefede Bir Sorun Olarak Nedensellik

Nedensellik ile ilgili temel sorun şudur: bizler biliyoruz ki, ya da en azından varsayıyoruz ki, bazı olaylar bazı başka olaylara neden olmakta, fakat neden oluşun (bir başka deyişle, nedenselliğin) tam olarak ne olduğunu tanımlayamıyoruz. Öyle gözüküyor ki sigara içmek akciğer kanserine neden oluyor, fast-food tüketimi obeziteye neden oluyor, bir genin eksikliği bir hastalığa neden oluyor, karbon salınımı küresel ısınmaya neden oluyor, ve benzeri. Dahası, bu tür olaylar öngörülebilme ve/veya olaylar üzerinde kontrol sağlanabilmektedir. Örneğin, eksik olan gen laboratuvarlarda yetiştirilerek hastaya enjekte edildiğinde hastalığı tedavi edebilmektedir veya diyet yapılarak obezite engellenebilmektedir. Öte yandan, durum her zaman bu şekilde değildir. Bir kişi fast-food tüketmemesine rağmen obez olmuş olabilir. Örneğin, obezite hormon salınımını fazlalığından kaynaklanabilir. Bu durumda fast-food tüketimini takiben obezitenin gözlemlendiği tüm vakaları yok mu saymalıyız? Aksine, eğer iki vaka için de nedenselliği varsaymalıysak nedensel ilişkileri tespit etme kriterimiz nedir? Bu sorulara verilen cevaplar farklılık göstermektedir, fakat bugün ne nedenselliğin tespit kriterlerine dair ne de tanımına dair herhangi bir uzlaşma bulunmamaktadır. Bilimsel pratikte, cevap genellikle istatistiksel çıkarımlar üzerinden verilmekte-

dir. Ancak felsefi tutum iki yönlüdür: metafiziksel ve epistemik. Nedensel bağların gerçekliğine dayalı nedensellik tanımları metafiziğin araştırma alanını oluştururken epistemik çalışmaların konusu nedensel ilişkilere yönelik bilgilerimizdir. Burada dikkat edilmelidir ki felsefi olarak ontoloji ve epistemoloji arasındaki çizginin nerede çekilmesi gerektiği başlı başına bir sorundur. Bu tezin tam da bu sözde çizgi üzerinde yürüdüğü iddia edilebilir: nedensel modeller nedenselliği temsil eden epistemik aygıtlar olarak incelenirken modeller ve modellenen şeylerin doğaları arasındaki ilişki sorgulanmıştır. Asıl problemin günümüzün nedensel modellerinde gömülü olan nedensellik tanımından kaynaklandığı iddia edilmiştir.

Felsefede, nedenselliğin tanımına dair sistematik akıl yürütmelerin izi Aristoteles'e kadar sürülebilir. Aristoteles tözlerde bulunan nedenler (materyal, formel, etken, nihai nedenler) sınıflandırması ortaya koymuştur. Bu Aristoteleci sınıflandırma Ortaçağ'da da kabul görmüştür, fakat burada Aziz Thomas Aquinas gibi skolastik düşünürlerinin sunduğu teolojik yorumlaması (Wallace, 1972) görülmektedir. Tanrı, bu anlamda, tüm 'şeylerin' son nedeni olarak görülmüştür ve Aristoteles'in hareketsiz hareket ettiricisinin yerini almıştır. Dünyadaki bütün varlıkların tek nedeni olarak Tanrı'yı gören Aranedencilik akımının yanı sıra, benzeri nedensellik açıklamaları sunulmuştur. O dönemde ortaya atılan tüm nedensellik felsefelerinin ortak zemini nedenselliğin zorunlu bir ilişki olması ve dolayısıyla bu zorunluluğu sağlayabilme yetisi yalnızca Tanrı'da olduğu düşüncesidir. Öteki yandan, nedenselliğin skolastik açıklamalarında ciddi bir sorun yer almaktadır: eğer Tanrı herşeyin nedeni ise geriye bedenlerin yapabileceği ne kalır? Bu düşünce mirasını devralan erken dönem modern filozofları daha çok nedenselliğin metafiziği üzerine çalışmalar yapmışlardır. Gerçekten de, erken modern dönem filozofları "Hobbes istisnasıyla, Tanrı hakkındaki bilginin doğayı ve doğa yasalarını anlamak için kritik bir öneme sahip olduğu düşüncesini paylaşmışlardır" (Clatterbaugh, 1999). Örneğin, kartezyen nedensellik anlayışı nedensel etkileşimleri sağlayan kanunların ancak bir kanun koyucu, yani bir Tanrı tarafından verilebileceği düşüncesidir. Fakat Spinoza'da Tanrı doğalayan doğa (natura naturans) olarak skolastik olmayan bir Tanrı tasviri vardır. Yine de diğer erken modern dönem filozoflarına benzer olarak, nedensel bağıntı bir tür mantıksal bağıntı olarak tam da gerçekliği oluşturan şeyin kendisi olarak görülmüştür. Tüm bunların yanında, modern dönemin çok daha başlarında yaşamış olan düşünür Francis Ba-

