The Causal Explanation in Molecular Biology

Balorda, Vito

UNIVERSITY OF RIJEKA

FACULTY OF HUMANITIES AND SOCIAL SCIENCES

Vito Balorda

THE CAUSAL EXPLANATION IN MOLECULAR BIOLOGY

DOCTORAL THESIS

Rijeka, 2024

UNIVERSITY OF RIJEKA

FACULTY OF HUMANITIES AND SOCIAL SCIENCES

Vito Balorda

THE CAUSAL EXPLANATION IN MOLECULAR BIOLOGY

DOCTORAL THESIS

Supervisor: Prof. Predrag Šustar, PhD

Rijeka, 2024

SVEUČILIŠTE U RIJECI

FILOZOFSKI FAKULTET U RIJECI

Vito Balorda

UZROČNO OBJAŠNJENJE U MOLEKULARNOJ BIOLOGIJI

DOKTORSKI RAD

Rijeka, 2024

ACKNOWLEDGMENTS

I would like to thank the many kind individuals who supported me throughout my PhD journey.

First and foremost, I would like to thank my supervisor, Predrag Šustar, for his consistent support, guidance, and mentorship from my undergraduate days to the completion of this research. His valuable comments and insightful feedback greatly influenced this thesis. His expertise, encouragement, support, and constant engagement with my research not only shaped this thesis but also significantly contributed to my growth as a scholar.

I would also like to express my gratitude to the members of the research project ThUMB, namely Zdenka Brzović, Martina Blečić, Zvonimir Anić, and Aleksandar V. Božić. Their collaboration, shared insights, and valuable comments on my research have greatly improved the quality of this thesis.

I am thankful for the F-427 crew, namely Tamara Crnko, David Grčki, and Ema Lalić, my dear colleagues and friends from the office. Their contributions to creating a fun, engaging, and pleasant working atmosphere made the writing process an enjoyable experience.

I express my thanks to Luca Malatesti for his helpful comments, support, and interest in my fellowship and grant applications. I also extend my thanks to all members of the Department of Philosophy for their warm welcome and the positive working atmosphere provided since the beginning of my employment.

I would like to thank both the Faculty of Humanities and Social Sciences Rijeka and the University of Rijeka for their ongoing support and the provision of resources throughout my PhD program.

I am grateful for the Croatian Science Foundation for funding this PhD project through the "Theoretical Underpinning of Molecular Biology" (ThUMB) project (grant numbers: IP-2018-01-3378 and DOK-2018-09-7078). Without their support I could not have participated in numerous international conferences and summer schools, offering opportunities for meaningful conversations and peer engagement that significantly contributed to my research.

I am also grateful for the Croatian Doctoral Fellowship program of the Wirth Institute for Austrian and Central European Studies and the University of Alberta for funding my research during the academic year 2022/2023. The members of the Institute, headed by Alexander Carpenter, warmly welcomed me, and provided support, offering numerous opportunities to improve my research. Special thanks to all the fellows, namely Ana Grgić,

Zbynek Mucha, Bartosz Jusypenko, Monika Horvath, and Balint Karolyi for creating a great working atmosphere, providing support, and making my days in Edmonton enjoyable.

I would like to thank my dear friends for their consistent encouragement and support throughout this journey. Additionally, I thank anyone else who may have played a role, no matter how small, in the completion of the thesis. Your support and interest are deeply appreciated.

Lastly, my biggest thanks and gratitude go to my family, my mom Jasna, my dad Aco, my sister Ines, and my nephew Luka. I also fondly remember my grandma Gracijela, whose influence remains significant even though she could not witness the conclusion of my PhD journey. Without their encouragement, support, and belief, I could not have come this far.

This journey has been both challenging and rewarding, and all of you helped me so much, for which I am forever grateful.

ABSTRACT

This thesis examines the causal explanation in molecular biology, particularly focusing on the predominant explanatory account in biology, namely, the new mechanistic account. According to this account, a phenomenon is explained by a mechanism, that is, a causal structure with a specific organization of entities and activities responsible for the phenomenon in question. For example, consider the mechanism of protein synthesis. It consists of entities (e.g., DNA) and activities (e.g., transcription) that are responsible for synthesizing proteins, that is, influencing the phenotypic traits of an organism.

The thesis aims to further advance this debate by emphasizing three points. *Firstly*, it aims to structure and present the debate on the issue, particularly by focusing on the background concepts related to the new mechanistic account, such as: *causation*, *explanation*, *mechanism*, *biological functions*, and *law of nature*. In particular, it outlines the interventionist and production accounts of causal explanation, the causal role account of biological functions, and the mechanistic approach to laws of nature.

Secondly, it presents various characterizations of the new mechanistic account, advocating for a consensus view on the concept of a mechanism. Additionally, it emphasizes alternative causal-explanatory structures, such as causal pathways and cascades. For each of these three structures, the thesis outlines their respective aspects regarding their corresponding explanation processes, namely the ontic, epistemic, and strategic aspects. The thesis argues that mechanisms, along with the aforementioned structures, are distinct in the strategic and epistemic aspects, while sharing certain features in the ontic aspect, asserting that mechanisms are the explanatory privileged causal structure.

Thirdly, it aims to apply the interventionist account of causal explanation and the three aspects of mechanisms and pathways outlined above to two case studies from scientific practice, namely: (i) the intervention in the glycolytic pathway to prevent the growth of cancer cells, and (ii) the characterization of natural selection in terms of causal-explanatory structures. Regarding (i), the thesis argues that both the pathway's epistemic aspect and the interventionist account can serve as useful conceptual frameworks. As for (ii), the thesis introduces a novel perspective, contending that natural selection could be partially characterized as a pathway.

KEY WORDS: causal explanation; mechanism; pathway; intervention; glycolysis; natural selection.

PROŠIRENI SAŽETAK

Doktorski rad razmatra *uzročno objašnjenje* u molekularnoj biologiji. Fokusira se na *novi mehanicistički pristup* objašnjenju koji je dominantan pristup u okviru biologije, posebice molekularne biologije. Prema ovom pristupu objašenjenju, fenomeni u biologiji objašnjavaju se putem uzročno-eksplanatorne strukture mehanizma koji se sastoji od entiteta i aktivnosti organiziranih na način da proizvode promatrani fenomen koji se želi objasniti. Primjerice, mehanizam sinteze proteina sastoji se od nukleinskih kiselina (npr. DNA) i aktivnosti (npr. transkripcije) koji su organizirani na specifičan način te u konačnici sintetiziraju protein, odnosno dovode do fenotipskih obilježja organizma.

Rad doprinosi raspravi ovom pristupu tako što: (1) donosi pregled rasprave o uzročno-eksplanatornim strukturama, kao što su mehanizmi i uzročni putevi (*pathways*); (2) zastupa konsenzualnu karakterizaciju mehanizama te izdvaja tri ogovarajuća aspekta za gore navedene strukture: *ontički, epistemički* i *strateški*, zastupajući tezu kako se mehanizmi i uzročni putevi razlikuju u epistemičkom i strateškom aspektu; i (3) primjenjuje intervencijsku teoriju uzročnog objašnjenja te aspekte iz doprinosa (2) na dva primjera iz prakse: liječenje tumora intervencijom u različite dijelove glikolize i slučaj karakterizacije prirodne selekcije, argumentirajući kako je ona djelomično uzročni put, a djelomično mehanizam.

Doprinos (1) očituje se u analizi novog mehanicističkog pristupa tako što rad uzima u obzir pojmove koji su mu temeljni. Ti pojmovi uključuju: *uzročnost*, *objašnjenje*, *mehanizam*, *biološke funkcije* i *zakone prirode*. U tom smislu, rad se isključivo fokusira na one teorije i pristupe koji su vezani uz navedene pojmove u kontekstu mehanizma, a to su: (i) kontrafaktičke teorije uzročnog objašnjenja (s posebnim naglaskom na intervencijsku teoriju); (ii) teorije usmjerene na ishode (*production accounts*, s posebnim naglaskom na fizikalnu i mehanicističku teoriju); (iii) pristup uzročnih uloga biološkim funkcijama (*causal role account)*; i (iv) mehanicistički pristup zakonima prirode.

Zatim, doprinos (2) očituje se u fokusu na sam novi mehanicistički pristup. U tom smislu, rad prezentira različite karakterizacije ovisno o autorima unutar obitelji promatranog pristupa. Rad izdvaja jednu definiciju za koju zaključuje da objedinjuje sve ostale te ju koristi kao opću u ostatku rada. Potom, rad strukturira raspravu novih mehanicista na način da izdvaja tri aspekta putem kojih možemo promatrati raspravu, a to su: ontički, epistemički i strateški. Rad zastupa tezu kako mehanicističko objašnjenje započinje strateškim aspektom, tj. heuristikama dekompozicije i lokalizacije, kako bi zahvatio mehanizam kao strukturu koja se

nalazi u prirodi (ontički aspekt) te, biva zaključen epistemičkim aspektom, tj. stvaranjem reprezentacije mehanizma koji se nalazi u prirodi (npr. stvaranje vizualnog modela).

U ovom dijelu rada, ističu se i recentne alternativne uzročno-eksplanatorne strukture kao što su uzročni put (*causal pathway*) i kaskada. Rad, osim definiranja istih, kao i kod mehanicističkog objašnjenja, izdvaja tri odgovarajuća aspekta za svaku strukturu. Rad argumentira kako se gore navedene strukture razlikuju od mehanizama u dva aspekta: strateškom i epistemičkom. Naime, uzročni putevi i kaskade koriste heuristike stvaranja mreža (*networks*) putem kojih se one zahvaćaju u prirodi (ontički aspekt). Epistemički aspekt razlikuje se u tome što navedene strukture koriste apstraktnije modele s izraženom vremenskom komponentom, tj. isticanjem koraka koji dovode do razultata, dok mehanizmi, osim te karakteristike, imaju i prostornu dimenziju, odnosno takva objašnjenja stavljaju naglasak i na specifičnu organizaciju entiteta unutar mehanizama. Upravo ističući navedenu kompleksnost vremenske i prostorne komponente kod mehanizama, rad zastupa tezu kako su mehanizmi eksplanatorno privilegirana struktura.

Konačno, u doprinosu (3), rad primjenjuje prethodno razmatrane uzročno-eksplanatorne teorije, kao što je intervencijska, te gore navedene aspekte procesa objašnjenja kod struktura, na dvije studije slučaja: (a) liječenje raka putem intervencije u uzročni put glikolize; te (b) karakterizaciju prirodne selekcije putem uzročno-eksplanatornih struktura. U okviru (a), rad zastupa tezu kako je intervencijska teorija uzročnog objašnjanje adekvatni konceptualni okvir promatranja intervencija u liječenju. Osim toga, rad zastupa tezu da studija slučaja (a) pretpostavlja ranije zastupanu tezu epistemičkog aspekta uzročnog puta. Što se tiče studije slučaja (b), rad predlaže karakterizaciju prirodne selekcije na novi način – tako što ju, u određenim segmentima tog procesa, promatra iz perspektive uzročnog puta, a u preostalima iz perspektive mehanizma.

KLJUČNE RIJEČI: uzročno objašnjenje; mehanizam; uzročni put; intervencija; investigativna strategija; glikoliza; prirodna selekcija.

Contents

INTRODUCTION

Terms like explanation and causation are commonly used in the everyday discourse. So commonly, in fact, that we rarely think about what they really mean and correspond to. For instance, when a car crash occurs, we seek to explain how it happened. Similarly, when we observe a rainbow, we seek an explanation for this natural phenomenon. Usually, these explanations involve some form of causation. When explaining a car crash, we aim to identify some factors that caused the car crash, such as flat tires, wet road, or the influence of alcohol that contributed to the incident. To explain a rainbow, we aim to find a scientific explanation, explaining this phenomenon by citing the causes of refraction and reflection of the sunlight entering a raindrop. It is evident that both terms are tightly linked together, and this thesis aims to examine the concept of one specific form of explanation that links both causation and explanation, that is, the *causal explanation*.

Although there exist other forms of explanation, for instance, in the form of an argument (e.g., unificationist and covering law theories) or non-causal explanation (e.g., mathematical explanations), this thesis specifically examines the causal explanation since it focuses on biological disciplines, especially molecular biology. In these disciplines, causal explanation is the predominant way of explaining things. For instance, when a biochemist aims to explain how glycolysis produces energy in the form of ATP molecules, the explanation typically involves citing causes in terms of the multiple steps preceding this energy production. To use philosophical terminology concerning explanation, the *explanandum*, or something that we want to explain, would be the energy production in the form of ATP molecules, and the *explanans*, or that what explains the explanandum, would be the sequence of steps leading to the production of energy.

In biology, particularly in molecular biology, the explanans often takes the form of a causal structure, such as, for instance, the form of a *mechanism* or a *pathway*, which then explains the explanandum, that is, the biological phenomena we wish to explain. Proponents of the new mechanistic account contend that what really explains biological phenomena are mechanisms. They characterize mechanisms in various ways but the most common characterization that most of the new mechanistic authors would endorse is as follows: mechanisms are causal-explanatory structures consisting of entities and activities organized in a way to be responsible for a specific phenomenon. Advocates of the new mechanistic account argue that nearly all biological phenomena can be explained through mechanisms. However,

some authors argue that not all biological explanations include mechanisms exclusively; rather, distinct structures, such as pathways and cascades, also play explanatory roles. This thesis aims to examine the relationship between these various causal structures and their roles in explanations within molecular biology. It seeks to outline their distinctions and delineate their respective ontic, epistemic, and strategic aspects of explanations, arguing that mechanisms hold a privileged explanatory status among other causal structures. Additionally, the thesis aims to apply the conceptual framework of the causal explanation, encompassing these structures, to scientific practice, namely cases in molecular oncology and the characterization of natural selection.

Aims of the Thesis

The thesis contributes to the causal explanation debate by outlining three points. *Firstly* (i), it systematically presents and structures the debate, with a particular focus on the concept of a mechanism. This involves an exploration of background concepts crucial for the debate in question: *explanation*, *causation*, *mechanism*, *biological function*, and *law of nature*.

Secondly (ii), the thesis advocates for a consensual characterization of the new mechanistic account, which, despite having multiple characterizations depending on various authors, aims to establish a unified perspective. It also outlines alternative causal-explanatory structures beyond mechanisms, such as pathways and cascades. Additionally, it argues for the distinction of three aspects in the explanation process for each of the aforementioned structures, namely ontic, epistemic and strategic aspects. Lastly, it outlines mechanisms as an explanatory privileged causal structure.

Thirdly (iii), the thesis extends its contribution by applying the previously examined causal-explanatory structures and their three aspects to scientific practice. This application is demonstrated through two case studies: the inhibition of the glycolytic pathway to prevent the growth of cancer cells and the characterization of natural selection in terms of causal-explanatory structures.

Regarding the contribution (i), in addition to exploring background concepts and structuring the debate, the thesis particularly outlines Woodward's (2003) interventionist account of causal explanation. It argues that this account provides an adequate conceptual framework for examining causal-explanatory structures in biology, especially within molecular biology. The choice of this account is motivated by its significance in the context of scientific practice. Woodward's interventionist account posits that it is heuristically valuable to

conceptualize explanatory and causal relationships as potentially exploitable for manipulation and control over a certain system. In this sense, the account emphasizes the importance of intervention and manipulation for the explanation of a phenomenon. Namely, for an explanation, it is crucial to assess whether manipulating causes can alter the effect, thereby changing the outcome of a process. It is also important to note that this account is commonly endorsed by proponents of the new mechanistic account.

In relation to contribution (ii), besides presenting various characterizations of the new mechanistic account, the thesis advocates for a consensus view on the concept of a mechanism. It endorses the new mechanistic consensus characterization following Illari and Williamson (2012), which states that a mechanism consists of entities and activities organized in a way to be responsible for the phenomenon of interest. The thesis endorses this view arguing that it shares three key features with other prominent characterizations proposed by various new mechanistic authors. These features include the identification of a phenomenon, the heuristic of decomposition of mechanisms, and the organizational aspect referring to entities and activities producing the phenomenon.

Additionally, the thesis systemizes the new mechanistic debate by delineating three aspects of the account. This approach aligns with Levy's (2013) similar framework but also incorporates Salmon's (1984) distinction between the ontic and epistemic aspects of explanations. The three advocated aspects are as follows: the ontic, epistemic, and strategic aspects. The ontic aspect pertains to a mechanism found in nature, representing the mechanism itself as present in the 'real' world, holding a degree of robustness. The epistemic aspect involves the representation of a mechanism, often through models, deriving explanations from these representations of mechanisms. The strategic aspect encompasses the investigative strategies of decomposition, i.e., a strategy referring to breaking down of a specific system or a structure to its constituents, and localization, i.e., a strategy of identification of entities and activities involved in the system or a structure in question, by which one captures mechanisms as found in nature. The advocated approach suggests that the process of a mechanistic explanation involves the strategic aspect aiming to capture the ontic aspect, that is, the mechanism found in nature. Consequently, the epistemic aspect of the explanation attempts to build representations of these mechanisms found in nature, with these representations serving as actual explanations.

In this contribution (ii), the thesis further distinguishes between different causal-explanatory structures beyond mechanisms. Drawing on Ross' (2021; forthcoming) characterization of structures, which provides a comprehensive analysis of pathways and

cascades in relation to biology, the thesis outlines their respective three aspects, mirroring the approach taken with mechanisms. Pathways are characterized as sequences of causal steps that are ordered in a specific way, leading to an outcome of interest (e.g., gene expression pathways; metabolic pathways). Cascades involve amplifying steps converting a small cause or trigger into a huge effect (e.g., blood coagulation; an avalanche).

The distinction between these structures is particularly evident in the strategic aspect. Unlike mechanisms, pathways and cascades do not rely on the heuristics of decomposition and localization; rather, they employ the strategy of building networks, that is, mapping or expanding out their processes. In this sense, scientists aim to create a map, that is, a network depicting available causal routes, serving as representations of potential and accessible routes for investigating new pathways or cascades (e.g., *WikiPathways* database). The difference is also evident in the epistemic aspect, where pathways and cascades use more abstract representations, providing less detailed information for explanatory purposes. However, the key point that the thesis emphasizes, lies in the difference between the *spatial* and *temporal* characteristics. Namely, mechanisms include both system and process-like characteristics, system-like referring to spatial characteristic, and process-like to temporal. The representations of mechanisms emphasize the specific orientation and organization of entities in space, which is responsible for a phenomenon. Additionally, they include the specific activities, that is, the order and duration of their components, also important for an explanation. However, pathways and cascades exhibit more of a process-like, that is temporal characteristics, where the aspect of connection between their steps is pivotal for the explanation of a phenomenon. Thus, different aspects are pointed out in their respective representations. The thesis argues that despite these differences, all three structures share similar features regarding the ontic aspect, which can be encompassed through the features of productive continuity and organization, features originally pertaining to mechanisms. Consequently, the thesis argues that mechanisms are the explanatory privileged causal structure, particularly because they encompass both spatial and temporal characteristics, corresponding to the system and process-like aspects.

In contribution (iii), the thesis aims to apply the abovementioned interventionist account of causal explanation and the three aspects of causal-explanatory structures in a novel approach to two case studies: (a) the intervention in the glycolytic pathway to prevent the growth of cancer cells, and (b) the characterization of natural selection as either a mechanism or a pathway. Case study (a) considers the case of glycolysis, a central metabolic pathway, as a targeted structure for scientists to intervene upon and hinder the growth of cancer cells. Namely, it has been discovered that cancer cells heavily rely on glycolysis as their primary

energy source. Thus, scientists are exploring various strategies to target glycolysis. The thesis outlines two potential targets, namely Glucose transporter 1 and Hexokinase 2. The thesis asserts that the conceptual framework of the interventionist account best accommodates this case study since it emphasizes the role of intervention and manipulation of causes to alter outcomes, as evident in this example. Additionally, it proposes a specific epistemic approach to observing pathways, distinguishing it from other perspectives on the issue, namely Ross' (2021) and Thagard's (2003). In this sense, the thesis argues that pathway representations involve a strong temporal characteristic, proving suitable for the case study in question.

Case study (b) examines the case of natural selection being characterized as a mechanism. In particular, the interpretation that it can be characterized within the framework of the new mechanistic account. In this sense, the thesis explores the natural selection schema proposed by Skipper and Millstein (2005). The thesis argues that the natural selection schema in question could be partially characterized as a pathway, asserting that some of its stages exhibit pathway characteristics, while others mechanistic.

In conclusion, the thesis has three primary aims, as follows: (i) structuring the debate, outlining the interventionist account of causal explanation, the causal role of functions, and the mechanistic approach to laws of nature as key conceptual frameworks in the debate; (ii) advocating a distinction between ontic, epistemic, and strategic aspects of mechanisms, pathways, and cascades, arguing that mechanisms are the explanatory privileged structure; and (iii) applying the conceptual framework of the interventionist account of causal explanation, encompassing the examined structures, to two significant case studies from scientific practice, namely cases in molecular oncology and the characterization of natural selection.

The Structure of the Thesis

The thesis is structured into three parts. Part I, consisting of three chapters, delves into crucial background concepts concerning the causal explanation in molecular biology and the new mechanistic account. In Part II, encompassing three chapters, the focus shifts to an exploration of the new mechanistic account and the alternative causal-explanatory structures, i.e., pathways and cascades. Part III, consisting of two chapters, applies the causal explanation conceptual framework, along with mechanisms and pathways, to two case studies.

In Part I, Chapter 1 examines the general concept of a mechanism. It provides a brief historical overview, beginning with tracing the origins of the mechanically perceived world, from the ideas of Democritus and the Epicureans to the contributions of key thinkers in the

field, such as Rene' Descartes and Emil du-Bois Reymond. The chapter then shifts the focus to contemporary authors, such as Bechtel and Richardson (1993), Glennan (1996), and Machamer, Darden and Craver (2000), who argued for a new conception of mechanisms, particularly relevant to discussions in the life sciences. Additionally, the chapter briefly discusses different meanings of mechanisms in the life sciences, with a particular emphasis on Nicholson's (2012) insights. A more in-depth discussion on this topic is reserved for Part II.

Chapter 2 examines the concepts of causation and explanation. Particularly, because they are intertwined within the context of biology. As mentioned earlier, biological phenomena are explained through the representation of a causal structure, such as a mechanism. The chapter explores those accounts of causal explanations that are particularly relevant for the life sciences. In this sense, counterfactual and production accounts are outlined. Within the family of counterfactual accounts, traditional accounts are briefly discussed (e.g., Lewis 1973a). However, the focus is directed towards Woodward's (2003) interventionist account of causal explanation, given its substantial endorsement by the new mechanistic account. Additionally, the interventionist account is emphasized for its strong connection with scientific practice. Namely, its core idea is that to explain phenomena, one needs to identify a cause that can be intervened upon and manipulated, thereby altering the effect. This account is further explored in Part III since its potential application to examples from scientific practice.

Concerning production accounts, Salmon's (1984), and Dowe's (2000) accounts, stating that, to explain phenomena one needs to have a nexus of causes leading to an effect, are examined. Moreover, Glennan's (1996) account, proposing that one needs mechanisms and causes within them leading to an effect, is considered. Although these accounts are also relevant to the new mechanistic account, it is the interventionist account that is thoroughly examined and endorsed throughout the thesis due to its alignment with scientific practice and the new mechanistic account.

Chapter 3 explores the concepts of biological functions and laws of nature. The discussion on biological functions emphasizes, besides the selected effects account of function (see Millikan 1984; Garson 2016), the causal role account, introduced by Cummins (1975). In particularly, because it was additionally advanced and modified by Craver (2001; 2013) considering the notion of a mechanism as proposed by the new mechanistic account. Specifically, asserting that the function of a trait depends on its current contribution to the system it pertains to. In other words, the function depends on the specific organization of entities and activities of a mechanisms producing a phenomenon. The examination of laws of nature delves into how new mechanists seek to diminish or replace the role of laws in

explanations within biology (see Andersen 2011; Glennan 2017). In particular, this perspective contrasts the role of laws in physics (e.g., Newton's first law of motion) with those in biology (e.g., Mendel's law of segregation). Contrary to laws of physics, which are considered as being defined in terms of universal antecedent statements, laws in biology often have exceptions and thus the proposed alternative are mechanisms, as structures better suited to capturing explanations of phenomena in the life sciences.

In Part II, Chapter 4 presents various characterizations of mechanisms depending on the three most prominent authors, namely Glennan (1996; 2002), Machamer, Darden, and Craver (2000), and Bechtel and Abrahamsen (2005). Although the thesis acknowledges their differences, it points out their similarities and argues for a consensus view about mechanisms, endorsing Illari and Williamson's (2012) and Glennan's (2017) perspective.

Moreover, drawing on Salmon (1984) and Levy (2013), the thesis distinguishes between three aspects of mechanisms, namely ontic, epistemic, and strategic aspects. The thesis advocates for a specific process of mechanistic explanation being initiated by the strategic aspect, aiming to capture the mechanism in nature, i.e., the ontic aspect. However, the epistemic aspect, that is, the representation of a mechanism, is the one that actually explains the phenomenon in question, grasping the ontic conception through that representation.

Chapter 5 shifts the focus to the recently re-emerged causal-explanatory structures, such as pathways and cascades (see Ross 2021; forthcoming). The chapter outlines the corresponding features and investigative strategies associated with these structures.

Chapter 6 provides the distinction between mechanisms, pathways, and cascades, and their respective aspects and processes of explanation. The thesis argues that the key differences among these structures lie in the strategic and epistemic aspects. Moreover, it asserts that mechanisms are the explanatory privileged causal structure, primarily because they encompass both spatial and temporal characteristics.

In Part III, Chapter 7 revisits the pathway concept previously explored and introduces Thagard's (2003) perspective on the concept, which aims to characterize biochemical pathways and elucidate their role in medical treatment. The chapter distinguishes between Ross's (2021), Thagard's (2003), and my own approach to the pathway concept by revisiting the three aspects of the pathway concept outlined earlier. It emphasizes the role of glycolysis in cancer treatment, as scientists explore strategies to target glycolysis and hinder the growth of cancer cells. The chapter argues that Woodward's interventionist account of causal explanation serves as a particularly useful conceptual framework in this particular case.

Chapter 8 addresses the case study of characterizing natural selection as a causal-explanatory structure. It specifically explores the natural selection schema presented by Skipper and Millstein (2005), dividing natural selection into seven stages. The chapter introduces a novel perspective to the debate by considering natural selection as a pathway that, partially, better characterizes natural selection.

PART I: The Causal Explanation: Background Concepts

INTRODUCTION. Part I

In this part of the thesis, I examine several background concepts related to causal explanation: mechanism, causation, explanation, law of nature, and function. I focus on their relationship with the new mechanistic account. These background concepts play a pivotal role in the detailed examination of the new mechanistic account in Part II.

Firstly, I examine the concept of a *mechanism*, widely used in diverse areas, such as, among others, politics, economics, technology, science, and philosophy. For example, the political mechanism of extremely general scope is brokerage described as "the joining of two or more previously less connected social sites through the intervention of third parties" (Tilly 2001: 26). The scientific mechanism, such as, the mechanism of protein synthesis, produces protein molecules (see Glennan 2017). Both abovementioned mechanisms aim to elucidate how things work, and by doing so, learning how to work with them.

The concept of a mechanism has historical roots, predating the 20th century and the rise of the new mechanistic account. We can trace the debate back to ancient philosophy, i.e., Democritus and the Epicureans. Moreover, early modern philosophers, such as Rene' Descartes (1596 – 1650), laid out the idea of providing mechanical explanations for all kinds of phenomena. Besides philosophers, Emil du Bois-Reymond (1818 – 1896) significantly contributed to biologists' understanding about what the search for mechanisms is (see Craver and Darden 2013: 4).

The concept of a mechanism holds, arguably, different meanings in the life sciences. Besides the characterization of mechanisms from the new mechanistic account, which originated in the 1990s (Bechtel and Richardson 1993; Glennan 1996; Machamer, Darden, and Craver 2000), other authors advocated for different perspectives on mechanisms. Among these authors, I outline Daniel Nicholson's (2012) approach to the issue.

Mechanisms are also crucial for some classic issues in the philosophy of science, such as, among others, *prediction*, *function*, *explanation*, *causation*, *scientific discovery*, and *control*, which I, to a limited extent, address in Part I. Each of these issues is examined in relation to the new mechanistic account, focusing on the concept of a mechanism.

Secondly, I examine the concepts of causation and explanation. These concepts are ubiquitous in everyday life as well as in scientific contexts. For instance, lifting weights in the gym *causes* muscle cells growth. That *explains* why individuals go to the gym for a muscular

physique. Moreover, motor cells in the sunflower pulvinus *cause* sunflowers to turn to the Sun and track its movement (see Briggs 2016). That *explains* why sunflowers follow the Sun. We search for causes and explanations to better understand phenomena around us and to manipulate and intervene on the causes to prevent or alter the effects.

In this part of the thesis, I examine the concept of causation and its relation to the concept of explanation by focusing on the accounts that are interesting for molecular biology, that is, especially for the leading account in the field, namely the new mechanistic account. In that sense, I emphasize two accounts of causation: (i) *counterfactual* accounts (see Lewis 1973a; Woodward 2003), and (ii) *production* accounts, that is, the physical account (see Salmon 1989; Dowe 2000), and the mechanistic account (see Glennan 1996). Both (i) and (ii) serve as a basis for their respective explanation accounts. Before exploring these accounts that are closely related to the causal explanation in molecular biology and the new mechanistic account, I discuss another account, arguably a causal explanation account as well, namely the *deductive-nomological* model (see Hempel and Oppenheim 1948).

Thirdly, I examine the concepts of *law of nature* and *function*, widely used across scientific disciplines and particularly in the life sciences. The relationship between laws, functions, and mechanisms is emphasized, with examples such as Gregor Mendel's law in genetics, which is used to describe regularities in inheritance.

Regarding the concept of law of nature and its connection to the new mechanistic account, some new mechanists avoid the role of laws for explanations (see Machamer, Darden and Craver 2000). On the other hand, some try to replace explanations that appeal to laws with those that appeal to the mechanistic account of causation (see Glennan 1996). New mechanists, especially Holly Andersen (2011), argue that "a great variety of scientific generalizations, some of which we call laws, are in fact explained by mechanisms" (Glennan 2017: 45).

Regarding the role of functions and its link to the new mechanistic account, two opposing views are outlined. On one side, Carl Craver's (see 2001; 2013) view on the relation between function and mechanism is discussed, specifically the relation between functional and mechanical description. Craver follows Robert Cummins' (see 1975; 2002) concept of function, known as the causal role account. On the other side, Justin Garson's (see 2013) perspective on the relation between function and mechanism is presented, emphasizing that mechanisms serve functions. Garson argues for the functional sense of mechanism, following the *selected effects* account of functions (see Millikan 1989; Neander 1991; Garson 2017), which stands in opposition to the *causal role* account.

Part I is structured as follows: in Chapter 1, the concept of a mechanism is examined, divided into three sections. Section 1 presents the pre-new mechanistic discussion. Section 2 examines the concept through the lenses of different perspectives in the life sciences. Section 3 explores some of the general issues concerning the concept of a mechanism, such as prediction and control.

In Chapter 2, causation and explanation are examined, also divided into three sections. Section 1 outlines the deductive-nomological model of explanation that predates the new mechanistic account. Section 2 explores counterfactual accounts of causation and explanation. Section 3 examines production accounts of causation and explanation.

In Chapter 3, the concepts of law of nature and function are examined. Section 1 explores the relationship between law of nature and mechanisms. Section 2 depicts Craver's view on the relation between function and mechanisms. Section 3 outlines Garson's view on the same issue.

Chapter 1: Mechanism

INTRODUCTION. Chapter 1

The term ‘mechanism’ finds frequent usage across various discourses, encompassing political, economic, technological, scientific, and other domains. For example, the political mechanism of broad scope is brokerage, i.e., “the joining of two or more previously less connected social sites through the intervention of third parties” (Tilly 2001: 26). In economics, a coherent mechanism explores commodity price fluctuations which, roughly, examines the influence of speculative storage on prices (see Deaton 2010). The technological mechanism is the mechanical watch movement mechanism, specifically the escapement, which regulates timekeeping accuracy (see Xu, Ko and Du 2011). In science, the biological mechanism is the mechanism of protein synthesis, the process by which protein molecules are produced (see Glennan 2017). All these mechanisms are aiming to present how things operate, facilitating a better understanding of how to work with them.

The primary focus of this chapter is to examine the concept of a mechanism. *Firstly*, to examine the concept of a mechanism in general and to discuss the pre-new mechanist discourse on the issue. The historical roots of the discussion trace back to ancient philosophy, that is, Democritus and the Epicureans. Early modern philosophers, such as Descartes, furthered the idea of providing mechanical explanations for various phenomena. Descartes envisioned a world of small corpuscles colliding with one another, proposing different models of mechanisms in *The World* to explain characteristics of both biological and non-biological realms. Besides philosophers, Bois-Reymond emerges as one of the central figures responsible for providing biologists with the needed insights into the search for mechanisms (see Craver and Darden 2013: 4).

Secondly, the chapter shifts its focus to the concept of a mechanism within the life sciences. In the late 20th and early 21st century, interest in the mechanistic concept in biology surged with the rise of new mechanists. The new mechanistic account originated in the 1990s with the publication of William Bechtel and Robert Richardson’s book “Discovering Complexity” (1993), and influential papers by Stuart Glennan (1996) and Peter Machamer, Lindley Darden, and Craver (2000). The chapter briefly discusses new mechanists and different meanings of mechanisms in the life sciences, particularly through the lenses of Nicholson (2012), reserving an in-depth discussion on the topic for Part II.

Thirdly, the chapter examines the significance of mechanisms for classic issues in the philosophy of science. The concept of a mechanism addresses various classic issues, including, among others, prediction, function, explanation, causation, scientific discovery, and control. However, this chapter provides a brief overview of prediction, control, the process of scientific discovery, and reduction, as other issues such as explanation, function, and causation are more extensively addressed in the subsequent chapters.

The chapter is structured as follows: In Section 1, the concept of a mechanism is examined by presenting the pre-new mechanistic discussion. Section 2 outlines the concept of a mechanism in the life sciences, with more in-depth coverage addressed in Part II. Section 3, briefly addresses the importance of mechanisms for classic issues in the philosophy of science, that is, discovery, reduction, prediction, and control.

SECTION 1. The Concept of a Mechanism: Pre-New Mechanistic Discussion

This section briefly outlines the pre-new mechanistic discussion on the concept of a mechanism, tracing its roots back to ancient Greeks. The mechanistic worldview originated with atomism, notably in the works of Democritus and the Epicureans. Democritus of Abdera (c. 460 – c. 370 BC) proposed that the world consists of atoms. According to him, atoms are ingenerated, imperishable, and homogeneous, with changes that are possible in atoms' respective spatial positions movement. All changes are reducible to combinations of atoms that are always in motion. Atoms differ from each other regarding the shape and size, which are important when, following their collisions, they form clusters. Also, different atomic shapes can determine perceptible qualities. For instance, according to Democritus, heat is caused by especially small and sharp atoms. Democritus perceived the world mechanistically in the sense that the world consists of solid particles and that their collisions, entanglements, and vibrations are responsible for the understanding of causation (see Popa 2017).[1]

The Epicureans embraced Democritus' and other early atomists' accounts and made their own original contributions to the atomistic approach as well. For instance, in his philosophical poem Lucretius (c. 99 – c. 55 BC) insisted on emphasizing order and regularity of nature. According to Tiberiu Popa (2017), Lucretius and thr Epicureans can be described as proponents of a mechanistic view because of their belief that all phenomena "are explainable through the causal relations between and within the vast realms of nature" (Popa 2017: 17).

Contrary to the atomism argued by Democritus, Lucretius and the Epicureans, Aristotle (384 – 322 BC) held a different view. He criticized early atomism for lacking teleology, which, he believed, led to contradictions.[2] However, in Aristotle's writings on, for instance meteorology, we can find limited mechanistic interest. Popa (2017) argues that Aristotle devoted his three books, Meteorology I-III, to a "description, and, especially, causal explanation of 'meteorological' processes" Popa (2017: 17). Aristotle believed that rainbows, rain, formations of minerals, etc., "are caused by the inherent nature of and also by the interaction between two exhalations (*anathumiaseis*) present in the sublunary world" (Popa 2017: 17). Moreover, according to Popa, their behavior is described in mechanical terms,

[1] However, some authors argue for a different perspective, contending that Democritus' physical theory is hardly mechanistic (see Berryman 2009).

[2] For more information on Aristotle's view, in particular, see Popa (2017). I do not address his view here since the aim of this section is to examine the mechanistic concept in general, particularly the pre-new mechanistic discussion, without focusing on specific periods, such as, for instance, antiquity.

predominantly by referring "to objects which are ejected under severe pressure" (Popa 2017: 17).

Besides the trace of mechanistic thinking found in the works of antic authors, early modern natural philosophers are responsible for introducing the term 'mechanical philosophy' and providing all sorts of mechanical explanations for various phenomena, such as gravity, magnetism, the motion of the heart, the circulation of blood, etc. (see Roux 2017: 26). Robert Boyle (1627 – 1691) is considered as the first scholar that coined the term 'mechanical philosophy'. During that period, i.e., the period of Scientific Revolution, intellectuals observed the natural world as a world of mechanisms and science as fundamentally constructed around the search for mechanisms (see Craver and Darden 2013: 3). These intellectuals, among others, were Galileo Galilei, Thomas Hobbes, Pierre Gasendi, and Descartes. To briefly demonstrate how they perceived the world through mechanisms, I examine Descartes, a philosopher often called the "arch-mechanist" (see Roux 2017: 41). I do so by examining his book *The World* in which he, partly, investigates mechanisms concerning the life sciences. Within *The World*, in the *Treatise on Man*, he argues about the composition of men.

Descartes (1664, in Gaukroger (ed.) 1998: 99-100) posits that "the body to be just a statue or a machine made of Earth".[3] He then illustrates this mechanistic perspective with an example of food digestion in the stomach, considering it as a part of the machine, i.e., the body. According to Descartes, the digestion process occurs "by the force of certain fluids" that drift through its parts. The fluid, rapidly pumped from the heart through the arteries, generates significant heat, leading to the breakdown and heating up of the food. Descartes' depiction of the digestion process closely aligns with the characterization of the mechanisms argued by new mechanists since it involves some activities and entities that are producing a certain outcome. In this example, the entity is the fluid, and the activities includes the separation, shaking, and heating of the food parts, culminating in the digestion outcome. However, a more detailed examination of the new mechanists' characterization is deferred to the following section and further explored in Part II.

Descartes emphasizes that the human body operates as a machine, with distinct parts responsible for various behaviors and outcomes. In his *Treatise on Man*, he presents detailed illustrations of mechanisms. For example, Figure 1 illustrates the eye, depicting the eyelids that are, according to Descartes, manipulated by two muscles. One that has its purpose to open the upper lid, while the other is responsible for both opening and closing both lids.

[3] Descartes refers to the element earth, which he describes in the *Treatise on Light.*

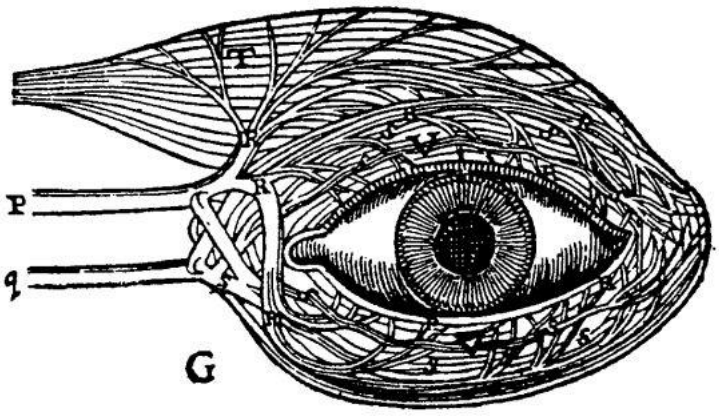

Figure 1: the eyelid muscles depicted in Descartes (reproduced from Descartes 1664, reprinted in Gaukroger (1998: 114).

As it is mentioned above, Descartes advocates for a mechanistic interpretation of the human body, highlighting the analogy between the body and a machine. This interpretation aligns with the prevalent seventeenth century characterization of the world, which often drew parallels between humans and human-made machines. However, as I outline in the next section, biological mechanisms often differ significantly from machines. On one side, machines are typically planned with pre-existing and organized connected parts. For instance, a mechanical watch, which movement mechanism was described in the introduction to this chapter, is an accurately assembled machine. On the other side, biological mechanisms evolve through the process of natural selection, which parts may be synthesized dynamically, undergo rapid degradation, or become more stable over time. Craver and Darden (2013: 15) aptly express this distinction stating that: "The blueprint of the typical biological mechanism is decidedly messier than the blueprint for even complicated machines". Contrary to machines with carefully crafted and organized structures, biological mechanisms often exhibit levels of complexity and messiness that is inherent in their evolutionary development. I further explore this distinction in the following section, as well as in Part II (see Chapter 4).

Besides philosophers, such as Descartes, I also emphasize Bois-Reymond as a key figure providing biologists with crucial insights into the search for mechanisms, particularly through his intriguing idea that natural selection is a mechanical process (see Craver and Darden 2013: 4). I examine this idea in more detail in Part III (see Chapter 8). Bois-Reymond, a German physiologist, is known for advocating the search of mechanisms within Charles Darwin's (1809 – 1882) worldview. He outlines that natural selection is a mechanism as evident in his following statement:

> "Afflavit Darwinius et dissipata est" would be a fitting inscription for a medal in honor of the Origin of Species. Now everything evolved from a few simple germs; now we did not require successive creations, just a single act that set matter in motion; now purpose in nature was replaced by a *mechanical* process, which is how we can regard natural selection; and now man finally took his proper place at the head of his brethren. (Bois-Reymond 1883: 557; italic added)

In this quote, Bois-Reymond explicitly identifies natural selection as a mechanical process. He believed that in the same way as Copernicus' heliocentrism had displaced the Earth from the center of universe, Darwin's theory of evolution had displaced man from the center of the Earth (see Finkelstein 2013). The ongoing debate among new mechanists about whether natural selection can be characterized as a mechanism was sparked by Robert Skipper and Roberta Millstein's influential paper in 2005. Various accounts either support (see Barros 2008; Illari and Williamson 2010; DesAutels 2016) or oppose (see Havstad 2011; Garson 2019) this idea. This debate is further addressed in Part III (see Chapter 8).

The pre-new mechanistic discussion regarding the concept of a mechanism and the mechanical point of view anticipated the impact of mechanistic perspectives in scientific disciplines. I tried to briefly present the historical summary of the above discussion, starting with the early atomists. Next, the focus shifted to the Scientific Revolution period. Lastly, I outlined the viewpoint of a 19th-century physiologist. Moving forward, the focus will shift to the concept of a mechanism in the contemporary setting, specifically within the life sciences, with a particular emphasis on molecular biology. Thus, I will engage with ideas on the issue found in the contemporary literature, which is, nonetheless, to a limited degree, an extension of the above historical pre-new mechanistic discussion.

SECTION 2. The Concept of a Mechanism: The Life Sciences

The concept of a mechanism, particularly among new mechanists, is commonly described as follows: a mechanism is constituted of entities and activities that are organized and responsible for a phenomenon.[4] However, it is important to note that the concept of a mechanism is not novel; it has been transformed throughout history to align with the changing understanding of causal forces in the world, such as, gravitational attraction, conservation of energy, etc. (see Craver and Tabery 2019). To examine the concept of a mechanism, various authors have terminologically fragmented the concept (e.g., see, among others, Allen 2005; Craver 2005; Ruse 2005; Andersen 2014). In this section, I present Nicholson's (2012) distinction between three different meanings of mechanism, as it incapsulates both the historical and causal meanings of the concept.[5]

Although Nicholson is not typically viewed as a proponent of the new mechanistic account, but rather as a critic of the account, I approach the first part of his examination of the issue in question as an analysis of the concept of a mechanism rather than a critique of the new mechanistic account. Thus, I explore his perspective on mechanisms and use it as a starting point in slightly altering the different meanings of the concept of a mechanism to align it with the framework of the new mechanistic account. This approach allows for a nuanced examination that integrates Nicholson's insights into the broader context of the new mechanistic account.

Nicholson's (2012: 153) three distinct meanings of mechanism are as follows: (i) *mechanism* as a philosophical thesis about the nature of life and biology; (ii) *machine mechanism*, referring to the workings of a machine; and (iii) *causal mechanism* involving a particular mode of explanation. Meaning (i) is often linked to *mechanistic philosophy*, or *mechanical philosophy* and it is usually identified with an atomistic, naturalistic, and deterministic views of nature. Its background is found in the work of famous philosophers and scientists, such as Galilei, Descartes, and Bois-Reymond. Meaning (ii) concerns machine-like

[4] New mechanists have provided slightly varied definitions of mechanisms (see, among others, Glennan 1996; Machamer, Darden, and Craver 2000; Bechtel and Abrahamsen 2005). However, the mentioned characterization is considered, to some extent, a consensus among new mechanists regarding the necessary conditions that encompass the concept of a mechanism (see Illari and Williamson 2012; Glennan 2017). The detailed examination of the definition and characterization of mechanisms is provided in Part II (see Chapter 4).

[5] In alignment with Nicholson (2012), Levy (2013) distinguishes three theses linked with the new mechanistic account, specifically addressing causation, explanation, and scientific methodology. However, for the purposes of this discussion, I examine Nicholson's perspective since I believe that it provides a more generalized examination of the concept of a mechanism. In contrast, Levy's distinctions within the new mechanistic account are examined in more detail in Part II (see Chapter 4) and Part III (see Chapter 8).

systems conceived in mechanical terms. For instance, organisms are conceived in analogy with machine mechanisms such as a clock or an engine. Machine mechanisms, both biological and technological, can be examined in isolation and decomposed into smaller machine mechanisms. Meaning (iii) relates to identifying causal relations in a mechanism. To examine the causal mechanism for a phenomenon of interest, one has to examine the causes that explain how that phenomenon is produced. The first two meanings were briefly addressed in the preceding section. The third meaning will be examined in the subsequent sections of this chapter and in more detail in Chapter 2.

Furthermore, Nicholson establishes a twofold relationship among these meanings, which is schematically represented in Figure 2.

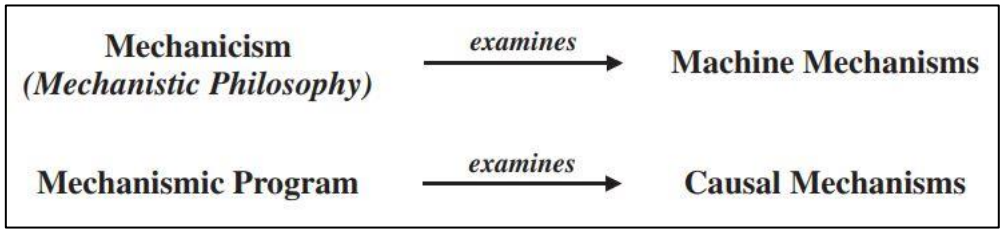

Figure 2: Nicholson's relationship between meanings of 'mechanism' (reproduced from Nicholson 2012: 155).

Following that figure, on one side, 'mechanicism' examines *machine mechanism*, and on the other side, 'mechanismic program' examines *causal mechanisms*. The terms 'mechanicism' and 'mechanismic' program are introduced by Nicholson to distinguish the new mechanistic discourse from the traditional view on mechanisms held by authors such as Galilei, Descartes, Pierre Gassendi, Boyle, and others during the Scientific Revolution. However, for the purposes of this thesis, I propose a slightly modified relationship between the meanings of mechanisms. For simplicity and consistency with established literature used throughout this thesis, I propose the following terms: *natural philosophy*, corresponding to Nicholson's 'mechanicism' program, and *new mechanistic account*, corresponding to Nicholson's 'mechanismic' program. These terms are commonly employed in the literature on mechanisms (see, for instance, among others, Craver and Darden 2013; Glennan 2017). I use Nicholson's distinction of two relationships between meanings of the concept of a mechanism to delineate the pre-new mechanist's discussion, i.e., the natural philosophy discussion, and the new mechanist's discussion. Thus, influenced by Nicholson's distinction and the terminology of

new mechanists, I present an alternate version of the relationship between different meanings of the concept of a mechanism in Figure 3.

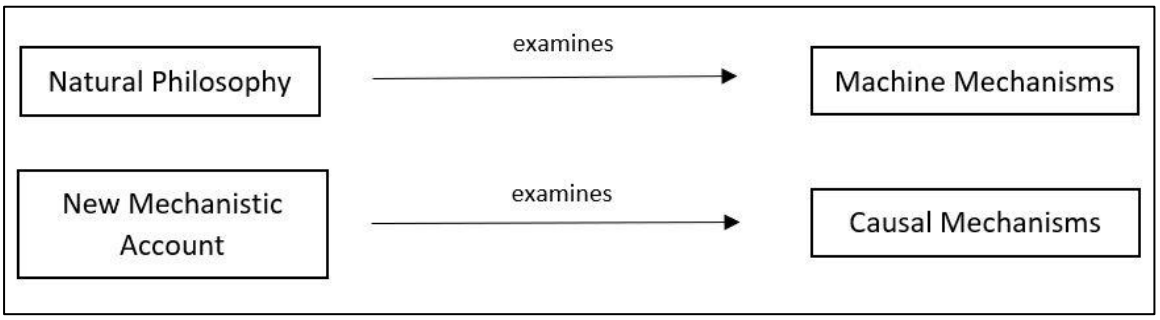

Figure 3: An alternative representation of the relationship between two meanings, aligning Nicholson's (2012) terminology with new mechanists.

Now, in alignment with Figure 3, I illustrate the following two meanings of the concept of a mechanism: (a) natural philosophy, which examines machine mechanisms, and (b) the new mechanistic account that examines causal mechanisms. Natural philosophy thrived during the Scientific Revolution, while the new mechanistic account is at its zenith in the contemporary philosophy of science, originating in the latter half of the 20th century. The previous section delved into meaning (a) highlighting, among others, early atomists, Descartes, and Bois-Raymond as exemplars of mechanists associated with this meaning. In particular, Descartes, who viewed the human body as a machine, was pointed out as a prime example. Following this literature, the preceding section also underscored the analogy between organism and machines, i.e., the comparison of the eye to a mechanical clock. Thus, the subsequent paragraphs will focus on meaning (b).

In the exploration of causal mechanisms within the field of philosophy of biology during the 20th century, scholars such as Stuart Kauffman (1970) and William Wimsatt (1972a) showed early interest. However, according to Nicholson, Robert Brandon (1985) is credited as the first to offer a comprehensive analysis of the significance of causal mechanisms for biological research. Brandon's perspective diverges from the common association of mechanism with reductionism often attributed to natural philosophy's machine analogy. According to him, "mechanism is not to be identified with reductionism in any of its forms; in fact, mechanism leads to a non-reductionist ontology" (Brandon 1985: 345). Contrary to the reductive stance linked to natural philosophy, "the appeal to causal mechanisms in scientific practice *does* not imply a commitment to the reductionistic agenda" (Nicholson 2012: 157).

Craver and Darden (2013: 12) contribute to this new mechanistic approach by asserting that biology encompasses various fields and subfields,[6] such as anatomy, physiology, cytology, epidemiology, genetics, evolutionary biology, etc. Although these fields operate somewhat independently, that is, in isolation from one another, they often require collaboration. That is evident regarding exciting discoveries related to mechanisms, which demand cooperation between researchers from different biological fields and the utilization of different experimental techniques. The elucidation of the mechanism of protein synthesis serves as a notable example. In this specific case, molecular biologists and biochemists collaborated across disciplines, working from different perspectives to elucidate protein synthesis. Molecular biologists, such as James Watson and Francis Crick, sought to understand the flow of genetic information, i.e., how the linear pattern in the DNA sequence of bases dictates the base sequence in RNA, ultimately producing the linear order of the amino acids in proteins. Simultaneously, biochemists were interested into the flow of matter and energy, examining aspects such as amino acids and the energy required for chemical bond formation. Although both fields approached the mechanism distinctively, these researchers converged on the common ground of RNA. Bridging their diverse research programs, they detailed the roles of three different kinds of RNA in the protein synthesis mechanism (see Craver and Darden 2013: 164-165).

In addition to the distinction regarding the reductionistic agenda, to distinguish meaning (a) from meaning (b), Nicholson argues that, in my terms, the new mechanistic account, unlike natural philosophy, "is not primarily concerned with biological ontology, but with the nature of biological explanations" (Nicholson 2012: 157). According to Nicholson, there are "few occasions" in which new mechanists "explicitly address matters of biological ontology" and this typically occurs when they seek to differentiate their views, i.e., causal mechanisms, from the natural philosopher's machine mechanism perspective. In this regard, new mechanists employ two distinct strategies. *Firstly*, they focus on the current usage of the term 'mechanism' in biology. Thus, their aim is to minimize the scope of merging the term's usage in the natural philosopher's sense in present scientific practice. For example, Bechtel and Adele Abrahamsen (2005: 422) state the following: "Perusing the biological literature, it quickly becomes clear that the term biologists most frequently invoke in explanatory context is *mechanism*".

[6] Craver and Darden (2013: 12) characterize a field as follows: "A field is unified by a common problem or set of questions, a shared vocabulary for describing the items in that domain, a set of experimental and observational tools to use for answering those questions, a set of accepted protocols for using those tools, and, finally, a set of concepts and theories in terms of which the problem might be solved".

Machamer, Darden and Craver (2000: 2) present the following claim: “Our goal is to sketch a mechanistic approach for analyzing neurobiology and molecular biology that is grounded in the details of scientific practice, an approach that may well apply to other scientific fields”. As demonstrated in the previous quotes, one of the new mechanists’ motivations behind the introduction of the new mechanistic account lies in the frequent use of the term ‘mechanism’ in scientific practice. This represents a significant difference in examining the term ‘mechanism’ compared to the machine mechanism point of view brought forward by natural philosophers, as mentioned in the previous section.

Secondly, new mechanists explicitly distinguish mechanisms, i.e., causal mechanisms, from machines, i.e., machine mechanisms, on various occasions. For instance, Craver and Darden (2013) offer the following statement:

> However, biological mechanisms are often quite unlike machines. A machine is a contrivance, with preexisting, organized, and interconnected parts. Paradigm examples are the mechanical clock, the water pump, the internal combustion engine, and the computer. Biological mechanisms have been tinkered together under mutual constraints through evolution by natural selection and through development […]. The blueprint of the typical biological mechanism is decidedly messier than the blueprint for even complicate machines. Craver and Darden (2013: 15)

As we can see in this quote, according to Darden and Craver, the meaning of causal mechanism is distinct from the machine mechanism view. They emphasize this to underline that biological mechanisms differ from machine mechanisms, particularly in that biological mechanisms are products of natural selection, whereas machines are constructed from preexisting and organized parts. However, not all new mechanists entirely abandon the distinction between machine mechanisms and causal mechanisms. As noted by Nicholson (2012: 157), Glennan (see 1996; 2002) insists on the notion of machine mechanisms in his new mechanistic account, even citing cells and organisms as prime examples of a ‘machine’. Thus, it appears that even within the ‘family’ of new mechanists, there are authors who are more inclined to draw comparisons between biological mechanisms and machines.

Although Nicholson (2012: 157) points out that new mechanists are not predominantly concerned with biological ontology, in contrast to natural philosophers, it is worth to highlight that some new mechanists argue that biological ontology is, in fact, an important issue in examining mechanisms. *Firstly*, as previously mentioned, Machamer, Darden and Craver

(2000) argued that a suitable characterization of mechanisms must meet standards of descriptive, epistemic, and metaphysical adequacy. Furthermore, Glennan (2017: 19) offers the following claim: "Moreover, to the extent to which successful mechanistic science must refer in some way to mechanisms as things in the world, methodological and metaphysical issues will inevitably be entwined". *Later*, he claims that his characterization of mechanisms, called "minimal mechanism", is an ontological characterization of what mechanisms are as things in the world. Thus, it seems inaccurate to categorize new mechanists as thinkers who do not engage in resolving ontological questions regarding mechanisms found in scientific practice. The new mechanistic account employs the concept of a mechanism to address a wide scope of various topics. Some of these topics, such as control, prediction, reduction, and discovery, are addressed in the next section, while others, such as causation, function, law and explanation, are the focus of the following chapters.

To sum up this section, I presented Nicholson's distinction of different meanings of 'mechanism' which encapsulates both the historical meaning of the concept of a mechanism and the causal meaning of the concept. I slightly altered his view to tailor it to fit into the new mechanistic framework, that is, the terminology used by the new mechanistic authors, which is more thoroughly addressed in Part II. Although Nicholson attempted to distinguish natural philosophy from the new mechanistic account by emphasizing the distinction between machine mechanisms and causal mechanisms on one side, and biological ontology and biological explanation on the other, I aimed to show that there are proponents of the new mechanistic account who emphasize the use of machine mechanisms and engage with biological ontology. In the following section, and even more in the subsequent chapters, this thesis attempts to address various topics found in the philosophy of science that are anticipated in the above meanings of mechanism, particularly causal explanation.

SECTION 3. The Concept of a Mechanism: Control, Prediction, and Other Issues

The significance of mechanisms in addressing classic issues within the philosophy of science, such as explanation, function, and causation, is thoroughly explored throughout the dissertation, particularly in the following two chapters. However, there are additional classic issues that new mechanists, to some extent, engage with, such as, control, prediction, reduction, and discovery. In this section, I provide a brief overview of these issues, with a slight emphasis on the issues of control and prediction due to their close connection to the issues examined in the following chapters.

Firstly, I provide a brief overview of reduction and scientific discovery as found in new mechanists' work. *Reduction* is a classic and extensively examined topic in the philosophy of science. For the purposes of this section, I briefly focus on one aspect of this debate, namely the example of the reduction of Mendelian or classical genetics to molecular genetics. One of the many authors that addressed this possible reduction of disciplines, i.e., a new mechanistic author, Lindley Darden (2005), emphasized the role of mechanisms in the reductionism debate. According to one of the many distinctions regarding reduction in philosophy of science, one can distinguish between theory, explanatory, and constitutive reduction.[7] I will begin by briefly defining theory reduction, as it is particularly relevant to the abovementioned example.

According to Sarkar (1992), theory reduction involves subsumption of concepts, explanations, and methods from one scientific discipline under those of another. Theory reductionists believe that when one theory is reduced to another, the explanation of the reduced theory is done by the reducing one. For instance, proponents of theory reduction might argue that explanations within Mendelian genetics could ultimately be reduced to those provided by molecular genetics. Kenneth Schaffner (1993) asserts that the successes in molecular biology indicate an ongoing reduction of Mendelian genetics to molecular genetics, and consequently, to biochemistry. Schaffner argues that, even though this reduction is still not complete, it is in principle possible, and will eventually take place. According to him, molecular biology analyzes the fundamental processes previously studied by Mendelian genetics, thereby paving the way for the reduction of Mendelian genetics to molecular genetics.[8]

[7] For more information on this particular distinction, see Sarkar (1992). Additionally, for more information on other distinctions related to reduction in philosophy of science, see van Riel and van Gulick (2023).

[8] Schaffner's position was challenged by several authors. For instance, David Hull (1976) argued that classical genetics was being replaced by molecular genetics instead of being reduced to it. Furthermore, Wimsatt (1976) advocated for a different version of reduction, namely for an explanatory reduction of Mendelian genetics to molecular genetics. Additionally, Philip Kitcher (1984) argued that Mendelian genetics lacked laws that could be reduced to molecular genetics, rejecting Schaffner's proposed reduction. Moreover, Sarkar (1992) argued that, at

However, Darden (2005) argues against the notion that Mendelian genetics has been reduced to molecular genetics, nor that it has been replaced by it. According to her, these two fields are best characterized as investigating "different, serially integrated, hereditary *mechanisms*" (Darden 2005: 349). These mechanisms "operate at different times and contain different working entities" (Darden 2005: 349). In Mendelian heredity, working entities are chromosomes, while in mechanisms of molecular genetics, working entities are larger and smaller segments of DNA and related molecules. Darden emphasizes that progress is not always made "by moving to lower size levels" (Darden 2005: 368). Although molecular genetics, along with its discoveries of DNA mechanisms, filled black boxes unilluminated by Mendelian genetics, the chromosomal mechanism of meiosis still explains the regularities captured in Mendel's law of segregation. Thus, Mendelian genetics and molecular genetics are seen as autonomous fields with their own distinctive contributions.

As we have seen in the abovementioned example, the new mechanistic account is not only relevant to the reduction debate in the philosophy of science but also plays a crucial role in the realm of *scientific discovery*. Logical empiricists, such as, Hans Reichenbach (1951) and Karl Popper (1959), argued that philosophers could not contribute to the understanding of how scientists generate ideas and that those so-called "a-ha" moments of creativity should be examined by psychologists and not philosophers.[9] However, Bechtel and Richardson (1993; 2010), Darden (2006), and Craver and Darden (2013), assert the importance of mechanisms as a framework for the process of scientific discovery. As Craver and Darden (2013: 65) highlight that: "the search for mechanisms, shapes the process of discovery". Bechtel and Richardson (2010: 23-24) emphasize that the process of searching for mechanisms starts with the characterization of a phenomenon and is followed by the strategies of *decomposition* and *localization*. Decomposition refers to the idea that one activity of the whole system is the product of a set of subordinated functions performed in the system. Localization involves the identification of different activities that are proposed by the task of decomposition. Darden (2006) and Craver and Darden (2013) argue that the idea of searching for mechanisms leads to the investigation of component parts, their entities and activities, and features of their organization. They further note that: "The idea that one is searching for mechanisms allows

least, the structure of explanations in molecular biology is reductionist. For a brief overview of critiques made towards Schaffner's theory reduction model, see Brigandt and Love (2022). For a more comprehensive overview of the reductionism discussion in molecular biology, particularly for the interplay between reductive and anti-reductive interpretations of Max Delbrück's role for the rise of molecular biology, see Balorda (2023).

[9] For more information on Scientific discovery in general, see Schickore (2022). For more information about scientific discovery in philosophy of science and mechanisms, see Craver and Tabery (2019).

one to work on the discovery project in a piecemeal fashion; one can work on one part of the mechanism at a time while leaving other parts as black or grey boxes" (Craver and Darden 2013: 65). Thus, this approach enables one to be more focused towards exploring the component parts and activities constitutive to a mechanism.

Besides reductionism and scientific discovery, *prediction* is another traditional issue in the philosophy of science. For the purposes of this section, I first briefly emphasize two different views on the issue and then proceed to address the new mechanistic perspective. One view comes from Gustav Hempel and Paul Oppenheim's (1948: 138) deductive-nomological model of explanation, according to which prediction is symmetrical to explanation. In other words, explanation is delivered in a form of an argument and prediction is symmetrically related to explanation. The other view comes from Wesley Salmon (1998: 126). He argues that both prediction and explanation are vital for scientific progress. According to Zvonimir Anić, Salmon argues that "predictions without explanations do not improve our 'scientific understanding' of the world" (Anić 2022: 155).

The new mechanistic perspective introduces a nuanced view on the relationship between explanation and prediction. While it does not endorse a symmetrical relation akin to Hempel and Oppenheim's model, it underscores a close connection between the two. New mechanists usually contend that both explanation and prediction are in correspondence through the process of the discovery of a mechanism and the construction of a model of that mechanism. The model is subsequently tested by scientists through intervention into mechanisms. If the predictions of outcomes of these interventions are failing, then it seems that there is something in the model of a mechanism that does not correspond to the mechanism found in the nature. Darden (2006: 30) describes the mentioned process as a two-step methodology: "Reasoning in the light of failed predictions involves, first, a diagnostic process to isolate where the mechanism schema is failing, and, second, a redesign process to change one or more entities or activities or stages to improve the hypothesized schema".

Correct predictions are crucial in science because they demonstrate the accuracy of representing entities and the interactions in the proposed model of a mechanism. This approach aligns with the new mechanistic emphasis put on empirical testing and refinement based on observed outcomes. When predictions based on a model of a mechanism fail, it triggers a revision of the model. A model of mechanism is revised depending on the outcomes of predictions. Darden aptly characterizes this discovery process in biological sciences as an "error-correcting process" or "iterative refinement" (Darden 2006: 272, 306). According to Craver and Darden (2013: 201): "[D]iscovery of a mechanism is a piecemeal iterative process,

not a linear march from constructing a schema to demonstrating its adequacy. Often anomalies turn up, and some require revision of a hypothesized mechanism schema". Following these quotes, the accuracy of predictions serves as a crucial criterion for the fidelity of a model to the real-world phenomenon it represents. It seems that the more closely a model of a mechanism corresponds to the actual mechanism in the world, the more accurate and reliable the predictions derived from it become.

The final issue I briefly touch upon in this section is *control*. The new mechanistic account emphasizes a strong connection between mechanistic knowledge and control. As Craver and Darden (2013: 186) point out: "When we know how a mechanism works, we know the buttons and levers inside it that might be pushed and pulled to make it work for us". In other words, this emphasis on mechanistic knowledge extends to the ability to manipulate and control outcomes by knowing the organization of entities and their respective activities. The control of a mechanism is especially important for science and even more so for medicine. The knowledge of physiological mechanisms, for instance, guides diagnostic reasoning, and enables scientists and physicians to target specific mechanisms for intervention. One illustrative example of the useful intervention is the intervention in the mechanism of glycolysis, which is addressed in detail in Part III (see Chapter 7). Here, however, I highlight another case, namely the intervention in the mechanism of cystic fibrosis.

Cystic fibrosis is a chronic disease that significantly impacts the respiratory system and digestive tracts. It is "attributed to the disrupted function" of the cystic fibrosis "transmembrane conductance regulator (CFTR) *gene*" (Wei et al. 2020: 4992). While primarily affecting the lungs, cystic fibrosis manifests as a multiorgan disease, impacting organs such as the pancreas, liver, and kidneys. This disease is generally a result of the deletion of phenylalanine at the 508th position of CFTR. CFTR is ubiquitous throughout the body. It is present, among others, in the epithelial cells in the kidney, pancreas, and airway. The knowledge of the molecular structure of CFTR helped with the discovery of the treatment methods. CFTR protein consists of "1,480 amino acids", and the length of its coding sequence is known, as well as its regulatory roles (Wei et al. 2020: 4993).

Three molecular therapies that are used in cystic fibrosis treatment are emphasized. These molecular therapies are based upon the knowledge of the mechanism of cystic fibrosis and the control of entities and activities organized in the mechanism. Molecular therapies are the following: supplement therapy, gene therapy, and modulator therapy. Supplement therapy uses the correct version of CFTR mRNA transfected or transduced into the respective target cells. Gene therapy uses nasal epithelium and adenovirus vectors containing CFTR gene to

restore CFTR function. Modulator therapy includes two groups: potentiators and correctors. “The potentiators act on the CFTR ion channels” and “the correctors serve a key role in the transportation of nascent proteins” (Wei et al. 2020: 4997). The diversity of treatment strategies in cystic fibrosis, each grounded in a comprehensive knowledge of the underlying mechanism, highlights the importance of knowledge and control of the mechanisms, which guides therapeutic interventions. By unraveling the mechanisms, scientists can tailor approaches that directly target specific parts of a particular causal structure to offer more effective and nuanced treatments.

CONCLUSION. Chapter 1

To sum up, in this chapter, I *firstly* examined the concept of a mechanism in general, beginning with the brief overview of the pre-new mechanistic discussion on the issue that traced back to ancient philosophers, such as Democritus and the Epicureans. The discourse extended to the examination of early modern thinkers, such as Descartes, who advocated for the idea of providing mechanical explanations for all kinds of phenomena. Besides philosophers, I also examined the role of Bois-Reymond, who emerged as a pivotal figure responsible for providing biologists with crucial insights into the search for mechanisms.

Secondly, I shifted the focus to the concept of a mechanism as found in the contemporary literature of the philosophy of biology. Here, the chapter briefly touched on the new mechanistic conception of mechanisms; however, I focused mainly on the concept of a mechanism in general, i.e., to situate the concept in a wider setting as well as examine it historically. In that regard, I outlined Nicholson's (2012) tripartite distinction between different meanings of mechanism. Primarily because it encapsulates, on the one side the historical meaning of the concept of a mechanism, and on the other the causal meaning of the concept.

Thirdly, I briefly examined the importance of mechanisms for some other classic issues in the philosophy of science, namely, reduction, discovery, prediction, and control. While reductionism and scientific discovery were touched upon. In particular the example of the possible reduction of Mendelian genetics to molecular genetics, particular attention was given to the issues of prediction and control, anticipating a further examination in the subsequent parts of the thesis, specifically for the interventionist account of causal explanation.

Chapter 2: Causation and Explanation

INTRODUCTION. Chapter 2

Causation and *explanation* are closely related and ubiquitous in everyday life, as well as in the sciences. For instance, lifting weights in the gym *causes* muscle cell growth. That *explains* why people, if they want to have a muscular physique, go to the gym. Moreover, motor cells in the sunflower pulvinus *cause* sunflowers to turn towards the Sun and track its movement (see Briggs 2016: 541-542). That *explains* why sunflowers follow the Sun. We search for causes and explanations to better understand phenomena around us. Additionally, we seek causes and explanations to manipulate and intervene, thereby preventing or altering the effects and outcomes.[10] Thus, it is important to examine what causation and explanation are, more specifically exploring their relationship.

In this chapter, I aim to examine the concept of causation and its relation to the concept of explanation. However, given the extensive debates and vast literature surrounding each of these concepts, it is impossible to cover all the accounts regarding them. Thus, I focus on accounts that are particularly relevant to molecular biology, specifically emphasizing the leading account in the field, namely the new mechanistic account. In this sense, I highlight two accounts of causal explanation as follows: (i) the counterfactual accounts of causal explanation, and (ii) the production accounts of causal explanation, namely the physical account of causation, and the mechanistic account of causation.[11]

Related to accounts (i), traditional counterfactual accounts are briefly discussed (e.g., see Lewis 1973a). However, the chapter focuses more on the advanced counterfactual analysis of causation, namely James Woodward's (2003; 2010) account of causation that links causation with the notions of *manipulation* and *intervention*. These notions are of critical importance for the subsequent discussion in this thesis. These discussions, in particular, revolve around the application of specific causal explanatory structures to molecular biology, including mechanisms and causal pathways. This becomes particularly relevant to the cancer treatment

[10] The latter search is of particular interest for Part III; nevertheless, I also examine the interventionist account in this chapter.

[11] Some authors, such as Glennan (see 2011: 791-793), categorize these two accounts as the concept of cause as productivity on one side and the concept of cause as relevance on the other. According to him, the counterfactual accounts encompass the concept of cause as relevance, while production accounts encompass the concept of cause as productivity. Glennan's distinction aligns with Sober's (see 1984) distinction between token and property (type) causation. Both distinctions bear similarities to the one presented above, namely the one between accounts (i) and (ii).

program, where interventions on the glycolytic causal pathway are explored in Part III (see Chapter 7).

Regarding accounts (ii), production accounts are briefly discussed. *Firstly*, Salmon's (1984) account is examined, alongside Phil Dowe's (2000) modification of Salmon's framework. Both accounts examine the concept of a causal process, proposing that causal connections in the world are best analyzed in terms of causal processes and interactions. *Lastly*, Glennan's (1996; 2017) version of the mechanical account of causation is explored. According to this account, causal claims revolve around mechanisms. Glennan's version of this account is particularly valuable for Part II, where his perspective on the new mechanism debate is discussed.

Nonetheless, before examining the abovementioned models of causation closely related to causal explanation in molecular biology and the new mechanistic account, I introduce another account that tightly links causation and explanation, namely the deductive-nomological model, proposed by Hempel and Oppenheim (1948).[12]

[12] Here, I would like to note that various accounts of explanations exist in the debate, including the unificationst account proposed by Michael Friedman (1974) and Kitcher (1981). Additionally, probabilistic accounts of causation and explanations are present (see Illari and Russo 2014). Furthermore, I would also like to note that not all explanations are causal. For instance, some argue that mathematical explanations are non-causal. However, this issue exceeds the scope of this thesis. For more information on non-causal explanations, see Woodward (2018). For a critical perspective on non-causal explanations and an extension of the causal explanation account, see Lina Jansson and Juha Saatsi (2019).

SECTION 1. The Deductive-Nomological Model of Explanation

Although the primary focus of this thesis is on causal explanation, specifically its implementation in the life sciences, another account, arguably also causal explanatory, dominated the discussion on the topic in the 1950s and 1960s, namely the deductive-nomological (DN) model of explanation. The DN model was developed by Hempel and Oppenheim in 1948. The name of the DN model is derived from two parts: *deduction* and *nomology*. According to Hempel and Oppenheim (1948), explanation is deductive because the phenomenon we seek to explain is deduced from the premises. Additionally, explanation is nomological because at least one premise of the explanation is a law. In this section, I briefly outline this model and its objections, which paved the way for other accounts to prevail. Specifically, more causally oriented accounts of explanations, such as the counterfactual account, and production accounts, such as the mechanistic and the physical accounts of causation.

Hempel and Oppenheim (1948: 136-137) abstracted the basic pattern of explanation from various examples of phenomena requiring explanation. For instance, consider a situation in which a mercury thermometer is rapidly immersed in hot water. The observation reveals that the mercury column first temporarily drops and then swiftly rises. This phenomenon can be explained by stating that hot water initially affects only the glass tube of the thermometer, which expands and thus creates more space for the mercury to drop. However, when the risen temperature reaches the mercury, it begins to expand, leading to the rise in the mercury level. This occurs because the coefficient of expansion of mercury is greater than that of glass.

According to Hempel and Oppenheim, two kinds of statements explain this phenomenon. On one side, there are certain antecedent conditions, such as the fact that the thermometer consists of a glass tube partly filled with mercury and being immersed in hot water. On the other side, there are certain general laws at play in this example, such as the laws governing the thermal expansion of mercury and glass.

Following the initial example of an explanation, Hempel and Oppenheim (1948) abstracted the structure of explanation by emphasizing two major constituents of an explanation: an *explanandum* and an *explanans* (see Figure 4). The explanandum is a sentence describing the phenomenon to be explained. The explanans is a class of sentences that are adduced to account for the phenomenon. The explanans is further divided into “two subclasses; one of these contains certain sentences C1, C2, …, Ck which state specific antecedent

conditions; the other is set of sentences L1, L2, …, Lk which represent general laws" (Hempel and Oppenheim 1948: 137).

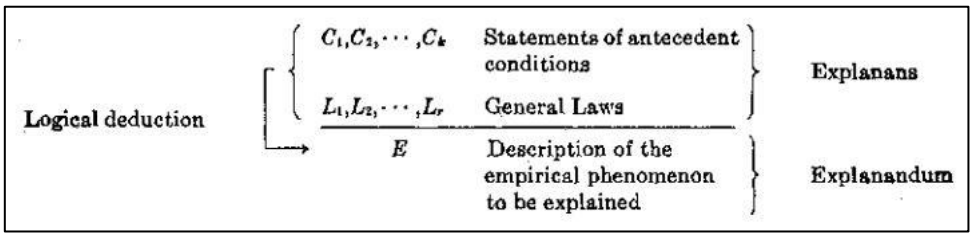

Figure 4: The structure of the DN model of explanation (reproduced from Hempel and Oppenheim 1948)

According to Hempel and Oppenheim (1948: 139), "if E describes a particular event, then the antecedent circumstances described in the sentences C1, C2, …, Ck may be said jointly to "cause" that event, in the sense that there are certain empirical regularities, expressed by the laws L1, L2, …, Lr, which imply that whenever conditions of the kind indicated by C1, C2, …, Ck occur, an event of the kind described in E will take place". Thus, the DN model described here is characterized as a type of causal explanation. In this sense, I emphasize the DN model as one type of a causal explanation that predates other accounts examined in later sections. However, the DN model type of explanation also involves subsuming the fact being explained under a general law or general laws; thus, this approach to explanation is also referred to as the *covering law model* of explanation.

Before briefly presenting counterexamples to this model, it is important to note that Hempel (see 1965) developed two additional models. While the DN model captures explanation via deduction from deterministic laws, the question of statistical laws and their explanatory status is also addressed. In this regard, Hempel introduced two types of statistical explanations, namely the deductive-statistical (DS) model and the inductive-statistical (IS) model. In the DS model, an explanation involves the deduction of a narrow statistical uniformity from a more general set of premises, including at least one general statistical law. This model follows a similar pattern to the DN model, as it includes the deduction of the explanandum from a statistical law.

In contrast, the IS model involves the subsumption of individual events under statistical laws, similarly to how the DN model subsumes particular events under general laws. One further distinction between the IS model and the DN model is that the DN explanations subsume events deductively, while the IS explanations subsume them inductively. However,

these two models are not directly relevant to the later discussion, so I am not examining them further.[13]

1.1. Counterexamples to the DN Model

Now, I present two counterexamples to the DN model that will directly lead us to the examination of models of explanations putting more emphasis to causation. The first counterexample is provided by Sylvain Bromberger (1966). Bromberger highlighted that many explanations exhibit asymmetric features, a criterion the DN model fails to address. For instance, consider measuring the length of the shadow cast by a flagpole. From information about the flagpole's height, the angle it makes with the Sun, and laws describing the propagation of light, we can deduce the length of the shadow (see Figure 5). According to the DN model, this qualifies as an example of an explanation. However, we can also deduce the height of the flagpole with information about the length of the shadow, the angle it makes with the Sun, and the same laws. Although this latter derivation meets all the criteria of the DN model, it does not explain why the flagpole has a certain height. Thus, the DN model faces an issue of asymmetry, which could potentially be addressed by placing greater emphasis on the role of causation in explanations.

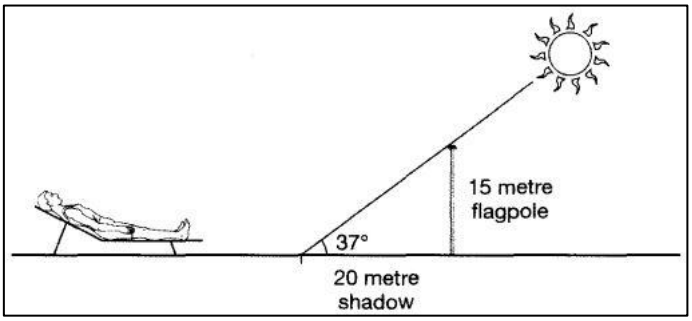

Figure 5: The flagpole counterexample to the DN model of explanation (reproduced from Okasha 2002)

The second counterexample I present is from Salmon (1971: 34) and focuses on the problem of explanatory relevance. He provides an example with the following structure: (i) a premise that all males who take birth control pills regularly fail to get pregnant, (ii) a premise that John Jones is a male who has been taking birth control pills regularly, and (iii) the

[13] For more information on the topic, see Hempel (1965), Salmon (1989), and Woodward (2003: 153).

conclusion that John Jones fails to get pregnant. This argument satisfies the criteria of the DN model for an explanation; however, the premises fail to explain why John Jones fails to become pregnant.

In response to the previous two counterexamples, new accounts of explanation emerged. The most prominent ones include the unificationist account, probabilistic accounts, and accounts that emphasize causation, such as the causal-mechanical model and the counterfactual account.[14] Given the relevance of the latter two for the following chapters of the thesis, particularly in the context of causal explanation in molecular biology, I focus on them in the next section.

[14] For more information on the unificationist account of explanation, see Friedman (1974) and Kitcher (1989). For more information on the probabilistic account of causation and explanation, see Phyllis Illari and Federica Russo (2014).

SECTION 2. Counterfactual Accounts of Causation and Explanation

The counterfactual accounts I emphasize in this section consist of two main accounts. The first account is David Lewis' (1973a; 1973b; 1986) counterfactual account of causation and causal explanation, which serves as the foundation for the second account that I examine in more detail, namely the interventionist account argued by Woodward (2003; 2008).

2.1. Explanation and Counterfactuals: Lewis' (1986) Causal Explanation

To better comprehend Woodward's account, which is predominantly used for causation in the context of examining the new mechanistic account,[15] I briefly outline Lewis' (1973a) counterfactual analysis. This analysis, in a way, serves as a starting point for Woodward's examination of causation.[16] Following this, I explore Lewis' (1986) perspective on causal explanation.

The idea behind counterfactual causation suggest that the meaning of causal claims can be articulated through counterfactual conditionals, phrased as "If A had not occurred, B would not have occurred" (see Menzies and Beebee 2020). The most prominent counterfactual account was developed by Lewis (1973a). Lewis (1973b) articulates his view in the following manner:

> We think of a cause as something that makes a difference, and the difference it makes must be a difference from what would have happened without it. Had it been absent, its effects, some of them, at least, and usually all – would have been absent as well. Lewis (1973b: 161)

Lewis' quote will be significant when I examine Woodward's account. For now, I focus on Lewis' counterfactual analysis.

The example Lewis (see 1973a: 1) emphasizes is the following one: "If kangaroos had no tails, they would topple over". This statement means that in any possible state of events in which kangaroos have no tails, and which resembles our actual state of events as much as kangaroos having no tails permits it to, the kangaroos topple over. Lewis acknowledges that

[15] Many new mechanistic authors, such as Craver (2007), Craver and Darden (2013), Glennan (2017), and Lauren Ross (2021), emphasize Woodward's (2003) account of causation serving as a basis for examining the concept of causal structures, particularly considering life sciences.

[16] Woodward (see 2003: 196) underlines that his account is closely tied to counterfactual information. In his book, he compares Lewis' (1973a) counterfactual account to his interventionist account and points out similarities and differences between the two (see 2003: 133).

counterfactuals are well-known to be vague, but nonetheless he attempts to provide a clear account of their truth conditions. His analysis is based on the possible-world semantics for intensional logic developed by authors such as, for instance, Saul Kripke (1963). The counterfactual analysis exceeds the scope of this thesis and merits its own discussion; however, the causal explanation analysis offered by Lewis is crucial for the subsequent development of Woodward's account.

Lewis (1986: 213-214) contends that any particular event someone wishes to explain is situated at the end of a complex and long causal history. According to him, an explanandum event has its multiple causes that act in conjunction. He illustrates that with an example of a car crash, a consequence of various causes. This specific car crash has its own causal history, including factors such as the icy road, the bald tire, the drunk driver, the blind corner, the approaching car, and more. These causes collectively caused the car crash. Without any one of these causes, the crash would not have happened, or at least, it would have been less probable than it was. Lewis notes that each of these causes has its own causes, potentially influencing the car crash as well. Thus, the car crash is a result of many different causal and converging chains. In this sense, the causal history has a tree-like structure where the chains may converge and diverge. For instance, the driver's roots in childhood could cause his disposition to get drunk.

According to Lewis (1986: 215-216), a causal history is also a relational structure that includes *relata*, in this case, events, namely specific local matters of fact, such as conversations, battles, flashes, falls, and so on. These events can be placed in various relations, such as spatiotemporal relations. The causal dependence found in the causal history of an explanandum is, to return to the counterfactuals, a counterfactual dependence, according to Lewis. Recall that the counterfactual sentence is the sort of a sentence where if the first event did not happen, the second event would never have existed.

Now, returning to explanation specifically, Lewis (1986: 216) argues that, in his words, "to explain an event is to provide some information about its causal history". According to him, when someone asks a why-question about a certain event or a phenomenon, the answer to that question requires explanatory information. In other words, a person explaining an event has to provide information about the causal history of that event. This information about the causal history may be specific or abstract; for instance, a person explaining a phenomenon might specify a certain causal chain that leads to an event or might highlight several causes that lead to a phenomenon.

Lewis' perspective on explanation primarily concerns particular events; however, he argues that his account can naturally extend to the explanation of general kinds of events. For instance, explaining why struck matches light in general is not different from explaining why a singular struck match lit. In that sense, he contends that all the events of a given kind have their histories, and those histories may, to some extent, be similar. Finally, Lewis proposes a list of seven desiderata that successful explanations should satisfy. However, for the purposes of this thesis I am not examining them further; instead, I move on to examine Woodward's take on causal explanation, which is partly based on Lewis' previously examined account.

2.2. Explanation and Counterfactuals: Woodward's (2003) Causal Explanation

Woodward's (2003) account of causal explanation, also known as the interventionist or manipulability account, shares many similarities with the counterfactual account of causation argued by Lewis. Both accounts attribute a central role to counterfactuals in exploring causal notions, setting them apart from production accounts of causation, which are examined in more detail in the next section. Before delving into the differences between Lewis' and Woodward's accounts, let me first present the interventionist conception of causal explanation.

Woodward (2003: 9) draws motivation for such an account because the manipulability and intervention conception plays a crucial role in the way scientists think about causal explanation. He specifically highlights molecular biology as a discipline in which explanations provide information that can be used for manipulation and control purposes. In molecular biology, new instrumental and experimental techniques significantly contribute to the progress of the discipline by enabling the potential to intervene and manipulate within biological systems, leading to results not previously achievable. Thus, according to Woodward (2003: 9), "we are in a position to explain when we have information that is relevant to manipulating, controlling, or changing nature". An explanation, or at least the beginning of one, emerges when factors or conditions are identified such as their manipulation or changes produce changes in the explanandum. For Woodward, explanatory information is information that is potentially relevant to manipulation and control.

Here, it is worth noting that Woodward follows the terminology used by Lewis regarding explanatory information, however in a slightly different way. He specifically emphasizes that for an explanation, information is only considered if it is potentially manipulative or controllable. Thus, as Woodward (2003) points out:

> [T]he notion of information that is relevant to manipulation thus needs to be understood modally or counterfactually: the information that is relevant to causally explaining an outcome involves the identification of factors and relationships such that *if* (perhaps contrary to fact) manipulation of these factors *were* possible, this would be a way of manipulating or altering the phenomenon in question. Woodward (2003: 10)

In other words, Woodward's idea is that any successful explanation should be linkable to a hypothetical or counterfactual experiment. This experiment demonstrates how manipulation of the factors mentioned in the explanans would serve as a means to manipulate or alter the explanandum. Thus, the explanation should be structured in a way that enables us to answer, as Woodward terms it, the *what-if-things-would-be-different* question. In this sense, the explanation should allow us to discern the potential differences in the explanandum if the factors in the explanans had been different in various possible ways. Therefore, similar to Lewis, there is a presence of counterfactual dependence between the explanans and the explanandum.

Nonetheless, let me now closely examine the interventionist account itself, focusing particularly on the notion of *invariance*, and then the characterization of causation. Afterward, I will highlight the differences between Woodward's account and the counterfactual account argued by Lewis, followed by some general critiques toward the interventionist account.

Woodward (2003: 25) argues that it is heuristically useful to think about explanatory and causal relationships as relationships potentially exploitable for the purpose of manipulation, and thus the control over a certain system. However, these relationships should also be invariant under interventions to be considered both causal and explanatory. Invariance, as Woodward (2003: 239) points out, is "the key feature a relationship must possess if it is to count as causal or explanatory". An invariant relationship is characterized as a relationship that remains stable or unchanged as changes occur. A generalization is deemed invariant or stable across changes if it holds up to a certain level of approximation across those changes. For instance, the ideal gas law and the gravitational inverse square law are considered invariant, according to Woodward. The ideal gas law states that the state of gas is determined by its pressure P, volume V, and temperature T: PV=nRT, where *n* is the number of moles of gas, and R is the universal gas constant" (Illari and Russo 2014: 102). Now, this generalization is invariant under a wide range of interventions on temperature. Consequently, the generalization accurately describes how manipulations or interventions on the temperature of the gas would influence the gas pressure while holding the gas volume fixed. Since this "generalization is

invariant, it is *potentially* exploited for manipulation and control" (Illari and Russo 2014: 102), implying that it is causally explainable.

Furthermore, Woodward argues for the concept of invariance to address the dilemma of whether much of what scientists call "explanation" is genuinely explanatory or if explanatory generalization in the special sciences qualify as laws. In this sense, invariance bypasses this dilemma by demonstrating that generalizations are potentially stable upon changes. Consequently, we can argue that certain generalizations are not purely accidental. Therefore, a generalization can play an explanatory role even if it holds only within a specific domain and has exceptions outside that domain. The notion of invariance is especially well-suited for explanations in the special sciences, which I examine more closely in the following section.

Woodard's interventionist account has predecessors in other manipulability accounts such as those developed by Douglas Gaskin (1955), Georg von Wright (1971), and Peter Menzies and Huw Price (1993). To better illustrate the manipulability account, Woodward distinguishes between two camps on causation, and those are: *impractical* and *practical* camps. The impractical camp consists of accounts that fail to elucidate how knowledge of causal relationships has any practical utility. Examples of such accounts are examined in this section, namely Lewis' counterfactual account and Salmon's and Dowe's production accounts. The practical camp consists of accounts that connect causal knowledge with a goal that has practical utility. Woodward's manipulability account falls into this camp. He argues that practical accounts are better than impractical, which he labels as incomplete. Practical accounts are superior because they provide a straightforward explanation of the practical value of causal knowledge that is crucial for improving our predictive abilities. For instance, if we know that C occurred and then we know that C causes an event E, the knowledge that C causes E improves our ability to predict whether E will occur. If E is an event that potentially affects the survival of an organism, then having this predictive information is beneficial.[17]

In this sense, Woodward's interventionist account of causation explains the role of experimentation in causal inference as follows: "experimentation is relevant to establishing causal claims because those claims consist in, or have immediate implications concerning, claims about what would happen to effects under appropriate manipulations of their putative causes" (Woodward 2003: 35). Thus, the link between causation and experimentation is built

[17] Woodward emphasizes that perceiving prediction as the distinctive goal of causal inquiry is problematic, and thus he explores this issue (see Woodward 2003: 31). However, this analysis surpasses the scope of this section.

in the content of causal claims. Woodward argues that such a connection is not found in other accounts of causation, such as Salmon's and Dowe's accounts, which are examined in the next section. This connection between causation and experimentation will be particularly crucial in Part III, where I emphasize Woodward's account of causal explanation in the cancer treatment program.