con'ın sistematik nedensel çıkarımlar için bir tür metodoloji sunduğu (Reiss, 2007) da not edilmelidir. Bacon (öyle gözüküyor ki) nedenselliğin metafiziksel yönüyle ilgilenmemiş, daha çok doğa üzerinde kontrol sağlanması yönünden nedenselliğin öneme vurgu yapmıştır. Geç modern felsefelerde nedenselliğe benzeri bir yaklaşımın olduğu iddia edilebilir. O dönemde, bedenlerin nasıl etkileştiğine yönelik metafiziksel sorulardan ziyade “nedensel bağıntının hakiki bir tespitinin” (Clatterburgh, 1999) sağlanması yönünde nedensellik çalışmalarına rastlanmaktadır.

David Hume, bu anlamda, nedensel olayların psikolojik deneyimdeki karşılığı üzerinden sağduyusal nedensellik anlayışına meydan okumuştur. Hume nedensellik üzerine yaptığı akıl yürütmelerinin sonucunda şunu görmüştür: her nedensel olaylar zincirinden bahsedildiğinde aslında (i) mekansal devamlılık ve (ii) zamanda önceliklik gösteren, (iii) sürekli rastlaşan olaylardan söz edilmektedir. Devamında gelen nedenselliği tanımlama denemelerinin tamamı, günümüzdeki çalışmalar da dahil olmak üzere, Hume'un bu nedensellik analizi fikrine dayanmaktadır. Bu çalışmalar, böylelikle, kabaca iki gruba ayrılabilir: nedensel realistler ve indirgemeciler. Nedensel realistler aynı zamanda karşı-Humecular olarak anılmaktadırlar. Çünkü Hume'un iddia ettiğinin aksine nedensel ilişkinin insan deneyiminin dışında da bir gerçekliğe sahip olduğunu düşünmektedirler. Buna karşın olarak nedensel indirgemecilik, nedenselliği nedensel-olmayan terimler üzerinden açıklamaktadırlar. Şuna dikkat edilmelidir ki, nedensel realist olup metodolojik olarak indirgemeci bir yol izlemek de mümkün. Aslında, günümüzdeki nedensel modellerin hemen hepsi bu yolu izlemektedirler.

Nedenselliği tarif etmeye yönelik indirgemeci çalışmalar önceleri mantıksal yöntemleri benimserken daha sonraları matematiksel analizler üzerinden gelişmiştir. Nedenselliğin mantıksal analizi John Stuart Mill tarafından ortaya konulmuş ve devamında John Leslie Mackie'nin çalışmalarıyla önem kazanmıştır. Mackie öne sürdüğü INUS koşulu ile yeterli ve zorunlu nedenlerin farklı olabileceklerini göstermiştir. Bu gelişmeleri takiben David Lewis'in karşıolguusal açıklamaları ile nedenselliğin mantıksal analizi bugünkü şekline kavuşmuştur. Buradaki temel fikir nedensel önermelerin koşul önermesi formunda analizini yapmaktır. Hatta Curt John Ducasse gibi bazı filozoflar bu fikri daha da ileri götürerek nedensel ilişkilerin ‘doğru’ bir tanımlamasının ancak ve ancak koşul önermeleri formunda verilebileceğini ve böylelikle Hume'da gördüğümüz sürekli rastlaşma özelliğinin bu formda yerinin olmadığını

(Sosa and Tooley, 1993) iddia etmişlerdir. Bu yaklaşımdaki en büyük problem ise birden çok nedenin bulunduğu (üstbelirlenim) vakalarda nedenselliği koşul önermeleri formunda temsil etmek neredeyse imkansız hale gelmesidir.

Nedenselliğin matematiksel analizi, bir diğer taraftan, nedenselliği ‘fonksiyonel bağıllık’ olarak ele almaktadır. Nedenselliği fonksiyonel bağıllıklar olarak temsil etme düşüncesi ilk kez Hans Reichenbach tarafından başlı başına bir çalışma konusu halini almıştır. Reichenbach aynı-zamanlı korele olayların ortak nedenler tarafından öncelenmesi gerektiği fikrinden yola çıkarak nedenselliği olasılıksal bağıllıklardan çıkarsamanın mümkün olabileceğini düşünmüştür. Böylece, korelasyonlardan nedensellik tespit edilebilirdi. Reichenbach’ın nedensellik analizinde birbirine neden olmayan iki faktörün, X ve Y , ortak bir neden olan C ’ye sahip olmaları durumunda $P(X.Y/C) = P(X/C)P(Y/C)$ olduğunu matematiksel olarak ‘Ortak Neden Prensibi’ ile tanımlanmıştır. Bugün bu prensibin birçok durumda kullanılamaz olduğu kanıtlansa da değişkenler arasındaki ‘koşullu bağımsızlıklar’ın formalize edilmesi fikrine ışık tutmuştur. Aslında, ‘nedensel Markov koşulu’ bu fikirden türetilmiştir (Spirtes ve Glymour, 1993). Nedensel Markov koşulu bir değişkenin kendisinin sonucu olmayan değişkenlerden olasılıksal olarak bağımsız olduğunu söyler. Buradaki koşullu bağımsızlık kavramına dayanarak daha sonraları nedenselliğin olasılıksal analizleri Patrick Suppes, Irving John Good, Wolfgang Spohn, John Williamson, Judea Pearl, Peter Spirtes, Clark Glymour ve Richard Scheines tarafından geliştirilmiştir. Bu tezde bahsi geçen nedensel modeller nedenselliğin hem mantıksal analizi (karşıolgusallık) hem de matematiksel temsilleri (olasılıksal bağıllıklar) konu edinmektedir.

Nedensellik ve Modelleme

Bugün elimizdeki modelleme gereçleri sistemlerin lineer davrandığı varsayımına dayanmaktadır. Fakat bazı sistemler hiç de lineer davranış biçimi göstermemektedirler ve bu sistemler, haliyle, lineer-olmayan sistemler olarak anılmaktadır. Dolayısıyla, bu sistemlere ait lineer-olmayan verilere lineer metodlar uygulanması halinde şeyler (entities) ile onların temsilleri arasında bir uyumsuzluk söz konusu olmaktadır. Bugüne kadar ortaya konulan nedensel modeller de lineer bir metodoloji üzerine kurulmuştur. Tezde bu modellerin kompleks (ve dolayısıyla, lineer-olmayan) sistemlere uygulanmasının kompleks sistemlerin anlaşılmasında yeterli gelemeyeceği ortaya konmuştur. Bu anlamda tezde güncel nedensel modellerdeki handikapları ortaya ser-