Now, with this conception of causation in mind, I present Woodward's (2010) characterization of what it is for X to cause Y:

> X causes Y if and only if there are background circumstances B such that if some single intervention that changes the value of X (and no other variable) were to occur in B, then Y or the probability distribution of Y would change. Woodward (2010: 290)

In this characterization, X and Y represent variables[18] of causal claims within the interventionist framework. Background circumstances refer to those circumstances not explicitly represented in the X-Y relationship. In an ideal experimental scenario, an intervention on X causes a change in Y in a way that is exclusively occurring through this change in X and not through any other means. Woodward illustrates this characterization of causation with an example of short circuits causing fire. Background circumstances, such as the presence of oxygen, mean that in these circumstances, intervening to change whether a short circuit is present or not will affect the outcome, i.e., whether the fire occurs.

From the examination of Woodward's account so far, it is evident that counterfactuals play a central role in elucidating causal notions, similar to Lewis' account. Now, let me briefly highlight the differences between Woodward's and Lewis' accounts. According to Woodward (2003: 133), Lewis' account aims only to be an account of token causation between particular events. In contrast, Woodward emphasizes that his account extends to cover type-causal claims as well.[19] Another distinction lies in the question of reduction. According to Woodward (2003: 136), Lewis' account is reductionist, aiming to characterize causation in terms of a more general notion of counterfactual dependence that does not itself presuppose causal notions. However, Woodward's interventionist account operates differently; one counterfactual claim always depends on other causal claims. In other words, if C causes E, the truth of counterfactual claims involving C and E, always depends on the truth of other causal claims involving other variables beyond C and E. Another difference lies in motivation. Lewis implicitly argues that

[18] A variable is a property, or a quantity, that is capable of at least two various values (see Woodward 2010: 290).
[19] Woodward proceeds to present his argumentation on the issue, however that difference is not relevant for purposes of this thesis (see Woodward 2003: 134).

his standards for similarity are adopted because they make counterfactual claims come true that we think, pretheoretically, ought to be true.[20] Woodward, however, sees Lewis' counterfactual account as impractical due to its complex and non-intuitive standards. In contrast, the interventionist account is more practical because it emphasizes the connection of causal knowledge with practical goals. In other words, Woodward's account is more intuitive, prioritizing the practical value of causal knowledge for improving our ability to predict and control.

Lastly, let me briefly address some general critiques of the interventionist account, which I do not examine further since they are not in focus of this thesis. For detailed responses to these critiques, see Woodward (2003: 103-105; 2016). The primary concerns include *anthropomorphism* and *circularity*. Anthropomorphism suggests that the interventionist account places excessive emphasis on human beings as the sole agents of intervention and manipulation in causal relationship. However, Woodward defends his view (see 2003: 104) by pointing out that his characterization of causation does not elevate humans in a superior position; rather, human interventions are considered events in the natural world, akin to any other activities. Circularity arises from the notion that concept of intervention itself must be characterized in causal terms, raising the question explaining causation with a concept already characterized in causal terms. Woodward (see 2016) argues that his account is not circular in the sense that there are other sorts of causal information, beyond the notion of intervention, that are relevant for characterizing a relationship as causal. For instance, in a relationship between X and Y to be considered causal, there needs to be, on one side, intervention on X that changes Y, and on the other side, additional correlational information, e.g., that X and Y remain correlated under this change.

Now that I have examined Woodward's account of causal explanation, as well as Lewis' counterfactual account, I proceed to examine other accounts of causation and explanation that are closely related to the new mechanistic account, namely production accounts.

[20] Lewis' standards of similarity are the criteria for evaluating similarity among possible worlds that are appropriate for analyzing counterfactuals to elucidate causal claims. However, the examination of these criteria surpasses the scope of this section. For more information, see Lewis (1986: 47).

SECTION 3. Production Accounts of Causation and Explanation

Production accounts encompass two main accounts. The first is the causal-mechanical (CM) account, proposed by Salmon (1984; 1989), and later refined by Dowe (2000), introducing the conserved quantity (CQ) account. The second is the mechanistic account, introduced by Glennan (1996; 2017). In the following subsection, I examine Salmon's CM account before examining Dowe's CQ account. Subsequently, I explore Glennan's (2017) mechanistic account of causation. Additionally, I highlight Peter Railton's (1980) advocacy for a mechanistically based account of causation.

3.1. Salmon's CM and Dowe's CQ Accounts

3.1.1. Salmon's Account of Physical Causation

Salmon's version of the production account is the CM account.[21] Roughly, it qualifies as a production account because it primarily considers causality as a property of individual processes.[22] His account, partly, is developed as a response to the deficiencies in the DN model of explanation, which could not address the previously mentioned counterexamples properly. Simply put, Salmon's account asserts that to explain a certain phenomenon, we have to identify one or more causes that produced the phenomenon. For instance, recall Bromberger's counterexample concerning asymmetry, i.e., the problem of measuring the height of a flagpole's shadow. Salmon's perspective resolves the asymmetry problem by highlighting that the shadow is caused by the interaction between the light from the Sun and the height of the flagpole. By emphasizing causation in explanation, we address the phenomenon entirely in one direction, thus eliminating the asymmetry problem. In this sense, Salmon's (1984: 139) account overcomes traditional challenges associated with the nature of causal relations by viewing causality as a continuous process rather than a relation between events. Salmon's account consists of two key concepts: *causal processes* and *causal interactions*. I first examine the former and then proceed to explore the latter, particularly exploring its relevance for explanations.

[21] Woodward argues that Salmon's account includes counterfactual notions, however, I emphasized it as a production account following Dowe's (2000) and Illari and Russo' (2014) approaches to Salmon's account.
[22] Salmon's account is partly influenced by Reichenbach's (1958; first published in German in 1928) and Bertrand Russell's (see 1948) views on causality. However, further analysis in that direction surpasses the scope of this chapter (for more information see Dowe 2000).

The CM account of explanation characterizes a causal process as a *physical process* (see Woodward 2003: 350). To illustrate this process, consider a baseball bat moving through the air. As the bat moves through space and hits the ball, Salmon identifies this as a causal process in which the baseball bat transmits a *mark* in a continuous way (e.g., spatiotemporal continuity). He characterizes a causal process as anything that displays consistency of structure over time (Salmon 1984: 144). In this sense, he introduces the notion of a 'mark', borrowed from Reichenbach's 'mark criterion', which asserts that a process is causal if it has the capacity to transmit a local modification to a structure (see Dowe 2000: 67).

In the case of the above baseball bat example, it qualifies as a causal process because the ball transmits the scuff mark from one location to the other (e.g., by hitting a wall). The notion of a 'mark' is crucial in Salmon's account as it distinguishes a causal process from a *pseudo process*, i.e., a process that is not causal and lacks the ability to transmit marks. For instance, a pseudo process could be the shadow of a moving physical object, such as a car. Now, if we attempt to mark the shadow by modifying its shape at one point, such as altering a light source or introducing another occluding object, this modification will not obtain, unless we constantly intervene in the process to maintain it as the shadow occupies successive spatiotemporal positions. Thus, the modification will not be transmitted by the structure of the shadow itself, and so, it will not be a causal process (see Woodward 2003: 250).

The notion of causal interaction, also referred to as an 'interactive fork', involves a spatiotemporal intersection between two causal processes that modifies the structure of both (Salmon 1984: 170). In other words, each process acquires features that it would not have had if the interaction had not occurred. To illustrate Salmon's view on both causal processes and causal interactions, consider a cue ball set in motion by the impact of a cue stick. This cue ball then collides with a stationary 8 ball which, sets it in motion, and alters the cue ball's direction. During the impact, the cue stick also transmits some chalk to the cue ball, which is then transmitted to the 8 ball upon impact. In this scenario, the cue stick, the cue ball, and the 8 ball are all causal processes. However, the collisions between the cue stick and the cue ball, as well as between the cue and 8 balls, are causal interactions. According to Salmon, by citing these causal processes and causal interactions, we explain the subsequent motion of the balls post-collision. In contrast, if one of these balls casts a shadow that moves across the others, this would be explanatory but causally irrelevant because the shadow constitutes a pseudo process (see Woodward 2003: 351). Having considered this example of a causal process and causal interaction, I now proceed to illustrate an explanation perceived by Salmon.

Following the CM account, an explanation of a certain event E involves tracing the causal processes and causal interactions leading up to E, constituting the *etiological* aspect of that explanation. In other words, the etiological aspect of the explanation cites the causal processes and causal interactions that occurred before the explanandum. However, according to Salmon, there is another aspect of the explanation, that is, the *constitutive* aspect. This aspect delineates the processes and interactions that constitute the explanandum itself. The constitutive notion holds particular significance for mechanistic explanations, which is addressed in more detail in Part II (see Chapter 4).

By focusing on this constitutive aspect of an explanation, we can elucidate how E fits into the so-called *causal nexus* (Salmon 1984: 9). The causal nexus represents a type of a network of causal processes and causal interactions wherein the relevant processes and interactions explaining the explanandum can be identified (see Figure 6). In other words, in the constitutive aspect of an explanation, the causal processes and causal interactions in the causal nexus serve as the explanans of the explanandum (see Glennan 2010: 254). In the Figure 6, causal processes are depicted as arrows from left to right, and points of intersection marked as black dots signify interactions between processes. Causal processes are then changed by these causal interactions. The vertical dimension represents levels of analysis. In other words, we can analyze a specific point of intersection and discern the causal processes and causal interactions that lead to the observed point of intersection.

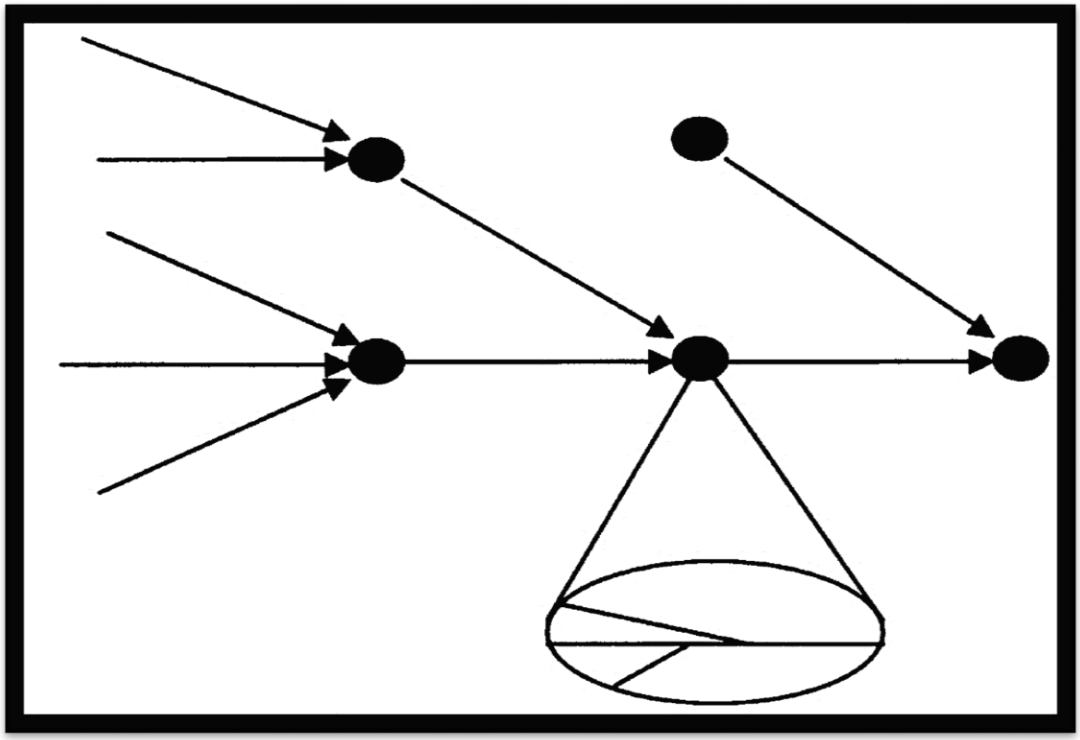

Figure 6: The causal nexus (reproduced from Glennan 2010:254)

According to Salmon, the explanation of events involves locating them within the causal nexus. However, due to the immense complexity of the causal nexus, which encompasses vast numbers of events extending indefinitely back in time, achieving a complete explanation becomes impractical. This represents one of the challenges in Salmon's account that might be addressed by acknowledging pragmatic considerations. These considerations could determine which portions of the causal nexus are actually described in response to a request for an explanation (see Glennan 2010: 254).

Another challenge to Salmon's account is raised by Christopher Hitchcock (1995), who critiques the problem of explanatory relevance. Recall the previously pool example. Now, consider this example differently and imagine a pool player who marks his cue stick with blue chalk. In this scenario, at the moment of striking the cue ball, the cue stick not only sets the cue ball in motion but also leaves a blue mark on the ball. The issue arises when we observe these two interactions, namely the force by which the cue stick moves the cue ball and the marking of the ball with chalk. According to Hitchcock, it becomes unclear which of these two interactions is relevant to the subsequent phenomenon of the interaction with the 8 ball.

Moreover, some authors have identified a circularity problem in Salmon's account (see Kitcher 1985; Sober 1987). These critiques, roughly, suggest that the definitions of a 'mark' and an 'interaction' may be mutually dependent, potentially rendering the account circular. The argument posits that the concept of a 'mark' involves the concept of an 'interaction'. In other words, a 'mark' is a type of a modification to a process introduced by a single 'interaction'. However, 'interaction' involves the concept of a 'mark', creating a potential circularity in the account. Salmon (1994: 298-299) attempts to address this concern by replacing 'interaction' with 'intersection'. However, s more in-depth discussion on this critique surpasses the scope of this chapter (for further details, see Dowe 2000: 72).

3.1.2. Dowe's Account of Physical Causation

In the preceding subsection, I examined Salmon's account of causation, highlighting some critiques of the account. It appears that a slightly different perspective of the production account, as presented by Salmon, is needed. Dowe (2000) seeks to refine Salmon's production account by advocating for an account of causal processes and interactions that adequately makes a distinction between causal and pseudo processes, namely the CQ account. Dowe modifies Salmon's account by introducing the concept of a *conserved quantity.* The core idea is that the possession of a conserved quantity, rather than the ability to transmit a mark, makes a certain process as a causal one.

According to Dowe (2000), the CQ account is articulated through two propositions. The first posits that "a *causal process* is a world line of an object possessing a conserved quantity" (Dowe 2000: 90). The second asserts that "a *causal interaction* is an intersection of world lines that involves exchange of a conserved quantity" (Dowe 2000: 90). It is evident that Dowe's CQ account shares similarities with Salmon's account, with the key distinction lying in the concept of the conserved quantity.

In Dowe's framework, an intersection refers to the overlapping in spacetime of two or more processes and occurs at the location comprising all the spacetime time points common to both (or many) processes. A conserved quantity, according to Dowe, is any quantity governed by a conservation law, such as, for instance, mass energy, linear momentum, and charge. These examples all qualify as conserved quantities. Conservation laws play a role in identifying which quantities are relevant for causation, and our current scientific knowledge and theories serve as the best guide to determining these conservation laws (see Dowe 2000: 91-92).

To return to Salmon's perspective on the issue of causation and explanation, it is important to note that he responded to criticism of his account and made revisions that are, partly, influenced by Dowe's account. In this regard, he adjusted his account and the concept of a causal process by substituting the 'capacity of a mark transmission' for the transmission of an invariant or 'conserved quantity'. Salmon (1994: 298) acknowledges that in making his revision, in which he draws significantly from Dowe's CQ account.

By examining the vocabulary used by both Salmon and Dowe, to support their accounts, it becomes evident that they are, to some extent, framing causation in the context of physics. For instance, this is particularly clear when Dowe employs the concept of a conservation law, which is notably controversial in the life sciences. I briefly address this issue in the next section of the thesis, where I explore the role of laws and functions in causal explanations in molecular biology.

Here, I wanted to emphasize Salmon's account and its variations, as expressed by Dowe, which have contributed to the ongoing development of concepts related to causation and explanation in the debate. Particularly, Glennan's position on the issue, which is explored in the following subsection, is influenced by these discussions. Glennan (2017: 174) criticizes Salmon's and Dowe's account for reducing production, i.e., causation, to one, or in some cases a few things that correspond to conserved quantities found in physics. However, Glennan argues that the new mechanistic account posits different kinds of production corresponding to different kinds of activities and interactions. While acknowledging that Salmon's and Dowe's accounts are not necessarily incorrect, especially in asserting that physical causation, to some extent, is foundational to an explanation, Glennan contends that without an account addressing the production of "higher-level" activities and interactions producing a phenomenon, we lack a comprehensive theory of causation.

3.2. Glennan's Mechanistic Account of Causal Explanation

Glennan (see 2017: 156) argues that for an adequate theory of causation, it is essential to have an account of the features in the world that make causal claims true. In this regard, he advocates for a mechanistic account of causation, which forms the basis of a mechanistic explanation.[23] According to the mechanistic account of causation, a statement of the form

[23] I would like to point out that Peter Railton (see 1978; 1980) also argued for a type of a mechanistic examination of causation. Railton introduced the concept of a mechanism for the purposes of explanation and causation. According to him, the DN model of scientific explanation (see Chapter 2, Section 1) is incomplete, and thus it is

"Event C causes event E" is deemed true only if there is a mechanism through which C contributes to the production of E. Glennan provides an illustrative example of such a statement. For instance, the statement "The heavy rain caused the basement's flooding" (see Glennan 2017: 156) is considered true only if there exists a mechanism by which the heavy rain contributes to the production of the basement's flooding.

Glennan develops this account to accommodate production accounts of causation and explanation, such as Salmon's and Dowe's accounts, with the new mechanistic account and the notion of mechanisms in the life sciences. Recall that production accounts argue that a cause is related to an effect through a series of causal processes and causal interactions. However, as I already previously mentioned, production accounts, in particular CM and CQ accounts, have fallen victim to objections of causal relevance (see Hitchcock 1995).

Moreover, another objection to the production accounts, that is, physical causation, involves causation by omission or prevention. Omission pertains to situations where the non-occurrence of certain event prevents a cause that leads (or at least allows) some effect to occur. In the case of omission, Glennan (see 2011: 797-798) illustrates this with an example of one's failing to turn off the alarm upon when one enters the house, causing the police to come. Prevention involves situations where the occurrence of some event prevents another from happening. Again, Glennan (see 2011: 797-798) provides an example where catching a vase as it topples off a shelf prevents it from breaking on the floor. The issue with objections related to omission and prevention is that either the supposed cause (in omission) or the supposed effect (in prevention) is a non-occurrence, posing a challenge for CM and CQ account. This is because there can be no set of processes linking non-occurring omissions to effects or preventive events to non-occurring effects.

Another objection for the production accounts highlighted by Glennan concerns their reductionist character. CM, and particularly CQ accounts, are production accounts that identify properties of causal connections by linking them to current physics. However, a lot of causal claims made in both ordinary and scientific discourse involve causal connections not described in the language of physics but in the languages of other disciplines, such as biology, psychology, economics, history, etc. These disciplines do not necessarily refer to the exchange

important to include mechanisms as vehicles of explanation. According to Railton (1978: 208): "Knowing enough to subsume an event under the right kind of laws is not, therefore, tantamount to knowing the how or why of it. As the explanatory inadequacies of successful practical disciplines remind us: explanations must be more than potentially-predictive inferences or law-invoking recipes". However, I am not examining his mechanistic account of causation and explanation since Glennan's account is more relevant to the new mechanistic account.

of conserved quantities, i.e., concepts drawn from physical theory. In this sense, some authors suggest there is autonomy in these higher-level causal claims from physical theory (see, e.g., Fodor 1974; Kitcher 1984). Thus, it seems that the proverb "the devil is in the details" may not be as relevant for causal and explanatory claims in the higher-level sciences, contrary to physics. In this sense, Glennan aims to accommodate production accounts for the purposes of the higher-levels sciences, such as biology, and thus develops the mechanical account of causation and explanation.

Glennan (2011) argues that the mechanical account's approach to causation is similar to the production account's approach to causation, however it avoids problems related to causal relevance. Additionally, Glennan (1996; 2011) asserts that two events are causally connected only if there is an intervening mechanism, which, as Glennan (2011: 798-799) acknowledges, sounds "very much like" the production account. However, the production account and the mechanistic account of causation and explanation are distinguished by their different conceptions of what a mechanism is. In the case of production accounts, a mechanism is simply a process of the sort described in their terms (see 3.1.1.). However, in the mechanistic account, as pointed out by Glennan, a mechanism is considered a *system*. Now, to delve into what a mechanism as a system entails, refer to Part II (see Chapter 4).

CONCLUSION. Chapter 2

To sum up, in this chapter, I focused on the concept of causation and its connection to the concept of explanation, with a specific emphasis on those accounts that are relevant to molecular biology, particularly the new mechanistic account. In this sense, I emphasized two families of accounts of causal explanation and these are as follows: (i) the counterfactual accounts, and (ii) the production accounts.

Here, I would like to highlight a common thread among the discussed accounts of causal explanation. According to the examined accounts, causation holds a more fundamental role in explanation, serving as the 'raw material' for the explanatory process (see Nathan 2020). In other words, these accounts of causal explanation assert that events are explained by identifying the causes that produce those events. All of the mentioned accounts in this chapter seek to establish a connection between causation and explanation, forming crucial frameworks for causal explanation that are particularly relevant to the special sciences, such as, among others, molecular biology. In particular, I outline Woodward's interventionist account of causal explanation, which holds that molecular biology is a discipline in which explanations provide information that can be used for manipulation and control purposes. Additionally, that intervention on causal information changes the effects, which is crucial for Part III, where I explore the causal explanation in biological practice.

Throughout the thesis, especially in Part II (see Chapter 4), I revisit and elaborate on these accounts, as they play a significant role in shaping our understanding of causation and explanation. However, I conclude Part I with Chapter 3 by examining two additional background concepts important for mechanistic conception, according to the new mechanistic account, and these concepts are law and function.

Chapter 3: Law of Nature and Function

INTRODUCTION. Chapter 3

The concepts of law of nature and function are ubiquitously used across various scientific disciplines, and the life sciences are no exception. The relationship between laws, functions, and mechanisms is particularly prominent. For instance, in genetics, Mendel's laws are used to describe regularities in inheritance. Additionally, conceptions of functions and mechanisms are frequently examined together in scientific literature (see Bartel 2004). In this chapter, I explore these two background concepts of causal explanation and examine their relationship with mechanisms, specifically within the framework of the new mechanistic account.

Firstly, I examine the concept of law of nature. As highlighted, partly, in the previous chapter, proponents of the new mechanistic account argued that mechanistic explanation surpasses Hempel and Oppenheim's DN model, which posits that an explanation requires at least one law of nature in the explanans. The mechanistic account seeks to minimize the role of laws in explanations (see Machamer, Darden and Craver 2000), or substitute explanations that rely on laws with those that appeal to the mechanistic account of causation (see Glennan 1996). New mechanists, in particular Andersen (2011), argue that a broad spectrum of scientific generalizations, some of which labeled as laws, are, in fact, explained by mechanisms (see Glennan 2017: 45). According to Andersen (2011: 325), "regularities are what laws describe and what mechanisms explain". In this sense, Glennan (2017: 45) emphasizes the example of the regularities described by Mendel's second law, specifically the law of independent assortment. This law relies on the mechanism of gamete formation. Other authors, such as Bechtel and Abrahamsen (2005), advocate for the new mechanistic account as the one that better generalizes explanations, aligning more closely with the actual research conducted by biologists.

Secondly, I explore the role of function. Here, I present two contrasting perspectives. On one side, Craver's view (see 2001; 2013) delineates the relationship between function and mechanism, specifically between functional and mechanical description. In this sense, a functional description serves as a means to integrate an item into a hierarchical nexus of mechanisms (see Craver 2013: 135). In other words, entities and activities that are parts of the mechanism (see Part II, Chapter 4) are those relevant to the function or the final product of that mechanism. Craver interprets relevance in Woodward's sense, where one can manipulate or

intervene in a part of a mechanism to change its outcome. In this sense, Craver aligns with Cummins' (see 1975; 2000) concept of a function, known as the causal role account. On the other side, Garson's view (see 2013) on the relationship between function and mechanism emphasizes that mechanisms serve functions, advocating for the functional sense of mechanism. For instance, the heart is not a part of a mechanism for circulating blood unless it has the function of circulating blood. In this sense, Garson follows a selected effects account of functions (see Millikan 1989; Garson 2017), which is in contrast with the causal role account.

The chapter is structured as follows: in Section 1, I examine the concept of a law of nature and its relation to the new mechanistic account by pointing out Andersen's (2011) and Glennan's (2017) positions, namely their assertion that generalizations are explained by mechanisms rather than laws. In Section 2, I explore the relationship between function and mechanism as articulated by Craver (2001; 2013). In this sense, I briefly outline the causal role account of function and the perspective that functional descriptions serve to integrate items into a hierarchical nexus of mechanisms. In Section 3, I examine the relationship between function and mechanism as presented by Garson (2013). Here, I provide a brief overview of the selected effects account of function and the viewpoint that mechanisms serve functions.

SECTION 1. Law of Nature and Mechanisms

New mechanists, particularly in their early work (see Machamer, Darden and Craver 2000; Bechtel and Abrahamsen 2005), have frequently draw comparisons between mechanisms and laws of nature. This comparison, developing in that early work, emerged from a growing consensus in the philosophy of biology suggesting that there may be few, if any, laws in biology (see Godfrey-Smith 2014). Roughly, the generalizations in biology often come with *ceteris paribus* clauses, namely they depend on background conditions that might not hold in certain circumstances. In other words, unlike laws in physics and chemistry, the so-called *ceteris paribus* laws in biology, are not universal and frequently have exceptions.[24] Thus, mechanisms, arguably, substituted for the role of laws in biology (see Craver and Tabery 2019).

According to Andersen (2011: 329), the issue with laws of nature, conceived as universal, exceptionless, and necessary, is that they perhaps do not adequately describe explanations provided by sciences such as, among others, biology, neuroscience, and psychology, contrary to sciences such as physics and chemistry. Thus, new mechanists argue that laws alone are insufficient for explanation, and both mechanisms and laws are needed in explanations. Moreover, that perspective is evident in the fact that new mechanists explicitly argue that mechanistic explanations are superior to those explanations proposed by Hempel and Oppenheim (see 1948; Chapter 2), i.e., explanations offered by the DN model that emphasizes the role of laws in the explanans. However, the new mechanistic account does not deny the existence of laws; instead, it argues that many generalizations, particularly in biology, are actually explained by mechanisms rather than laws (Glennan 2017: 44-45).

In this sense, Andersen (2011: 325) argues that "regularities are what laws describe and what mechanisms explain".[25] In her paper, she presents a counterargument to Bert Leuridan (2010). Leuridan (2010) asserts that mechanisms are not an alternative to laws of nature as models of explanation in the sciences. Thus, he advocates for Sandra Mitchell's (see 2000) pragmatic account of laws of nature. In other words, Leuridan claims that models of mechanisms in sciences depend on regularities and, consequently, on the pragmatic account of

[24] For instance, Mendel's laws do have certain exceptions, unlike Newton's or Einstein's laws (for more information on laws of nature, see Carroll 2020).

[25] Here, I would like to briefly touch upon the broader debate on laws of nature and the positions of Andersen (2011) and Glennan (2017) within that framework. Laws are sometimes conceptualized as things in nature and, on one side regularities of certain kinds, and on the other side, some sort of dependency in the world that accounts for regularities. Furthermore, laws are considered as generalizations, i.e., statements that describe those regularities. Andersen (2011) and Glennan (2017) advocate for the approach that considers laws as generalizations, asserting that laws describe regularities that are the behaviors of mechanisms (see Glennan 2017: 45).

laws of nature. Mitchell's (2000) pragmatic account of laws accommodates for the critique that the existing criteria for lawfulness are too strict (see Nagel 1961; Hempel 1965). She aims to refine the concept of a law of nature by asserting that generalizations qualify as laws if they can be employed for purposes of prediction, explanation, or manipulation. Since these generalizations may not always be considered universal, particularly in the strict sense of lawfulness, it is crucial to discern the specific contexts in which these pragmatic laws hold. Although Mitchell's account and Leuridan's perspective are, to some extent, related to mechanisms, for the purposes of this section, I focus on Andersen's view since she engages more comprehensively with the new mechanistic approach to causal explanation.

Andersen believes that mechanisms are not an alternative to regularities but rather an alternative to laws of nature. She argues for this view by referring to Machamer, Darden and Craver's (2000) paper, which characterizes mechanisms as explanatory structures that display regularities (see also Part II, Chapter 4). For instance, mechanisms explain phenomena that regularly occur under specific circumstances because their activities and entities are regularly linked and regularly produce outcomes. In this sense, Andersen (2011: 327) claims that "mechanisms are not supposed to be alternative to regularities; they are an alternative to laws as an explanation of regularities". In other words, if a regularity is an explanandum, then a mechanism would serve as an explanans.

Andersen (2011: 328) proceeds to emphasize that regularities are integral to both law-based and mechanism-based accounts when attempting to explain phenomena. According to her, the presence of stable regularities in nature is crucial; without them, there would be no need for either mechanisms or laws. She contends that stable regularities serve as a starting point for scientific inquiry, and mechanisms offer avenues for researching these regularities.

To illustrate Andersen's perspective on the relationship between regularities, laws, and mechanism, Glennan (2017) uses Mendel's law as an example. Mendel's second law, known as the law of independent assortment, describes the regularity as follows: in a diploid organism, "the probability of transmitting a particular allele at the first locus is independent of what occurs at the second locus" (Glennan 2017: 45). For example, in an organism with genotype AaBb, the probability of transmitting the dominant alleles (A and B) is independent, namely P(AB) = P(A) = P(B). Combining this with Mendel's segregation law, "which states that the probability of transmitting each allele from a given locus is equal", results in the following regularity: "P(AB) = P(Ab) = P(aB) = p(ab) = .25" (Glennan 2017: 45).

Now, the regularities described by Mendel's second law are, in fact, dependent on the mechanism of gamete formation. This mechanism involves genes located on paired

chromosomes, each with two alleles at a single locus. In gamete formation, half of the genetic material is copied to each of the two gametes. However, Mendel's laws are not strict, in the sense that they are exceptionless. The regularities described by these laws have many exceptions. For instance, "linked genes do not assort independently, violating the second law" (Glennan 2017: 45). Invoking mechanisms as an alternative to laws, new mechanists characterize regularities in a way that these regularities hold only as long as "the mechanism does not break down" (Glennan 2017: 46). Thus, in the example of Mendel's second law, the law holds true only "in the absence of segregation distorters and for non-linked loci" (Glennan 2017: 46). According to Glennan, the new mechanistic approach to laws is deflationary, as there are various laws, and all of them have restricted scope.

There is another new mechanistic perspective on the relationship between laws and mechanisms, presented by Bechtel and Abrahamsen (2005). According to them, a crucial aspect of scientific explanations is the ability to generalize. In this sense, laws are considered as suitable candidates since they are often represented in universal antecedent statements, i.e., meaning that generalizations are automatic as they apply to every situation where the antecedent of the conditional is satisfied. To illustrate this point, Bechtel and Abrahamsen (2005: 437) provide an example of Newton's first law of motion, stating that the momentum of a body will remain the same if there is no force acting on it. However, mechanistic explanations appear to operate differently. It seems that these kinds of explanations are context-dependent. To illustrate this, Bechtel and Abrahamsen offer an example of the distinction between the mechanism responsible for oxidative phosphorylation in liver cells versus heart cells found in cow.

To accommodate that kind of context dependency found in the mechanistic explanations, Bechtel and Abrahamsen consider discussions about concepts and categorization. In this sense, they emphasize that, traditionally, most philosophers and psychologists, up until the early 1970s, construed concepts with necessary and sufficient conditions. However, Ludwig Wittgenstein (1953) argued against this idea, contending that it was impossible to provide a definition even for the concept of a game because instances of a concept may not share any distinctive common properties; they may simply resemble each other, similarly to how family members resemble each other.

In addition to Wittgenstein's perspective, psychologist Eleanor Rosch (1978) argues for an empirical basis for Wittgenstein's idea. She suggests that one can rate the typicality of instances by appealing to a category. For example, in the category of birds, robins are highly

typical, and chickens are atypical exemplars. Thus, membership in a category is a matter of degree. In other words, an item is an exemplar of a category if it is similar to the prototype.

The prototype and exemplary accounts mentioned above, as outlined by Bechtel and Abrahamsen, offer a way to approach the challenge of generalizing mechanistic explanations. In this sense, different mechanisms can share similar relations to each other without being exactly the same. To illustrate this point, they provide the canonical example of protein synthesis, where the mechanism may exhibit similarities in different organisms or even in different cell types within the same organism without being the same.[26]

Following this discussion, Bechtel and Abrahamsen argue that the mechanistic account offers a superior characterization of how explanations are generalized, as scientists frequently focus on a specific exemplar in their investigations, particularly in the initial stages of developing their accounts. In the life sciences, those exemplars are called *model systems*. For example, molecular biologists, in the development of the mechanism of protein synthesis, often preferred to work with bacteria and bacteriophages (see Balorda 2023).

After utilizing a model system in the initial stages of their investigations, biologists then need to experimentally determine the level of generalization of their accounts by examining the counterpart mechanism in other organs and species. This is where the difference between the DN model, in which the conditions of application of a law depend on refining its antecedent, and variations found in the mechanism itself becomes apparent. For instance, two variations on the same mechanism can be identified. This variation might be in some minor part of a mechanism that has an impact on the working of the entire mechanism. Such findings are common in the life sciences, and it seems that the mechanistic framework accommodates these types of explanations better than an account based on explanations provided via laws (see Bechtel and Abrahamsen 2005: 439).

In this section, I explored various perspectives on the relationship between laws and mechanisms. In general, new mechanists have argued that mechanistic explanations better suit the life sciences than Hempel and Oppenheim's DN model of explanation, particularly because of the greater level of flexibility for explanations, provided by the concept of a mechanism. Some new mechanistic authors attempt to diminish the role of laws in explanations (see Machamer, Darden and Craver 2000), while others aim to replace explanations based on laws

[26] Bechtel and Abrahamsen (2005: 438) highlight that appeals to similarity have been criticized in the past (see Goodman 1955). However, they note that scientists continue to make judgements about similarity without always specifying precise dimensions and metrics, which is a practice that is arguably important when appealing to similarity.

with those appealing to the mechanistic account of causation (see Glennan 1996). Andersen (2011) asserts that a great variety of scientific generalizations, often labeled as laws, are, in fact, explained by mechanisms. She contends that regularities are what laws describe, but mechanisms are what actually explain.

Finally, Bechtel and Abrahamsen (2005) advocate for the new mechanistic account as better suited to the life sciences, as it aligns with the actual research conducted by biologists, particularly relying on model systems that are especially important in the development of scientific accounts. Although I have attempted to encompass a comprehensive range of the discussion on this topic, it appears that, in general, new mechanists have not extensively addressed the connection between explanations via laws and explanations via mechanisms. It seems that there is still considerable room for investigation into whether there is a stronger connection between the two. However, the limited attention to this connection may stem from the new mechanists' focus on characterizing actual practices in life sciences, which diverge from the reliance on laws as it is evident in the field of physics. Thus, they have moved beyond employing models such as the DN model, which is not as suitable for the life sciences as it is for physics.

SECTION 2. Causal Role Account of Function and Mechanism

In this section, I examine the causal role account of function and its relationship to the new mechanistic account. Firstly, I characterize the causal role account and provide a brief overview of its historical development. Then, I present Craver's (2001; 2013) perspective on the connection between the causal role account of function and the new mechanistic account.

The causal role account of function asserts that the function of a part within a system depends on its contribution to the overall system-level effect, specifically an effect that is of particular interest to scientists. For instance, the function of the heart is to pump blood because without the circulation of blood our organism would cease to exist. In other words, the function of the heart, i.e., to pump blood, contributes to the survival of the organism, that is, a system. This account aligns with the notion that biologists attribute functions to items by demonstrating how these items contribute to a larger system (see Garson 2016: 81). Again, consider the heart as a system comprised of specific parts, such as four chambers, that is, upper left and right atria and lower left and right ventricles. The heart is also enclosed in a protective sac. Each of these parts plays a role in the larger system, i.e., the heart, thus having a certain function. Consequently, the function of the heart is to pump blood, sustaining the larger system, i.e., the organism.

However, besides the heart's primary function of pumping blood, scientists may identify additional functions of the heart that contribute to some system-level effects. For instance, the heart also produces sound as its valves close during the pumping process. This sound could potentially contribute to detection of certain diseases or malfunctions of the heart. Hence, one could argue that the heart's ability to produce a sound is another function of the heart. To put it in a formula, a function X contributes to a system S while performing a process Y.

The causal role account belongs to the family of *dispositional* or *forward-looking* accounts.[27] These accounts are opposed to the selected effects accounts positioned within the family of *etiological* or *backward-looking* accounts (which I cover in more detail in the subsequent section). The philosophical framework underpinning the dispositional or forward-looking family of accounts encompasses at least two criteria to identify what counts as a target capacity within a functional relationship. From these criteria, relevant norms can be deduced.

[27] Prominent proponents of such accounts include Cummins (1975; 2002), Christopher Boorse (2002), and John Bigelow and Robert Pargetter (1987), among others. In addition to the causal role account, I also included proponents of the fitness-contribution accounts, which are arguably forward-looking accounts (for more information on the fitness-contribution accounts, see Garson 2016: 67).

In this sense, each of the accounts in the dispositional family approaches the criteria differently. The first criterion, namely the *naturalized* criterion, argues that to identify what effect qualifies as a function, it should be grounded in certain constitutive features of the system. The second criterion, namely the *appropriate* criterion, posits that to identify what effect qualifies as a function, it should conform to scientific practice (see Mossio, Saborido, and Moreno 2009: 816).

In this section, I examine the causal role account of function due to its close association with the new mechanistic agenda, particularly through the contributions of Craver (see Craver 2001; 2013). However, the first proponent of the causal role account was Cummins (1975).[28] Cummins initiates his account by critiquing the work of Ernest Nagel (1961) and Hempel (1965) concerning functional analysis and explanation. He provides his own characterization of the issue, prioritizing the biologists' perspective. Cummins contends that biologists occasionally attribute functions to traits without necessarily considering the benefits to the species. For instance, he emphasizes that flight, as a capacity, requires an explanation based on its anatomical functions, irrespective of its impact on species survival (see Cummins 1975: 756). Here, it is evident that Cummins' notion of a function does not require the attribution of a function to species history, unlike the selected effects account, which is examined in the next section. The causal role account of function characterizes functions in terms of their contribution to the system they are a part of. Thus, to use the example with the heart again, according to Cummins, stating that the function of the heart is to pump blood is not an attempt to explain the heart's existence but to explain blood circulation and its significance for the system, i.e., the organism (see Garson 2016: 82).

Cummins distinguishes between two types of explanation. The first is the *subsumption* aspect of an explanation. This aspect is akin to Hempel and Oppenheim's (1948) DN model (see Chapter 2). For instance, when explaining why an item X exhibits a disposition Y, one does so by subsuming X under a certain physical law, i.e., a law of physics or chemistry. The second, and more important for this section, is the *functional* aspect of an explanation. A prime example of a functional aspect is the previously mentioned function of the heart. The structure of the functional aspect of an explanation is the following one: first, a scientist selects some capacity of a certain system (e.g., the heart's capacity to circulate blood). Next, the scientist decomposes that system into various sub-capacities (e.g., different parts of heart with their own

[28] It is worth noting that Cummins' causal role account has been refined and examined by numerous authors, including Elizabeth Prior (1985), Craver (2001), Predrag Šustar (2007), and Šustar and Zdenka Brzović (2014), among others.

capacities). Finally, the scientist demonstrates how those sub-capacities are organized to collectively produce the overall capacity of the entire system. Thus, the function of a given item is characterized by the role it plays within a functional explanation (see Garson 2016: 83).

However, Cummins' account faces a challenge when a specific system plays a role in multiple different systems, as seen with the example of the heart's role in the organism. In this scenario, the heart serves more than one function. Beyond its function of circulating blood, the heart contributes to diagnostics by producing sounds that doctors can interpret as signs of heart malfunction. Additionally, the heart plays a role in polygraph tests by indicating whether someone is lying through spikes in blood pressure. Thus, it seems that Cummins' account is overly liberal in the sense that it becomes challenging to pinpoint a single, most important function for a given item. For instance, asserting that the sole function of the heart is its capacity to pump blood. In this sense, Cummins acknowledges this issue and emphasizes the idea that functions depend on one's own interests and perspectives. Thus, functions are always subject to a specific viewpoint, shaped by the focus of a particular scientific inquiry (see Cummins 1975: 762).

Now, let me move to the further development of Cummins' causal role account, a particularly important aspect for the new mechanistic account. Craver (2001; 2013) aims to further refine Cummins' framework within the new mechanistic perspective. Specifically, by clearly examining some key elements in Cummins' account, such as organization, complexity, and capacity. Moreover, Craver's contribution extends to adapting Cummins' terminology to align with the new mechanistic framework. In this Craver's updated framework, a system is a mechanism, comprising components, namely, parts and activities (Garson 2016: 84).

Moreover, Craver (2013) advocates for a perspectivalist view on functional analysis. This perspective posits that functions are dependent upon observers' decision regarding what aspects matters or are of interest in the system within their respective fields of study. Let me first examine Craver's (2001) position, and later address his perspectival approach to functions and its relationship to mechanisms.

Craver (2001) asserts that the discovery of mechanisms, particularly developing by discovering the role functions of their components, proves beneficial for various scientific disciplines. In this regard, Craver aligns with Cummins' account and his examination of understanding capacities of various systems by analyzing them through the capacities of their components. Craver aims to synthesize Cummins' causal role account with the new mechanistic agenda. Craver contends that this synthesis holds significant benefits for science. Specifically, he argues that the discovery of a mechanistic role of a component serves as a

means of integrating that mechanism into a multilevel mechanism/system. Consequently, this integration into a higher-level mechanism results in a "*contextual* variety of causal/mechanical explanations" (Craver 2001: 54).

According to Cummins (1975), the structure of function-ascribing sentences are as follows:

> X functions as a φ in S (or the function of X in S is to φ) relative to an analytic account A of S's capacity to ψ just in case X is capable of φ-ing in S and A appropriately and adequately accounts for S's capacity to ψ by, in part, appealing to the capacity of X to φ in S. Cummins (1975: 762)

Now, here, S is a system that has a capacity to ψ. X is a component of S that has the capacity to φ. To illustrate this structure, consider, again, the example of the heart. The heart X functions as a blood pump φ in the circulatory system S relative to an analytic account A of the circulatory system's S' capacity to deliver oxygen and calories to body tissues ψ. This holds true only if the heart X is capable of pumping blood φ-ing in the circulatory system S, and the analytic account A adequately accounts for the circulatory system's S ability to deliver oxygen and calories to body tissues ψ by appealing, in part, to the heart's X capacity to pump blood φ in the circulatory system S (see Craver 2001: 55).

Craver initially clarifies Cummins' stance on the structure of function-ascribing sentences using the example of the heart's function. However, it is important to note that A, i.e., the analytic account, is an explanation. This explanation operates by analyzing the capacities of the system into the capacities of their component parts. For instance, when explaining the circulatory system's S' capacity to deliver oxygen and calories to body tissues ψ, the analytic account A explains that by analyzing S' component parts (Xs) and capacities (φs) relevant to S' capacity to ψ. Thus, to explain S' capacity ψ one has to decompose the system S into its parts Xs. In the case of the heart, decompose it to parts, such as arteries and valves, and capacities φs, that is, to pump, to convey, to filter, and connecting these parts and capacities together to demonstrate the system S' capacity ψ (see Figure 7). This process aligns with the decomposition of the system into its constituent parts to provide a comprehensive analysis of its functional capabilities.