ilerek kompleks sistemlerdeki nedensellik ilişkilerinin modellenmesi için alternatif yollar tartışılmıştır. Bu amaçla, ilk olarak sistemleri temsil etmedeki temel yöntemler tartışılmıştır. Tartışmalar sistem teriminin anlamından başlayıp sistemleri nasıl modellediğimize kadar uzanmaktadır. Konun bağlamı modellemeler olması dolayısıyla sistemlerin matematiksel temsilleri tartışılmıştır. Sistemlerin matematiğine bağlı olarak modeller, sistemin davranışına (sistemin çıktı üretme şekline) göre durağan ve dinamik olmak üzere iki ayrı gruba ayrılmaktadırlar. Durağan sistemler bu tezin bağlamı dışında kalmaktadır, çünkü bu sistemler hiç bir aktivitenin olmadığı veya herhangi bir çıktı üretiminin söz konusu olmadığı bir tür denge durumundadırlar. Öte yandan dinamik sistemler lineer veya lineer-olmayan yollardan çıktı üreten sistemlerdir. Burada dikkat edilmelidir ki tez boyunca deterministik (rastgele olmayan) sistemler konu edilmiştir. Eğer sistemler toplanabilirlik özelliği gösteriyor ve homojenite söz konusu ise sonuç olarak bu sistemlerin tepkisi (yani, bir sonraki durumu) önceki durumların lineer kombinasyonu olarak belirlenmektedir. Bu sistemlere lineer sistemler denilmektedir. Lineer kombinasyonlar matematiksel olarak analitik çözümlere sahiptir. Bu demektir ki, her bileşen aslında birbirinden bağımsızdır ve bileşenleri toplamak veya çıkarmak bileşenlerin doğasında bir değişikliğe yol açmaz. Buradaki matematiksel elemanlar aslında, temsil edilen şeyin bileşenlerine ayrılmış halini temsil etmektedir. Dolayısıyla lineer modeller analiz edilebilen şeyleri (örneğin, elektronik aletleri veya saatleri) temsil etmek için çok uygundur. Lineer modellerde toplanabilirlik ve homojenite sayesinde parçaların aranjmanı bu parçaların lineer kombinasyonunun ta kendisidir. Lineer-olmamaklık, öte yandan, toplanabilirlik ve homojenite özelliklerinin olmamasıdır ve bir fonksiyon ile temsil edilmektedir. Bu özelliklerin olmaması, matematiksel olarak bu fonksiyonların analitik çözümlerinin olmaması anlamına gelmektedir. Yine de bu demek değildir ki lineer-olmayan biçimlerde davranan hiçbir sistemin analiz edilemez. Örneğin, biyolojik sistemler analiz edilebilmektedirler. Lineer-olmamaklık daha çok modelleme anlamında bazı sınırlandırmalara tabidir. Fakat sistemleri modellemek için önce veriye ihtiyaç vardır.

Veri bir şeyin temsil edilmesindeki ilk basamak olarak bazı prosedürler ile edinilir. Bu süreç genellikle iki aşamalıdır: veri toplama ve verinin yorumlanması. Veri edinim sürecinde birtakım istatistiksel varsayımlarda bulunmaktadır. Herhangi bir pratik hatanın yokluğu gibi varsayımlar bir yana konulduğunda, tezde, bu istatik-

sel varsayımların lineerliği teşvik ettiği ortaya serilmiştir. Bunu göstermek önemlidir çünkü söz konusu istatistiksel varsayımlar yüzünden lineer davranmayan sistemler hakkında toplanan veriler yorumlanırken sistemler lineermiş gibi düşünülmektedir. Tez boyunca kompleks sistemleri temsil etmek için lineerleştirme işlemlerinin zorunlu olup olmadığı tartışılmıştır. Veri yorumlama işlemleri söz konusu istatistiksel varsayımlara dayalı olarak çıkarım yapılmasını sağlayan birtakım prosedürden oluşmaktadır. Dolayısıyla veriden ne okunacağı bu varsayımlara göredir. Örneğin, eğer pozitif korelasyonun nedensel ilişkinin göstergesi olduğu varsayılmışsa bu durumda ilgili değişkenlerin nedensel olarak bağımlı oldukları çıkarımı yapılacaktır. Bu anlamda korelasyonun nedenselliği belirttiği tartışmaları veri oluşturma sürecinin tam da bu aşamasında karşımıza çıkmaktadır.