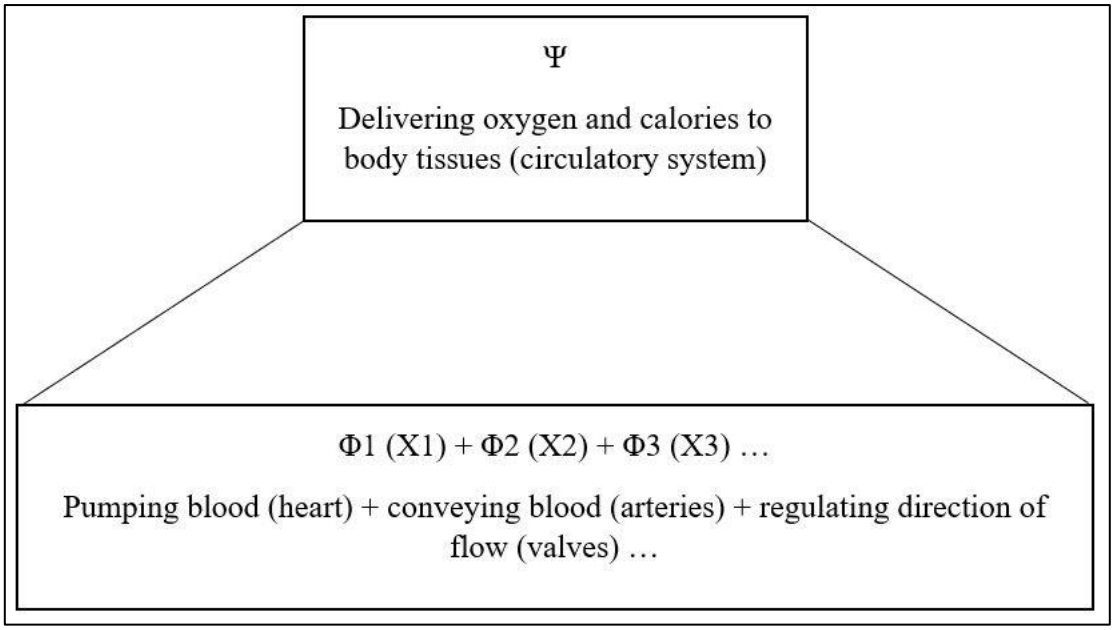

Figure 7: Illustration of Cummins' functional description (reproduced from Craver 2001: 56)

However, Cummins highlights that "not all analytic explanations are interesting" (Craver 2001: 56), and he introduces three criteria to evaluate these explanations. Firstly, the analyzing capacities (φs) should be "less sophisticated" (Craver 2001: 56) than the analyzed capacity (ψ). Secondly, the analyzed capacities (φs) should be "different in type" (Craver 2001: 56) from the analyzed capacity (ψ). Lastly, the analyzing components (Xs) and (φs) should exhibit a complex organization such that they work together to ψ (see Cummins 1975: 764). Although Cummins laid out these criteria, Craver (2001: 57) contends that Cummins "hasn't said enough" about what systems are or how analyzing components are organized to distinguish between interesting and uninteresting explanations. Craver aims to synthesize the new mechanistic account and its terminology with Cummins' perspective on explanations. In this sense, he attempts to enrich Cummins' ideas by incorporating insights from the new mechanistic account.

Although there are various types of systems, including formal, procedural, and representational, Craver emphasizes one particular type of systems, namely, mechanisms. This focus aims to bring a certain precision to the idea of a mechanistic role function by characterizing mechanisms, especially their specific organization. I examine mechanisms in Part II; however, for now, it is important to keep in mind that one of the characterizations of mechanisms is that they consist of entities and activities organized to produce a certain phenomenon. According to Craver, the entities in this characterization correspond to Cummins' component parts (X) and they are the physical parts of the mechanism, such as the heart and

the veins. Activities, on the other hand, are the actions that entities do and align with Cummins' capacities (φ). Thus, the description of a mechanism involves characterization of how entities (Xs) and activities (φs) are organized to produce a phenomenon (ψ).

Craver synthesizes Cummins's causal role view with the new mechanistic agenda by examining the concept of organization. This synthesis involves distinguishing between *spatial* and *temporal* organization.[29] Craver argues that one understands mechanisms only if one discovers its component's entities and activities and by learning how their activities are spatially and temporally organized. Consequently, aspects of this kind of organization are crucial for evaluating ascriptions of mechanistic role functions (see Craver 2001: 60).

According to Craver (2021: 60), the spatial organization of mechanisms includes characterizing entities in terms of their sizes, shapes, orientations, and locations, permitting them to engage into their activities effectively. For instance, the size of the heart is suitably related to the size of aorta, and the heart's shape facilitates its function of pumping blood through its auricles and ventricles. Temporal organization, on the other hand, includes characterizing activities in terms of their order, rate, and duration. For instance, blood enters the heart through the vena cava, and it returns through the pulmonary vein. There are specific sequences of stages, and the order of these stages cannot be changed without affecting the heart's activities. Each stage has characteristic durations and rates crucial for its proper working. According to Craver, to assert that a mechanism is organized means emphasizing that its components have active both spatial and temporal relations to one another, resulting in a production of an outcome. Thus, contrary to Cummins' analytic account, Craver argues that an analytic account for a mechanism goes beyond merely listing entities and activities; rather, it entails describing how they are organized to produce a particular outcome.

The examination of the notion of organization in mechanisms is closely related to functions. To specify a certain mechanistic role for a certain component X, one has to describe how X is organized in relation to other components and, consequently, how they collectively contribute to producing an outcome. In summary, Craver's examination suggest that attributions of mechanistic role functions describe an item in terms of, on one side, entities, and activities contributing to the working of a mechanism, and on the other side, the mechanistic organization through which it makes that contribution (see Craver 2001: 61).

[29] This distinction is of particular interest for Part II, Chapter 6, where I examine the relationship between various causal explanatory structures, such as mechanisms, pathway, and cascades. Morevoer, I consider this distinction in Part III, in Chapters 7 and 8.

I emphasize another point made by Craver regarding the connection between the causal role account and the new mechanistic agenda. Craver (2001; 2013) explores the relationship between functions and mechanisms by demonstrating how functional descriptions contribute to the search for mechanisms, and he terms his view as a *perspectivalist* view. Basically, Craver (2013: 143) argues that some biologists, depending on their interest, may focus on various aspects, such as cognition, social bonding, or ecological systems. Their interest might also extend to the mechanistic arrangement and functions contributing to the fitness of a certain organism. The differences in these perspectives are rooted in the diverse interests of biologists. Before examining into details hi perspectivalist view, I point out Craver's stance on explanation and causation, which is important for understanding his viewpoint.

Craver (2013: 144) aligns with Salmon's production account, specifically the CM account of explanation (see Chapter 2, Section 2). In a brief recap of this perspective on explanation, to explain a phenomenon involves situating it within the causal nexus. However, it is important to note that Craver does not fully endorse Salmon's and Dowe's views on causation. To recall their perspectives again, briefly, the causal nexus consists of causal processes interacting with each other and exchanging conserved quantities. Craver acknowledges the value of this view and its alignment with embodying the statements of early mechanical philosophy (see Chapter 1, Section 1). However, he deems it less than ideal when applied to sciences, particularly the life sciences. For instance, the description of the mechanism of transcription of DNA is too complex to describe it in terms of simple and "tidy" causal processes proposed by Salmon and Dowe. Thus, Craver suggests adopting a different account when considering causation in life sciences, i.e., one that provides more flexibility in understanding the causal nexus. He turns to Woodward's interventionist account (see Chapter 2, Section 3). Following Woodward's account, causation is understood in terms of the relationship between activities, where the value of one variable depends upon the value of another. So, intervening to change the value of the second variable allows one to alter the value of the first variable. Thus, the causal nexus perceived through Woodward's notion of causation, is represented as a set of variables related by generalization that remain stable, i.e., invariant when one intervenes to change the value of a certain variable in the generalization (see Craver 2013: 144).

Furthermore, Craver contends that Woodward's account of causation aligns with actual scientific practices, particularly when conducting experiments to test causal claims. He also maintains that this perspective on causation remains consistent with Salmon's overall vision, emphasizing that, to explain a phenomenon, one has to situate it in the causal structure of the

world. It is crucial to note that Salmon distinguishes between two aspects of explanation, namely the *etiological* and the *constitutive* aspect. In the etiological aspect, an explanation involves tracing antecedent causes that precede a phenomenon. On the other hand, the constitutive aspect involves explaining a phenomenon by revealing its internal causal structure.

Building upon these two aspects of explanation, Craver develops his perspectivalist view on explanation and its relation to functions. In Figure 8, Craver outlines three explanatory perspectives, aiming to move beyond Salmon's dualistic model of explanatory perspective. These three explanatory perspectives are as follows: *etiological*, *constitutive*, and *contextual*.

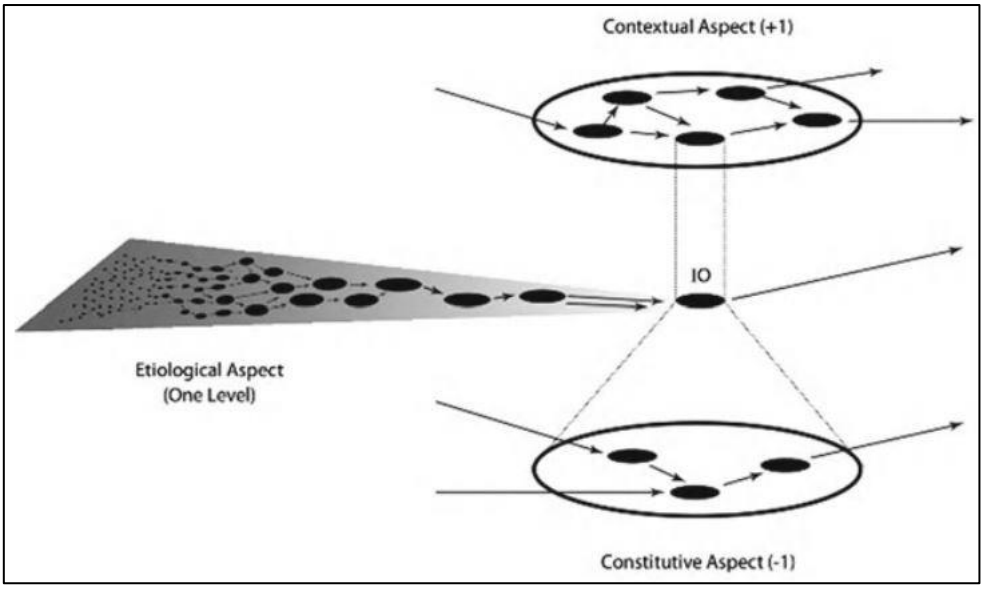

Figure 8: Three explanatory perspectives proposed by Craver (reproduced from Craver 2013: 155)

Within the figure, a complex function[30] is positioned in the center. On the left side, we find the past portion of the causal nexus, while the right side represents the future portion of the causal nexus. Following each type of the explanation, explanandum E is some aspect of the complex function in the center, either an etiological, constitutive, or contextual aspect.

Etiological explanation, illustrated on the left side in Figure 8, involves tracing the pathway of entities and activities, finishing in E, and explaining how E came to pass. Etiological explanations are aiming to answer questions regarding the origins of some item, i.e., its historical trajectory. These kinds of explanations are also termed *backward-looking*,

[30] In the figure, as well as in his paper, Craver (2013: 149) characterizes a function as a mapping from inputs to outputs in accordance with a rule. Specifically, he refers to IO (input-output) functions, which Cummins describe as "capacities" (Cummins 1983: 53). However, for the purposes of this section, where the focus is on the connection between mechanisms and functions, I omit the IO aspect and focus solely on the „function" part.

and I further examine them in the next section. Here, it is important to note that Craver specifically links etiological explanation to adaptational explanation, considering it as one type within the etiological explanation. In exploring the concept of functions as adaptation, Craver references Larry Wright's (1973: 161) characterization of a function where the function of X being Z means that: (i) X is present because it performs Z, and (ii) Z is a consequence (or result) of X being there. One of the biological interpretations[31] of how (i) comes to be is the interpretation that natural selection is a process[32] in which a certain effect, i.e., a heritable trait X, by virtue of performing Z, increases the likelihood of the organism bearing X to survive and reproduce. Thus, in turn, leads to an increased prevalence of trait X in a population. The advantage of linking biological functions to adaptations lies in aligning with the intuition that a trait's function explains its presence. Explanations utilizing natural selection as an explanandum necessitate an understanding of genes, organs, organisms, populations, and ecosystems. They are particularly suited for addressing the question how a certain explanandum E came to be.

Constitutive explanation aims to explain how an explanandum E works and is considered *downward-looking*. In this sense, it situates E within the lower portion of the causal nexus, or as Craver points out, the lower level in the hierarchy of mechanisms, which I examine in more detail in the next section. By looking into this lower level, one can analyze different entities and activities comprising a certain system, i.e., mechanism. While complex functions, such as the IO function in Figure 8, are useful for describing E without looking into its details, they also provide a framework for investigating E's mechanistic arrangement and, consequently, the mechanistic explanation. Hence, the significance of functions is also present in constitutive explanations that I explore further in Part II.

Contextual explanations are *upward-looking,* and they are the ones that Craver emphasizes and with whom he advances Salmon's depiction of explanations. These explanations position E in respect to the portion of the causal nexus in a higher level of hierarchy of mechanisms. It is important to note that, according to Craver (2013: 155), citing E's role function is explanatory in this type of explanation. Contextual explanations not only seek to address the question of how E came to be but also provide insights into how E is related to other entities and activities situated in higher-level mechanisms.

[31] Various interpretations surround part (i) of Wright's characterization, and I examine some of these interpretations in the next section.

[32] Characterization of natural selection is a distinct debate. In Part III, Chapter 8, I examine whether natural selection can be characterized as a causal structure, such as a mechanism or a pathway.

To sum up this section, it is important to know that, according to Craver, functional and mechanistic descriptions collaboratively contribute to bringing understandable order to complex systems. When identifying functions within a certain system, one can approach the system with a specific set of interests and perspectives in mind. These perspectives might involve understanding how an organism works, how a certain organ operates, or how a specific system may break or malfunction, which I asses in the next section. However, the identification of functions is a pivotal step in the discovery of mechanism, regardless of the chosen perspective.

SECTION 3. Selected Effects Account of Function and Mechanism

In this section, I examine the connection between the selected effects account of function and the new mechanistic account. Firstly, I characterize the selected effects account. Then, I briefly map the history of the selected effect account and highlight some of the criticism directed towards it. Finally, I examine Garson's (2013) perspective on the relationship between function and mechanisms.

The selected effects account of function posits that a function of a trait is determined by whatever it was selected for through natural selection or some other selection process. The motivation behind this account is rooted in the idea that functions, at least in some situations, mean to be explanatory. For instance, when stating the function of an item, the intention is to explain why that item exists. In this sense, this aligns with a type of a causal explanation, wherein the aim is to address questions regarding the item's presence. To support this perspective, Garson (see 2016: 34), cites an example by Tim Caro et al. (2014) in which a team of biologists aimed to answer the question of why zebras have stripes. They assert that zebra stripes have the function to deter biting flies. They argued so because of five different functional hypotheses which suggested that, when stating the function of something, a causal explanation for its existence is inherently provided. Another important aspect is that Caro and colleagues also provided historical data that support their claims, not solely emphasizing the current benefits of stripes for zebras but also delving into the etiological pathway that causally explains the origin of zebra stripes (see Garson 2016: 34).

However, a challenge arises after considering the function of zebras' stripes: "How can an effect of a trait explain its own existence?" (Garson 2016: 35). Certainly, for a zebra to deter flies, it must already possess stripes. The answer to this challenge is found in the concept of natural selection. Zebras have stripes because, in the past, there existed a population of ancestors of modern zebras, some with stripes and others without. Those zebras with stripes held an advantage over their non-striped counterparts, leading the trait, i.e., stripes, to endure in the population.

Expanding on the example of zebra stripes, let me emphasize some key aspect of the selected effects account. *Firstly*, this account aims to distinguish between a function and an accident. For instance, in the case of zebras, the stripes serve a function, i.e., to deter flies, rather than being a result of aesthetic appeal. *Secondly*, the reliance on natural selection when assigning a function to a certain trait entails providing a causal explanation for the existence of that trait. This approach, often referred to as the etiological account of functions, allows one to

trace the causal history of the trait.[33] *Thirdly*, the selected effects account introduces a normative dimension by illustrating that a particular trait token can *malfunction*, i.e., meaning it has a function that fails to perform. This emphasis on the trait's causal history, rather than its current abilities within a specific system, namely an organism in the case of zebra's stripes, sets the selected effects account apart the other accounts of function, such as the previously examined causal role account (see Garson 2016: 35).

Another important aspect of the selected effects account is its teleological aspect. According to Garson (2016: 36): "*a teleological explanation is one that purport to explain the existence of an entity (such as an organism, trait, or behavior), in terms of some effect the entity brings about*". Garson contends that the selected effects account provides *teleological explanations*. For instance, explaining the current existence of zebra stripes by stating that they were selected in the past for deterring flies constitutes citing an effect produced by the stripes as part of an explanation for their existence, that is, an inherently teleological explanation. To further reinforce this perspective, Francisco Ayala (1970) and Wimsatt (1972b) emphasize that "if functional statements are selectionist explanations, and selectionist explanations are teleological explanations, then function statements are teleological explanations, too" (Garson 2016: 36). However, whether something is a teleological explanation is subject to another debate, thus I do not examine it further, as it lies outside the scope of this thesis.

The selected effects account developed in the latter half of the 20th century, initially spurred by observations made by George Williams (1966) regarding the relationship between function, particularly its purpose and goal, and selection. The systematic examination of the relationship between function and selection gained momentum with the contributions of Ayala (1970) and Wimsatt (1972b). However, I particularly underscore Wright's (1973) development of the etiological account, briefly discussed in the previous section. Although Wright acknowledged the link between function and natural selection, he attempted to avoid defining function solely in terms of it. His aim was to create a unified framework for the analysis of both biological and artificial functions, thus rejecting the notion that artificial functions undergo a selection process. For example, the function of a hammer's claw is to pull out nails. However, that function is not present now because once there existed a population of hammers with and without claws undergoing a selection process, rather because of its designed purposes. Wright (1973) articulates his definition of function as follows:

[33] Recall that Craver (2013) also emphasizes the significance of the etiological aspect of an explanation and its connection to the functional role of a mechanism, as discussed in the previous section.

The function of X is Z means that:

a) X is there because it does Z,

b) Z is a consequence (or result) of X's being there. Wright (1973: 161)

In the life sciences, (a) point, in the previous Wright's definition, holds true when X was selected for Z.

However, Wright's definition of functions faced substantial criticism, thus another refined account emerged that now clearly defines functions in terms of selection, and the now formed selected effects account of function supplanted Wright's etiological characterization. Both Karen Neander (1983; 1991) and Ruth Millikan (1984; 1989) concluded that, to define biological functions, one has to appeal to natural selection. Although Neander and Millikan agree on the fundamentals of the selected effects account, they differ in some details regarding the account. Neander characterizes her account through "a conceptual analysis of modern biological usage" of the term 'function', whereas Millikan characterizes it "as a theoretical definition" (see Garson 2016: 41). However, their accounts are not examined in detail here since they do not establish a direct connection between mechanisms and functions. In subsequent years, the selected effects account continued to develop, giving rise to several new variations. Among these, David Buller's (1998) weak etiological account, Brzović and Šustar's (2020) refined version of the weak etiological account, and Garson's (2017) generalized selected effects account are notable examples.

Here, I highlight some of the major criticism directed at the selected effects account, particularly those articulated by Cummins (1975; 2002), as discussed in the previous section, and Paul Davies (2001). This is done to underscore the distinctions between the previously examined causal role function and the selected effects account.

Firstly, Cummins (1975) argued that selected effects functions are not truly explanatory because proponents of the selected effects account misinterpret what natural selection actually explains. Cummins asserts that natural selection does not explain why a specific individual possesses a particular trait, for instance, a zebra having stripes. Instead, he contends that it explains the current frequency of that trait in a population. Thus, according to Cummins, natural selection can explain why most zebras have stripes, but it cannot explain why a particular zebra has stripes. The ongoing debate centers on whether this criticism is valid, or if proponents of

selected effects have a satisfactory response to this issue, although, this is not the primary focus of this thesis.[34]

Secondly, according to Garson (2016: 47), Cummins (2002) presents another critique of the selected effects account, echoing the first one, as it questions the understanding of how natural selection works. For instance, consider a functional statement like "the function of the eye is to see". According to the selected effects account, this statement holds true if, in the past, there was a population of organisms with variations for having eyes, and those with eyes were selected over those without. However, Cummins contends that natural selection does not operate in this manner. Instead, he argues that natural selection favors slight improvements in functionality and does not involve a direct competition between organisms possessing a trait and those entirely lacking the same trait. Further counterarguments to this critique are not explored here (for more information, see Garson 2016: 47).

Davies (2001) attempts to criticize the selected effects account by arguing that it is redundant. He posits that the selected effects account is merely a subset of the causal role account. To illustrate his position, consider again the function of zebra's stripes, namely the function of deterring flies. If stripes have the function of deterring flies, then in the past, they must have had a causal role function of deterring flies. However, according to Garson (2016: 49), some argue that the selected effects account is not solely a subset of the causal role account because it possesses *normativity*. In other words, it accounts for the fact that a certain trait can *malfunction*. For instance, according to the selected effects account, a trait can malfunction as follows: consider a heart with the condition called tricuspid atresia, where the heart has only three chambers. Proponents of the selected effects account would assert that the heart has the function of pumping blood, but it cannot do so effectively, indicating that the heart is malfunctioning. On the other hand, proponents of the causal role account struggle to address the concept of malfunctioning, contending that the three-chambered heart does not have the function of pumping blood.

3.1. The Functional Sense of Mechanism

After the examination of the selected effects account and the briefly addressed criticism, let me now examine the connection between the selected effects account of function and the new mechanistic account. This analysis is based on Garson's (2013) perspective on the issue.

[34] For more information on this issue, see Garson (2016).

Garson aims to present an alternative viewpoint when approaching the concept of a mechanism, namely, what he terms the functional sense of mechanism. According to Garson, in this functional sense of mechanism, mechanistic characterization is constrained. In his view, "mechanisms serve functions" (Garson 2013: 318). That implies that there are no mechanisms for pathology per se, as pathologies result from the disruption of mechanisms for function. For instance, the heart is considered a mechanism *for* the circulation of blood; however, it is not viewed as a mechanism *for* heart disease. Moreover, natural selection is not regarded as a mechanism since it lacks a specific function.

According to Garson (2013: 320), mechanisms serve functions[35] in the following way: "where X is a system and Y is an activity of X, X is a (part of a) mechanism for Y, only if X has the function Y". To illustrate this proposed formula of mechanisms serving functions, consider once again the example with the heart. The heart is considered a part of a mechanism for circulating blood only if it possesses the function of circulating blood. According to this view, there are no mechanisms for heart disease, rather, pathologies arise as a result of broken or disrupted mechanisms. Although there are instances of pathologies that perform functions, where a certain biological phenomenon can be functional to one system and pathological to another, particularly in the field of cancer research (see Garson 2013: 321), these examples do not contrast with the functional sense of mechanism. In such cases, a mechanism still has a function for one system (e.g., a tumor) and not for the other (e.g., an organism).

Furthermore, Garson provides the example of natural selection as a phenomenon that is not a mechanism for evolution because it does not serve function. Although, according to the selected effects account, something has a function only when it is selected for by a certain selection process; however, natural selection itself is not selected for. I particularly address this issue in Part III, Chapter 8; thus, for now, I do not examine further this issue.

Lastly, Garson outlines that the functional sense of mechanism is ubiquitous in biology because scientists commonly discuss about mechanisms in terms of functions. He posits that this is done because mechanisms are frequently employed in a normative fashion, i.e., it is possible for a certain mechanism to be a mechanism for an activity even though the same mechanism is unable to perform that same activity. For instance, the function of a heart is to

[35] Garson explicitly asserts that, in the functional sense of mechanism, he adopts a modest pluralistic stance. In other words, this means that he believes many accounts of function align with the functional sense of mechanism, specifically, selected effects and goal-contribution accounts. However, he excludes the causal role account, deeming it to too permissive (see Garson 2013: 319). The relationship between Garson's functional sense of mechanism and the causal role account is further addressed towards the end of this section.

pump blood, although there are instances of hearts that cannot perform that activity, indicating malfunction.

In this normative sense, the acknowledgement that something can break or malfunction is fundamental. Garson believes that the concept of mechanisms that can break is widespread in biology. Additionally, he contends that the functional sense of mechanism is useful, and biologists should explain mechanisms in terms of function. This functional sense of mechanisms ultimately leads to "better generalizations", as there are many more states of an organ or various systems that are compatible with disease than with health. The former kinds of states can be explained as a result of the breaking of mechanisms for the latter kinds of states of a mechanism (see Garson 2013: 326).

3.1.1. The Functional Sense of Mechanism vs. the Causal Role Account of Function

Let me now address the differences between the two views regarding the relationship between functions and mechanisms, particularly between Craver's view influenced by the causal role account of function and Garson's view primarily influenced by the selected effects account. Both perspectives highlight a significant connection between functions and mechanisms, asserting that assessments of mechanisms presuppose judgements about function. Although their differences have been partly addressed in the previous two sections, I further discuss them in the following paragraphs.

Let me first briefly outline the first view and then expand it with Lenny Moss' (2012) similar approach to the issue. Craver (2013) contends that judgements about mechanism presuppose judgements about function because whether a system qualifies as a mechanism for something depends on which capacity is selected as the function of that system. In other words, when analyzing a system from a mechanistic perspective, a decision has to be made regarding what counts as the system's function (Craver 2001).

To support his position, Craver develops his own version of the causal role account, adapting it to accommodate the mechanistic approach. As discussed in Section 2, he argues for a perspectivalist approach, suggesting that the function of a system, considered as a whole, is relative to the research community interested in examining the system. Additionally, the function of each part of the system includes the contribution to the function of the system as a whole. Garson (2013: 323) highlights that this causal role version is embraced by many proponents of the new mechanistic account, who, in part, engage with the debate about

functions, including Machamer, Darden, and Craver (2000), Craver (2001; 2013), and Glennan (2002), among others.

The consequence of Craver's view, as outlined by Garson (2013), is that it does not impose substantive restrictions on the kinds of biological activities for which there can be a mechanism.[36] This leads to an important difference from Garson's view, that is, that Craver's approach to the relationship between functions and mechanisms recognizes mechanisms for pathologies, such as mechanisms for drug addiction or Alzheimer's disease. This recognition stems from the idea that "the only context in which a researcher would talk of a 'mechanism for' drug addiction is a context in which drug addiction is the system's conventionally chosen function" (Garson 2013: 323). In this sense, Craver asserts that a mechanism breaks just when it cannot perform its function, and thus the relevant functions are causal role functions and not selected effects functions. On the other hand, Garson's view acknowledges dysfunctions, in contrast to the causal role account of function. This distinction allows Garson to place substantive restrictions on the kinds of system that can be depicted as mechanisms, a position I elaborate on later.

Moss' (2012: 165) perspective is also different than Garson's view. He argues that there are two main senses of 'mechanism' that are important for the present discussion, namely, a strong and a weak sense. According to the weaker sense, asserting that there is a mechanism for a phenomenon means claiming that there is a physical explanation for it, that is, categorizing it as being within the scope of empirical investigation. On the other hand, in the stronger sense, a mechanism for a biological phenomenon relates to the goals or ends of the biological system being investigated. When one explains a part of a system as a mechanism for something, one explicitly conceives of the system itself as having goals or purposes. Although at first glance, it may seem that Moss implies that something counts as a mechanism only if it serves a function, following the strong sense, Garson (2013: 324) argues that Moss does not claim that mechanisms must serve functions because Moss accepts the weaker sense of mechanism. Thus, similar to Craver, Moss accepts that there are mechanisms for pathologies.

On the other hand, Garson (2019: 246) strongly opposes the idea that there are mechanisms for pathologies. In other words, he does not believe that mechanisms can break. Here, Garson attempts to critique the causal role account and their characterization of, i.e., their lack of examination of dysfunction. Garson (2019: 249) argues that there is a conceptual benefit

[36] However, once a research community assigns a function to the system, there are constrains on the functions that can be assigned to its components.

to using his functional sense of mechanism because it explains how mechanisms break. Specifically, in scientific practice, biologists often employ a rich lexicon when describing how mechanisms break, and one of the purposes of medicine is to elucidate how this break happens.

Garson further argues that the normativity of functions preserves the normativity of mechanisms because, as he points out, "a mechanism breaks just when it can't perform its function".[37] According to him, the notable aspect about functions is that they persist, i.e., although the corresponding disposition is no longer present, the idea of the function endures. Thus, Garson describes functions as hooks on which one can "hang the idea of a broken mechanism" (Garson 2019: 250). To sum up his view, "mechanisms are the sort of things that can break because functions are the sort of thing that can fail" (Garson 2019: 250).

In other words, a crucial benefit of the functional sense of mechanism is that it aids biologists and medicine in explaining diseases, particularly when diseases involve dysfunctions. For instance, Alzheimer's disease involves dysfunction, and to quote Garson (2019: 252): "When we know, or have good reason to think, that a disease involves a dysfunction, we shouldn't try to find a mechanism for it. Instead, we should show how the disease is caused by a breakdown of a mechanism". In this sense, according to Garson, the functional sense of mechanism "holds the upper hand" in the relationship between functions and mechanisms because the functional sense of mechanism can accommodate the normative aspect of characterizing mechanisms, which is not the case with the causal role account of function and, in particular, Craver's variation of the mentioned account that is linked with the new mechanistic account.

Garson (2019: 257-258) outlines another way in which the functional sense of mechanism can address philosophers' problems. In this sense, he explores the issue of whether natural selection can be characterized as a mechanism, a topic raised by various authors, most notably, Skipper and Millstein (2005), Benjamin Barros (2008), Illari and Williamson (2010), and Joyce Havstad (2011). According to Garson, natural selection is not a mechanism of evolution because it does not have a function, i.e., it is not itself selected for anything. However, I extensively address this issue in Part III, Chapter 8, where I challenge Garson's perspective on whether natural selection is a mechanism or some other causal explanatory structure.

[37] Garson (2019: 248) argues that scientists and philosophers acknowledge that mechanisms can break; however, the new mechanistic characterization of mechanisms do not make sense of that fact. Thus, he contends that the functional sense of mechanism can help in that regard, i.e., by introducing the normative aspect. Different mechanistic characterizations are examined in Part II, Chapter 4.

CONCLUSION. Chapter 3

To sum up this chapter, I briefly examined the connection between laws of nature and mechanisms, particularly in the context of the new mechanistic account. In that sense, I highlighted that the new mechanistic account aims to sidestep the role of laws in explanations (see Machamer, Darden and Craver 2000) or replace explanations that rely on laws with those grounded in the mechanistic account of causation (see Glennan 1996). However, some authors, such as Andersen (2011), argue that a broad range of scientific generalizations, including some labeled as laws, are actually explained by mechanisms (see Glennan 2017: 45).

Additionally, I examined the role of functions and their status within the new mechanistic account. I particularly focused two contrasting views, namely Craver's (see 2001; 2013) perspective on the relationship between function and mechanism, specifically between functional and mechanical descriptions, and Garson's (see 2013; 2019) view on the relationship between function and mechanism. I outlined that Garson emphasizes that mechanisms serve functions and advocates for the functional sense of mechanism. Moreover, I compared the two opposing views and emphasized their differences, in particular regarding the concept of normativity, which is encompassed in the selected effects account of function. In both Craver's and Garson's characterizations the relationship of functions and mechanisms is highlighted, specifically by asserting that assessments of mechanisms presuppose judgements about function. However, Craver's view is more frequently endorsed by new mechanists since it aims to link the causal role of functions with the use of functions in scientific practice.

CONCLUSION. Part I

To sum up Part I, I initially examined the concept of a mechanism, which finds frequent usage in various areas, such as, among others, politics, economics, technology, science, and philosophy. I discussed its historical roots, tracing back to ancient philosophy, i.e., with figures like Democritus and the Epicureans. Additionally, I outlined different meanings of the concept, with putting a particular emphasis on Nicholson's (2012) approach to the issue, laying the foundation for the subsequent in-depth analysis of the new mechanistic account that is addressed in Part II, Chapter 4. I highlighted the significance of mechanisms in addressing some classic issues in the philosophy of science, such as, among others, prediction and control.

In Chapter 2, I further examined the concept of causation and explanation, focusing on accounts relevant to molecular biology, particularly within the framework of the new mechanistic account. In this sense, I emphasized two families of accounts of causal explanation, namely: (i) the counterfactual accounts of causation, and (ii) the production accounts of causation, i.e., the physical account of causation, and the mechanistic account of causation. In particular, within the accounts (ii), I outlined Woodward's (2003) interventionist account, which is primarily endorsed by new mechanists. Moreover, I depicted another account that preceded the debate on causation and explanation, namely the DN model proposed by Hempel and Oppenheim (1948), which is, as I argued, a causal explanation account as well.

Furthermore, in Chapter 3, I examined the concepts of law of nature and function, addressing the new mechanists' perspectives on the relationship between laws and mechanisms, particularly Glennan's (1996) and Andersen's (2011) views. Here, I emphasized how some new mechanists aim to diminish the role of laws in biology, while others attempt to replace their role with mechanisms. Regarding the role of functions and their connection to the new mechanistic account, I outlined two opposing views. On one side, Craver's (see 2001; 2013) perspective on the relationship between functions and mechanisms, and, on the other, Garson's (see 2013) view on the same relationship. I particularly emphasized Craver's account on this relationship as the one that is mostly endorsed by new mechanists. Specifically, by highlighting that the causal role of functions prevails in scientific practice, which is the focus of new mechanistic authors. This sets the stage for a more in-depth examination of these concept and their interplay in Parts II and III.

PART II: The Causal Explanation in Molecular Biology

INTRODUCTION. Part II

The dominant account of causal explanation in molecular biology is the new mechanistic account, which considers mechanisms as the causal structures that explain phenomena. Although the new mechanistic account encompasses various characterizations of mechanisms, with distinctions among authors, there is a consensus that mechanisms are characterized as causal explanatory structures involving entities and activities organized to produce a particular phenomenon (e.g., protein synthesis; DNA replication) (see Illari and Williamson 2012; Glennan 2017). However, alternative causal structures, such as pathways and cascades, have been introduced in the debate. Ross (2021; forthcoming), among others, prominently and comprehensively advocates for both pathways and cascades as potential alternatives to mechanisms. Pathways are characterized as a sequence of steps ordered in a specific manner leading to an outcome (e.g., glycolysis). Cascades are characterized as structures that involve amplifying steps initiated from a small cause resulting in a huge effect (e.g., blood coagulation; nuclear fission). In this part of the thesis, I focus on the abovementioned causal-explanatory structures.

Firstly, I examine the new mechanistic account. In particular, its various characterizations, with a specific focus on the three most influential ones, that is, Glennan's (1996), Machamer, Darden and Craver's (2000), and Bechtel and Abrahamsen's (2005). I highlight their features and aspects, such as entities, activities, productive continuity, and organization. Furthermore, I examine the concept of a phenomenon, as well as how mechanisms are represented (e.g., through models). Additionally, I emphasize the investigative strategies employed in identifying mechanisms, i.e., decomposition and localization. Moreover, I advocate for a characterization that I believe should be consensual, namely the one proposed by Illari and Williamson (2012), which is also built upon in Glennan (2017). To summarize the new mechanistic account debate, I develop three aspects of mechanisms, modifying Levy's (2013) three kinds of mechanism to incorporate the distinction between the *ontic, epistemic,* and *strategic* aspects. Namely, I argue that this distinction is not always clear in the debate. Finally, I argue for a mechanistic explanation as a process in which scientists identify the mechanism as found in nature (i.e., the ontic aspect of a mechanism) using investigative strategies of decomposition and localization (i.e., the strategic aspect of a

mechanism), and then build a representation of that mechanism, e.g., a model, ultimately explaining the phenomenon in question (i.e., the epistemic aspect of a mechanism).

Secondly, I examine alternative causal-explanatory structures, namely pathways and cascades, with a specific focus on Ross' (2021; forthcoming) characterizations of these structures. I emphasize their distinctive features and aspects (e.g., connection aspect, flow, and amplification). Moreover, I highlight their investigative strategies, namely, the 'mapping' and 'expanding out' strategies. In this sense, I point out that both pathways and cascades share some features and aspects with mechanisms. Particularly when considering Ross' depiction of these structures in contrast with the characterizations of the new mechanistic account. However, I argue that they differ in terms of investigative strategies. Namely, mechanisms are identified through decomposition and localization, whereas pathways and cascades involve 'mapping' and 'expanding out' strategies. Lastly, similar to the case with mechanisms, I delineate three aspects of the corresponding causal structures. I emphasize their respective ontic, epistemic, and strategic aspects, along with their unique processes of explanation.

Thirdly, I highlight another distinction between mechanisms, pathways, and cascades, namely the *system/process* distinction borrowed from Glennan (2017), which I examine while considering pathways and cascades as well. I argue that mechanisms exhibit both system-like and process-like characteristics, whereas pathways and cascades primarily have process-like characteristics. The main difference between these characteristics lies in the *spatial* and *temporal* aspects, with systems having a spatial aspect, while processes emphasize more of a temporal aspect. Lastly, I argue that mechanisms hold a position of explanatorily privilege. Firstly, this stems from the abovementioned system/process distinction, where mechanisms possess both system and process-like characteristics. Secondly, I consider the dimensions of explanatory power introduced by Ylikoski and Kuorikoski (2010). I specifically focus on one dimension, namely *mechanistic detail*, in which mechanisms are explanatorily privileged since they possess more depth in their explanations, i.e., they entail more 'what if' questions and statements in the *explanans – explanandum* relationship compared to pathways and cascades.

The structure of Part II is as follows: in Chapter 4, I examine the new mechanistic account. Section 1 explores its canonical characterizations, starting with Machamer, Darden, and Craver's (2000). Section 2 focuses on Bechtel and Abrahamsen's (2005) characterization, and Section 3 delves into Glennan's (1996; 2002; 2017). Section 4 summarizes the account by providing a consensus view and outlining three aspects of mechanisms.

In Chapter 5, I outline alternative causal explanatory structures, specifically pathways and cascades. Section 1 focuses on the pathway concept proposed by Ross (2021), while Section 2 examines the cascade concept brought forward by Ross (forthcoming).

In Chapter 6, I explore the relationship between all the abovementioned causal-explanatory structures. Section 1 argues for three aspects of the pathway concept along with its respective explanation process. Section 2 presents three aspects regarding the cascade concept. In Section 3, I advocate for mechanisms as a privileged causal-explanatory structure when compared to pathways and cascades.

Chapter 4: The New Mechanistic Account

INTRODUCTION. Chapter 4

The new mechanistic account encompasses a group of similar characterizations of mechanisms within the life sciences. While Chapter 1 in Part I addressed the general meaning of mechanisms, this part of the thesis specifically examines mechanisms as causal-explanatory structures in the life sciences, focusing on molecular biology. In this sense, there are several canonical characterizations of biological mechanisms,[38] with the three most prominent being those put forth by Glennan (1996), Machamer, Darden and Craver (2000), and Bechtel and Abrahamsen (2005).

In this chapter, I emphasize their analysis of mechanisms, highlighting both their similarities and differences. Additionally, I examine their perspectives on representing mechanisms, i.e., the issues of abstraction and specification, as well as the strategies used to discover mechanisms, namely, decomposition and localization. Moreover, I explore Illari and Williamson's (2012) characterization of mechanisms, which aims to synthesize the abovementioned canonical new mechanistic characterizations and establish a consensus view. According to this view, mechanisms are causal-explanatory structures consisting of entities and activities organized in a way that they are responsible for a certain phenomenon of interest. For instance, DNA replication serves as a prominent example of a mechanism, involving the production of two identical replicas of DNA from one original DNA molecule. The process of replication involves the unwinding (represents an activity) of the DNA double helix (which represents an entity) and the bonding (activity) of new component parts (entities) to both parts of the unwound DNA helix (see Craver and Darden 2013: 17).

After examining the three most prominent characterizations, I summarize the debate by advocating for the consensus view presented by Illari and Williamson (2012). I argue that features argued for by various proponents of the new mechanistic account converge into this canonical characterization, encompassing crucial components, such as productive continuity and organization. Additionally, I outline three aspects of the new mechanistic account, namely, ontic, epistemic, and strategic, and situate these aspects within the mechanism's explanation

[38] Bechtel (2011: 534) phrases the term *basic mechanistic account* referring to canonical new mechanistic accounts. However, I do not adopt his terminology since it is not largely endorsed by other proponents of the new mechanistic account.

process. Particularly, to align these aspects with Salmon's (1984) and Levy's (2013) perspectives on causal explanation and mechanisms.

The structure of this chapter is the following one: in Section 1, I examine Machamer, Darden, and Craver's (2000) characterization of mechanisms. In Section 2, I outline Bechtel and Abrahamsen's (2005) view and its further ramifications. In Section 3, I first present Glenann's early view (see 1996; 2002) and then delve into his recent characterization (2017). In Section 4, I outline the consensus view offered by Illari and Williamson (2012), and then delineate between the three aspects in the new mechanistic account, leading to the assertion of the mechanism's explanation process.

SECTION 1. Machamer, Darden and Craver's (2000) Characterization of Mechanisms

The canonical example of a mechanism is protein synthesis (see Figure 10). The mechanism consists of DNA, RNA and protein and is usually abstractly depicted in the following sketch: DNA → RNA → protein.

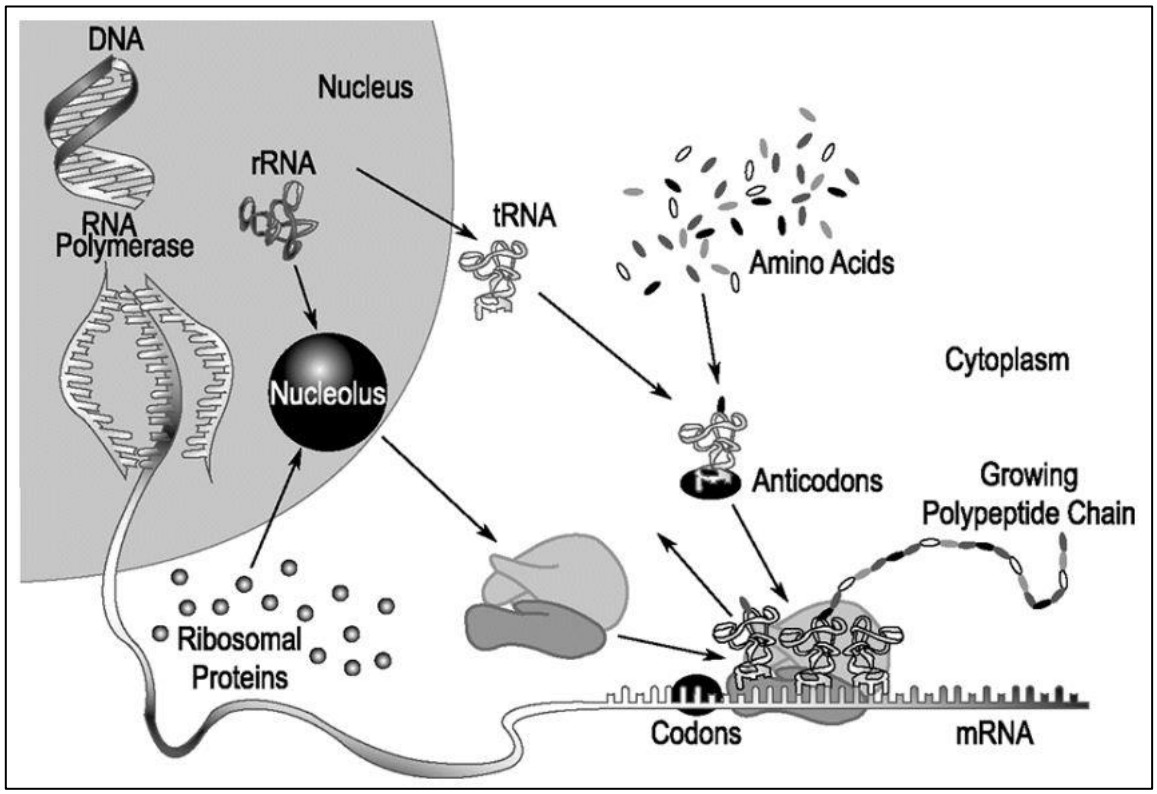

Figure 9: The mechanism of protein synthesis (reproduced from Craver and Darden 2013: 33)

The mechanism involves transcription from DNA to messenger RNA and translation from messenger RNA to the arrangement of amino acids in a protein. It comprises additional details, namely component parts and different activities, which I will examine later.

Mechanisms, such as the protein synthesis mechanism, serve as causal-explanatory structures in the life sciences, particularly molecular biology. These mechanisms are characterized differently by various authors within the new mechanistic account. The first characterization that I examine is introduced by Machamer, Darden and Craver (2000). Their characterization is structured as follows:

> Mechanisms are entities and activities organized such that they are productive of regular changes from start or set-up to finish or termination conditions. (Machamer, Darden and Craver 2000: 3).

According to them, mechanisms explain how a phenomenon comes about or how some significant process works. For instance, following the above-quoted definition, consider the mechanism of DNA replication, where the DNA double helix *unwinds* and new component parts *bond* to both parts of the unwound DNA helix. In this example, we can observe entities, such as DNA and component parts, and activities, such as unwinding and bonding. Explaining a certain phenomenon, such as the replication of DNA, involves providing a description of a mechanism for that phenomenon, that is, explaining how that phenomenon is produced. I first examine the features and aspects of this characterization of mechanisms, and then I explore the characterization of a phenomenon and the way of presenting descriptions of mechanisms.

1.1. Features and Aspects of Mechanisms

Let me first examine the components and features of the above-quoted definition, such as: (i) entities and activities, (ii) finishing conditions, (iii) productive continuity and regularity, and (iv) organization.

(i) Broadly construed, "entities are the things that engage in activities", and "activities are the producers of change" (Machamer, Darden and Craver 2000: 3). In other words, entities are the parts in the mechanism, while activities are the things that entities do. For instance, an enzyme phosphorylates a protein, or a neuron releases a neurotransmitter. In those examples, entities are an enzyme, a protein, a neuron, and a neurotransmitter. Activities are phosphorylating and releasing. Here, we can already observe a difference between entities and activities; activities are described using active verbs, and entities are described using nouns. For instance, pushing, pulling, bonding, releasing, opening, or blocking are kinds of activities. On the other hand, organisms, cells, enzymes, or molecules are kinds of entities. Activities require the existence of entities. Entities have properties, such as, among others, structure, and orientation. Entities then engage in activities. Mechanisms include entities and activities organized together to produce something that individual components cannot produce on their own.

(ii) Finishing conditions concern the working of a mechanism from starting conditions to finishing. Those mechanisms are called linear. In other words, these mechanisms progress linearly from beginning to end. For instance, consider the protein synthesis mechanisms, which is a linear mechanism starting with DNA and ending with the

protein. However, not all mechanisms operate linearly from start to finish. Some may have endpoints in rest, equilibrium, neutralization of a charge, etc. Some mechanisms are organized in cycles. For instance, such a mechanism is the Krebs cycle, namely, a step in the metabolism of sugar. Additionally, "some mechanisms are more clearly described as *underlying* a phenomenon rather than producing it from some earlier state" (Craver and Darden 2013: 19). For instance, the mechanism of action potential "underlies or implements the phenomenon of action potential" (Craver and Darden 2013: 19). Thus, one can say that mechanisms come in various patterns; however, they all have a feature of set up or starting conditions and some kind of an end state or finishing conditions.

(iii) Productive continuity corresponds to the continuity between stages in the mechanism, meaning how mechanisms are productive and bring about a certain process or changes to an end state. In other words, to illustrate how a mechanism works, one has to showcase the different stages in the mechanism, and, particularly, with each stage making a difference to what happens at the subsequent stages. In that sense, one stage of a mechanism produces the other, and it is crucial to depict those stages to present the whole mechanism with its stages from the beginning to the end of the process. The feature of regularity is linked to productive continuity because regularity is exhibited in the way that mechanisms run consistently from the beginning to the end. Moreover, they do so by being regular, i.e., always working in the same way under the same conditions. For instance, consider again the example of DNA replication. The mechanism starts with a single double helix and finishes with two. "One double helix unwinds, and each half provides a template along which complementary bases are aligned, yielding two helices at the end" (Craver and Darden 2013: 19). The two helices can be more or less like the parent one, which depends on the reliability of the copying mechanism that operates. In more stressful environments, for instance, some copies of DNA are produced with more mutations in the daughter helices.

(iv) Organization is a feature of mechanisms that distinguishes between mechanisms being a mere sum of the properties of their comprising parts and mechanisms being what they actually are, i.e., having entities and activities organized in such a way that produce a phenomenon. Those entities and activities are organized *spatially* and *temporally*. "*Spatial* organization includes such things as the locations, sizes, shapes, and orientations of entities. *Temporal* organization includes the orders,

> rates, and durations of the stages" (Craver and Darden 2013: 20). Depending on the spatial and temporal organization, mechanisms act differently. A crucial point to note regarding the feature of organization is that there is a presence of active organization, which includes facts about which components make a difference to others and how those differences are made, in particular, by which activities are those differences made.

After examining the abovementioned four features of mechanisms, I also emphasize that there are two other important aspects of Machamer, Darden and Craver's (2000) understanding of mechanisms. These aspects are further modified and adjusted in their future work regarding the concept of a mechanism (e.g., see Darden 2005; Craver 2007; Craver and Darden 2013). The two aspects are the following: *levels of mechanism* and the *mechanistic context*.

Mechanisms found in the life sciences often span multiple levels. For instance, some biologists study higher levels, such as ecosystems and populations, while others examine organisms, organs and cells, and some focus on macromolecules, molecules, and ions. Different biological fields of study explore different levels of mechanisms, i.e., ecologists study ecosystems, cell biologists examine cells, and molecular biologists study molecules. Thus, levels of mechanisms can be "defined in terms of the relationship between the behavior of a mechanism as a whole and the behavior of a component in that mechanism" (Craver and Darden 2013: 22). Here, I introduce one important aspect when examining mechanisms and that is *decomposition,* which is crucial for other characterizations of mechanisms (see Bechtel and Abrahamsen 2005; Bechtel and Richardson 2010). Craver and Darden (2013: 21) use decomposition to present the aspect of a mechanism in which, by decomposing a mechanistic hierarchy, one includes another level in the description of a mechanism. For instance, to understand the food chain, "one has to understand the relationships among many different items in an ecosystem: the behaviors of individual organisms, the physiological function of biological systems within organisms that mediate those behaviors, the operations of organs composing those systems, the activities of cells within them, etc." (Craver and Darden 2013: 21). In this sense, one observes that the description of a multilevel mechanism tops off in some highest-level phenomenon of interest and bottoms out in some lowest-level mechanisms. Where mechanisms top off depends on one's interest and explanatory questions (see Part I, Chapter 3, Section 2). For instance, structural chemists may not be interested in higher-level mechanisms. Regarding the bottom-out point for a description of a mechanism, Craver and Darden (2013: 22) believe that our knowledge about bottom-out mechanisms might be limited by the scientific

level that we are currently at or that the further emphasis of details is not required or even irrelevant for a certain explanatory project a scientist is engaged in. In other words, bottoming-out is defined as reaching to the lowest level mechanisms in the hierarchy and the component parts that are accepted as "fundamental or taken as unproblematic for the purposes of a given scientist, research group, or field" (Machamer, Darden and Craver 2000: 13).[39]

The second important aspect of mechanisms is also that they are located in wider contexts. For instance, when one wants to situate a mechanism in a wider temporal context, one has to find mechanisms that occur before it and provide some of its start conditions. That is particularly important when examining mechanisms in an evolutionary context where the origin (or origins) of life began a temporal chain of biological mechanisms that spanned through the present day. Following this reasoning, products of final conditions of a mechanism may provide starting conditions for the subsequent mechanism. Thus, it is important to locate mechanisms in wider hierarchical and temporal biological contexts, which then, consequently, helps with the examination of further mechanisms.

1.2. Characterizing a Phenomenon and Representing Descriptions of Mechanisms

1.2.1. What is a Phenomenon?

Before moving on to other characterizations of mechanisms, I would like to examine the characterization of a phenomenon, or an *explanandum*. I do so here because I believe that it is important to address this issue in the beginning to clarify what the subject of a mechanistic explanation is. In particular, I examine what phenomenon means according to various new mechanistic authors, beginning with Machamer, Darden, and Craver (2000).

Their characterization perceives a mechanism as mechanism *of* a given phenomenon. This statement is also found in Kauffman (1970) and Glennan (1996). What a phenomenon is for new mechanists is laid out in Craver and Tabery (2019), where they characterize it in the following way: "The phenomenon is the behavior of a mechanism as a whole". For instance, the mechanism of protein synthesis synthesizes protein, i.e., macromolecules composed of

[39] When one mentions levels of mechanisms and the multi-level mechanistic aspect, it inevitably needs to mention functions, namely the contribution of parts to the mechanism as a whole as well as the function of a mechanism as a whole. I already covered that topic in Part I (see Chapter 3, Sections 2 and 3), thus I do not examine that issue further.

amino acids. In this sense, the limits of mechanisms, that is, the components of a mechanism that are a part of a mechanism, are specified by the phenomenon that mechanism explains. In other words, those entities, activities, and organizational features that are in the mechanism, are those that are relevant to the phenomenon that one aims to explain. As Craver and Darden (2013: 52) point out: "mechanisms are the mechanisms of the things that they do".

Glennan (2017) emphasizes that, according to his minimal mechanism characterization (see this chapter, Section 3), "all mechanisms are mechanisms for some phenomenon" (Glennan 2017: 23). According to him, that is consistent with the scientific usage of the term mechanism, in the sense that scientists identify a mechanism by finding out what it is that the particular mechanism does or produces (e.g., the mechanism of protein synthesis is the mechanism identified for producing proteins). In other words, one uses a phenomenon to identify its mechanism.

Glennan (2017: 24) also explores the close relationship between the phenomenon of a mechanism and the idea of function. According to his minimal characterization of a mechanism, he argues that not all mechanistic phenomena have functions. Mechanisms that do have a function include those made with a purpose or possessing characteristics acquired through natural selection. Instances in which a mechanism does not have a function are the side effects of certain mechanisms (e.g., engines produce heat through a mechanism, but heat is not the function of the mechanism). Additionally, these instances are phenomena produced by mechanisms independently, without a design or a selection process (e.g., volcanoes that are mechanisms for spitting lava, but there is no design or function involved in their eruption).

In his earlier characterizations, Glennan (see 1996; 2002) referred to a mechanism's phenomenon as a *behavior*. In his more recent characterization (see 2017), he clarifies that a phenomenon can also be a behavior, or a pattern for which a mechanism is responsible. By this, he means that phenomena, behavior, or patterns can be observable, i.e., seen. Lastly, he specifies that phenomena can be either regular or non-recurrent, in other words, 'one-off' phenomena. He provides the example of the continual beating of a heart as a regular phenomenon and a heart attack that is a "one-off" phenomenon.

Tudor Baetu (2019) approaches the definition of a phenomenon within the context of the new mechanistic account in a slightly different manner. He contends that phenomena are commonly conceived as both constitutive elements of empirical reality and objects of explanation. According to his account, *data* satisfy this description, thereby making data phenomena to be explained. Baetu's perspective is motivated by the way scientists perceive phenomena. He describes his task as the one introducing an alternative view of what a

phenomenon is. According to his view (2019: 7), "empirical research in contemporary biology is primarily a matter of conducting controlled experiments in order to generate data structured in such a way as to make possible inferences about the causal structure of the world". In contrast, the opposing view considers mere observations as phenomena.[40] Baetu argues that this perspective is motivated by the implicit assumption that data correspond to what logical positivists refer to as 'observation', i.e., end products of measurements. Upon examining data presented in scientific papers, Baetu contends that data are not mere observations, and he argues that such an assumption is mistaken.

Returning to more traditional new mechanistic views, it is important to recognize that characterizing a phenomenon for explanation is integral to the process of discovering mechanisms. Describing a phenomenon, in this sense, involves characterizing it using the language specific to a given field. Additionally, it is important to highlight that the characterization of a phenomenon implicitly prompts the use of explanatory concepts such as entities, activities and organizational structures that are familiar to the field at that time. These concepts are then employed to construct a representation of a mechanism.

1.2.2. How is a Mechanism Represented?

Scientists describe mechanisms in several ways, for instance, in visual, textual, diagrammatic, or mathematical approaches. Craver and Darden (2013: 30), as well as Machamer, Darden and Craver (2000), use terms like 'mechanism schemas' or 'mechanistic models' to refer to representations of mechanisms. These representations, or schemas, serve scientists in describing, explaining, exploring, predicting, and controlling phenomena. The abovementioned diverse ways of representing mechanisms can be illustrated through the example of depicting a human heart (see Figure 10). On one side, a textual explanation can be provided, detailing how the heart works, outlining its relevant parts, such as the right and left atria, right and left ventricles, pulmonary and aortic valves, pulmonary artery, and pulmonary vein, along with the activities of these parts. On the other side, a video representation can showcase how these heart components work and contribute together, depicting the reception of blood by the right and left atria and the pumping of blood by the right and left ventricles. Such a video might portray the heart in three dimensions, revealing spatial relationships

[40] For more information on the traditional view, originating with logical positivists, about phenomena being perceived as observables, see Jamse Bogen and Woodward (1988).

between its parts, or it might emphasize temporal relations and the sequential order of activities and interactions of the parts included. Such a representation of the heart could be detailed and very informative. However, the heart can also be represented visually, in the form of a picture, as commonly found in medical textbooks and other educational materials. This representation includes some component parts and might indicate certain activities and interactions of these parts. These causal relations are usually represented with arrows to signify the directions of activities and interactions (e.g., the flow of blood). Although this depiction might not capture all interactions and activities in detail, it provides information not necessarily present in textual representations. In Figure 10, certain component parts are visible, and while most interactions and activities are omitted, arrows indicate the direction of flow.

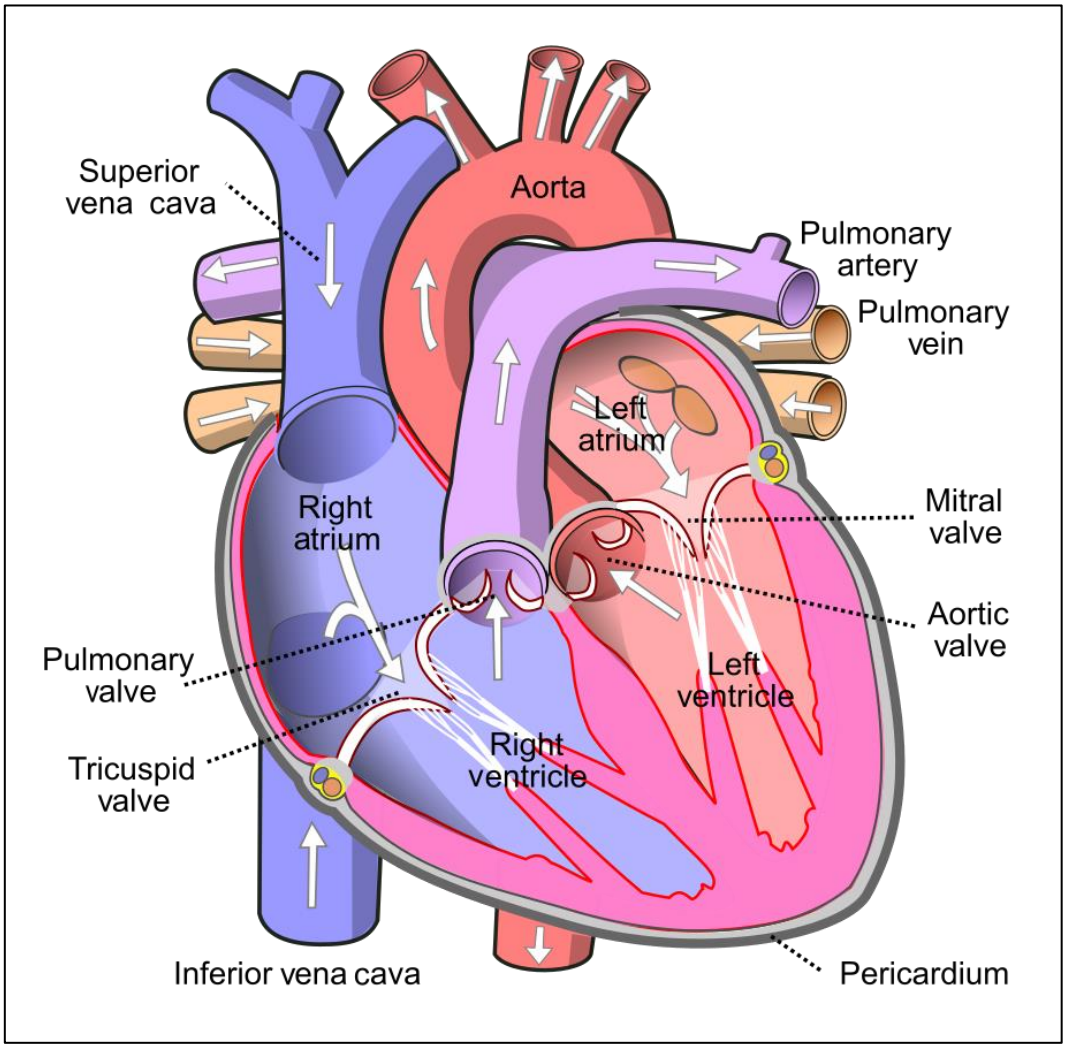

Figure 10. The model of the human heart[41]

[41] By Wapcaplet – own work, CC BY-SA 3.0, https://commons.wikimedia.org/w/index.php?curid=830253

This kind of a representation offers some insight into the spatial organization of parts and at least this type of activity.[42]

I would like to emphasize another important distinction between different dimensions of mechanism schemas, offered by Craver and Darden (2013: 30): (i) *completeness* (sketch to schema), and (ii) *detail* (abstract to specific).[43]

According to dimension (i), representations of mechanisms range from a basic mechanism sketch to a comprehensive schema suitable for purposes of scientific inquiry. A mechanism schema is defined as a detailed description of a mechanism, including known mechanistic features such as entities, activities, and organizational features. The schema's details can be filled in further based on the need for additional information in subsequent scientific inquires. Contrary to a schema, a sketch is an incomplete representation of a mechanism. In other words, it characterizes some parts, activities, and features of organization but also includes gaps typically depicted as black boxes or question marks. Moreover, these gaps can be masked using filler terms, such as 'to activate', 'cause', 'inhibit', and so on. Black boxes, question marks, and filler terms play a significant role by highlighting areas where scientists should focus their research efforts, aiming to progressively enhance the representation of the mechanism, ultimately reaching the status of a complete and comprehensive mechanism schema.

Craver and Darden (2013: 31) nicely illustrate the crucial scientific task in discovering mechanistic models as follows: "to transform black boxes (for which neither components nor functions are known) into grey boxes (for which component functions are specified) into glass boxes". They use the central dogma of molecular biology as an abstract example: DNA → RNA → protein. This schema represents protein synthesis, and if necessary, one can fill this abstract illustration with details, based on research goals or educational purposes.

In 1952, James Watson presented just a piece of paper featuring the abovementioned sketch of protein synthesis. At that time, little was known about the structures and types of RNA and their roles in the protein synthesis mechanism. Additionally, biochemists did not unanimously agree that DNA and RNA belonged in the protein synthesis sketch. However, today, we

[42] Mechanisms can also be described using sets of equations or in the form of causal Bayesian nets, serving as representations of the causal organization within the mechanisms. While these models may omit certain features of the mechanism, such as spatial and temporal relations, as well as numerous other details of its parts and organization, they provide a representation of causal organization that, perhaps, textual, pictorial, and other forms of representations may not convey. These representations are not particularly addressed by new mechanists, at least not by the characterization offered by Machamer, Darden and Craver (2000).

[43] Craver and Darden (2013: 30) introduce two additional dimensions of mechanism schemas, namely support (from how-possibly to how-actually), and scope (from narrow to wide). However, these dimensions are not extensively explored in the new mechanistic literature, and as a result, the emphasis in this discussion is placed more on the first two dimensions.

understand that Watson's sketch accurately depicts the mechanism of protein synthesis. In other words, the initial sketch, which had numerous black boxes, i.e., representing parts and functions of the mechanism that are unknown, evolved into a schema with glass boxes, i.e., a fully articulated mechanism that allows one to look inside and observe the relevant components of the protein synthesis mechanism.

According to dimension (ii), a crucial aspect of mechanism schemas is the level of detail, namely the *abstraction* and *specification* processes. Abstraction is the process of simplifying a schema by omitting certain details, while specification involves the process of adding details to enhance specificity. A comparison between Figure 9, representing the mechanism of protein synthesis, and the previously mentioned DNA → RNA → protein sketch illustrates this distinction. It is obvious that Figure 9 contains more details, i.e., making the schema more specific, whereas the sketch is more abstract.

It is important to distinguish abstraction from a similar process called *idealization*. Both abstraction and idealization are features of mechanisms models and are not mechanisms themselves. They are methods that are used to represent mechanisms. The key difference between them lies in the fact that abstraction involves intentional omission of details in a model, while idealization intentionally misrepresents a certain phenomenon. In other words, idealized models are not accurate depictions and do not exist in the real world. They are tailored specifically for the model's purpose (see Anić 2022: 107). On the other hand, abstraction is characterized as the level of detail in a model, where it can be understood as a form of incompleteness (see Jones 2005; Godfrey-Smith 2009).[44] In other words, models with high degree of abstractions are true but simplified (see Potochnik 2017; Levy 2018). This distinction will be revisited in the following section when presenting Bechtel and Abrahamsen's (2005) characterization of mechanisms.

[44] For instance, Jones (2005: 175) characterizes idealization as "assertion of falsehood" and abstraction as "omission of a truth".

SECTION 2. Bechtel and Abrahamsen's (2005) Characterization of Mechanisms

Bechtel and Abrahamsen (2005) proceed to characterize mechanisms in the following way:

> A mechanism is a structure performing a function in virtue of its component parts, component operations, and their organization. The orchestrated functioning of the mechanism is responsible for one or more phenomena. (Bechtel and Abrahamsen 2005: 423)

Besides this characterization, Bechtel and Abrahamsen (2005) expand on it by highlighting that the component parts of a mechanism are those that are actively involved in producing a phenomenon of interest. Moreover, each component operation within the mechanism involves at least one of these component parts. Additionally, operations can also be organized in a temporal sequence. However, biological mechanisms tend to be organized in a complex fashion. Finally, mechanisms may operate at multiple levels, aligning with Machamer, Darden and Craver's (2000) characterization (see Bechtel and Abrahamsen 2005: 424).

Before examining their characterization further, let me briefly outline Bechtel and Abrahamsen's motivation for considering mechanisms as a causal structure suitable for explaining phenomena in the life sciences. They argue that the widely accepted view of scientific explanation, namely the DN model (see Part I, Chapter 2, Section 1), is not applicable in the life sciences. Most explanations in the life sciences often do not rely on laws, as implied by the DN model. This becomes particularly evident when examining biological literature, where the term 'mechanism' is frequently used in explanations. According to Bechtel and Abrahamsen (2005: 422), to explain the *why*, biologists explain the *how*. They contend that the examination of a causal structure, such as a mechanism, was overlooked in the philosophy of science, largely due to the dominance of physics and its emphasis on explanations including laws. However, with the growing interest in the life sciences in recent decades, philosophers have turned their attention to mechanistic explanations. Bechtel and Abrahamsen aim to provide a basic characterization of mechanisms within the framework of the new mechanistic account.

Let me now examine their approach in more detail. Consider the model of the human heart (see Figure 10). Following that model and Bechtel and Abrahamsen's (2005) characterization, a component part might be, for instance, a ventricle, and a component operation could involve the contraction and relaxation of chambers and valves. These

component parts are organized both spatially and temporally, creating a sequence where blood flows from the atrium to the valve, then to the ventricle, another valve, and finally into the aorta or pulmonary artery, continuing into the rest of the circulatory system. Importantly, the component operations have to be precisely timed to produce a coordinated effect (see Bechtel and Abrahamsen 2005: 424).

After characterizing mechanisms in the abovementioned terms, Bechtel and Abrahamsen present an approach to mechanistic explanation. Here, they reference Salmon's (1984) distinction between *ontic* and *epistemic* approaches to explanation, a distinction not covered in Part I where Salmon's production account of causation was presented (see Chapter 2) since it is more closely connected to Bechtel and Abrahamsen's perspective, which is addressed here. The ontic approach implies that the mechanism itself, as found in nature, serves as the explanation. In other words, an ontic explanation appeals to the actual mechanism in nature. On the other hand, the epistemic approach implies that an explanation derives from our mental activities, appealing to derivations from laws, models, etc. In this sense, Bechtel and Abrahamsen's (2005) account of mechanistic explanation is often regarded as epistemic (see Glennan 2017).[45] They argue that mechanisms are real systems in nature, but they emphasize that "it is crucial to note that offering an explanation is still an epistemic activity and that the mechanism in nature does not directly perform the explanatory work" (Bechtel and Abrahamsen 2005: 425). In other words, they argue that the explanation is a cognitive activity, which is particularly evident in cases where scientists provide explanations for mechanisms that are not currently operating in nature, or for mechanisms that have been operating long before scientists provided explanations. In these cases, although there is no current mechanism operating in nature, we still appeal to explanations. This perspective aligns with Bechtel's later work (see Bechtel 2011; 2019), where the focus is on representations of mechanisms. The distinction between epistemic and ontic approaches to explanations will be further addressed in Section 4 of this chapter.

Bechtel and Abrahamsen (2005: 439) encapsulate their characterization by outlining that a mechanistic explanation is a cognitive activity involving representing and reasoning about nature. They specifically underscore the importance of visual presentations of

[45] In contrast, Craver (see 2001; 2007; 2013) is commonly considered as an author pertaining to the ontic explanation. However, he rejects that label (see Glennan 2017; Craver and Kaplan 2020) by emphasizing his previous work, namely that Machamer, Darden and Craver (2000) and Craver and Darden (2013) discussed thoroughly the epistemic approach, particularly representations of mechanisms, i.e., mechanism schemas (e.g., models). Constructing schemas is considered an epistemic activity. In this sense, Craver and Kaplan (2020) assert that they have put an emphasis on both ontic and epistemic approaches.

mechanisms as vehicles for enhancing explanations. This aspect is further explored in the following subsection. Additionally, they emphasize the significance of investigative strategies, such as decomposition and localization, in the process of discovering mechanistic explanations. This aspect is examined in the next subsection as well.

2.1. Representing Mechanisms

Representations of mechanisms is a widely discussed topic in the new mechanistic account (see Section 1.2.2.). Usually, when new mechanists discuss representations, they refer to schemas, diagrams, linguistic description, equations, etc. In literature, particularly in the case of Bechtel and Abrahamsen (2005), these representations are referred to as *models* of mechanism. A model of a mechanism represents the relevant component parts and operations of a mechanism, along with their organization in the system that leads to the production of a phenomenon. The aim of the model is to depict relevant aspects of the mechanism operating in the world.

Consider the model of a heart (see Figure 10). In this model, relevant parts of a heart found in the human organism are represented. The heart model illustrates relevant parts that operate and are organized in a way to maintain blood circulation throughout the organism. For instance, the heart model shows that blood is being pumped simultaneously from two chambers to two ventricles. Thus, models, and in this case a diagram, are of particular value in biology. Biologists use diagrams to evaluate and revise their accounts of phenomena in nature and the mechanisms producing those phenomena (see Sheredos et al. 2013).

The main distinction between linguistic representations and diagrams is that the latter convey both *spatial* and *temporal* characteristics. Spatially, a diagram shows the parts of a mechanism and their relationship, including colors, and shapes that can enhance the accuracy of the information. Temporally, a diagram can represent time using arrows to indicate relations. According to these advantages, particularly the ability to perceive parts and operations of mechanisms simultaneously, visual representations are often found in biological papers and handbooks that include mechanistic explanations.[46] Furthermore, Sheredos et al. (2013: 932)

[46] There is also present an emerging interest in attempting to characterize networked-based approaches to mechanistic explanations (see Bechtel 2019). Systems biology represents an approach that tries to, roughly, explain phenomena by understanding a "larger picture", i.e., by putting pieces together (e.g., cells, parts of organisms, tissues, etc.). It is perceived as a rival approach to the reductionist one that does the opposite thing, namely take pieces apart. Thus, in a way it is also viewed as a possible alternative to the new mechanistic

argue that diagrams play a central role in biology due to two key tasks as follows: (i) displaying phenomena at various levels of detail; and (ii) constructing mechanistic explanation for those phenomena. In the next few paragraphs, the focus is on point (i), while point (ii) will be examined when discussing investigative strategies central to Bechtel and Richardson (2010) and my further argumentation for the three aspects of mechanisms, particularly the strategic aspect (see Section 4).

Now, regarding point (i), the issue of specification and abstraction is further examined here as it is closely connected to Bechtel and Abrahamsen's (2005) characterization of mechanisms and its later ramifications. Since the introduction of the new mechanistic account, authors such as Machamer, Darden, and Craver (2000), Darden (2006), and Craver (2007), often emphasize the importance of completeness and specificity in mechanistic explanations.[47] In other words, it seems that the advantage of mechanistic explanations is that it contains the needed details used to explain phenomena. Some authors have criticized this approach, arguing that the new mechanistic account lacks abstraction (see, in particular, Ross 2021).[48]

Bechtel and Abrahamsen (2005) did not explicitly emphasize whether the new mechanistic account exhibits more specification or abstraction. However, Levy and Bechtel (2013) attempted to extend this approach to the new mechanistic account by asserting that 'less is more', i.e., that abstraction is an important feature in the new mechanistic account. According to them (2013: 242), "abstraction is the omission of detail". In other words, an abstract description intentionally leaves things out and leaves matters open. Abstraction also exists in degrees, i.e., implying that a certain description is abstract when a more detailed description is available. When we deal with the topic of causal structures (e.g., mechanisms), abstraction in description pertains to representing patterns of causal connections in a system and the process of omitting structural features of the system and other details deemed irrelevant for the current description.

Abstraction also plays a role in explanation. Levy and Bechtel (2013: 258) argue that abstraction is especially important in the process of identifying relevant causal organization

reasoning. System biologists usually represent their data using networks that lay out a more system-wide perspective than traditional mechanistic approaches.

[47] Levy and Bechtel (2013: 257-258) address specifically the mentioned authors stance towards completeness, i.e., they argue that those authors (Darden 2006; Craver 2007) suggest that mechanisms schemes are just templates for explanation, not explanatory themselves. Levy and Bechtel (2013) argue that abstraction both serves the virtue of identifying relevant causal organization and for the purposes of generalization.

[48] I would like to point out that Craver and Kaplan (2020) argue for a correction in the discussion in the sense that Craver's earlier papers, as well as the new mechanistic account as a whole, does not argue exclusively for specification and completeness, rather for both specification and abstraction as each of them has its own advantages in representing mechanisms.

and generalizing. They describe generality virtue as a quality that emphasizes a common underlying causal structure. In other words, these same causal features play similar roles in diverse systems. In summary, they point out the importance of abstraction for certain explanatory purposes, particularly those related to identifying causal organization. The following quote nicely summarizes their view:

> It is always possible, and we argue, often desirable to overlook the more concrete aspects of a system and represent its organization abstractly as a set of interconnections among its elements, oftentimes such a detail-poor representation will be well suited for the explanatory purposes at hand. (Levy and Bechtel 2013: 255)

I proceed to demonstrate this virtue of abstraction argued by Levy and Bechtel (2013), introducing the example of the central dogma of molecular biology, along with its representation.

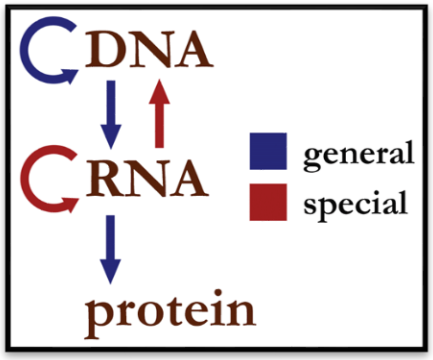

Figure 11: Abstract representation of the Central Dogma of Molecular Biology

The central dogma was first introduced by Francis Crick in 1958. According to Crick (1970: 561), "the central dogma of molecular biology deals with the detailed residue-by-residue transfer of sequential information. It states that such information cannot be transferred from protein to either protein or nucleic acid." In this definition, the term 'information' is mentioned twice. The issue of what constitutes information in the central dogma requires a research on its own, as a significant philosophical debate surrounds it (see, for instance, Stegmann 2005; Šustar 2007b; Blečić 2022). For the purposes of this section, I focus solely on the representation aspect of the central dogma of molecular biology.

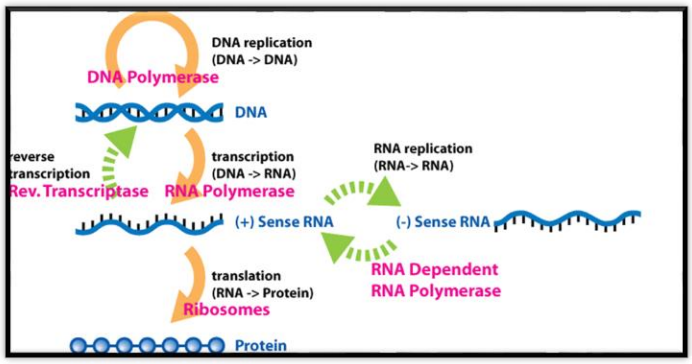

Figure 12: Detailed representation of The Central Dogma of Molecular Biology

The central dogma can be represented in several ways,

depending on the degree of abstraction. On one side, as shown in Figure 11,[49] it can be represented at the highest degree of abstraction. On the other hand, it can be depicted as seen in Figure 12,[50] i.e., more comprehensively, with more emphasis on details. Observing the former representation of the central dogma (see Figure 11), three parts are highlighted, namely DNA, RNA, and protein. Arrows indicate the direction of information flow through the central dogma, that is, from DNA to RNA to protein but not vice versa. This representation provides a general idea of how the central dogma operates, highlighting the operating parts, such as DNA, RNA, and protein, and the flow of information between them with different directions.

However, looking at the second representation (see Figure 12), it is more complete, i.e., containing more details than the Figure 11. Here, DNA replication (DNA to DNA), transcription (DNA to RNA) and translation (RNA to protein) are present. Enzymes such as DNA Polymerase and RNA Polymerase, as well as ribosomes, are highlighted as parts of the central dogma. This representation offers more information, including the enzymes and molecules involved in the process, the components, and their interactions. Arrows indicate the direction of information flow through the process.

Both representations depict the central dogma but with different degrees of abstraction. The choice of representation depends on the circumstances. For instance, the first representation (see Figure 11) may be more suitable and sufficient for someone enquiring about the central dogma without requiring in-depth knowledge. It allows one to grasp the general idea even with a highly abstract representation. The second representation (see Figure 12) is more suitable for individuals seeking a deeper understanding of the process, including the names of its parts, their interactions, etc.

To sum up this subsection, let me return to Bechtel and Abrahamsen (2005), and their emphasis on the significance of visual presentations. They argue that diagrams offer advantages over linguistic presentations, primarily due to their utilization of space to convey relations and interactions between component parts. Consider, for instance, the heart model (Figure 10). The spatial layout and organization of component parts play a crucial role in effectively conveying mechanisms, particularly given that various operations may occur in different locations. Diagrams present these spatial relations more completely.

[49] Figure 11 is reproduced from Narayanese – own work, CC BY-SA 3.0, https://commons.wikimedia.org/wiki/File:Centraldogma_nodetails.png
[50] Figure 12 is reproduced from Dhorspool – own work, CC BY-SA 3.0, https://commons.wikimedia.org/wiki/File:Extended_Central_Dogma_with_Enzymes.jpg

Another advantage of diagrams lies in their capacity to include different colors and shapes, allowing for the emphasis of the most important parts and operations within a mechanism. Additionally, temporal relations, which are equally important for explaining mechanisms, can be depicted through the strategic use of arrows. Lastly, as highlighted in Bechtel and Abrahamsen (2005: 428), the most apparent advantage of diagrams is that all parts and operations are available for examination simultaneously. Although there are various ways to depict a mechanism, e.g., linguistically, or through diagrams, it seems, at least for Bechtel and Abrahamsen (2005), that visual representations of mechanisms clearly offer advantages.

2.2. Investigative Strategies: Decomposition and Localization

Investigative strategies, such as decomposition and localization, play a pivotal role in the scientific discovery of mechanisms. Before examining the specifics of these strategies, a brief overview of scientific discovery is warranted, namely, another significant topic within the new mechanistic account, as it is connected with investigative strategies, i.e., with the identification of mechanisms.

According to Bechtel and Richardson (2010), scientific discovery stands out as a key area in the philosophy of science where mechanisms are prominent. However, in the initial part of the 20th century, logical empiricists, such as Hans Reichenbach (1951) and Carl G. Hempel (1965), largely overlooked the topic of scientific discovery. The works of Karl Popper (1959) and Ernst Nagel (1961) also focused on the logical analysis and structure of scientific explanation and theories. Scientific discovery was deemed as a topic for psychologists rather than philosophers.

However, a significant shift occurred in the latter half of the 20th century. Scholars such as Thomas Kuhn (1970) and Paul Feyerabend (1975), and especially in the later decades with authors such as Paul Thagard (1988) and Lindley Darden (1991), recognized that the history of science presented numerous examples that did not align with logical positivists and empiricist perspective on science. These authors turned their attention to investigating the process of scientific discovery, specifically by delving into the history of science. In this sense, Bechtel and Richardson (1993; 2010) align themselves with these authors, focusing on the reasoning strategies employed by scientists engaged in mechanistic research, i.e., into how these strategies contribute to the discovery of mechanisms.

The two heuristic investigative strategies employed in the discovery of mechanisms are decomposition and localization. *Decomposition* involves considering the behavior of a system, i.e., its activity, as the product of subordinate functions performed within the system. In other words, the activity of a system is a result of interactive functions that collectively contribute to the system's behavior. The assumption of a system being decomposable is evaluated retrospectively. Thus, while it may lead to a false explanation, the decomposition strategy serves as an initial approach to explaining and understanding systems. Despite the potential failures, Bechtel and Richardson (2010: 24) argue that such failures can be enlightening, since they often lead to the discovery of additional influences on the behavior of a system. *Localization* is a strategy involving the identification of different activities proposed during the process of decomposition within the system. In other words, this can take the form of a scientist pinpointing a physical part of a system and localizing various component functions. Alternatively, various functional tools can be employed to determine that there are parts without identifying them, namely, this may involve inhibiting the operation of parts and observing the consequences of system's behavior.

To illustrate these strategies, the authors present examples such as a specialized enzyme that is responsible for catalyzing a certain chemical reaction within a cell and a cell nucleus that is responsible for genetic control. In other words, Bechtel and Richardson (2010: 24) note that "decomposition and localization assume that a single component within the system is responsible for some range of phenomena by the system". These kinds of simple assumptions serve as the initial steps in constructing explanatory models. However, there is also a presupposition inherent in these two strategies. To assume that a certain system is decomposable, it has to exhibit a modular character, where each component depends on other components. According to Bechtel and Richardson (2010: 25): "each component is dependent at most upon inputs from other components, influences other components only by its outputs, and has a specific, intrinsic function". On the other hand, localization presupposes a modular organization of a system. This means that components can be analyzed separately, namely, assuming that each component has its own intelligible function in isolation that can be investigated independently.

Additionally, the authors introduce distinctions for both strategies. Concerning localization, they distinguish between *simple* or *direct* localization and *complex* or *indirect* localization. The former involves straightforward assumptions, such as attributing the responsibility for vision to the posterior cerebral lobes, focusing on single components and few constraints. However, simple assumptions such as this one often proves insufficient, as the

behavior to be explained, that is, an *explanandum,* is frequently produced by more than one component. In this sense, complex or direct localization focuses on localizing a set of component parts along with their functions. It assumes complex constraints and organization in the interaction between components within such complex systems. This approach acknowledges that behaviors often arise from the interplay and coordination of multiple components, requiring a sophisticated understanding of their relationships and functions.

Regarding decomposition, the authors make a distinction between *aggregative* systems and *composite* systems. An aggregative system is, for instance, a fluid flow or the movement of a herd, which, according to authors, "*approximates* aggregative motion" (Bechtel and Richardson 2010: 25). These systems are *simply* decomposable in the sense that a system behavior in such an example is a linear or aggregative function of component behavior. However, if the relevant systemic properties are at least partially determined by the organization of a system, the system can no longer be considered aggregative. To address this, the authors introduce composite systems, which are further distinguished between *component* systems and *integrated* systems. In the former, the behavior of the parts of the system is intrinsically determined, and it is possible to determine the component properties in isolation even though those component parts interact. In the latter, systemic organization significantly determines more constituent functions, and in such a system, dependence of components may obscure the distinction between them. Bechtel and Richardson illustrate this concept with the example of mitochondria, which was once an independent organism but is now integrated into the cell, being a part of it, and playing a significant role in cell metabolism. In other words, understanding mitochondria requires considering its integration with the cell.

In sum, both decomposition and localization strategies serve as tools for addressing explanatory challenges in complex systems. However, for these strategies to be effective, Bechtel and Richardson (2010: 27) emphasize the importance of considering hierarchy and organization. This issue, along with decomposition, was briefly touched upon in Section 1, where I discussed Machamer, Darden, and Craver's (2000) characterization of mechanisms, which shares some similarities with Bechtel and Richardson's proposed strategies.

The success of decomposition and localization strategies is influenced by hierarchical structure of systems. In this sense, Bechtel and Richardson (2010) consider components and whole systems as parts of nature that incorporate a hierarchy of levels. Here, the authors distinguish between two kinds of processes, namely, *horizontal* and *vertical* processes. A horizontal process occurs when parts of a system interact with each other. In other words, interaction occurs between units on a single level. On the other hand, a vertical process is

present when attention is given to the components combined into larger units that can interact with other larger units, highlighting relationships between different levels.

I emphasize that there are similarities between the views presented by Bechtel and Richardson (2010) and Machamer, Darden, and Craver (2000), particularly in the sense that both share emphasis on the role of decomposition and the hierarchy of levels. The hierarchy of levels of mechanisms in Machamer, Darden, and Craver (2000) is characterized as the relationship between the behavior of a mechanism as a whole and the behavior of its components. In this sense, decomposition is employed to elucidate the aspect of a mechanism by decomposing a mechanistic hierarchy, adding another level to the description of a mechanism. For instance, understanding the food chain requires comprehending the relationships among various elements in the ecosystem, such as, among others, the behaviors of individual organisms, the operations of organs within those organisms, and the activities of cells within them. Bechtel and Richardson (2010) explore these issues further, as outlined in the abovementioned paragraphs, by introducing two distinct strategies and examining their roles in more detail. However, I want to emphasize that, in both characterizations of mechanisms, the role of these strategies is paramount, serving as a means for scientists to discover mechanisms and build mechanistic explanations. These strategies will be revisited in Chapter 5, where pathway features and their investigative strategies, arguably different, will be explored.

SECTION 3. Glennan's (1996; 2002; 2017) Characterization of Mechanisms

I have structured this section into two parts. The first part delves into the earlier versions of the new mechanistic account developed by Glennan (see 1996; 2002). The second part of the section examines Glennan's most recent analysis of mechanisms (2017). I already explored, to some extent, Glennan's approach to the concept of causation (see Part I, Chapter 2) and the law of nature (see Part I, Chapter 3, Section 1). Thus, in this section, I particularly examine his approach to mechanisms.

3.1. Glennan's (1996; 2002) Earlier Characterization of Mechanisms

Glennan (1996) characterizes mechanisms as follows:

> A mechanism underlying a behavior is a complex system which produces that behavior by of the interaction of a number of parts according to direct causal laws. (Glennan 1996: 52)

Glennan characterizes a mechanism as a mechanism that underlies a behavior and argues that one cannot identify a mechanism without acknowledging what that mechanism does. According to this characterization, a mechanism is a complex system, capable of performing multiple functions simultaneously, with numerous mechanisms underlying its behaviors. By stating this, the concept implies a hierarchal structure of levels, akin to ideas from previous authors mentioned earlier. Glennan illustrates this concept with examples of complex system, such as cardiovascular and respiratory systems found in human bodies. These systems exhibit various mechanisms producing different behaviors, such as pumping blood or inhaling oxygen. Glennan asserts that these systems are composite, as they interact with each other. Each system comprises different parts (e.g., cardiovascular system having heart, veins, and arteries). Notably, some parts overlap with other systems. For instance, arteries and veins, present in both cardiovascular system and the respiratory system (e.g., in lungs). In this sense, Glennan not only describes what he considers a complex system but also introduces the concept of decomposition, aligning with the perspectives of other new mechanists.

Moreover, Glennan contends that the effectiveness of decomposition is dependent on the context, i.e., on whatever someone aims to explain. However, he asserts that descriptions of mechanisms are valuable to the extent that they accurately depict what really exists in the world. This emphasis is particularly important because decomposition can often imply a kind

of an anti-realistic or relativistic position. Recall that Bechtel and Richardson (2010) employ the heuristics of both decomposition and localization, which are, arguably, closely tied to an epistemic approach to the notion of a mechanism.

In this sense, Glennan also underscores the importance of an ontic approach. Thus, he suggests that the characterization of a mechanism involves both the heuristics of decomposition and localization (an epistemic approach), and the grounding of mechanisms, i.e., their parts, in the 'real' world (an ontic approach). In other words, he argues that the parts of a mechanism possess a degree of robustness and reality, suggesting that these parts of a mechanism can be considered as objects in various contexts. I will address further this issue in the next section when examining three aspects of new mechanisms.

Furthermore, expanding on his characterization, it is also important to note that, according to Glennan, the interaction of parts is controlled by laws. This view is unique among other authors characterizing mechanisms. He employs laws in the sense proposed by Goodman (1947), as generalizations supporting counterfactuals. However, I do not address further this point here since I already addressed it, to some extent, in Part I (see Chapter 3). It is noteworthy to highlight that in Glennan's later characterizations of mechanisms, the role of laws governing interactions between parts is replaced with the notion of *invariance* (see Woodward 2003).

Glennan (2002) revises his earlier characterization as follows:

> A mechanism for a behavior is a complex system that produces that behavior by the interaction of a number of parts, where the interactions between parts can be characterized by direct, invariant, change-relating generalizations. (Glennan 2002: S344)

The most significant change in this characterization of a mechanism is the replacement of direct causal laws, as mentioned in Glennan's earlier characterization, with invariant, change-relating generalizations. Glennan (2002: S345) elucidates this shift by clarifying that his prior characterization employed laws in a different sense than is commonly conceived by philosophers. That is, the term 'law' often carries the connotation of being exceptionless and universal, which leads to misunderstandings regarding the context of his characterization of mechanisms. In this sense, he aims to articulate this aspect differently. Thus, he adopts the concept of invariance and change-relating generalizations introduced by Woodward (2000; 2003) in his interventionist account of causal explanation. I have previously discussed the concept of invariance elsewhere, so I do not address it further here (see Part I, Chapter 2).