Peki lineer-olmamaklık nerede yer almaktadır? Bunu tartışmak için önce şu iki ayrım yapılmalıdır: lineer-olmayan sistemlerin kompleksite gösterip göstermemeleri ve kaotik olup olmamaları. Lineer-olmayan davranışlara sarkaç gibi basit sistemlerde de rastlanılmaktadır. Bu anlamda teknik bir terim olarak kompleksite, kompleks sistemler bağlamında açıklık getirilmesi gereken bir konudur. Bölüm 2.3.1’de tam da bu konuya açıklık getirilmiştir. Böylelikle berimsel anlamdaki kompleksite terimi kompleks sistemler teriminden farklı bir kavram olarak ele alınmıştır. Diğer taraftan bu iki terim arasındaki ilişki tartışılmıştır. Açıklık getirilmesi gereken diğer bir konu da kaos ve kompleks sistemler arasındaki ilişkidir. Kaos fenomeni, gerçekten de, doğası gereği lineer olmayan bir fenomendir fakat bu demek değildir ki tüm kompleks sistemler lineer olmayan fenomenler oldukları için aynı zamanda kaotik olmak zorunda. Bu zorunlu olmayışın sebepleri bölüm 2.3.2’te açıkça tartışılmıştır. Bu ayrımların ışığında kompleks sistemlerin karakteristikleri genel olarak tartışılmıştır. Bahsedilen karakteristiklerin aslında kompleks sistemlerin lineer olmayan ilişkileri sayesinde ortaya çıktıkları tartışılmıştır. Dolayısıyla lineer olmayan ilişkiler ile kastedilen şeyin ne olduğu da ayrıntılı bir tartışmayı gerektirmiştir. Bu bağlamda lineer olmamaklık, kompleks sistemlerdeki davranış örüntüsünü ortaya çıkaran bileşenler arasındaki ilişkilerin türü olarak konu edinilmiştir. Düzenin stabil olmayan bir durumdan ortaya nasıl yeniden kurulduğu bölüm ise 2.3.4.1’in ana tartışma konusu olmuştur. Devamında kurulan (nedensel) ilişkilerin bu sistemlerde nasıl ortaya çıktığı tartışılmıştır. Bu anlamda bölüm 2.3.4.2’deki amacım kompleks sistemlerde il-

işkiselliğin kritik önem taşıdığını gerekçeleriyle ortaya koyarak vurgulamaktır. İkinci bölüm kompleks sistemlerin temel özellikleri ile lineerlik varsayımlarını karşılaştıran bir liste ile bitirilmiştir. Burada lineer metodların dayattığı özellikler ile kompleks sistemlerin özellikleri arasındaki uçuruma dikkat çekilmiştir. Sonuç olarak lineer metodların kompleks sistemleri temsil etmede yetersiz oldukları gösterilmiştir. Üçüncü bölüm, bugüne kadar öne sürülmüş nedensel modellerde referans gösterilen nedensellik teorilerinin analizine yer verilmiştir. Bu analizin sunulmasındaki temel amaç ise nedensellik teorileri ile nedensel modellerin (ve/veya modelleme tekniklerinin) karşılıklı ilişkisine işaret etmektir. Böylelikle nedensel ilişkilerin temsilleri, bu temsillerin gösterdiği ontolojik bağlanımlara göre kategorize edilmiştir. Bağlılık ve Süreç kategorileri nedenselliğin temsil çerçevelerini oluşturan iki temel kategori olarak ele alınmıştır. Bu sınıflandırma güncel literatüre göre yapılmış olsa da bazı detaylarda güncel sınıflandırmalardan farklılık göstermektedir. Bu detaylardan biri yeni bir nedensel açıklama sisteminin eklenmiş olmasıdır. Bu yeni nedensellik önerisi nedensel belirimcilik olarak anılmaktadır. Erik Hoel, Larissa Albantakis ve Giulio Tononi tarafından ortaya atılmış bu yeni görüş süreçsel nedensellik başlığı altında ele alınmıştır. Bu başlık altında ele alınmasının sebebi Russo ve Illari'nin sınıflandırmasına benzer olarak bu görüşün bir tür enformasyon-teorisi temelli nedensellik açıklaması olmasıdır. Aslında bu iki nedensellik temsil kategorisi arasındaki sınır çok keskin değildir. Benzer olarak Hoel aslında enformasyon teorisi temelli açıklamaların bağlılık üzerinden yapılan nedensellik açıklamalarını da kapsayacak kadar geniş olabileceğini belirtmektedir. Gerçekten de bu tartışmaların çok önem arz etmesine karşın bir yüksek lisans tezinde tartışılmayacak kadar zorlu bir başlık olduğuna da dikkat edilmelidir. Bu nedenle bu tezde enformasyon teorisi temelli nedensellik açıklamaları ayrıntılı bir şekilde ele alınamamıştır. Dolayısıyla, güncel felsefi literatürüne bağlı kalınarak nedensel modellerden bahsedilirken kastedilen şey yapısal denklem modelleri (YDM) ve Judea Pearl'ün öne sürdüğü nedensel grafikler olacaktır. Bölüm 3.2'de gösterildiği üzere Pearl'ün nedensel grafikleri aslında YDMnin geliştirilmesiyle oluşturulmuştur. Lineer sistemleri modellemekteki üstün başarılarına karşın bu nedensel modeller, kompleks lineer-olmayan sistemleri modellemede yetersiz kalmaktadırlar. Kanıtlar bölüm 3.3'te listelenerek öne sürülen nedensel modellerin aslında lineerlik varsayımlarına sıkı sıkıya bağlı oldukları gösterilmiştir. Bir başka deyişle, bölüm 2.2'de tartışılan lineer varsayımların aslında nedensel modellerde de gömülü