When examining scientific explanation, Glennan distinguishes between two versions, i.e., explanations of singular events and explanations of general regularities.[51] The mechanistic explanation of a regularity involves describing a mechanism whose behavior aligns with that regularity. To illustrate this, Glennan refers to the example of Mendel's law of segregation, where that mechanistic explanation describes the meiotic mechanism producing gametes. To accommodate this, he introduces his perspective on what constitutes a mechanical model. He characterizes it as a description of a mechanism that encompasses both the description of mechanism's behavior, and the description of a mechanism that accounts for that behavior. The former description pertains to the description of the *external* behavior of a mechanism, i.e., what the system does, while the latter pertains to a description of the *internal* structure, i.e., how the system performs that behavior. Glennan also terms this internal aspect as "the guts of the mechanism" (Glennan 2002: S347).

The example that Glennan provides of such a model is the input-output mechanism, i.e., a complex system existing in an environment with characteristic environmental events, namely inputs that trigger interactions between parts of the mechanism leading to an outcome, namely outputs. For instance, he illustrates this with the example of a computer running a word processing program. In this system, key presses are inputs, and characters appearing on the monitor represent outputs. In this instance of an explanation, the *explanandum* is a generalization, and the description that provides information about the input event and the interactions between parts leading to an output event constitutes the *explanans* part of an explanation. In this sense, Glennan highlights that a mechanical description can take the form of an argument, where the argument consists of premises representing statements in the mechanical description, and the conclusion is the conjunction of statements in the behavioral description. However, Glennan (2002: S348) emphasizes that the explanation is not solely rooted in the logical relationship between these descriptions but also in causal relationships between the parts of the mechanism producing the described behavior. I do not examine the causal relationship here, as I addressed it elsewhere (see Part I, Chapter 2).

When comparing earlier characterizations of mechanisms by Glennan with those from Machamer, Darden and Craver (2000) and Bechtel and Abrahamsen (2005), certain similarities and differences become apparent. For instance, parts in Glennan's characterization correspond

[51] I do not examine explanations of singular events since they are not pertinent to mechanistic explanations within molecular biology. Glennan (2010) extensively argues for this perspective, while other new mechanistic authors focus on regularities observed in the life sciences. The common example of an explanation of a singular event is the beginning of World War 1, which started with the assassination of Archduke Franz Ferdinand.

to entities and component parts in the abovementioned characterizations, while interactions align with activities and component operations. Similarly, behavior corresponds to the function of a mechanism and the phenomena as an *explanandum*. Differences arise in that Glennan highlights that mechanisms are complex systems, whereas other characterizations either do not specify that relation (Machamer, Darden, and Craver 2000), or simply assert that mechanisms are structures (Bechtel and Abrahamsen 2005). By outlining that a mechanism is a complex system, Glennan implies a hierarchy of levels, signifying that a complex system includes many mechanisms that can overlap in producing certain behaviors. Although the hierarchy of levels is not explicitly stated in other characterizations, those authors acknowledge the existence of levels of mechanisms, a topic explored in previous sections.

Additionally, by highlighting that a mechanism is a complex system, Glennan may implicitly acknowledge the specific organization inherent in mechanisms, which is explicitly emphasized in other characterizations but not directly in Glennan's. Besides the abovementioned distinction, there is also a unique emphasis on the role of laws, and later on, the role of invariance, which is not present in other characterizations. By invoking the role of invariance, Glennan explicitly outlines the role of causality within mechanisms, linking his characterization to Woodward's (2003) interventionist account of causal explanation. However, in a recent revision, Glennan has developed a minimalist characterization of mechanisms that can accommodate a broad range of systems, i.e., extending beyond those found in the sciences (e.g., physics and biology) or engineering fields (e.g., a flush mechanism).

3.2. Glennan's (2017) Recent Characterization of Mechanisms

In his recent characterization of mechanisms,[52] Glennan (2017) develops a minimal characterization as follows:

> A mechanism for a phenomenon consists of entities (or parts) whose activities and interactions are organized so as to be responsible for the phenomenon. (Glennan 2017: 17)

[52] Before presenting his minimal mechanism characterization, Glennan already begun to explore different kinds of mechanisms, i.e., extending beyond mechanisms as complex systems (e.g., see Glennan 2010); however, in this subsection, I particularly examine his latest comprehensive characterization, where he fully develops his minimal mechanism account.

Here, one can already observe that he includes Machamer, Darden and Craver's (2000) terminology regarding entities and activities. However, parts and interactions still play a role in his recent characterization. He also incorporates organization as an essential component when characterizing mechanisms. With this characterization, Glennan's motivation is to accommodate for a wide variety of commonalities across different fields and the mechanisms found in them (e.g., physics, chemistry, biology, psychology, sociology, history, engineering, etc.). Thus, following this minimal characterization, there are limited requirements for something to be considered a mechanism.

His minimal mechanism characterization is grounded in Illari and Williamson's (2012) framework, which I examine in the next section. Both characterizations represent a consensus view among new mechanists considering necessary conditions for something to be considered as a mechanism. Before moving on to examine the features of this characterization, it is important to note that this characterization is more suitable for addressing metaphysical issues concerning the ontology of the causal structure of the world. In this sense, in alignment with Levy's (2013), and Andersen's (2014) distinctions, Glennan's minimal mechanism characterization corresponds to a weaker sense of mechanisms, linked to the ontic approach. A more restrictive sense of mechanisms becomes pertinent when addressing issues in epistemology and methodology.[53] Glennan (2017: 19) acknowledges these distinctions, emphasizing that his minimal characterization of mechanisms can serve as a basis to which additional conditions can be added when accommodating epistemological or methodological issues. By articulating this characterization, Glennan aims to emphasize the necessary conditions regarding mechanisms as an ontological characterization of what mechanisms are as things in the world. In this sense, he specifically addresses the features, i.e., necessary conditions that mechanisms entail. I examine these features to a limited degree since I have already addressed them elsewhere (see Section 1). In particular, I highlight specific aspects differing from other characterizations.

Entities and activities are terms describing that mechanisms are compounds composed of items performing actions. As mentioned in the previous subsection, various new mechanistic authors use different terms for the same concept, i.e., entities and activities correspond to parts and interactions or components and operations. Recall that entities are parts that constitute mechanisms and that perform actions to produce the phenomenon for which a mechanism is responsible. Entities are referred to as nouns (e.g., proteins, organisms, etc.), and activities as

[53] I address this distinction in more detail in the following section.

verbs (e.g., pushing, bonding, etc.). Glennan in particular emphasizes that entities and activities are not abstract, i.e., "they are fully determinate particulars located somewhere in space and time: they are part of the causal structure of the world" (Glennan 2017: 20). In addition, he outlines that entities and activities are inherently linked, i.e., one cannot exist without the other. For instance, if there is a bonding activity, there must be something that is being bonded.

Although, at the first glance, the relationship between entities and activities seems similar to the case of Machamer, Darden and Craver (2000), Glennan (2017: 21) makes a slight distinction. He phrases the characterization in the sense that mechanisms consist of entities *whose* activities and interactions are organized to be responsible for the phenomenon. On the other hand, Machamer, Darden and Craver in their characterization phrase the referred part as entities *and* activities, implying that entities can exist without activities. Thus, Glennan highlights that his characterization better captures the notion that entities and activities are inseparable.[54]

Concerning other features, in Glennan's minimalistic mechanism view, organization is understood in the following way. By looking at Figure 13, a general schema of mechanisms, one can observe that there are two dimensions, i.e., a horizontal and a vertical.

[54] However, he points out that there is still an ontological agreement among new mechanists, since it is evident that, terminological differences put aside, entities and activities are strongly linked (see, in particular Glennan 2017: 21).

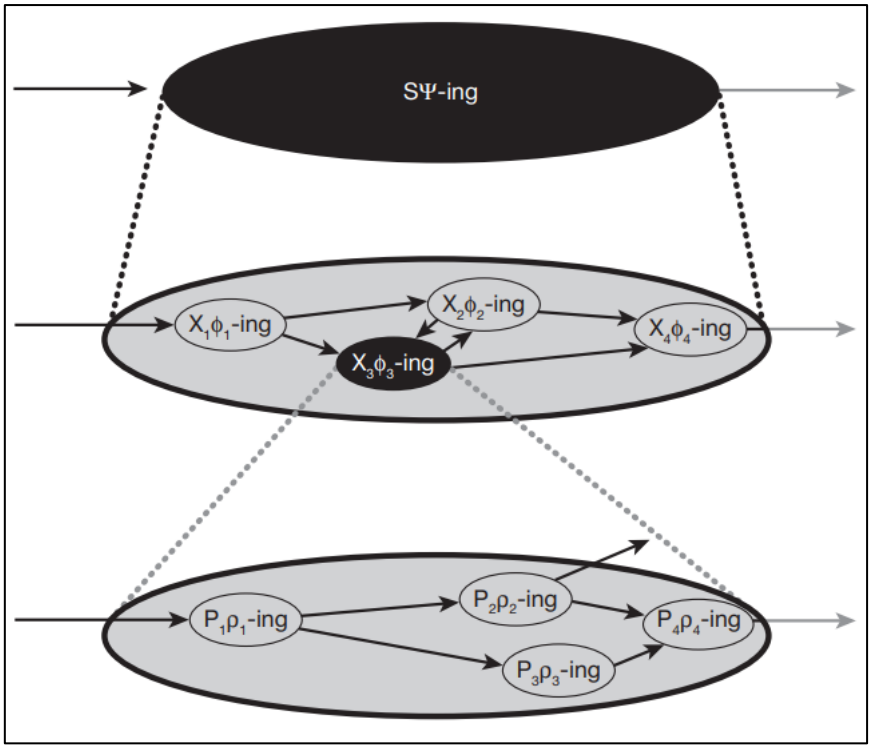

Figure 13: Mechanism schema (reproduced from Povich and Craver (2017) in: Glennan and Illari (eds.) (2017)).

The horizontal dimension is spatio-temporal and causal. In that dimension, there are represented entities Xs engaged in activities Φ-ing. The vertical dimension pertains to the relationship between a mechanism as a whole and the collective organized activities of its parts. Thus, the mechanism as a whole is a complex system S engaged in an activity ψ-ing. The mechanism's activity is constituted by the organized activities of its entities. Organization implies that mechanisms do not depend solely on their parts and activities but on how those parts and their activities are arranged. For instance, a car engine will not run if all the parts are just laid out unless they are arranged in a certain way. In the mechanism schema (see Figure 13), organization is represented via arrows between entities performing activities. Glennan (2017: 23) points out that these arrows primarily indicate the causal nature existing in a mechanism, i.e., that the primary form of organization in mechanisms is causal. Since I already examined Glennan's view on causation earlier (see Part I, Chapter 2), I do not focus on this issue further.

Lastly, I address his distinction between systems and processes, which, I believe, is not entirely clarified in the previous new mechanistic characterizations of mechanisms and is sometimes used to described mechanisms (in particular by Glennan, see 1996; 2002).

The metaphysical discussion about processes and systems is, to some extent, present in philosophy in general (e.g., see Rescher 1996), and in the philosophy of the life sciences (e.g.,

Dupre' 2012; Nicholson and Dupre' 2018). Glennan (2017: 25) examines them by taking into account the new mechanistic characterizations of mechanisms. However, he first delineates them in general terms. He outlines that systems have two features. Firstly, they are wholes made up of parts. Secondly, they do things, i.e., there exists an interaction between parts. He illustrates a system by providing an example of an ecosystem that consists of many parts, such as plants, animals, water, air, and soils. Those parts interact with each other by, among other things, providing energy to the system. In this sense, Glennan argues that systems are constituted of their part-whole organization and their persistent causal structure. On the other side, Glennan argues that processes are organized in a temporal and causal fashion. He provides an example of the development of an infant into an adult. The primary difference between processes and systems is that processes are organized usually into stages. Processes, contrary to systems, do not have to be regular or repeatable (e.g., a genetic mutation).

Following this general distinction between systems and processes, Glennan introduces the distinction between mechanistic systems and processes. The former considers systems that regularly engage in mechanistic processes, while the latter involves compound processes that emphasize how the outcome of one process depends on the interaction between parts and the organization responsible for that outcome. Glennan proceeds to offer some further distinctions. Firstly, systems differ from mechanisms in that systems do not always act, whereas mechanistic phenomena involve the occurrence of activities and interactions. Systems can be perceived as compounds and entities that include other entities and systems and can be part of mechanisms including further mechanisms. Secondly, systems depend on many mechanisms, e.g., a mouse is a system that produces waste and eats food, has a digestive mechanism, etc., but the mouse itself, according to Glennan, is not a mechanism. Lastly, not all mechanistic processes are actions of mechanistic systems. In this sense, Glennan (2017: 27) provides an example of kidneys as systems that, among other things, remove waste from blood and concentrate them in urine, which is a process.

To illustrate this system/process distinction in more detail, I present the diagram of variates of mechanism-phenomenon relations introduced by Craver and Darden (2013: 66) and further elaborated by Glennan (2017: 28).

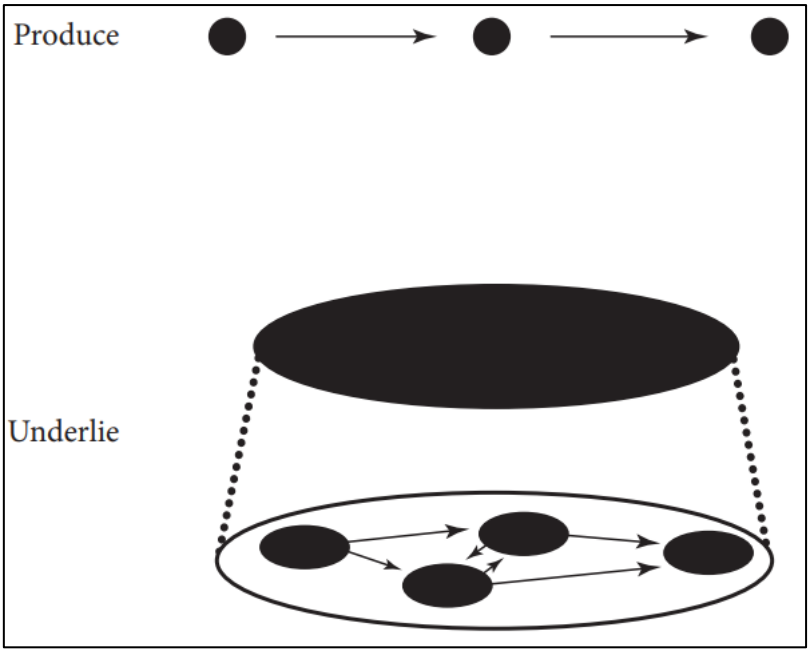

Figure 14: Mechanism-phenomenon relations (reproduced from Craver and Darden (2013: 66) and Glennan (2017: 28)).

In Figure 14, one can observe mechanisms producing phenomena and mechanisms that underlie phenomena. Mechanisms that produce phenomena do not have a higher level involved, but only the horizontal dimension and causal organization laid out. Mechanisms that underlie phenomena are represented in vertical levels in which the lower level represents the mechanistic constitution of the phenomenon. As Glennan points out, "mechanistic processes that underlie phenomena are always mechanisms operating within systems" (Glennan 2017: 28).

The abovementioned characterization by Glennan is closely connected with other characterizations I examined earlier. He develops his minimal mechanism characterization by merging the terminology and features found in other characterizations, and, to some degree, including features from his earlier characterizations. As I already mentioned, Glennan's minimal mechanism characterization is based on Illari and Williamson's (2012) characterization, which I examine in more detail in the next section.

SECTION 4. The New Mechanistic Account Summed Up

4.1. The Consensus View

In this section, I present the general characterization, namely the consensus view of the new mechanistic account. I believe that this characterization encompasses all the important features when one perceives mechanisms. I outline this new mechanistic consensus characterization (NMCC) as the one that I refer to in the rest of the thesis. Illari and Williamson (2012) characterize mechanisms in the following way:

> A mechanism for a phenomenon consists of entities and activities organized in such a way that they are responsible for the phenomenon. (Illari and Williamson 2012: 123)

The NMCC is motivated by both identifying similarities among the various characterizations of mechanisms provided by new mechanistic authors and accommodating an understanding of the commonalities of mechanisms across multiple scientific fields, such as, physics, chemistry, etc. I first examine two differences between the NMCC and the previously mentioned characterizations, followed by an outline of three features that NMCC shares with them.

Before examining two differences between various characterizations, I clarify Illari and Williamson's (2012: 120) view on the ontic/epistemic distinction mentioned throughout this chapter. They are primarily interested in providing a characterization of mechanisms *themselves*, i.e., an ontic approach similar to Glennan's. While acknowledging the importance of the epistemic approach, Illari and Williamson (2012: 121), along with other new mechanists like Craver and Bechtel, argue that "both ontic and epistemic explanation require real *mechanisms*".

The first difference between previously mentioned characterizations (excluding Glennan's 2017) and NMCC lies in the omission of characterizing mechanisms as systems or structures. This omission stems from the inflexibility of the concepts of structures and systems, as they may not always accommodate mechanisms as Illari and Williamson perceive them. To illustrate mechanistic behavior that defies the concepts of structures and systems, they present Darden's (2006) example of protein synthesis where the mechanism can create its own entities as needed (e.g., mRNA is made and broken down when needed). They also provide an example from astrophysics, where supernovae are described as mechanisms with violent and sudden

changes from one structure or system to another. The second difference is the absence of Machamer, Darden, and Craver's (2000) feature that mechanisms have "start or set-up" or "finish or termination conditions". Illari and Williamson believe that this is a feature of some mechanisms but not all. For instance, the Krebs cycle is a mechanism operating in a cyclical fashion.

Three features that NMCC shares with other characterizations are as follows: (i) identification of a phenomenon, (ii) decomposition into entities and activities relevant for the phenomenon, and (iii) organization of entities and activities producing the phenomenon. Features (i) and (ii) correspond to investigative strategies found in Bechtel and Richardson (1993; 2010) of localization and decomposition. Additionally, I have already mentioned in the previous sections that other new mechanistic authors also consider these strategies. Feature (iii) corresponds to the arrangements of entities and activities or parts and interactions between them, as found in all the previously mentioned characterizations.

Regarding feature (i), Illari and Williamson (2012: 123) use the terminology of Bechtel and Abrahamsen (2005: 422), asserting that mechanisms are *responsible for* a phenomenon. This terminology is adopted because a mechanistic explanation is deemed successful once the mechanism is discovered and described as the one responsible for the phenomenon. Additionally, to accommodate the diversity of mechanisms, considering that they perform different tasks (e.g., regulation) and exhibit various behaviors (e.g., growth). For instance, they illustrate this with the example of the mechanism of metabolism, which can perform more than one task, such as metabolizing glucose normally and metabolizing lactose in the absence of glucose. Moreover, they include the notion of responsibility in their characterization to emphasize counterfactual relation, acknowledging that the phenomenon can be both something actual and something modal, as exemplified in the mentioned case.

Concerning feature (ii), Illari and Williamson (2012: 125) adopt the terminology of both Machamer, Darden and Craver (2000), referring to entities and activities to describe component parts and operations within mechanisms, and the investigative strategy of decomposition found in Bechtel and Richardson (2010). Since these aspects have been extensively examined in previous discussions, I do not delve into them further. However, I would like to note that Illari and Williamson prefer this terminology (i.e., entities and activities) primarily because they consider both entities and activities as equally important in mechanisms, particularly in the production of a phenomenon. Although other characterizations use different terms to describe a similar relationship within mechanisms, Illari and Williamson (2012: 126) argue that entities

and activities provide the strongest rhetorical resistance to "default entity-bias", i.e., emphasizing that the language's rhetorical impact matters to scientists and philosophers.

Regarding feature (iii), Illari and Williamson (2012: 127) adopt Machamer, Darden and Craver's (2000) terminology, using the term 'organization' instead of Bechtel and Abrahamsen's (2005) term 'orchestrated functioning'. They do so because they consider 'orchestrated functioning' as a 'too strong' term, suggesting a tight integration and a more robust form of organization, which is prominently seen in designed systems. However, Illari and Williamson aim to broaden the scope of what mechanisms encompass; thus, they use the term 'organization' to describe distinct and particular relationships between entities and activities within mechanisms. They emphasize the significance of organization, along with other authors, primarily because organization is the final element in the process of producing a phenomenon. Only entities and activities organized in a certain way will produce a phenomenon, i.e., if organized differently, they would produce a different phenomenon.

By outlining these three features, Illari and Williamson aim to account for a wide variety of mechanisms across different scientific disciplines. They advocate for a consensus among new mechanistic characterizations, suggesting that NMCC captures this consensus. Subsequent works in the new mechanistic literature (e.g., see in particular Glennan 2017; Ioannidis and Psillos 2017), often refer to Illari and Williamson's consensus view, building upon it.[55] Thus, I consider NMCC as the consensus view when addressing certain issues related to the new mechanistic account in the following chapters, particularly concerning the similarities and differences when delineating alternative causal-explanatory structures, such as pathways and cascades. I conclude Chapter 4 by summarizing the new mechanistic account, arguing for three aspects present in the debate, as well as the process of a mechanistic explanation.

4.2. The Three Aspects of the New Mechanistic Account

I summarize the debate on the new mechanistic account by presenting a framework in which I address several issues discussed throughout this chapter. I advocate my own

[55] Ioannidis and Psillos (2017) argue that Illari and Williamson (2012) advanced the debate by advocating for a minimalistic view on mechanisms. However, they believe that even such an account deserves to be more 'minimal' and include less metaphysical issues (e.g., modified entities and activities), to accommodate scientific practice.

perspective on how these issues can be systemized, identifying three main aspects, namely ontic, epistemic, and strategic aspects.[56]

Previously, particularly in Sections 2 and 3, I discussed the ontic and epistemic distinction that is often prominent in the new mechanism debate. Recall that ontic implies that the mechanism itself, as found in nature, explains phenomena. In this sense, an ontic explanation relies on the actual mechanism in nature. In other words, the grounding of mechanisms, i.e., their parts, is in the 'real' world and possesses a certain robustness and reality.[57] The Epistemic approach suggests that an explanation stems from our mental activities, i.e., from building models of mechanisms. Although authors may assert that mechanisms are real systems or structures in nature, the act of offering an explanation is portrayed as an epistemic activity that serves the explanatory purpose.

In addition to these two distinct issues found in the new mechanistic debate, I emphasize a third one, namely investigative strategies. Recall that the strategies commonly found in new mechanistic literature are decomposition and localization. Decomposition is a strategy by which, roughly, one can break down certain system or a structure into its constituents. Localization, again roughly, is a strategy by which different activities proposed during the decomposition strategy are identified in the system or structure. In the following paragraphs, I address each of these aspects, namely, (a) the ontic aspect, (b) the epistemic aspect, and (c) the strategic aspect.

All the previously mentioned characterizations address all three aspects, with (a) being the central focus for each. Authors articulate (a) by delineating the constituents of mechanisms, i.e., highlighting the crucial features such as entities and activities, components parts and components operations, or simply the parts and interactions of a mechanism. In addition, a recurring feature in every characterization is organization, namely, the specific arrangement or composition of these entities and activities.

Recall that characterizations argued by Machamer, Darden, and Craver (2000), Bechtel and Abrahamsen (2005), and Glennan (1996; 2002) share these features and incorporate additional elements. However, NMCC, and later Glennan (2017), particularly emphasize the three core elements of mechanisms, and these are: entities, activities, and organization. I

[56] Levy (2013) draws a similar distinction by delineating three kinds of new mechanism, i.e., causal mechanism, explanatory mechanism, and strategic mechanism theses. While my distinction builds upon Levy's, it incorporates slight modifications based on the characterizations explored in this chapter. I do not address Levy's distinction here, as I do so in Part III (Chapter 8), where I examine whether natural selection can be characterized as a mechanism. Additionally, Anić (2022: 76) also highlights a similar distinction in the debate.

[57] In that sense, one can argue that new mechanists holding this view are realists about the world; however, the realism/antirealism debate is out of the scope of this thesis, so I do not address that further.

propose an approach suggesting that these three elements constitute (a) and correspond to the mechanism itself, as found in nature. In other words, these elements ground mechanisms, i.e., their parts, in the 'real' world, holding robustness through causal relations, as previously discussed in both Part I (see Chapter 2) and Part II. Nonetheless, I contend that models, i.e., epistemic activities, explain, rather than the actual mechanism found in the real world, which I address in the next paragraph.

New mechanistic authors address (b) by highlighting the challenge of representing mechanisms. They usually do so by emphasizing the significance of mechanism schemas and sketches (see Machamer, Darden, and Craver 2000; Craver and Darden 2013), or models (Bechtel and Abrahamsen 2013; Bechtel 2019). The emphasis stems from scientific practices where scientists describe mechanisms through various representations, either visual, textual, diagrammatic, or mathematical. These schemas are crucial to describe, explain, explore, predict, and control phenomena. For instance, consider the model of a human heart (see Figure 10). The role of such a model is to represent the relevant entities and activities of the mechanism, as well as their organization, leading to the production of a phenomenon.

The aim of a model is to portray relevant aspects of a mechanism operating in the world, effectively representing a mechanism found in nature. In this sense, a model aims to represent a particular mechanism, and that model serves to explain the occurring phenomena. Here, I in particular align with Bechtel and Abrahamsen's (2005) perspective, emphasizing mechanistic explanation as a cognitive activity involving representing and reasoning about nature. I support this view because I believe that visual presentations of mechanisms play a crucial role in scientific practice, serving as a vehicle for explaining phenomena. Consequently, scientific models are often simplified for presentation in textbooks to communicate with the nonscientific community. These models vary in their degree of abstraction, a topic extensively addressed in both Sections 1 and 2.

Lastly, (c), i.e., decomposition and localization, is most prominently discussed by Bechtel and Richardson (2010). However, as previously highlighted, other authors, to some extent, endorse these strategies. In the process of discovering mechanisms, scientists incorporate these strategies to identify mechanisms. Decomposition is a method for initiating explanations and comprehending structures or systems, while localization is a strategy that aids scientists in identifying and pinpointing entities and activities.

Aspects (a), (b), and (c), are present and intertwined in the debate surrounding the new mechanistic account. New mechanistic authors address these aspects based on their focus on scientific practice and mechanisms in general. Some authors focus more on the ontic aspect,

others on the epistemic, and some on the strategic. In conclusion, after examining the new mechanistic account and various mechanistic characterizations, I would like to highlight the mechanism's explanation process as I perceive it (see Figure 15). I do this by considering the new mechanistic account through the lens of these three aspects. I argue that mechanistic explanation is a process wherein scientists identify the mechanism as found in nature (a) by utilizing investigative strategies of decomposition and localization (c). Subsequently, they construct a representation of that mechanism, i.e., a model, that ultimately explains the phenomenon in question. In this sense, in Figure 15, I have placed aspect (a) at the center of the process, with aspect (c) on the left as the starting point for identifying the mechanism, and aspect (b) on the right as the final product of the process, i.e., representations (e.g., models) that explain phenomena.

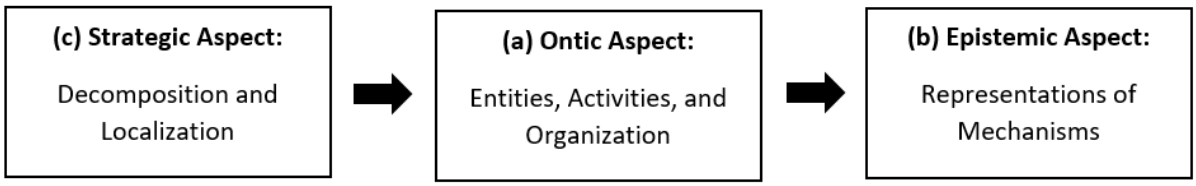

Figure 15: Mechanism's explanation process.

Let me illustrate this process with the example of the identification of the translation process in the mechanism of protein synthesis (see Figure 9). Namely, the elucidation of translation began in the 1970s, and by the end of the 1980s, most components of the translation mechanism had been identified (see Tahmasebi et al. 2019). This can be described as aspect (c), where scientists aim to identify the mechanism in nature, corresponding to aspect (a). Consequently, the activities of the identified components were reconstructed, leading to the elucidation of translation. This, in turn, enabled scientists to create a representation of translation, namely a model, related to aspect (b). This final aspect of the process explains translation, enhancing our understanding of both translation and the mechanism of protein synthesis.

CONCLUSION. Chapter 4

This chapter extensively examined three widely discussed characterizations of biological mechanisms, that is, Glennan's (1996; 2002), Machamer, Darden and Craver's (2000), and Bechtel and Abrahamsen's (2005). My focus was on highlighting the similarities and differences between these characterizations. Subsequently, I aimed to synthesize them into a consensus view, introducing NMCC, a perspective built on Illari and Williamson's (2012) and Glennan's (2017) framework. I argue that NMCC encompasses features found in all commonly used characterizations. Specifically, by emphasizing the role of entities and activities, organization, and the mechanism's responsibility for producing a specific behavior or phenomenon.

Additionally, I attempted to summarize the new mechanistic account by differentiating between three aspects, i.e., ontic, epistemic, and strategic aspects. The ontic aspect involves the use of entities, activities, and organization as the constituents of mechanisms. The epistemic aspect involves the use of representations, such as models, as vehicles for explanations. The strategic aspect encompasses strategies such as decomposition and localization. I argued that the mechanism's explanation process begins with the strategic aspect, where scientists decompose and localize to capture the mechanism as found in nature (ontic aspect). Finally, scientists construct representations of these mechanisms, i.e., the epistemic aspect, which serves as the final product of this process and explains phenomena.

Chapter 5: Alternative Causal-Explanatory Structures

INTRODUCTION. Chapter 5

In this chapter, I explore alternative causal-explanatory structures in molecular biology, such as pathways and cascades. In particular, Ross (see 2018; 2021; forthcoming) argues that these structures are distinctive from mechanisms.[58] According to Ross (2018: 552), the pathway concept[59] "refers to a group of causal factors, which are ordered in a sequence that leads to some final outcome of interest" (e.g., gene expression pathways, cell-signaling pathways, metabolic pathways, etc.). Additionally, according to her (forthcoming), cascades "involve amplifying steps that convert a small signal into a huge, explosive effect" (e.g., blood coagulation, nuclear fission, trophic cascades in ecology, etc.).

Pathways and cascades are distinct structures with unique features and investigative strategies that set them apart from mechanisms. Interestingly, new mechanists often overlooked these differences, interpreting various causal-explanatory structures within the mechanistic framework.[60] However, Ross (forthcoming) emphasizes that scientists "rely on a wide variety of causal concepts in their explanations". She argues against examining different causal structures solely through the lens of mechanisms. Instead, she advocates for recognizing other distinctive causal structures besides mechanisms, such as pathways and cascades. These structures have their own features, investigative strategies, and significant roles in scientific explanations. However, by arguing so, Ross does not dispute the role of mechanisms in scientific explanations; rather, she asserts that pathways and cascades, among others, also contribute significantly to scientific explanations, highlighting the diverse landscape of causal structures.

I address both of these structures, starting with pathways. For each, I delineate their features and their respective investigative strategies. In doing so, I draw parallels between those

[58] There are other authors, such as Thagard (2003) and Schaffner (2016) that examined pathways; however, Ross (2018; 2021) provides a more comprehensive examination of their role and features, particularly taking into consideration the new mechanistic account. I examine Thagard's account in more detail in Part III, Chapter 7, where I explore the role of pathways (e.g., glycolysis) in cancer treatment. In particular, I examine his perspective on the role of biochemical pathways in disease treatment. There, I outline the differences between Thagard's, Ross', and my own view on the distinction between pathways and mechanisms.

[59] Ross (forthcoming) uses the terms causal 'concept' and causal 'structure' interchangeably, indicating that the concept refers to the structure in the world. Consequently, I follow her interchangeable use of those two terms.

[60] For instance, for pathways, see Levy and Bechtel (2013). For cascades, see Craver (2007) and Brigandt (2013).

structures and mechanisms. Additionally, I introduce potential criticism towards pathways and cascades. Namely, by highlighting similarities between mechanistic features and the features of pathways and cascades. However, I address the relationship between them more extensively in the next chapter, where I advocate for different aspects of these structures as well as their specific hierarchy.

The chapter is structured as follows: in Section 1, I examine the pathway concept, in particular by outlining its four features along with specific investigative strategies. Additionally, I introduce a potential critique regarding pathway delineation. In Section 2, I outline the cascade concept, specifically focusing on its three features and investigative strategies. Moreover, I present several points that a new mechanist might argue against cascades.

SECTION 1. The Pathway Concept

Ross' (2021) motivation for distinguishing between different causal structures stems from observations in scientific practice. Namely, in the life sciences, scientists commonly explain phenomena using various concepts, including, among others, pathways. New mechanists often do not differentiate between these potentially distinct causal structures; rather, they argue that these concepts fall under the umbrella of a mechanism (see, e.g., Craver 2007; Robins and Craver 2009). However, Ross (2021) contends that it is unclear whether this singular concept adequately captures the diversity of causal structures in the life sciences. This uncertainty arises because biologists explore concepts such as gene expression pathways, cell-signaling pathways, anatomical pathways, and ecological pathways. The difference between pathways and mechanisms in these instances lies in the possibility "that a single pathway can be instantiated by different mechanisms, that distinct pathways can have similar mechanisms, and that pathways can be discovered without any knowledge of the mechanisms that underlie

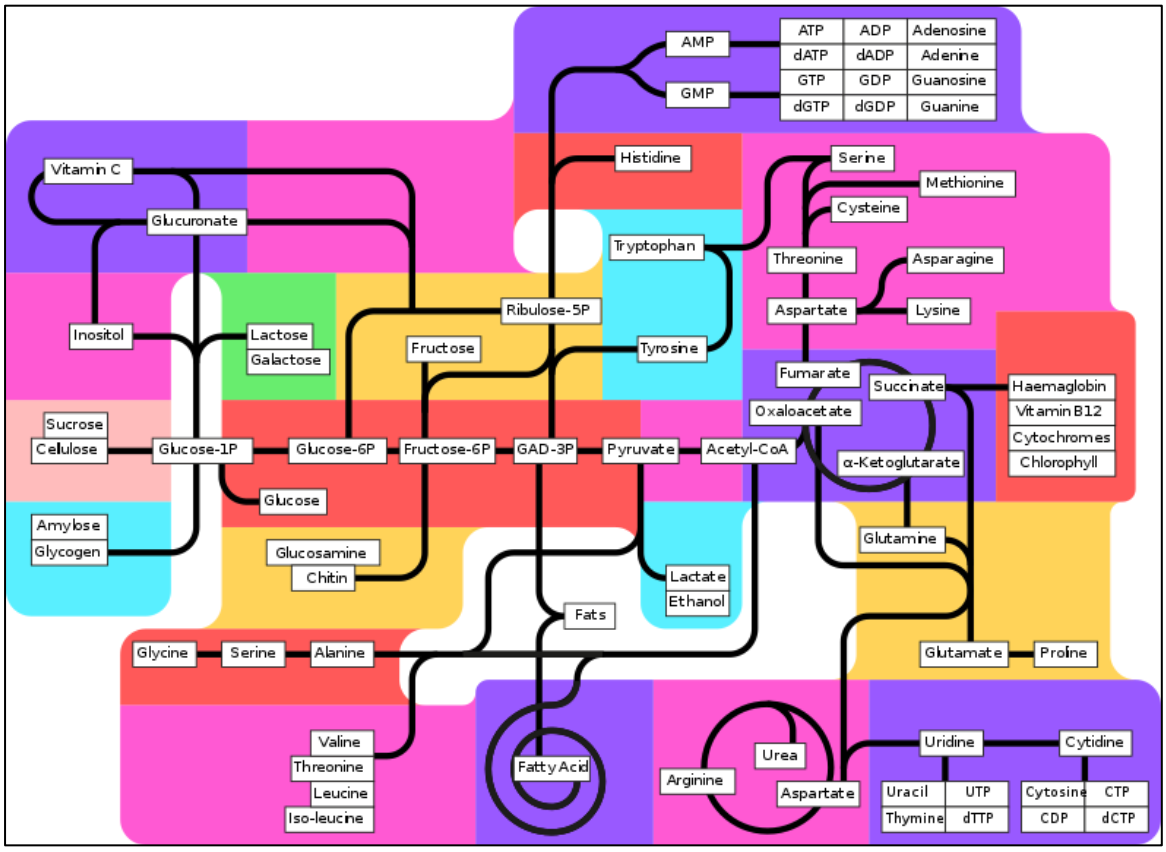

Figure 16: Metabolic pathways. The metabolic pathways are shown in a "metro-style" map and depict the complexity of pathway networks. In the map, there are several molecules emphasized, such as Glucose, Lactose, and Pyruvate, each being depicted as a sequential step in a certain pathway, that is, a starting point, namely, an initial substrate, or an outcome.

them" (see Ross 2021: 132). Thus, her aim is to provide an alternative understanding of nature and the limits of mechanistic explanation by introducing alternative causal structures.

Ross (2021: 137) characterizes the pathway concept as one that "refers to a sequence of causal steps that string together an upstream cause to a set of causal intermediates to some downstream outcome". For example, metabolic pathways (see Figure 16)[61] depict sequences of steps in the chemical conversion of an initial substrate to a final outcome. She emphasizes an important distinction between mechanisms and pathways concerning how biologists use analogies. In the case of pathways, analogies often imply that a certain system can be understood in terms of roadways, highways, or city streets, contrary to mechanisms where biologists typically utilize more machine-like analogies. In addition, she identifies four features that pathways encapsulate, as follows: (i) a sequence of causal steps; (ii) tracking the 'flow' of some entity or a signal through a system; (iii) abstraction from details; and (iv) an emphasis on the 'connection' aspect of causal relationships.

Besides these features, Ross outlines investigative strategies that differ from mechanistic ones, i.e., methodological considerations taken into account by scientists when identifying causal structures. These considerations include, for instance, scientific goals and features of the structures. In the case of the pathway concept, the identification of causal connections across entities is involved, without necessarily pointing out a specific effect of interest or a causal starting point. In other words, the strategy encompasses expanding or mapping out causal connections. The creation of a map of causal connections is then used to generate new causal connections and potential starting points, as depicted in Figure 16 where metabolic pathways are intertwined. Here, the same molecule can be a step in the sequence of a pathway or a starting/ending point of it. Ross draws an analogy between these maps with available freeways that a vehicle can travel along.

[61] Figure from: Fred the Oyster – Own work, CC BY-SA 4.0, https://en.wikipedia.org/wiki/Metabolic_pathway#/media/File:Metabolism_pathways_(partly_labeled).svg

1.1. The Pathway Concept Features

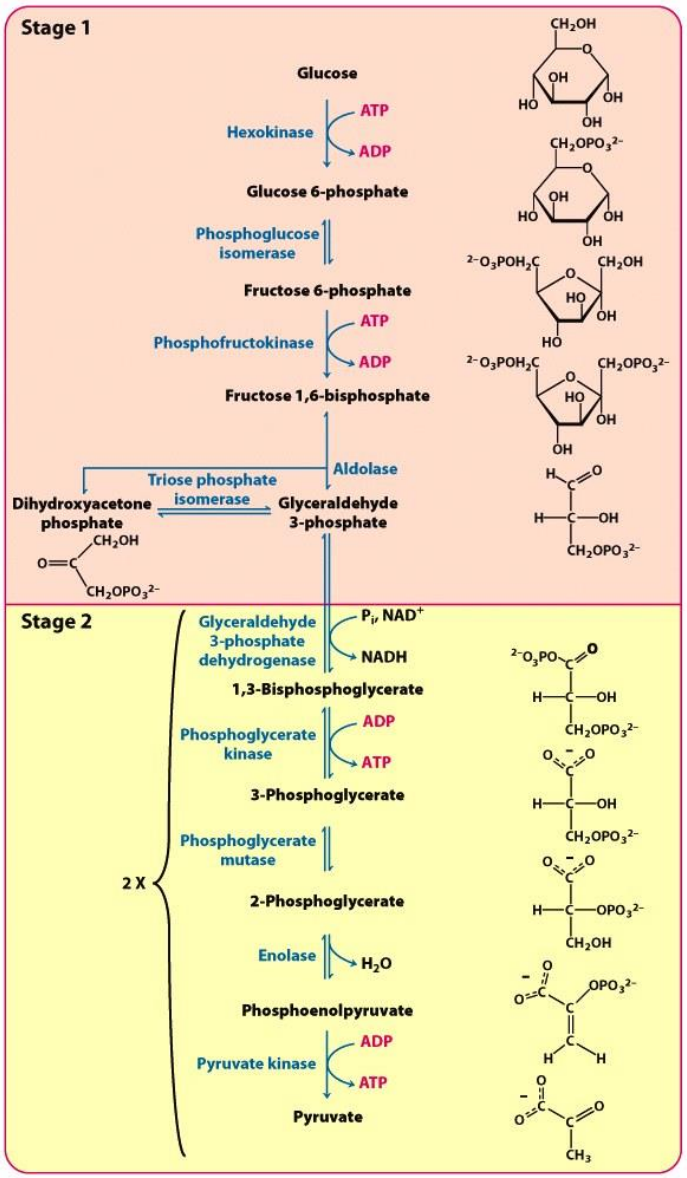

Figure 17: The glycolytic pathway (Reproduced from Stryer et al. 2012)

The first feature of pathways[62] involves considering them as processes that capture a sequence of causal steps. In particular, each step 'sets up' the following one, meaning that each step reflects the outcomes that should occur before and after subsequent steps in the process, leading to a final outcome. For instance, consider the glycolytic pathway (see Figure 17). Glycolysis consists of 10 sequential steps, starting with the first step where a glucose molecule is broken done via the Hexokinase enzyme. The outcome of that step is the next step, namely Glucose 6-Phosphate (G6P). G6P is followed by Fructose 6-Phosphate (F6P), and so on. Following this order of steps, the ultimate outcome of the process is the production of two pyruvate molecules.[63]

In pathways, as evident from the glycolysis example, there is "a fixed order of causally related entities" (Ross, 2021: 140). In other words, a causal chain exists in which every downstream product (e.g., F6P) depends on an upstream entity (e.g., G6P). Thus, an upstream entity serves as a causally relevant factor for a downstream product, i.e., G6P is causally relevant for F6P in the glycolytic pathway.

[62] It is worth noting that Ross (2018) introduced a similar list of features concerning the causal structure of pathways, especially in biochemistry. The features in that list include: (i) *causal control*, (ii) *material continuity*, and (iii) *fixed order*. While there is some overlap with Ross' (2021) list, particularly in the characterization, I focus on the most recent list as these features are more closely related to the comparison with mechanisms.
[63] I present glycolysis in more detail in Part III (see Chapter 7).

Although this feature is highlighted as characteristic for pathways, it does not actually capture the true difference between various causal structures, such as pathways and mechanisms. Thus, I do not examine it in detail here but later on when I compare pathways to mechanisms. Ross (2021) acknowledges this by stating that: "this first feature does not capture a true difference between the pathway and mechanism concepts because both can be understood in terms of causal steps" (Ross 2021: 140).

The second feature is more specific to the pathway concept, emphasizing a sense of "flow" (Ross 2021: 141-142) within pathways. This flow pertains to an entity or a signal passing through a certain system. In the example of glycolysis (see Figure 17), the metabolic pathway traces the flow of a chemical substance through intermediate stepwise changes. Ross argues that this feature is unique to pathways, particularly in comparison to other features on the list that oppose mechanistic ones. Particularly, because this notion refers to entities that are carried across the steps of a pathway. In other words, 'flow' involves the continuity of an entity passing though causal steps leading to an outcome. Ross draws a comparison with pathways found in ordinary life, such as freeways and plumbing systems. For instance, an entity that continuously 'flows' through a highway is a vehicle, and water is an entity that continuously 'flows' through a pipeline in the plumbing system.

Ross emphasizes that the notion of 'flow' in pathways is not just a metaphor for scientists; it highlights objective and physical features, i.e., demonstrating how pathways operate in the world. Scientists employ tracing and tagging techniques, wherein they tag a specific entity and observe its progress throughout the process. In this way, entities 'flowing' in a system can be manipulated to change outcomes. For example, if there is a blockage in a certain pathway, entities can be rerouted along 'bypass' routes, as seen in some therapeutic instances addressing pathologies.[64] Ross concludes this feature by pointing out that "something more is present in these pathway cases that is not found in all causal relationships" (Ross 2021: 142). According to her, this feature is not present in mechanisms.

However, Brzović, Balorda, and Šustar (2021) argue that "there are some difficulties with such a characterization of the flow feature: (i) it is, in our view, vulnerable to the same kind of difficulties as the notion of genetic (biological) information" (Brzović, Balorda and Šustar 2021: 7). In the sense that biologists usually think of the 'flow' of (genetic) information

[64] For more information about these therapeutic instances, see Ross (2021: 141).

involving ‘something more’ than mere causal relationships between macromolecules. They suggest that similar features to the ‘flow’ can be found in the new mechanistic account, particularly when considering the notion of ‘productive continuity’ in mechanisms, as pointed out by Machamer, Darden, and Craver (2000). ‘Productive continuity’ implies a sense of flow, with different stages in a mechanism making a difference for subsequent ones (see Craver and Darden 2013: 19). Thus, Brzović, Balorda and Šustar (2021: 7) argue that “the notion of ‘productive continuity’ also implies a sense of ‘flow’”. However, Ross highlights that the pathway flow “involves the permanence or continuity of something that travels along causal connections” (Ross 2021: 141). Namely, ‘something more’, an entity, is potentially found in pathways, which is not captured by the notion of ‘productive continuity’.[65]

The third feature of pathways, and the one that is, according to Ross, clearly distinctive from mechanisms, is dealing with their abstract nature of representations. Namely, pathway representations avoid including a large number of causal details and are presented in an abstract fashion. Ross argues that this feature sets pathways apart from mechanisms, as mechanistic representations often include a significant amount of detail. According to Ross, “one way that pathways do this is by representing only those causal factors that capture flow through a system as opposed to representing the entirety of factors that support or are causally relevant to this flow” (Ross 2021: 142). An example of this abstraction is seen in metabolic pathways that exclusively represent the ‘flow’ of metabolites and thus omitting other factors regulating that flow itself, such as enzymes, coenzymes, accessory substrate, temperature, pH etc. However, it is important to note that the new mechanistic account, as discussed earlier in this chapter, approaches this issue differently. It argues that mechanisms can be represented both in detail and abstractly, with the level of abstraction depending on the explanandum, i.e., varied based on the corresponding issue. Namely, omitting causal factors in mechanistic representations is a regular procedure. The same can be applied to pathways. For instance, considering the glycolysis example, representations may vary in detail based on the format, i.e., if it is for a high school textbook or a scientific paper. The glycolytic pathway can be also represented in more detail, since it is also divided in three distinct phases: (i) the energy investment phase or priming phase; (ii) the splitting phase; and (iii) the energy-generation phase (see Akram 2013). Scientists also target glycolysis in cancer treatment strategy, a topic explored in Part III, Chapter 7.

[65] For a comprehensive critique on this feature, see Brzović, Balorda, and Šustar (2021: 7-8).

The fourth feature considers the ‘connection’ aspect present in pathways. In other words, the aim of a pathway is to illustrate the causal connection between a sequence of steps, rather than delving into the details of ‘how’ those steps are connected. This feature is closely related to the previous ones, assuming that pathways have an abstract characterization and are structured as a sequence of steps. Namely, the aim of a pathway is to capture the ‘connection’ between these abstract steps. Consider the glycolysis example (see Figure 17). The glycolytic pathway aims to illustrate the causal connection occurring between sequential steps leading to an outcome. According to Ross (2021: 144), this feature specifically opposes a mechanistic feature using causal relationship such as ‘force’, ‘action’, and ‘motion’. In other words, mechanisms aim to show ‘how’ X causes Y, while pathways aim to show ‘that’ X causes Y. However, I examine the specific differences between pathways and mechanisms in the following subsection, pointing out similarities between them and their respective investigative strategies.

1.2. Differences Between Pathways and Mechanisms

Differences between causal structures such as pathways and mechanisms have already been addressed, to some extent, in Brzović, Balorda, and Šustar (2021). However, here, I specifically draw comparisons between the previously examined mechanistic features (see Chapter 5) and the four abovementioned pathway features discussed by Ross. I argue that two mechanistic features, namely (a) productive continuity and (b) the abstract representation of mechanisms, are shared with Ross’ pathway features. This leads to two possible outcomes. On one hand, the pathway list of features should be adjusted, and consequently, other specific features should be highlighted. On the other hand, mechanisms, as captured by new mechanistic authors, include pathways, meaning pathways are not a distinct causal structure; rather, mechanisms encompass pathways. I argue for the former solution, suggesting that pathway features should be adjusted if one wants to claim that pathways share the same explanatory status as mechanisms. However, I advocate that pathways should be, at least to some degree, distinctive from mechanisms, and I attempt to demonstrate that by examining their specific investigative strategies.

First, let me highlight some similarities between mechanisms and pathways. Ross (2021) identifies three features that mechanisms have, as follows: (i) mechanisms characterized in a constitutive manner, (ii) mechanisms as systems described in detail, and (iii) the mechanistic concept involves the use of terms such as 'force', 'action', and 'motion' in describing causal relationships. I briefly present these features and then argue that they are not representative. In other words, mechanisms, as depicted by new mechanistic authors, include many more features, even those that Ross highlights as distinctive for pathways, such as abstraction and productive continuity.

Feature (i) pertains to the constitutive nature of mechanisms as causal structures, i.e., "involving particular systems with higher-level behaviors that can be decomposed into lower-level causal parts" (Ross 2021: 134). This constitutive nature implies the use of traditional new mechanistic heuristics presented in the previous section, namely, decomposition and localization. In this sense, scientists identify parts of mechanisms, their locations, and interactions related to a behavior or an outcome of the explanandum in question. In other words, mechanisms are delineated based on parts that causally interact and produce a certain phenomenon. As Ross points out: "those causal factors that produce this behavior make up the mechanism and those that are not involved in this production are not mechanism components" (Ross 2021: 134). However, it is important to note that new mechanistic authors argue that mechanisms also refer to a sequence of causal steps occurring at the same level, i.e., they do not necessarily involve a system with higher-level behaviors that can be decomposed into lower-level causal parts. These kinds of mechanisms correspond to etiological mechanistic explanations (see Salmon 1984; Craver 2007; Craver and Tabery 2019). For example, new mechanists highlight the *temporal* organization for mechanisms, namely, the order, rate, and duration of successive component-parts' activities are all important features of a mechanism (see Craver 2001: 60). Furthermore, new mechanists emphasize the etiological characterization of mechanisms in the sense that it displays "a sequence of stages from beginning to end, and it would not be possible to change their order without gumming up the works (or making it a different mechanism entirely)" (Craver 2001: 61). Additionally, the etiological characterization of mechanisms is addressed in the idea of perceiving mechanisms as producing, underlying, or maintaining the phenomenon (see Craver and Darden 2013). For instance, the idea of production in the new mechanistic characterization of mechanisms is understood as causal

sequences terminating in a determined end-product (see Craver and Tabery 2019).[66] I return to this point later when examining feature (a), namely the productive continuity feature, which, as I argue, could capture both pathways and mechanisms.

Feature (ii) highlights the inclusion of a significant number of details when depicting mechanisms. According to Ross, biologists need complete descriptions of mechanisms, which are full of information and lack abstraction, to explain a certain phenomenon (see Ross 2021: 134-135). However, I point out that some new mechanistic authors emphasize the importance of abstraction. Ross acknowledges this by stating that: "some mechanistic philosophers subscribe to this 'abstract mechanism view'" (Ross 2021: 143) but she argues that the majority of new mechanists understand "mechanisms as highly detailed". However, mechanistic models do not always have to be highly detailed. I have examined this issue extensively in the previous section and I will return to this point later when examining feature (b), namely the abstract representation of mechanisms, which also captures pathways.

Feature (iii) is connected to the predominant emphasis placed by the new mechanistic account on the notions of 'force', 'action', and 'motion' to characterize causal relationships within mechanisms. Ross clarifies this feature by drawing an analogy with machines in ordinary life. Machines, she notes, have parts, such as levers and hammers that actively perform tasks and utilize 'force'. However, I argue that feature (a), namely the productive continuity feature, encapsulates the 'connection' aspect mentioned earlier when describing pathway features. I will revisit this point in the following paragraphs.

Now, let me turn to feature (a), productive continuity, and feature (b), the issue of abstract representations. According to new mechanists (see Chapter 4), more specifically Craver and Darden (2013: 19), feature (a) refers to the continuity between stages in a mechanism. Particularly, it demonstrates how mechanisms are productive and bring about a certain process or changes to an end state. Thus, to illustrate how a mechanism works, one has to depict each stage of the mechanism, emphasizing the stages that make a difference to what happens at subsequent stages. In other words, one stage of a mechanism produces the next. It

[66] To illustrate the etiological characterization of a mechanism, Brzović, Balorda and Šustar (2021: 5) present the simplified representation of the mechanism of protein synthesis. In this process, DNA molecules are transcribed into mRNA molecules, which are then translated into the corresponding sequence of amino acids, ultimately forming the resulting protein. According to their perspective, the causal chain typically represented as 'DNA→RNA→amino acid sequence' can be interpreted as an etiological causal chain.

is important to outline these stages comprehensively to present the entire mechanism from initiation to completion of the process, including all relevant stages.

Besides productive continuity, the term 'regularity' is closely associated with it. Regularity corresponds to the consistent way that mechanisms operate from the beginning to an end under the same conditions. Consider the example of DNA replication. The mechanism begins with a double helix, and through unwinding and bonding activities, it concludes with two helices. The fidelity of the copying mechanism influences the resemblance of the helices to the parent one. While some copies are more faithful, others may contain mutations, depending on the environmental stress during the process. DNA replication is an example of a linear mechanism.[67] Linear mechanisms consist of distinct stages where each stage leads to the next. All stages have a certain connection between them and consequently leading to an outcome. In the example of DNA replication, there are entities, such as DNA, and activities, such as unwinding and bonding, that are connected, i.e., in a relationship of a productive continuity. This relationship between stages can be described more in the notion of 'connection' than in the notion of a 'force', 'action', and 'motion'. Thus, it seems that the pathway's fourth feature, i.e., the "connection" feature, is not exclusive to pathways. Moreover, this example, and the notion of productive continuity, highlights that mechanisms also include connected steps and stages leading to an outcome. Consequently, the features (i) and (iv) of the pathway concept can be described in terms of productive continuity, which is a mechanistic feature.

New mechanists, particularly Levy and Bechtel (2013), advocate for feature (b) by pointing out the principle of 'less is more'. According to them (2013: 242), "abstraction is the omission of detail", i.e., abstract descriptions intentionally leave things out, allowing for openness. Abstraction comes in degrees, signifying that labeling a certain description as abstract implies the existence of a more detailed alternative. For example, a mechanistic description may represent some patterns of causal connections within a system while omitting other structural features and details considered irrelevant for the description at hand.

In this sense, abstraction plays a crucial role in explanation, particularly in identifying relevant causal organization and the generalization process. Levy and Bechtel describe generality virtue as a quality that highlights a common underlying causal structure, i.e., by

[67] Besides linear mechanisms, there are also present non-linear types of mechanisms, such as cyclical ones, e.g., the Krebs cycle in biochemistry.

abstracting representations of mechanisms, one can potentially observe that similar causal features play comparable roles in diverse systems. Hence, it appears that the role of abstraction is vital for mechanisms, extending beyond specificity, which Ross' claims as a defining feature of mechanisms. Thus, pathway feature (iii) requires revision, particularly because even in some instances of pathways, such as glycolysis, there are representations with varying levels of detail depending on the context. The glycolytic pathway, for example, can be depicted by dividing it into three distinct phases, such as, the energy investment phase or priming phase, the splitting phase, and the energy-generation phase (see Akram 2013).

In examining features (a) and (b), Ross emphasizes a specific interpretation of the mechanism concept, overlooking broader mechanistic characterizations encompassed by various new mechanistic authors. I have specifically highlighted examples such as productive continuity and abstract representation of mechanisms, which are also inherent in pathways, i.e., are pathways features that Ross delineates. Thus, I argue that it is important to draw clearer distinctions between pathway and mechanism features; otherwise, it might appear that pathways largely overlap with the features already established for mechanisms.

Now turning to the investigative strategies, which I believe differ between mechanisms and pathways. Mechanistic investigative strategies, namely decomposition and localization have been extensively explored in my previous examination (see Chapter 5), thus I do not delve further into them here. Ross contends that pathways employ a distinct investigative strategy. This strategy begins by "identifying causal connections across entities in some domain without specifying either an effect of interest or a causal starting point" (Ross 2021: 145). Notably, this strategy differs from mechanistic ones, specifically because it does not require identifying the so-called locus of control, i.e., whether it be a system or an effect of interest (see Bechtel and Richardson 2010: 35).[68] Instead, the pathway strategy involves creating a "road map" or a "network" of available causal routes. By developing such a map, scientists construct networks representing "available or potential causal connections that are relevant to a variety of explanatory outcomes and causal starting points" (Ross 2021: 145). In other words, this strategy opposes mechanistic strategies by 'expanding out', generating possible new points and routes for pathways. Contrary, mechanistic strategies focus on identifying a specific

[68] According to Bechtel and Richardson (2010: 35), the term "locus of control" in their terminology is linked to identifying systems that perform functions and the determination of what functions these systems perform initially. The identification of the locus of control precedes the application of the heuristics of decomposition and localization, i.e., it is a necessary step before initiating the process of decomposing a system or localizing its constituent parts.

explanatory target and then initiating the process of decomposition and localization. Ross compares these 'expanding out' strategies, i.e., causal maps, to "a set of available freeways that some vehicle can travel along" (Ross 2021: 145). This stands in contrast to mechanistic strategies, which she compares to decomposing parts of, e.g., engines or clock mechanisms.

Examples of these causal maps are evident in the already mentioned metabolic pathways, especially those found in databases, such as *WikiPathways*. Such databases serve scientists as networks of pathways upon which they can build new ones. In this sense, Boniolo and Campaner (2018) also argue that numerous pathway databases, such as, among others, the *Kegg Pathway* and the *Reactome*, highlight the prominent use of pathways in scientific practice. The existence of databases emphasizes the need to consider pathways more extensively in philosophical debates concerning molecular biology issues. I will return to this point again in the next chapter.

I argue that the investigative strategy mentioned above is distinctively suited for causal explanatory structures such as pathways. To illustrate, consider again the case of glycolysis. Glycolysis, a metabolic pathway, underwent a discovery process that spanned nearly 300 years. While a more detailed examination of its history is reserved for Part III (see Chapter 7), I briefly highlight its discovery process here as an exemplar of this investigative strategy.

The discovery of glycolysis commenced in 1747 when glucose was first isolated from raisins by Andreas Marggraf. However, it was not until the 1940s that all the steps of glycolysis were known, i.e., leading to the identification of the Embden-Meyerhof-Parnas (EMP) pathway. The EMP pathway is named after three distinguished biochemists, namely Gustav Embden, Otto Meyerhof, and Jakub Karol Parnas. The research on glycolysis, in particular its long discovery process, led to numerous breakthroughs in biology. These breakthroughs included the revelation of enzymatic activities, the identification of cofactors, the discovery of ATP, and so on. What is particularly noteworthy is that the glycolytic pathway continues to drive new insights in contemporary biology. For instance, despite being often portrayed in textbooks as a static sequence consisting of 10 steps leading to pyruvate, the EMP pathway has generated new interpretations. For instance, that under different conditions, pyruvate can undergo further metabolism (e.g., in as being converted to lactate in anaerobic conditions). Thus, EMP pathway example demonstrated its dynamic characteristic and a specific status of being in the intersection between various modes of metabolism and its adaptability across different organisms (e.g., where pyruvate can be further metabolized into ethanol in some

microorganisms) (see Gruening and Ralser 2021). Moreover, the dynamic process of discovering glycolysis, coupled with its central role in metabolism, has contributed to understanding the regulations occurring in metabolism, particularly in connected pathways. For instance, mathematical models for glycolysis, developed by, among others, Bruno Hess, described the oscillatory behavior of the glycolytic pathway, which influenced feedback and feedforward regulation pathways in glycolysis (see Hess and Boiteux 1968). These examples highlight a distinction between the investigative strategies of pathways and mechanisms. In other words, the pathway strategy appears to build networks, i.e., maps, emphasizing potential causal connections used to develop new pathways, explanatory outcomes, or causal starting points. Thus, unlike mechanistic strategies, these networks 'expand out', creating new causal points and routes for pathways.

I will examine this issue in greater detail in the next chapter. However, I highlighted it here to initiate an early distinction between mechanisms and pathways. I underscored that pathway features lack a clear distinction from mechanistic features, particularly noting two of them, namely productive continuity and abstract representation. Additionally, I outlined the differentiation in investigative strategies between pathways and mechanisms, using the example of glycolysis to illustrate its discovery process and networking abilities inherent in pathways.

SECTION 2. The Cascade Concept

Ross (forthcoming) introduces another causal structure, namely, a cascade. According to her, cascades possess its own features, analogies, and investigative strategies. As noted in relation to pathways, she derives the concept of cascades from scientific practice. In other words, the cascade concept encompasses causal elements found across various sciences, such as biology, physics, chemistry, psychology, and economics. Ross (2021; forthcoming) contends that scientific practice employs diverse causal concepts, emphasizing that mechanisms should not be the sole framework, i.e., causal structure, subsuming all others, such as pathways and cascades.

The cascade concept, according to Ross (forthcoming: 5-6), "refers to a unique causal pattern that occurs in variety of domains". In biology, examples include blood coagulation cascades and cell-signaling cascades; in physics, collision cascades occur; and in chemistry, there are oxidative cascades, among others. Cascades represent causal systems that differ from mechanisms and pathways, possessing unique features of their own. These features are as follows: (i) the involvement of an initial trigger, (ii) sequential amplification, and (iii) a stable progression from start to finish. Ross specifically emphasizes a crucial aspect of cascades, noting that they encompass "amplifying steps that convert a small signal into a huge, explosive effect" (Ross forthcoming: 1). Thus, the cascade concept is compared to avalanches or snowball effects, i.e., where a small cause triggers a substantial outcome.

According to Ross, cascades and mechanisms differ significantly, and these differences can be delineated as follows: (a) cascades lack constitutive elements in the mechanistic sense, (b) the study of cascades involves distinct investigative strategies, (c) cascades lack fine-grained causal detail, and (d) cascades exhibit clear and precise boundaries, i.e., their beginning and end are not context-dependent – they are objective features. First, I outline the three features of cascades. Next, I examine the four abovementioned distinctions between cascades and mechanisms. Lastly, I provide some critique regarding Ross' clear-cut differentiation between cascades and mechanisms.

2.1. The Cascade Concept Features

The first feature of the cascade concept is its incorporation of an initial trigger, often referred to as "a single main cause" (Ross forthcoming: 8). This initial trigger can be likened to the mechanism in a firearm responsible for releasing a bullet, the trigger of a firearm, a switch that initiates subsequent steps towards a specific outcome. In cascades, the causal trigger serves as the starting point, enabling a sequence of steps that ultimately lead to an outcome. Ross compares causal triggers to binary 'switch-like' values, emphasizing their binary nature – either 'on' or 'off'. Once these causal triggers are 'on', the likelihood of initiating subsequent steps, and, consequently, effects, is significantly high. In this sense, this attribute lends causal triggers considerable explanatory power, in particular because they start a certain causal process. Thus, exerting a significant influence on the entire sequence, culminating in producing large effects. In this sense, the cascade concept mirrors an avalanche, where a small snowfall triggers a massive effect, i.e., a huge surge of snow down a hill slope.

The second feature is sequential amplification.[69] According to Ross (forthcoming), "amplification refers to a situation in which a small amount of cause produces an amplified or large amount of effect" (Ross forthcoming: 9). She distinguishes between two types of amplification: a single-product and multi-product. The former involves amplifications with an increased amount of a single effect, while the latter entails an increased amount of different effect types. For example, a single-product amplification is seen in the blood coagulation cascade, whereas a multi-product amplification is evident in natural disasters. In the blood coagulation process, the enzyme X produces an increased quantity of a single product, such as enzyme Y. In contrast, during a natural disaster, such as a hurricane, a single cause generates several effects, including infrastructure destruction, floods, and devastation of natural areas. However, both types of amplification capture the basic characteristic of cascades, namely, that "every single unit of cause produces many units of effect" (Ross forthcoming: 10). Additionally, cascades involve a sequence of steps, each containing amplification, i.e., representing a continuation of amplifying steps. For instance, if enzyme X produces a hundred units of enzyme Y, and enzyme Y, in turn, produces a hundred units of enzyme Z, the overall system's gain equals the gain acquired at each step.

[69] Ross deems this feature as perhaps the most important one for depicting cascades.

The third feature is stable progression. This feature entails that, once triggered, cascades maintain "a strength that extends through their sequence of steps" (Ross forthcoming: 11). Namely, this means that once a cascade is set in motion, there is a high likelihood that its sequence will continue. In other words, cascades 'gain momentum' once 'ignited'. This feature also draws parallels to a chain-reaction, i.e., a reaction in which the effect of one step propels the next step, sustaining the process in a self-sustaining manner. Thus, once initiated, cascades can be challenging to stop, akin to a snowball effect.

According to Ross (forthcoming: 12), cascades are deemed "powerful causal systems". The abovementioned features can be summarized as follows: "cascades produce huge, expensive outcome and they do so in a way that is difficult to stop" (Ross forthcoming: 12). In other words, once this type of causal process is triggered, it generates an inevitable and substantial outcome that is basically unstoppable. Thus, the identification of cascades, in particular their triggers, becomes crucial in order to control the outcomes that, once initiated, unfold inevitably.

I concur with Ross that the abovementioned features are unique to cascades, setting them apart from any mechanistic characterizations I examined in Chapter 4. I return to these features in the next chapter, where I explore the relationship between mechanisms, pathways, and cascades. Before elucidating that relationship, I address some differences between mechanisms and cascades highlighted by Ross.

2.2. Differences Between Cascades and Mechanisms

Before delineating distinctions between cascades and mechanisms as presented by Ross (forthcoming), it is important to acknowledge her portrayal of mechanisms. In both Ross (forthcoming) and Ross (2021), mechanisms are depicted as having a constitutive nature, detailed representations, and described in mechanical language, i.e., featuring terms such as 'force', 'action', and 'motion'. I have previously examined and critiqued this portrayal, emphasizing its narrowness and failure to encompass the diversity of new mechanistic characterizations explored in Chapter 4. However, I do not reiterate this point here; rather, I focus on outlining the differences between cascades and mechanisms.

Firstly, Ross (forthcoming) highlights difference (a), namely that cascades lack a constitutive nature, i.e., that they do not adhere to a specific hierarchy. In other words, cascades exhibit 'level-agnostic' behavior concerning their causes and effects, which can be situated at the same level.[70] Moreover, this 'level-agnostic' behavior allows cascades to "spill over from one system to another as they ripple through some domain" Ross (forthcoming: 5). In other words, cascades exert a causal influence that moves both upstream and downstream, not strictly adhering to a bottom-up, level-specific progression.

Secondly, Ross addresses difference (b), asserting that cascades are investigated differently than mechanisms due to the absence of lower-level causal parts. Unlike mechanisms, cascades cannot be decomposed in a similar manner, as their causal factors extend across different levels. Consequently, investigative strategies for cascades require a distinctive approach. Additionally, Ross contends that fixing an explanatory target, as one does with mechanisms, is challenging for cascades since these targets "are not always relative to single outcomes" (Ross forthcoming: 5). This is evident, especially in cases of multi-product amplification, as previously discussed. Thus, an effective investigative strategy for cascades involves commencing with the trigger and then expanding outward, particularly by focusing on the effects produced by the cascade. Ross (forthcoming: 6) notes that: "this required a system-wide outlook, which contrasts with the more local focus on discrete mechanisms". Furthermore, investigative strategies for cascades emphasize the importance of focusing on the causal trigger. As mentioned earlier, the trigger serves as the starting point for the entire process, gaining momentum and often undergoing significant amplification. Thus, prioritizing the trigger is crucial for the purposes of manipulation, i.e., particularly due to the potential to control, prevent, and predict cascades. Ross illustrates this point by citing developmental cascades in psychology, where triggers for a certain behavior are rooted in early childhood. In this sense, manipulating such triggers enables control over several outcomes that may develop once the causal process is set in motion.

Thirdly, Ross (forthcoming) addresses difference (c), emphasizing that cascades lack fine-grained causal detail in their representations. She contends that these representations do not need to capture intricate details because "cascades capture 'that' a cause amplifies its effect, but they need not detail 'how' this amplification takes place" (Ross forthcoming: 6). In this sense, representations of cascades that serve as explanations require less detailed information.

[70] For more information on the example of these different levels, see Ross (forthcoming, footnote 21).

To illustrate this point, Ross offers the example of the cascade-like spread of the COVID-19 disease. She argues that we used "the same branching diagram to represent and explain the amplified spread of COVID-19 in various contexts, even though the mechanisms of spread differ drastically across these contexts" (Ross forthcoming: 7). For instance, the disease in question can spread through various mechanisms of airborne transmission, such as coughing, talking, sneezing, or contact with infected objects like door handles and groceries. In this sense, Ross emphasizes that the cascade structure is useful in highlighting the amplified spread of the disease, whereas mechanistic reasoning would delve into specific causal relationship for each transmission method. Thus, cascade structures prove to be explanatory, especially in the example of explaining the amplified transmission of COVID-19 across different contexts. Through cascades, one can demonstrate that a disease is more easily transmitted and challenging to control, outlining the significance of managing the early stages of disease spread.

Lastly, Ross addresses difference (d), contending that cascades possess clear and precise boundaries, namely, their beginning and end are not context-dependent, as she argues is the case with mechanisms. According to her, mechanistic philosophers, such as Craver (2009) and Bechtel (2015), who suggest that mechanism boundaries are context-dependent, i.e., are influenced by pragmatic factors tied to the goals and interests of an explanatory target. However, Ross argues that cascades exhibit specific boundaries, i.e., a cascade commences with "a small causal trigger", and concludes with "a large downstream effect" (Ross forthcoming: 8). Thus, cascades, contrary to mechanisms, are characterized by their lack of context dependency and instead feature distinct boundaries with a clear beginning and end.

To avoid redundancies, I do not readdress differences between cascades and mechanisms, namely differences (a) and (c), as I have previously highlighted that new mechanists do not consistently characterize mechanisms as constitutive and lacking abstraction. I also do not address difference (b) here, as I align with Ross in acknowledging that cascades employ different investigative strategies compared to mechanisms and pathways, a topic I explore further in the next chapter.

However, I briefly address difference (d). In particular, in some instances mechanisms do exhibit clear boundaries. For example, consider the mechanism of protein synthesis (see Figure 9), which has distinct starting and ending points. The mechanism is initiated with DNA replication and concluded with the synthesis of the protein. Moreover, Glennan (2017)

explicitly argues that "mechanisms have boundaries – starting points and ending points, insides and outsides – and so too do entities, activities, and interactions that make them up" (Glennan 2017: 36).[71] He distinguishes between three kinds of boundaries, and these are as follows: system/object boundaries, boundaries of processes, and boundaries of mechanisms themselves. System/object boundaries involve two kinds – the first one is about the boundary between a system and its environment, and the second one is about the separation of system into parts. Boundaries of processes include the boundaries between entities engaged in a process, i.e., temporal boundaries between steps and stages of the process. Here, Glennan (2017) again distinguishes two kinds of boundaries – the first one regarding the temporal boundaries of the beginning and the end of mechanical processes as wholes, and the second one about the temporal boundaries in the parts of the process, i.e., activities and interactions of the process. Third boundaries – the ones regarding mechanisms themselves, can be, according to Glennan (2017: 37), observed "in nature", e.g., think about the human body which contains many systems, each responsible for a specific behavior or activity (e.g., cardiovascular, respiratory, and nervous systems). In light of Glennan's distinctions, it becomes evident that mechanisms can indeed have clear boundaries. Therefore, it seems that this feature is not exclusive to cascades. It appears that features (a), (c), and (d) may not serve as clear distinctive points between cascades and mechanisms. I will delve deeper into this in the following chapter.

[71] However, Glennan (2017: 36) notes that it is "empirically and conceptually challenging to decide what goes inside or outside" when considering mechanism boundaries. He uses the term 'carving problem' that is previously outlined by Laura Franklin-Hall (2016).

CONCLUSION. Chapter 5

In this chapter, I explored alternative causal explanatory structures in molecular biology, namely pathways and cascades. I delineated their features and investigative strategies, as argued by Ross (2021; forthcoming). She asserts that both features and investigative strategies are distinct from mechanistic explanations. However, I challenged that view by arguing that only investigative strategies are distinctive, suggesting a need for revision of both pathway and, to some extent, cascade features, aligning them more closely with mechanistic features. I particularly pointed out two mechanistic features, that are now closely related to pathway and cascade's features, namely features (a) – productive continuity, and (b) – abstract representation of mechanisms.

Moreover, I emphasized the need for clearer distinctions in cascade features, particularly regarding the concept of clear-cut boundaries. While mechanisms, to some degree, exhibit clear starting and ending points (e.g., protein synthesis, see Glennan 2017), I contended that pathways and cascades should be more distinctive in this regard. Despite these considerations, I argued that pathways and cascades possess distinctiveness in their investigative strategies. Pathway structures involve building networks to highlight potential causal connections and 'expanding out' their respective connections, differing from mechanistic strategies that focus on decomposition and localization. Similarly to pathways, cascade investigative strategies center around 'expanding out', i.e., starting with the trigger and focusing on expanding out, emphasizing the effects produced by the cascade. Thus, this approach contrasts with mechanistic strategies, outlining the unique aspects of investigating cascades.

Chapter 6: The Relationship of Causal-Explanatory Structures

INTRODUCTION. Chapter 6

In this chapter, I examine the relationship among the previously mentioned causal explanatory structures: mechanisms, pathways, and cascades. Each of these structures has been delineated with distinct features, investigative strategies, and analogies. I draw parallels between them and highlight both their similarities and differences.

In this sense, I *firstly* examine three aspects of causal explanatory structures, previously discussed in relation to the new mechanistic account (see Chapter 4, Section 4). However, within this chapter, I expand this perspective and argue for the three aspects relevant to both causal structures, namely pathways and cascades. Additionally, I advocate for their respective explanation processes, which slightly differ from the mechanistic explanation process. Particularly, I highlight the distinction between pathways and cascades, on one side, and mechanisms on the other, by emphasizing the strategic aspect. This involves considering the distinct investigative strategies present among these causal structures. Specifically, mechanistic investigative strategies encompass decomposition and localization, while pathways and cascades employ 'mapping' or 'expanding out' investigative strategies.

Secondly, I focus on the specific hierarchical relationship among these structures. The aim of this part of the chapter is to highlight the specific relationship between these structures and particularly to outline mechanisms as the explanatorily privileged structure, i.e., the one that possesses the highest degree of explanatory power. The approach is twofold. Firstly, I present the distinction (a) between systems and processes, arguing that mechanisms exhibit both features. Secondly, I examine (b), namely, what explanatory power is, advocating for mechanisms as the privileged explanatory structure within the tripartite structure distinction. The distinction (a) is, to some extent, anticipated by Glennan (2017); however, I specifically emphasize this distinction in the context of the relation between the abovementioned tripartite distinction. In distinction (a), I advocate that mechanisms exhibit both *system* and *process*-like characteristics, encompassing *spatial* and *temporal* aspects. In contrast, pathways and cascades demonstrate more of a process-like characteristic, demonstrating a temporal aspect.

In examining (b), I draw on the dimensions of explanatory power outlined by Ylikoski and Kuorikoski (2010), namely *non-sensitivity* and *mechanistic detail* dimensions. Their

approach proves particularly valuable in this discussion as it introduces features pertaining to the interventionist account of causal explanation. I assert that within the tripartite causal structure's distinction, all structures are non-sensitive, that is, they encompass a strong relationship between an explanation and its background conditions. However, the difference lies in the mechanistic detail dimension. Here, I argue that mechanisms hold the advantage, establishing them as an explanatorily privileged structure. This assertion is based on the provision of more detailed explanations by mechanisms, evident through the explanans-explanandum relation containing a broader set of *what-if questions*. This, in turn, offers a deeper pool of statements that could potentially be false. Thus, I argue that mechanisms stand as a privileged explanatory structure in comparison to pathways and cascades.

The structure of the chapter is as follows: in Section 1, I argue for the three aspects of the pathway concept and the three aspects of the cascade concept. I illustrate them schematically and compare their similarities and differences. In Section 2, I examine the specific relationship among all structures and advocate for mechanisms as the privileged explanatory structure.

SECTION 1. The Three Aspects of the Pathway and Cascade Concepts

In this section, I introduce three aspects, namely ontic, epistemic, and strategic aspects for both pathways and cascades. I begin the examination with pathways. Ross' analysis of the pathway concept primarily encompasses "methodologically important considerations including scientists' goals, available strategies, and features of the system under the study" (Ross 2021: 133). Ross focuses on mechanisms and pathways as her 'system under the study'. Thus, it seems that she emphasizes the epistemic and strategic aspects of an explanation. Nevertheless, I contend that the ontic aspect of Ross' examination of the pathway concept can be identified in some of her outlined pathway features, such as features (i) and (ii), namely the pathway as a sequence of steps and the feature of 'flow' throughout the pathway. I argue this because Ross emphasizes that: "they highlight objective, physical features of these systems that reveal how they operate in the world and how we can best study, discover, and understand them" (Ross 2021: 141). Here, she particularly outlines the feature of flow in pathways. Thus, I begin the examination with the core elements found in the ontic aspect.

The ontic aspect of pathways encompasses two main features. *Firstly,* a pathway consists of a sequence of causal steps, meaning that "this sequence captures a fixed order of causal relationships that reflect which outcomes need to occur before and after others in the unfolding of a causal process" (Ross 2021: 139). According to this feature, I argue that the pathway found in nature, i.e., the one captured by pathway models, consists of a specific sequence of steps with causal relationships. Consider, for instance, glycolysis, a central metabolic pathway. This pathway follows a fixed order of steps, initiating with the molecule of glucose catalyzed by a specific enzyme, such as Hexokinase, followed by Glucose 6-phosphate, which is further catalyzed, leading to the subsequent step. This sequence reflects the fixed order of causal relationships that ought to happen in the glycolytic pathway.[72]

Secondly, I posit that a feature of flow encapsulates the ontic aspect of a pathway, in the sense that it refers to an entity carried throughout the pathway, that is, from one step to the other. This feature emphasizes an entity that is a constant in the process, i.e., an entity without which a pathway would not exist. For instance, metabolites are entities consistently present throughout metabolic pathways. Thus, this feature highlights an *objective,* physical feature inherent in a pathway.[73]

[72] This feature can be viewed as very similar to the corresponding mechanistic feature. I addressed this issue in the previous section, thus I will not analyze it further here.

[73] The issue with this feature arises when considering whether 'flow' refers to the entities flowing through the pathway or to the entire signal of entities flowing through the pathway. Moreover, Brzović, Balorda, and Šustar

The epistemic aspect of a pathway includes two main features of a pathway concept concerning how the pathway is represented. Namely, feature (iii), the abstract nature of the pathway, and feature (iv), the 'connection' aspect of the pathway. Feature (iii) highlights that representations of pathways abstract from details. That is, a pathway representation captures a specific order of causal steps relevant to the notion of a 'flow' of an entity through the pathway. For instance, representations of metabolic pathways focus solely on the flow of metabolites, excluding other factors, such as enzymes, temperature, pH, and so on. Additionally, pathways are represented with an economy of causal steps. For example, representations of developmental pathways condense the entire life cycle of certain organisms into just a few steps, typically ranging from four to twelve, which can be further subdivided into more detailed causal steps. Thus, Ross argues that pathways "are not intended to be exhaustive descriptions" (Ross 2021: 142). Feature (iv) emphasizes the 'connection' included in the causal relationship in the representation of pathways. In other words, pathways demonstrate 'that' X causes Y, without delving into the fine-grained details of 'how' they are connected. This feature is linked with the previous one, namely, pathways are represented abstractly by outlining the 'connection' between a sequence of steps in a particular pathway.

The strategic aspect of a pathway involves various investigative strategies and analogies within the pathway concept. Ross argues that pathways are associated with the strategy of "identifying causal connections across entities in some domain without specifying either an effect of interest or a causal starting point" (Ross 2021: 145). In this sense, scientists aim to create a map, i.e., a network or a landscape, depicting available causal routes. These maps serve as representations of potential and accessible routes for investigating new pathways, as seen in pathway databases such as *WikiPathways*. This process is thus 'expanding out', identifying a certain set of entities and then tracing their causal connections. This investigative strategy can be termed 'mapping out' since scientists develop a network of causal routes to further expand and potentially discover new pathways. To illustrate this investigative strategy, one can draw an analogy between pathways and freeways. Pathways can be envisioned as highways on which vehicles (e.g., entities) travel. These highways can further branch into new routes. Thus, a pathway network can be compared to a roadmap.

(2021) argue that Ross' 'flow' feature does not clearly specify whether entities are altered through the overall process, i.e., whether they retain their identity. Additionally, according to them, the notion of 'flow' faces similar challenges to the concept of the 'flow' of genetic information, involving 'something more' than causal relationships, similar to Ross' conception. Nevertheless, while this issue is important, it is not relevant for the above feature being perceived as an ontic aspect since I do not intend to address the status of the 'flow' concept in general, rather its status as an ontic aspect.

Let me now address the three aspects of cascades, beginning with the ontic aspect. The ontic aspect encompasses three features advocated by Ross (forthcoming), namely, the initial trigger, sequential amplification, and stable progression. The initial trigger represents a single cause that initiates the cascade, that is, a concept akin to the trigger of a firearm, as mentioned in the previous chapter. This causal trigger sets off subsequent steps in the cascade. Amplification refers to the large increase in the produced effect that follows the causal trigger. Additionally, subsequent steps in the cascade involve further amplification, resulting in a significant cumulative effect originating from a single cause. Stable progression pertains to the feature of a cascade whereby, once triggered, its sequence continues with high likelihood. Once initiated, the cascade gains momentum, leading to a chain reaction. Thus, cascades can be characterized as processes involving amplifying steps that convert a small cause into a large effect.

The epistemic aspect, similar to the pathway epistemic aspect, posits that cascades are represented abstractly, i.e., lacking fine-grained causal detail. The primary focus of a cascade representation is to convey the idea that a cause amplifies its effect; thus, as previously mentioned in the preceding chapter, Ross (forthcoming) argues that cascade representations demand less detailed information to be explanatory. To illustrate that, she uses the case of the amplified spread of COVID-19, as presented earlier. This aspect highlights a common feature with the pathway epistemic aspect, namely, the abstraction of representations. However, the representation of cascades particularly outlines the initial cause, i.e., a causal trigger that amplifies a specific effect. As it is evident in the earlier example of blood coagulation.

The strategic aspect concerns the investigative strategy of initially identifying the trigger of a process and subsequently expanding from the trigger to discern its effect(s). Specifically, the investigative focus is directed towards the trigger as the starting point of the entire process. The significance of the trigger lies in its role as the initiator of a process that typically undergoes large amplification, resulting in huge effects. Thus, for the purposes of control, prevention, and prediction, identifying the trigger becomes crucial. Following the identification of the trigger, the strategy involves investigating the expansion, i.e., the amplification subsequent to the trigger that leads to effects. Notably, this strategy shares some resemblance, to a certain extent, with the pathway investigative strategy, particularly in its emphasis on the temporal aspect of the investigation process.

Although Ross (2021; forthcoming) does not explicitly delineate between these three aspects, I argue that these aspects are inherent in her conceptualization of both concepts. In this sense, I would like to illustrate my view on the explanation processes of these concepts with

models similar to the one previously presented when summarizing the mechanistic explanation process (see Figure 15). In the models of both pathways and cascades, I also emphasize that the ontic aspect is central, where scientists aim to capture these causal structures as found in nature. However, the entire process in both structures begins with the strategic aspect. In the case of pathways, this involves initiating the search for potential causal routes in a network, akin to a roadmap. For cascades, it begins with the identification of the trigger and then tracks its expansion and large effects, resembling the strategy of 'expanding out', as present in the pathway strategic aspect. Taking this into account, the examination of a network serves as the initial step in the process by which scientists seek to grasp pathways and cascades in nature. The final aspect of the process is the epistemic one. Pathway representations are constructed by highlighting the 'connection' between steps of a pathway, emphasizing crucial steps through which entities 'flow'. In cascade representations, the focus is on emphasizing a cause that amplifies its effect. As mentioned earlier, these representations are abstract and typically depict these structures in an economical fashion. In pathways, emphasis is on crucial steps between entities, and in cascades, it lies in highlighting the initial trigger and the amplification producing large effects.

In Figure 18, I illustrate the explanation processes of both structures. By placing them one above the other, I aim to provide a comparative visual representation of the explanation processes for pathways and cascades. In both figures, arrows represent the temporal aspect, where the strategic aspect (c) initiates the explanatory process by aiming to capture the ontic aspect (a), i.e., the structure in nature, using their respective investigative strategies. The epistemic aspect (b) is the final step, where the structures are represented abstractly.

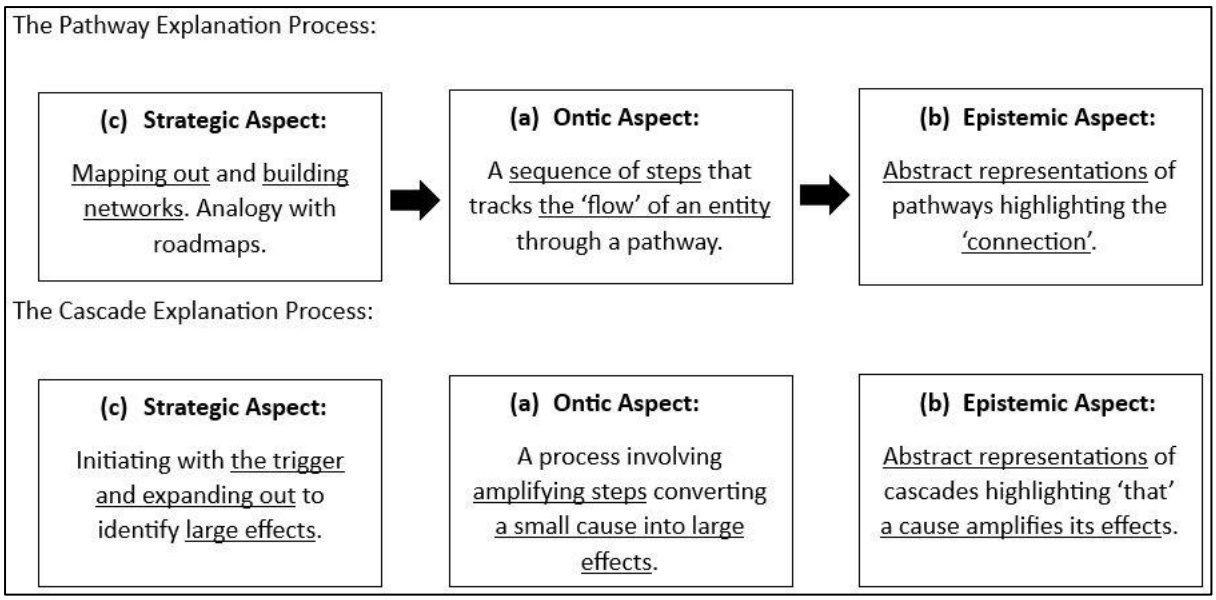

Figure 18: The Pathway and Cascade Explanation Processes

The aspects depicted in this figure exhibit notable similarities. By contrasting them, I aim to discern the nuanced differences in their respective explanatory processes. In terms of (c), both pathways and cascades involve mapping and expanding out investigative strategies. However, a distinction arises with the emphasis on the initial trigger in cascades. Pathways employ mapping and expanding out strategies resembling networks and roadmaps, while cascades prioritize the initial trigger as a crucial element in their investigative strategy. Moving to (a), pathways and cascades differ in how their causal relationships are structured. Pathways are structured as a sequence of steps with the flow of an entity. On the other hand, cascades are structured including an initial trigger leading to a large effect. Regarding (b), both structures are represented abstractly. However, there is a difference where pathway representations aim to capture the connection between steps, emphasizing the flow of an entity. In contrast, cascade representations aim to highlight that a cause amplifies its effects, focusing on the initial trigger and subsequent amplification leading to large effects.

Now, I intend to highlight the main difference between the explanation processes of mechanisms on one side, and the pathway explanation process on the other.[74] The difference between these processes lies mainly in the strategic aspect, specifically in how scientists approach the ontic aspects of causal structures in their practice. Although Ross (2021), based on her recent account, which I extensively covered elsewhere (see Chapter 5), might argue that

[74] Here, I use the pathway explanation process rather than the cascades' explanation process since their processes are fairly similar. Moreover, I continue to use the pathway concept in the subsequent chapters.

all these aspects are distinctive in comparison to mechanistic aspects, I advocate for an approach where only the strategic aspect is distinctive. Recall my earlier critique of Ross' pathway features (i) to (iv), which I included in both ontic and epistemic aspects. I argue that both of those aspects share features with their mechanistic counterparts. However, it is worth noting that pathway representations do tend to be more abstract than mechanistic ones.