oldukları gösterilmiştir. Eğer gerçekten de lineer metodlar kompleks sistemleri temsil etmede yetersizlerse, lineerliği temel alan nedensel modellerin kompleks sistemlerdeki nedensel ilişkileri temsil etmede yeterli oldukları nasıl savunulabilir? Savunulamayacağını gösteren bir liste, bölüm 2.4'teki liste ile uyumlu bir şekilde sunulmuştur. Böyle bir karşılaştırma yapılmasının sebebi ise nedensel modellerdeki lineerlik varsayımlarının gözettiği özellikler ile kompleks sistemlerin özellikleri arasındaki uyumsuzluğu ortaya sermektir. Böylelikle, nedensel modellerdeki ontolojik bağlanımlar ile kompleks sistemlerin ontolojisi arasındaki ayrıklık gözle görülür hale getirilmiştir. Dördüncü bölüm ise nedensel ilişkilerin kompleks lineer olmamaklık durumuna uygun olarak temsil edilebilmesi için olası çözümleri tartışılmıştır. Modellerde temsil etmek için lineerleştirme işlemi zorunlu bir işlem midir? Kompleks sistemlerdeki nedensel ilişkiler için lineer olmayan bir nedensellik açıklaması sunmak mümkün müdür? Eğer mümkünse hangi adımlar atılmalıdır? Eğer değilse bunun sonuçları nelerdir? Bu bölüm boyunca bu sorular tartışılmıştır. Bölüm 4.2'de ise ilişkilerin ontolojik statüleri üzerinden nedensellik modellemeleri için alternatif teoriler tartışılmıştır. İlişkileri önceleyen bir nedensellik temsili teorisi, bugünkü modellerde yapılan nodların öncelenmesi düşüncesine nazaran, umut vaadedici gözükmemektedir. Fakat temsil problemi henüz çözülememiştir. Neden çözülemediği ise kapanış tartışması olarak beşinci bölümde ele alınmıştır. Temel sorun olarak lineer olmayan kompleks sistemlerde nedenselliğin nasıl işlediği ele alınmıştır. Bu sorun kolayca cevaplanamayacağından soruna kompleks sistemlerdeki ilişkilerin doğası tartışılarak yaklaşmıştır. Kompleks sistemlerin bileşenleri öyle şekillerde bağlantılar kurmaktadır ki bir bileşenin aktivitesi bağımsız olamamakta ve dolayısıyla aktivite sonucu birden fazla sonuç doğmaktadır. Tam da bu noktada lineer nedensellik yaklaşımı kullanılamaz hale gelmektedir. Ancak aynı zamanda büyük bir problem karşımıza çıkmaktadır: böylesi bir sonuçlar yığını içinden nedenselliği nasıl tespit edebiliriz (ve/veya modelleyebiliriz)?

Nedenselliğe lineer yaklaşım bağlam üzerinde belirli bir sınırlandırmayı şart koşar ve bu sınırlandırma üzerinden uzlaşa gerektirir. Bu bağlamdaki sınırlılığın felsefedeki karşılığı ceteris paribus koşuludur. Bu ceteris paribus koşulları altında yapılan temsiller de, dolayısıyla, bileşenler çevrelerinden izole edilmiş olurlar. Bu da kompleksitenin ortadan kalkması anlamına gelmektedir. Lineerlik şartları altında nedensellik

yalnızca bileşenlere bağımlıdır ve böylelikle ilişkilerin kendi başlarına bir dinamiklik taşıması durumu söz konusu değildir. Bileşenler ancak belirli tip ilişkileri kurmakla yükümlüdür. Bir başka deyişle, ilişkisel dinamikler göz ardı edilirken bileşenler fazlaca önemsenmektedir. Öte yandan, kompleks sistemlere aynı bileşenlerin farklı kompozisyonlarda yer almasıyla farklı karakteristiklerin ortaya çıktığını görmekteyiz. Nedensel ilişkilerin, ilişkiselliğin kendisine odaklanan bir temsil yapısı ile kompleks sistemlerin oluşum ve gelişim süreçlerini modelleyebilmek mümkün olur muydu? Bu sorunun yanıtı hakkında tartışmalar yapılmıştır. Sonuç olarak matematiksel bir altyapı sunulamasa da otokatalitik kümeler teorisinin bir çıkış yolu olabileceği sonucuna varılmıştır. Otokatalitik kümelerde görülen kendi üretim sürecini katalize eden reaksiyonların bizzat ilişkisel dinamikler üzerinde nedensel etkide bulunması fikri kompleks sistem modellemelerinde baz alınabilir. İlişkisel dinamiklerin temsil edilmesi kritik önem taşımaktadır, çünkü kompleks sistemlerin kompleks olmalarının sebebi, yani belirlimlilik gibi karakteristikleri göstermesi tam da bu ilişkisellik biçimleri sayesinde. Tezde kompleksiteye yol açan en önemli ilişkisellik dinamiği olarak pozitif geridönüt döngüleri işlenmiştir. Pozitif geridönüt döngüleri literatürde ‘döngüsel nedensellik’ veya ‘karşılıklı nedensellik’ kavramları olarak da karşımıza çıkmaktadır. Lineer olmayan dinamikleri oluşturan bu döngüler modellemelerde ciddi problemlere yol açtığı için genelde ya lineerleştirilerek temsil edilmekte ya da tamamen yok sayılmaktadır. Bunun nedeni ise tam da ilişkisel dinamikliği temsil edebilecek temsil gereçlerimizin olmayışındır. Bu gereçleri edinebilmek içinse Prigogine ve Stengers (1984)’in belirttiği gibi yeni düşünce araçlarına ihtiyaç duyulmaktadır.

İlişkisel dinamikleri felsefi olarak temellendirebilmek için ontolojik bir sorgulama gereklidir. Varlıkların doğasına ilişkin bu sorgulama yolu günümüzde iki kola ayrılmaktadır: Yeni-tözcülük ve Ontik Yapısal Realizm. Yeni-tözcülük, temel olarak, bileşenlerin veya bileşenleri oluşturan alt-bileşenlerin tözsel bir varlık statüsünde olduklarını öne sürer. Tözün kendinde potansiyel olarak taşıdığı bir başka şey(lere) neden olma eğilimleri nedensel ilişkiler olarak tecelli olur. Dolayısıyla ilişki var değildir, ancak bileşenlerin tözleri ve töz olmağının getirdiği birtakım eğilimler vardır. Ontik Yapısal Realizm ise aksine, tikellikten yani bağımsız olarak var olabilen tözlerden söz etmenin mümkün olamayacağını herşeyin ilişkilerden ibaret olduğu düşüncesine dayanır. Metafiziksel olarak ciddi sorunlarla karşılaşsa da ontik yapısal realism,

kompleks sistemleri modellemek adına uygun bir ontolojik altyapı sağlayabilir gibi gözükmektedir. Bunun temel gerekçesi ise ilişkisel dinamiklerin temsil edilebileceği uygun bir zemin ancak ilişkileri ön plana çıkaran bir ontolojiyle mümkün olabilir gibi gözükmektedir.



B. TEZ FOTOKOPİ İZİN FORMU

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YAZARIN

Soyadı : Kocaoğlu

Adı : Başak

Bölümü : Felsefe

TEZİN ADI (İngilizce) : The Representation Problem of Causal Relationships in Complex Systems Modeling.

TEZİN TÜRÜ:

Yüksek Lisans ☐

Doktora ☐

1. Tezimin tamamı dünya çapında erişime açılsın ve kaynak gösterilmek şartıyla tezimin bir kısmı veya tamamının fotokopisi alınsın. ☐
2. Tezimin tamamı yalnızca Orta Doğu Teknik Üniversitesi kullanıcılarının erişimine açılsın (Bu seçenekle tezinizin fotokopisi ya da elektronik kopyası Kütüphane aracılığı ile ODTÜ dışına dağıtılmayacaktır.) ☐
3. Tezim bir (1) yıl süreyle erişime kapalı olsun. (Bu seçenekle tezi-nizin fotokopisi ya da elektronik kopyası Kütüphane aracılığı ile ODTÜ dışına dağıtılmayacaktır.) ☒

Yazarın imzası:

Tarih