In mechanisms and pathways, the strategic aspect is distinctive in that, in the mechanistic explanation process, scientists capture the ontic aspect by localizing and decomposing a targeted system. On the other hand, in the pathway explanation process, scientists map out, i.e., build networks used in the discovery of new pathways. I emphasize Boniolo and Campaner's (2018) perspective, highlighting their view that pathways are distinct from mechanism in scientific practice. Specifically, they note that scientific literature often includes precise mentions of pathways, usually equivalent to processes. In this sense, there are present precise characterizations. For instance, regarding molecular pathways, I refer to the definition from the Dictionary of Cancer Terms of the National Cancer Institute (NCI), which is as follows: "A series of actions among molecules in a cell that leads to a certain end point or cell function" (https:// www.cancer.gov/publications/dictionaries/cancer-terms?cdrid=561605, accessed: July 10th, 2022). Boniolo and Campaner highlight that this characterization underlines four different aspects of a pathway, as follows: (i) "any pathway is the representation of a series of causal actions among molecules"; (ii) "any pathway is the representation of a series of actions among molecules that end in a particular function"; (iii) "any pathway is the representation of a number of actions among molecules, where such a number can vary from n=1 (there is just one action leading to one molecular bond) to n=m (there are m bonding steps, depending on the length of the pathway considered)"; (iv) "any pathway is the representation of a temporally continuous process of bonding steps" (Boniolo and Campaner 2018: 6).

Since these four aspects of pathways are, to some extent, similar to Ross' (2021) depiction of pathways (apart from the mention of functions), I do not examine them further. Particularly, to ensure that Ross' (2021) concept of a pathway remains the focus of this thesis as I proceed to examine the explanatory hierarchy of all aforementioned causal structures.

SECTION 2. The Explanatory Hierarchy of Causal Structures

In this section, I examine the relationship among the previously discussed causal-explanatory structures, namely mechanisms, pathways, and cascades. Additionally, I aim to establish an explanatory hierarchy, positioning mechanisms at the top. Here, I summarize the issues discussed in the previous chapters, focusing on two main differences: (a) the system/process distinction, i.e., the spatial/temporal characteristics of strategic concepts, and (b) the degree of explanatory power among the structures. I begin with (a), specifically, the distinction between systems and processes, which was touched upon in Chapter 4. I emphasize this because I argue that the strategic aspect of these causal structures, the one aspect that I contend is distinctive between them, inherently carries this system/process distinction at its core.

In relation to difference (a), I argue that when considering the mechanistic strategic aspect, the concept of a system is paramount. On the other hand, when considering the strategic aspect of pathways and cascades, the concept of a process should take precedence. In this sense, I reintroduce Glennan's (2017) distinction between systems and process. Glennan argues that there exist both *mechanistic systems* and *mechanistic processes*. However, he also presents this distinction in a broader manner, which I apply to the discussion about strategic aspects of pathways and cascades. Roughly put, I argue that pathways and cascades are processes, while mechanisms are both systems and processes, i.e., there are mechanistic systems and processes, pathway processes, and cascade processes, and they can be differentiated through their respective investigative strategies.[75]

Recall that Glennan (2017) makes a general distinction between systems and processes by emphasizing two features of systems, namely, they are wholes made up of parts, and they do things, i.e., indicating an interaction among the parts. An example of a system is an ecosystem, which consists of parts such as plants, animals, water, soils, etc. These parts interact with each other and contribute to the energy of the system. In this sense, systems can be conceptualized as having a spatial dimension, i.e., involving various parts and a part-whole organization.[76] To illustrate this in a biological context, consider the heart as a system. It

[75] I do not cover the difference between mechanism systems and mechanism processes since I have already addressed that in Chapter 4. Here, I focus on the general distinction of systems and processes because it is applicable to pathways and cascades.

[76] I use the part-whole term since Glennan uses it too; however, one can think about it in the term of the hierarchy of levels, a term that is more prominent in Craver's work. Both of these terms acknowledge that some mechanisms have the structure of levels, i.e., upper and lower level – a mechanism consists of parts. I already mentioned this

exhibits a part-whole organization, with various parts (e.g., ventricles) that causally interact to produce an effect. Thus, a system, in this sense, represents a causal structure with a part-whole organization. On the other side, Glennan argues that processes are temporally organized yet maintain a causal dimension similar to systems. Processes are mainly manifested as *causal chains*, i.e., sequences of steps or stages involving activities and interactions leading to an outcome. An example he uses to illustrate this concept is the development of an infant into an adult.

In this sense, I argue that pathways and cascades are causal structures characterized by features aligning more with processes than systems. Considering the two main examples of these structures mentioned throughout the thesis, namely, glycolysis as a pathway and blood coagulation as a cascade, it becomes evident that they lack the part-whole organization typical of systems. Instead, they are organized as sequences of steps (e.g., glycolysis involves a sequence with more steps, i.e., ten). Thus, I emphasize the temporal characteristic inherent in processes. In other words, for pathways and cascades, the temporal characteristic is prominent, unlike systems where the spatial characteristic holds greater importance. The main difference among these three structures, namely mechanisms, pathways, and cascades, lies in the fact that mechanisms exhibit more spatial, i.e., system-like characteristics, while pathways and cascades exhibit more temporal, i.e., process-like characteristics.

This distinction is evident in the strategic aspect, i.e., in investigative strategies. For instance, cascades lack the part-whole feature, indicating an absence of a hierarchical structure with lower-level interactions and parts contributing to higher-level outcomes. Cascades should be approached temporally, i.e., investigating them by starting with a trigger – a single cause – and then expanding out towards the effects. As Ross points out: "Cascades have causal influence that is better understood as 'relationships between distinct' factors, as opposed to a 'whole and its parts'" (Ross forthcoming: 17). Pathways follow a similar approach, employing an investigative strategy of 'expanding out', i.e., building maps and networks of pathways. In this sense, pathways exhibit a characteristic different from the strategic aspect of mechanisms, which involves the heuristics of decomposition and localization. These heuristics require that the examined structures possess a certain hierarchy of levels, where a whole includes parts, and a system can be decomposed into its constituent parts. I revisit this distinction in Part III, where

mechanistic feature in Chapter 4. On the other hand, Ross (see forthcoming) argues that cascades do not have a hierarchy of levels.

I argue that natural selection can be characterized, at least partially, as a pathway, by referencing the more prominent temporal characteristic in causal structures such as pathways.

I illustrate the system/process distinction in Figures 19 and 20, where I emphasize the process characteristics of pathways and cascades. To discern the system and process characteristics of mechanisms, see Figures 13 and 14. There, one can observe that a phenomenon, i.e., an explanandum, can be decomposed into parts situated at lower levels, forming the underlying components of the explanandum. These parts are spatially organized in a specific way to produce the phenomenon (recall the NMCC characterization of mechanisms). Moreover, there are interactions among these parts, depicted as processes (again, see Chapter 4 for a reminder of the mechanistic system/process distinction).

However, I argue that pathways and cascades lack this system-like characteristic, and I outline their process-like characteristics in Figures 19 and 20. In particular, I highlight their respective strategic aspects of 'expanding out', namely building networks in the case of pathways and expanding post-trigger with amplification leading to effects in the case of cascades.

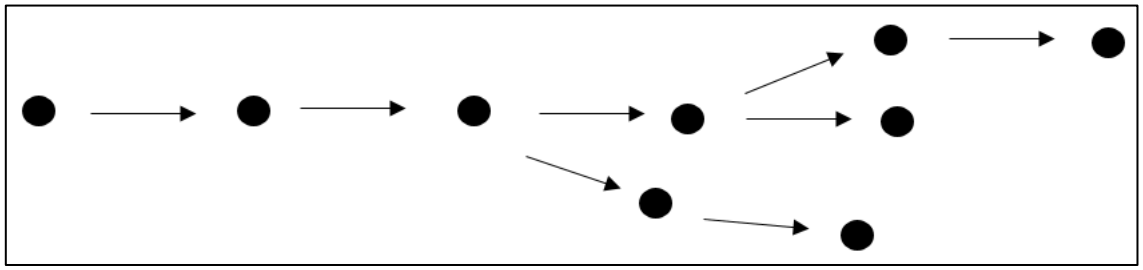

Figure 19: The pathway process-like strategic scheme.

In Figure 19, I represented causes as dots, and causal processes as arrows. This process exhibits branching, i.e., the development of a network of new pathways. In this sense, I aimed to visually convey the 'expanding out' strategic aspect inherent in the investigative strategy of pathways.

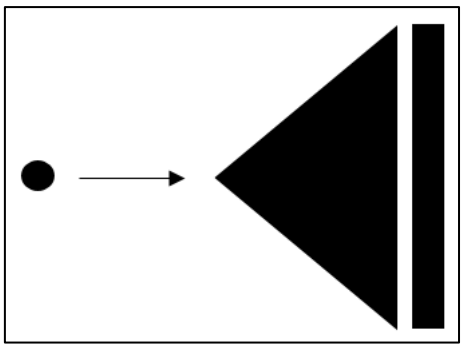

Figure 20: The cascade process-like strategic scheme.

In Figure 20, I depicted the causal trigger as a dot and the causal process as an arrow. The triangle represents the amplification and stable progression inherent in the cascade. Additionally, the rectangle represents the effects that are the outcome of the cascade (either single or multi-product outcomes).

Now, let me consider the difference between the mentioned structures (b), namely the degree of explanatory power among these structures. Here, I draw on Ylikoski and Kuorikoski's (2010) analysis of understanding explanatory power. I do so because they, as well as I, analyze it from the standpoint of the interventionist account of causal explanation (see Woodward 2003). Recall that in this framework, causal explanation involves tracing "objective relations of dependence" (Ylikoski and Kuorikoski 2010: 204). In other words, dependencies are objective because they are independent from our theorizing, conceptualizing, and thinking processes. Thus, explanation revolves around counterfactual dependence. For instance, "X explains Y if Y depends on X in the sense that if X had not happened, then Y would not have happened either" (Ylikoski and Kuorikoski 2010: 204). Here, it is important to emphasize the significance of w-questions (*what-if-things-would-have-been-different* questions), covered in Part I (see Chapter 2). These w-questions are especially important for practical purposes, i.e., related to potential interventions on causes to manipulate effects. In this sense, scientists can predict possible outcomes depending on specific interventions. Having covered this account of causal explanation and with further exploration planned in Part III, I now turn to the role of explanatory power, particularly in the context of the hierarchy of causal-explanatory structures.[77]

[77] A hierarchy of causal structures in molecular biology has previously been proposed by Brzović, Balorda, and Šustar (2021) aligning with the argument that mechanisms are at the top of the hierarchy of causal structures. They advocated such a view through the case study of the *de novo* origin of genes theory. However, in this thesis, I take a more general approach to the hierarchy, i.e., exploring the system/process distinction and the varying degrees of explanatory power among different causal structures, namely by focusing on the non-sensitivity and mechanistic detail dimensions of explanatory power.

Ylikoski and Kuorikoski assert that 'explanatory power' is a quality evaluated in the context of competing explanations, i.e., where one explanation is deemed more 'powerful' than another. Although they acknowledge that "explanatory power can be an attribute of both theories as well as of individual explanations" (Ylikoski and Kuorikoski 2010: 206), I focus on the latter as it is more closely tied to the issue of the relationship between different causal structures. According to them, explanatory power comprises of five dimensions, as follows: non-sensitivity, precision, factual accuracy, degree of integration and cognitive salience. Additionally, there is an extra property associated with explanatory power, namely mechanistic detail. For the argued hierarchy of causal explanatory structures, I focus on two dimensions that I consider the most relevant for the relationship between structures, as follows: non-sensitivity and mechanistic detail. I highlight non-sensitivity as a dimension through which the mentioned causal structures are similar to each other, carrying a similar degree of explanatory power. I outline mechanistic detail as a dimension through which they differ, placing mechanisms at the top of the hierarchy.

Sensitivity is related to the relationship between an explanation and its background conditions. When sensitivity increases, i.e., when an explanation is influenced by a change in its background conditions (e.g., pH, temperature, etc.), it is considered weaker. However, when sensitivity decreases, i.e., indicating that a change in background conditions does not affect an explanation, the explanation is stronger, namely it possesses more explanatory power.[78] Thus, according to Ylikoski and Kuorikoski, an explanation is "less sensitive if it would continue to hold under a larger set of interventions" (Ylikoski and Kuorikoski 2010: 209) on the background conditions.

Now that I have introduced 'intervention' in examining non-sensitivity, I revisit Woodward's (2003) interventionist account of causal explanation, particularly in relation to sensitivity (see Part I, Chapter 2). According to Woodward:

> [A] causal claim is sensitive if it holds in the actual circumstances but would not continue to hold in circumstances that depart in various ways from the actual circumstances. A causal claim is insensitive to the extent to which it would continue to hold under various sorts of changes in the actual circumstances. (Woodward 2006: 3)

[78] Some authors might use the term 'robust' to describe an explanation that exhibits non-sensitivity (see Wimsatt 2007; Glennan 2017). However, it is important to note that robustness is a concept that requires its own analysis. In this thesis, my focus is specifically on the concept of explanatory power, and a detailed exploration of robustness is not within the scope of this discussion.

Here, one can observe how Woodward, similar to Ylikoski and Kuoriskoski, characterizes sensitivity. He ties sensitivity to his counterfactual theory of causal explanation by noting that causal claims have a relationship with background circumstances. He argues that one tends to "regard causal claims that are highly sensitive as defective, nonstandard, or at least importantly different from less sensitive causal claims" (Woodward 2006: 2). Thus, it seems that this dimension is crucial when distinguishing explanations from one another. In other words, more sensitive explanations would be deemed less explanatory, while the less sensitive ones would be considered as possessing more explanatory power. This distinction is particularly pertinent in sciences such as molecular biology. I examine three examples of causal explanatory structures, as follows: protein synthesis for mechanisms, glycolysis for pathways, and blood coagulation for cascades.

Protein synthesis is a mechanism that can be described as non-sensitive, i.e., it is fairly stable as a causal explanatory structure. Mistakes in this process are typically prevented by various structures, such as, for instance, ribosomes. During one of the stages of protein synthesis, namely translation, the genetic code is precisely translated into amino acids forming proteins. This process, that is, the pairing of codons with anticodons, is monitored by ribosomes, often referred to as the cell's protein-synthesis factory. While occasional mistakes can occur in this complex process, resulting in non-functional proteins, the error rate is approximately one mistake per 20 000 amino acids (see Frederick and Ibba 2009). This low mistake rate indicates that protein synthesis is a relatively stable and a non-sensitive causal structure. Although cells can experience potential forms of 'stress', such as change in the abovementioned background conditions (e.g., temperature change and oxidative damage), various control patterns, used by ribosomes, exist to halt the mechanism of protein synthesis, and thus prevent the waste of energy consumed during the process of synthesizing a protein, thus preventing the formation of a damaged protein. For instance, translational control is one such pattern that responds to stress by halting the elongation stage[79] of the translation of protein synthesis mechanism. In this sense, this translational control responds to stress by maintaining the intracellular protein homeostasis (see Liu, Han and Qian 2013). Thus, protein synthesis, as a complex mechanism controlled by several processes, can be described as a fairly stable and non-sensitive causal structure that explains the phenomenon of protein production in cells.

Glycolysis is a pathway that can be described as fairly non-sensitive, although with some distinctions from the previously described mechanism. Namely, enzymes, such as

[79] Three stages exist in mRNA (messenger RNA) translation, i.e., initiation, elongation, and termination.

Hexokinase, which play a crucial role in catalyzing the process, could be potential targets for treatment purposes, a topic further explored in Chapter 7. Glycolysis is a metabolic pathway present in almost all organisms, i.e., it evolved in three dimensions of life – Bacteria, Archaea, and Eukarya. This widespread of the glycolytic pathway already suggests the importance of the pathway and its non-sensitivity since it is present across diverse organisms with each having their own environmental conditions. Its flexibility, i.e., capacity to persist in such a wide scope of organisms, is evident through its occurrence in both aerobic, that is, in the presence of oxygen, and anaerobic, that is, without the presence of oxygen, conditions. Although they have slightly different outcomes – pyruvate enters the citric acid cycle in aerobic conditions and converts into lactate in anaerobic conditions. However, during both aerobic and anaerobic glycolysis the same amount of energy is released (four ATP molecules) (see Chaudhry and Varacallo 2022). Thus, the glycolytic pathway, similar to the protein synthesis mechanism exhibits a degree of non-sensitivity, i.e., demonstrating flexibility across various environmental contexts (e.g., the degree of complexity and controlling patterns in protein synthesis, and the variety of organisms in which glycolysis is found).

Blood coagulation is a part of hemostasis,[80] i.e., the process that stops the bleeding after injuring the body. The clotting cascade is responsible for sealing vascular damage. It is triggered through two pathways, i.e., the tissue factor pathway and the contact pathway. For the purposes of describing the clothing cascade, I focus solely on the former pathway. This pathway is named after the protein triggering it, i.e., the tissue factor (TF). It is also known as the extrinsic pathway, as it requires contact between the plasma, i.e., the liquid part of the blood, and an external element, i.e., outside the circulatory system, to trigger it. The TF pathway, in turn, activates the clotting cascade, involving several reactions including different enzymes (e.g., serine proteases). The outcome of the cascade is the formation of clots that halt the bleeding (see Smith, Travers and Morrissey 2015). This process is tightly regulated to confine clot formation to the injury site and prevent the formation of clots elsewhere in the circulatory system. There are several anticoagulant mechanisms in place that regulate this formation (see Gale 2011). Thus, it seems that this causal structure is having a fair degree of non-sensitivity, as well as the previous structures. Particularly, because of the regulatory mechanisms in place, as well as pathways that are triggering the formation of clots. However,

[80] Thrombosis is another aspect where the clotting cascade comes into play, leading to pathological conditions. In thrombosis, the clotting cascade can be triggered within blood vessels, leading to the formation of blood clots, known as thrombus. These clots have the potential to impede the normal flow of blood within vessels, posing a health risk (see Smith, Travers and Morrissey 2015).

the difference between these structures lies in the level of detail needed for their explanation, i.e., the mechanistic detail dimension.

According to Ylikoski and Kuorikoski (2010), mechanistic detail serves as both an evidential and an explanatory virtue, primarily because "people prefer detailed explanations to mere sketches of explanation" (Ylikoski and Kuorikoski 2010: 215). It is crucial to emphasize that this preference for detail pertains specifically to relevant information for an explanation.[81] Mechanistic detail is an evidential virtue because detailed explanations are considered more likely to be true. Detailed hypotheses, when presented, are often more easily tested, and potentially falsified. On the other hand, mechanistic detail is predominantly an explanatory virtue for several reasons. Firstly, the sheer amount of detail connecting explanans and explanandum enhances the richness of an explanation. Furthermore, the increased detail of the causal system or a process account for additional information about why the causes led to a particular effect, thereby expanding the understanding of the 'why' and 'how' questions related to a certain phenomenon. In this sense, this broadening of understanding facilitates the exploration of a wider range of *what-if* questions that one can answer if provided with the additional information about a certain phenomenon. This aligns with the significance of *what-if-things-had-been-different* questions, discussed earlier in Part I (see Chapter 2). Moreover, the additional information gained through increased detail means more *what-if* questions contributing to the illumination of possible outcomes if some parts of the structure were altered. Furthermore, the amount of added information improves the previously mentioned dimension of sensitivity. Particularly, a more detailed understanding of the link between the explanandum and the explanans allows for better exploration of what-if questions concerning the relationship between the background conditions and the explanation.

Following the analysis of the system/process distinction and the dimensions of non-sensitivity and mechanistic detail, I argue that mechanisms hold a privileged position among causal explanatory structures when compared to pathways and cascades. Particularly, in relation to difference (a), namely the system/process distinction, since mechanisms exhibit both spatial and temporal characteristics, distinguishing them from pathways and cascades, which primarily display temporal characteristics. The spatial characteristic is especially significant when considering the importance of mechanistic detail emphasized in difference (b).

[81] Salmon (1998) addresses the question whether irrelevant information renders an explanation unexplanatory. I do not address this issue because it surpasses the scope of this examination.

By examining two dimensions, namely non-sensitivity and mechanistic detail, I aimed to illustrate that the former is important in highlighting that all of the mentioned structures carry explanatory power, and the latter is important to emphasize that mechanisms carry more explanatory power. In particular, mechanisms are privileged because of their spatial characteristic that has potential for mechanistic detail. This richness in details provides more information about the relationship between explanans and explanandum, allowing for the exploration of a broader range of *what-if* questions. In conclusion, I argue that mechanisms are at the top of the hierarchy of causal structures in terms of explanatory power. Pathways and cascades, as distinct causal structures, possess comparatively less explanatory power than mechanisms.

CONCLUSION. Chapter 6

In this chapter, I examined the relationship among mechanisms, pathways, and cascades. Particularly, by outlining the parallels and distinctions between these causal explanatory structures. I examined three aspects related to pathways and cascades. I advocated for their respective processes of explanation, which differ slightly from the mechanistic process of explanation. I specifically highlighted the main difference between all the structures, which lies in the strategic aspect, i.e., revealing different investigative strategies utilized in pathways and cascades.

In addition, I focused on the specific hierarchy among these causal-explanatory structures. I argued that mechanisms hold a position of explanatorily privilege, i.e., mechanisms are the structure possessing the highest degree of explanatory power. I advocated for this view by outlining two key differences. *Firstly,* the distinction between systems and processes was explored. I advocated that pathways and cascades exhibit more of a process-like characteristic, particularly because of their temporal emphasis. On the other hand, mechanisms encompass both system and process-like characteristics, incorporating the spatial component as well. *Secondly,* I examined the difference in the explanatory power between these structures. I analyzed Ylikoski and Kuorikoski (2010), namely their proposed two dimensions, that is, non-sensitivity and mechanistic detail. I emphasized the latter one as more significant for deeming mechanisms as explanatorily privileged structure. This is particularly because mechanisms provide more detailed explanations, evident in the richer relationship between explanans and explanandum, and the capacity to address a broader range of *what-if* questions. Consequently, considering the notion of explanation, mechanisms stand out by offering a deeper pool of potentially false statements, establishing them as the privileged structure compared to pathways and cascades.

CONCLUSION. Part II

In Part II, I examined the new mechanistic account, the leading account of causal explanation in molecular biology. Additionally, I examined alternative causal-explanatory structures in molecular biology, such as pathways and cascades. Considering the new mechanistic account, I particularly examined three canonical characterizations of mechanisms. I argued for a consensus perspective, namely NMCC, which I contend is the one encompassing features from the preceding canonical characterizations. Moreover, I provided three aspects of mechanisms, namely ontic, epistemic, and strategic aspects. I drew a schema of the mechanism's process of explanation, arguing that scientists aim to identify the mechanism as found in the nature (i.e., the ontic aspect) by employing investigative strategies of decomposition and localization (i.e., the strategic aspect), and then create a representation of that mechanism, e.g., a model, which explains the phenomenon in question (i.e., the epistemic aspect).

Regarding the alternative causal-explanatory structures, namely pathways and cascades, I examined their features, investigative strategies, and analogies as advocated by Ross (2021; forthcoming). I contended that these structures share some features and aspects with mechanisms, particularly pertaining to the ontic aspect. However, they differ from mechanisms if one considers their respective investigative strategies, namely the strategic aspect. This strategy involves 'mapping' and 'expanding' out methods, contrary to decomposition and localization strategies employed by mechanism. Moreover, I outlined pathway and cascade's ontic, epistemic, and strategic aspects, along with their unique processes of explanation.

Lastly, I pointed out an important difference between mechanisms, pathways and cascades, namely the system/process distinction. I argued that mechanisms have both system-like and process-like characteristics, while pathways and cascades have process-like characteristics. The main difference between the two characteristics lies in the spatial and temporal characteristics, with systems having a spatial aspect, and processes having more of a temporal characteristic. Furthermore, I argued that mechanisms are an explanatorily privileged structure. Apart from the previously mentioned system/process distinction, I considered two dimensions of explanatory power introduced by Ylikoski and Kuorikoski (2010), namely non-sensitivity and mechanistic detail. I outlined one dimension, namely mechanistic detail, by which mechanisms hold an explanatorily privileged status among causal structures since they

possess more depth in their explanations, that is, more *what if* – questions and statements in the explanans – explanandum relationship.

PART III: The Application of Causal-Explanatory Structures

INTRODUCTION. Part III

In Part III, I examine pathways and mechanisms within the context of two case studies, namely *glycolysis* and *natural selection*. The former is a pathway that can be exploited for the purposes of treating cancers. The latter is, arguably, a mechanism of evolution. I explore both structures, aiming to emphasize the significance of, on one side, the causal explanation in scientific practice (e.g., for cancer treatment) and, on the other side, the role of characterizing certain biological processes as causal explanatory structures (e.g., the characterization of natural selection as a causal explanatory structure).

Firstly, I revisit the pathway concept previously explored in Part II (see Chapter 5). Particularly because here I introduce another approach, namely Thagard's (2003), which aims to characterize biochemical pathways and elucidate their role in medical treatment. I distinguish between Ross (2021), Thagard's (2003), and my own approach to the pathway concept by revisiting the three aspects of the pathway concept outlined earlier, namely ontic, epistemic, and strategic. In particular, I emphasize the epistemic aspect, arguing that pathway representations involve a strong temporal characteristic, which proves suitable for the case study of inhibiting the glycolytic pathway for cancer treatment. Glycolysis is the central metabolic pathway responsible for the cellular breakdown of glucose. It involves ten steps and two stages, namely investment and pay-off stages, resulting in the breakdown of a molecule of glucose to two molecules of pyruvate. Energy in form of ATP is released during this process, catalyzed by various enzymes. Glycolysis is used by normal cells to acquire energy; however, it has been discovered that cancer cells use glycolysis as their 'fuel' at a much higher rate than normal cells – up to 200 times more (see Gill et al. 2016). Scientists are exploring strategies to target glycolysis to hinder the growth of cancer cells. I outline two potential targets, namely Glucose transporter 1 and Hexokinase 2.

I argue that Woodward's interventionist account of causal explanation serves as a particularly useful conceptual framework for these case studies. Thus, I assert that it is important to comprehend the information in pathways, i.e., their causal explanatory structure, especially to intervene on potential targets, that is, parts of the pathway. This is evident in the case of glycolysis, where one can disrupt the process by intervening on certain causes, such as enzymes, to prevent the growth of cancer cells.

Secondly, I examine natural selection, a process of evolution. Natural selection has been characterized in various ways, depending on different authors, including as a concept of force, a statistical concept, and as a causal structure (e.g., a mechanism). I am interested on the characterization of natural selection as a causal structure, particularly considering the ongoing debate on perceiving natural selection as a mechanism, especially within the framework of the new mechanistic account. However, I introduce another perspective by considering natural selection as a pathway, that is, a distinct causal structure that I argue can, at least partially, better characterize the nature of natural selection. In this sense, I explore the specific schema of natural selection provided by Skipper and Millstein (2005), which divides natural selection into seven stages. I contend that Stages II, III, and IV exhibit *pathway-like* characteristics, while the remaining stages display *mechanistic-like* characteristics. Moreover, I advocate for specific pathway and mechanistic aspects, specifically their respective *strategic* aspects (see Part II, Chapter 5).

The structure of this part of the thesis is as follows: in Chapter 7, I examine the role of the glycolytic pathway in treating cancer, specifically how this pathway can be targeted to cut off the energy supply to cancer cells. The chapter is divided into three sections. The first section presents Thagard's (2003) conception of a biochemical pathway. The second section examines the glycolytic pathway in general, providing its interesting historical perspective and discovery process. This section emphasizes the importance of understanding causal explanatory structures, such as the glycolytic pathway, for treating various effects (e.g., cancers). The third section links the case study of glycolysis and tumor growth with the interventionist account of causal explanation.

In Chapter 8, I address the issue of characterizing natural selection. The chapter is divided into two sections. The first section explores the debate on whether natural selection should be perceived as a mechanism. The second section argues that the natural selection schema, proposed by Skipper and Millstein (2005), partially exhibits pathway-like characteristics.

Chapter 7: Pathways in Molecular Biology: Glycolysis and Cancer Treatment

INTRODUTION. Chapter 7

In this chapter, I examine the case study of inhibiting the glycolytic pathway to prevent the growth of cancer cells. I address the elucidation of the pathway's representation and highlight its distinctions to mechanisms. In this context, I delineate between Ross' (2021), Thagard's (2003), and my views on this distinction. Additionally, I outline the conceptual framework of the interventionist account of causal explanation, which underlies the case study in question.

Firstly, I focus on Thagard's (2003) concept of a biochemical pathway, which differs somewhat from Ross' (2021) pathway concept, extensively discussed elsewhere (see Part II, Chapters 5 and 6). I aim to distinguish between pathways and mechanisms, focusing on three aspects thoroughly examined in Chapter 6, namely, the strategic, ontic, and epistemic aspects. The strategic aspect relates to investigative strategies, the ontic pertains to the features of these causal structures in nature, and the epistemic to their representations. I aim to clarify that my view differs from Ross' stance by arguing that pathways and mechanisms share similarities in the ontic aspect. In contrast to Thagard, who advocates for a close similarity between pathways and mechanisms in general, I argue for a distinction between them in the realms of the strategic and epistemic aspects.

Secondly, I examine the relevance of the glycolytic pathway for cancer treatment. Glycolysis, a central metabolic pathway, consists of ten sequential steps. During this process, a glucose molecule is broken down into two molecules of pyruvate, releasing ATP molecules in the course. Since the 1930s, following Otto Warburg's discovery, it has been established that cancer cells extensively rely on glycolysis as their primary energy source for growth. Consequently, specific segments of the glycolytic pathway are considered as potential targets for medical intervention, aiming to inhibit the pathway and hinder the growth of cancer cells. I highlight glycolysis as a promising pathway for cancer treatment purposes. In this sense, I outline Woodward's (2003) interventionist account of causal explanation, which serves as a conceptual framework for explaining interventions in the pathway. Consequently, various intervention methods can inhibit glycolysis, thus effectively preventing the growth of cancer cells.

The chapter is structured as follows: in Section 1, I present Thagard's (2003) pathway concept and compare it to Ross' (2021) depiction of pathways. I emphasize the distinction

between our perspectives on pathways and mechanisms. In Section 2, I shift the focus on glycolysis. I provide a brief overview of its history and structure. This is followed by an exploration of its relevance in cancer treatment. In Section 3, I outline the significance of pathways, as well as Woodward's causal explanatory conceptual framework, underscoring their importance in scientific practices within molecular biology.

SECTION 1. Thagard's (2003) Pathway Concept and the Three Aspects of Pathways

Besides Ross' (2021) characterization of the pathway concept, Thagard (2003) argues for an alternative perspective. As mentioned in Part II, Chapter 5, my focused leaned towards Ross' characterization since it is more comprehensive and it examines pathways across various biological disciplines. In contrast, Thagard's characterization has a narrower scope, focusing on *biochemical* pathways and their roles in diseases and medical applications.

According to Thagard (2003: 235), "a biochemical pathway is a sequence of chemical reactions in a biological organism". This sequence of chemical reactions explains "how cells carry out their major functions by means of molecules and reactions that produce regular changes" (Thagard 2003: 235). Recall the example of glycolysis. Glycolysis is a pathway comprising ten steps, each involving a specific reaction catalyzed by enzymes. For instance, the first step refers to the molecule of glucose being catalyzed by the enzyme Hexokinase. I will describe glycolysis in more detail in the next section.

Beyond glycolysis there are other examples of pathways that, as in the case of glycolysis, can be linear, that is, progressing from one step to the next (e.g., from one molecule to the other) or cyclical, i.e., involving a loop that produce initiating chemicals (see Thagard 2003: 236). According to Thagard (2003): "pathways are crucial to explaining how cells carry out their major functions, including energy acquisition, division, motion, tissue formation, signaling, and apoptosis (cell death)" (Thagard 2003: 236). For example, glycolysis explains cell's energy acquisition process. To analyze these pathways, scientists rely on their representations. In this sense, I examine how pathways are represented, aiming to distinguish between Thagard's, Ross', and my approaches to the pathway concept. This analysis considers the three aspects of pathways discussed earlier in Part II (see Chapter 6), that is, the strategic, ontic, and epistemic aspects of pathways.

Thagard (2003: 237) emphasizes the epistemic aspect of pathways by pointing out that scientists utilize "both external representations via print and computer screens and internal mental representations". For the purposes of the subsequent section where glycolysis will be described, I focus more on external representations. Two prevalent models of external presentations are verbal and visual. Both are used in textbooks, which commonly incorporate two-dimensional visual representations of pathways illustrating the sequence of steps, molecules, and interaction between these steps. Additionally, three-dimensional representations, employed by various databases, also play a significant role alongside these representations.

These representations *explain* by emphasizing different parts of the pathway, depending on the level of abstraction used in their representations. According to Thagard (2003: 237), "biochemical pathways are a kind of mechanism", providing a mechanistic explanation. He argues for that following Machamer, Darden, and Craver's (2000) account of mechanisms. Recall that they outline that entities and activities within mechanisms are organized in order to produce a phenomenon, exhibiting regular changes, set-up conditions and ending products. Thagard (2003) argues that pathways share the same features. In the case of glycolysis, "the entities are the molecules such as the initial glucose and the terminal pyruvate, as well as the numerous molecules produced and transformed during the ten chemical reactions in the pathway" (Thagard 2003: 238). Furthermore, these molecules are organized in a specific way to produce regular changes in cells, i.e., repeating production of pyruvate from glucose. Additionally, one can identify set-up conditions, that is, molecules initiating the pathway, and terminating conditions, i.e., molecules produced by the pathway. Consequently, pathways exhibit features akin to mechanisms.

Thagard (2003: 238) asserts that "biochemical pathways explain by showing how changes within a cell take place as the result of the chemical activities of the molecules that constitute the cell". Moreover, he (2003: 239) points out that: "discovery of a pathway provides a mechanism that describes the productive activity". Thus, Thagard establishes a link between pathways and mechanisms, positing that pathways are a part of a mechanistic explanation due to sharing common features.

Here, to some extent, I align with Thagard's perspective of how pathways are represented within a cell. Specifically, I focus on the epistemic aspect of pathways, i.e., the one regarding the vehicles of explanations (e.g., representations). While I have previously discussed this distinction in Part II (see Chapter 6), I now reconsider it in light to Thagard's viewpoint on the issue of representations. The difference I emphasize is between the temporal and spatial characteristics of mechanisms and pathways, which is particularly evident in their representations.

Namely, pathway representations, as seen in the case of glycolysis, predominantly rely on depicting the 'connection'[82] aspect between steps in a sequence. This representation puts an emphasis on the *activities* between steps, including its direction (e.g., line of operating), order (e.g., its positioning in the sequence), and duration (e.g., the approximate time required for a certain step to advance). These specific sequences and the order of steps needs to operate in an

[82] For more information on this aspect in Ross (2021), see Part II, Chapter 5.

orchestrated fashion, which is crucial for interventions in a pathway to control its outcome. Thus, in pathway representations, the highlighted characteristic is the temporal characteristic, involving organization based on the activities of the steps in a specific pathway. This emphasis will become particularly evident in the next section and the case of cancer treatment.

On the other hand, mechanisms' representations include, in addition to a temporal characteristic, a spatial component. As pointed by Craver (2001) this involves characterizing its entities in terms of their various sizes and shapes, as well as their specific locations within their boundaries. I have previously discussed this in both Part I and II (see Chapter 3; Chapter 6). Since this chapter focuses on pathway representations, I do not delve further into the spatial characteristic.

Now, let me further address the differences between Thagard's, Ross', and my views on the pathway and mechanism distinction. Besides these representational differences in pathways and mechanisms, pertaining to the epistemic aspect, another distinction, as previously highlighted in Part II (see Chapter 5), lies in the strategic aspect. Ross (2021) argues that pathways are distinct from mechanisms, encompassing both ontic differences, i.e., different features, and strategic differences, i.e., investigative strategies. Contrary to Ross' view, as argued in Part II (see Chapter 6) I contend that pathways, as conceptualized by Ross, lack distinct features compared to mechanisms, thus sharing the ontic aspect. However, they involve different investigative strategies, thus having a distinctive strategic aspect.

To briefly recap the above difference between the ontic and strategic aspect, recall that Ross (2021) identifies four features of pathways, namely, a pathway is a (a) sequence of causal steps, that (b) tracks the 'flow' of some entity or a signal through a system, (c) abstracts from details, and (d) puts an emphasis on the 'connection' aspect of causal relationships. I previously addressed each of these features in Part II (see Chapter 5, Section 1), arguing that Ross' pathway features align with the two features of mechanisms present in the new mechanistic account, namely productive continuity and the abstract representation of mechanisms. I presented two possible outcomes regarding the similarity issue: either adjusting the pathway feature list to highlight other pathway features that differ from mechanistic ones, or asserting that the new mechanistic account encompasses the pathway concept. In line with Thagard's perspective, pathways, as he points out (Thagard 2003: 237), can be considered "a kind of mechanism". The latter outcome aligns with Thagard's advocacy, as highlighted above.

Ross' (2021) argues that the pathway concept encompasses various features. Similar to Thagard, I advocate that the pathway concept shares features with the mechanistic concept, particularly features (a), (c) and (d), which can be described in terms of the abovementioned

notions of productive continuity and abstraction. However, I diverge from Thagard's perspective by asserting that the distinction between these two causal structures lies in their different investigative strategies, a point elucidated in Ross (2021).

Recall that the investigative strategies associated with pathways involve building networks (e.g., maps). In this approach, scientists seek potential connections within these networks, subsequently using them to develop new pathways. This strategy is characterized by the 'expanding out' of networks, that is, creating new causal points and routes, which is a distinctive approach compared to mechanistic investigative strategies, involving decomposing and localizing. While Thagard (2003: 243) does not explicitly delineate this investigative strategy as distinct from mechanistic ones, he highlights the use of networks and, refers to them as "subway maps", which is a concept similar to Ross' 'expanding out' strategy mentioned above.

In summarizing the differences between our perspectives on pathways and mechanisms, particularly concerning the strategic, ontic, and epistemic aspects, I argue that pathways possess distinct strategic and epistemic aspects. Thagard's viewpoint aligns with mine insofar as it outlines the similarity between the ontic aspects; however, he extends that similarity to the epistemic aspect by proposing that pathways are a type of mechanism. In contrast, Ross provides a distinction in all three aspects. With her view, I particularly align with the difference in the strategic aspect but emphasize the temporal characteristic of pathways in the epistemic aspect. This temporal characteristic is crucial in the representation of pathways, particularly important for scientific practices, in this case, cancer treatment.

SECTION 2. The Glycolytic Pathway and Cancer Treatment

Thagard (2003: 245) asserts that "an understanding of pathways often leads to new medical treatments", particularly by either enhancing or inhibiting pathways. This knowledge of pathways can be instrumental in treating diseases. Thagard proposes that treatment can involve either enhancing pathways by repairing defective ones or inhibiting pathways by blocking destructive ones. I focus on the latter since the case study I deal with concerns the inhibition of glycolysis to prevent the growth of cancer cells. The inhibition of pathways entails the ability to block those responsible for a certain disease. In this regard, drugs are developed to intervene in pathways and suppress particular molecules (e.g., enzymes) and reactions. I proceed to examine the abovementioned case of inhibiting glycolysis.

The glycolytic pathway (see Figure 17) represents the central metabolic pathway found in nearly all organisms, evolving across the three domains of life – Bacteria, Archaea, and Eukarya. Glycolysis results in pyruvate molecules and the release of energy in form of ATP molecules. It is a pathway consisting of ten steps divided into two stages, i.e., the *investment* and *pay-off* stages. The investment stage initiates with the rearrangement of the glucose molecule, catalyzed by the enzyme Hexokinase, into glucose 6-phosphate (G6P). It proceeds with the change into fructose 6-phosphate, fructose 1,6-biphosphate, and ultimately, glyceraldehyde 3-phosphate, resulting in two phosphate-bearing three-carbon sugars. The phosphate groups, modifying the initial glucose molecule, are derived from ATP molecules, thus, in the process, two ATP molecules are used in this stage, hence its designation as the investment stage.

In the pay-off stage, each three-carbon sugar undergoes a series of conversions leading to the production of pyruvate molecules. These steps include the transformation from 1,3-bisphosphoglycerate to 3-phosphoglycerate, then to 2-phosphoglycerate, and finally phosphoenolpyruvate. This stage earns its name from the production of four ATP molecules and two NADH molecule, that is, equivalent to two ATPs and an NADH for each phosphate-bearing three-carbon sugar produced in the first stage. Every step of the pathway is catalyzed by enzymes, such as, among others, Hexokinase and Phosphofructokinase, which accelerate reaction rates (see Gill et al. 2016: 88). Moreover, the glycolytic pathway can operate either *aerobically*, i.e., in the presence of oxygen, or *anaerobically*, in its absence. In the presence of oxygen, "pyruvate enters into the tricarboxylic acid (TCA) cycle (also known as the citric acid or Krebs cycle) within the mitochondria to produce ATP" (Zhou et al. 2022: 3). In the absence of oxygen, pyruvate converts into lactate.

The discovery of glycolysis unfolded over a 300-year span of research. It originates in the 18th and 19th centuries with a focus on the wine and brewery industry's quest to maximize alcohol production. However, Grüning and Ralser (2021) assert that the isolation of glucose marks the beginning of glycolysis research, representing a pivotal milestone in the inception of what we now recognize as biochemistry. Andreas Sigismund Marggraf isolated glucose from raisins in 1747. In 1838, Jean Baptiste Dumas assigned the name 'glucose' to the "sugary white powder" (Grüning and Ralser 2021: 1) derived from this isolation, with the term originating from the Greek word *glykys* that translates to 'sweet'. Afterward, glucose became associated with fermentation, a biological process wherein microorganisms, such as bacteria and yeast, break down a certain substance (see Teillefer and Sparling 2015). In the 1850s, Louis Pasteur begun the research into the fermentation process. Consequently, the French alcohol industry sought Pasteur's assistance in resolving "the question why the products sometimes turn distasteful" (Grüning and Ralser 2021: 2). Pasteur concluded that yeast has an important role in fermentation, contrary to then attributed passiveness to yeast's role in fermentation. Subsequent research, involving various scientists, discovered the role of enzymes in the fermentation process. These enzymes catalyze the process and play an important role in breaking down glucose.[83]

Lastly, in this brief history of glycolysis, I would like to emphasize the roles of biochemists Otto Meyerhof, Gustav Embden, and Jakub Parnas, who introduced the 10-step sequence of glycolysis. This sequence is known as the EMP pathway, an acronym derived from the first letters of their surnames. In 1918, Meyerhof discovered that the biochemical process involving abovementioned enzymes in yeast also occurs in muscles. This led him to argue that metabolic processes could be similar across "the living world" (Grüning and Ralser 2021: 5). In 1927 and 1928, Embden played a crucial role in the isolation of the ATP molecule. Parnas discovered phosphate groups in glycolysis, which are crucial for connecting glycolytic intermediates, completing the glycolytic sequence. As Grüning and Ralser (2021: 5) point out, "eventually, all metabolic steps from glucose to pyruvate were elucidated until the 1940s".

Now, what is particularly interesting regarding glycolysis and cancer research is that the rate at which cancer cells utilize glycolysis for energy requirements is significantly higher

[83] The discovery of enzymes played a pivotal role in debunking vitalism, particularly concerning the process of fermentation. Before this revelation, some scientists believed that *vis vitalis*, or vital force, was necessary to break down glucose. While the debate on vitalism is not in the focus of this thesis, it is noteworthy that the discovery of enzymes contributed significantly to challenging and revising these earlier notions.

than that of normal cells.[84] Specifically, "glycolysis rate is 200 times higher in tumor cells than in the normal cells" (Akram 2013: 1). This phenomenon, known as the *Warburg effect*, was discovered by Otto Warburg in 1930. Cancer cells[85] predominantly use glycolysis "to fulfill their bioenergetic and biosynthetic demands to support their proliferation" (Gill et al. 2016: 88).

Contrary to normal cells, which, in the presence of oxygen, derive their energy mainly from oxidative phosphorylation (about 70% of their energy demands), cancer cells opt for the glycolytic pathway even in the presence of oxygen. This is due to their excessive need for consuming glucose and synthesizing lactate, a product of glycolysis. Although oxidative phosphorylation can yield more energy (more than 30 ATP molecules per cycle), cancer cells prefer glycolysis because of its much faster production rate. Moreover, tumor cells are overreactive and create an abundant number of cells. In particular malignant tumor cells, which are detaching "from neighbouring cells to translocate to distant locations for metastasis" (Zhou et al. 2022: 3). To support their rapid cell division, invasion, and migration, cancer cells require a lot of energy and biosynthetic precursors.

As a malignant tumor is expanding, i.e., migrating from the initial locus, "it grows beyond the diffusion limit of the local blood supply", thus leading to hypoxic conditions, i.e., conditions without the presence of oxygen. Under these conditions, cancer cells undergo a 'metabolic reprogramming' and heavily rely on glycolysis (see Hsu and Sabatini 2008). Additionally, according to Ganapathy-Kanniappan and Geschwind (2013: 3): "cancer cells require further metabolic intermediates and precursors that are critical for the biosynthesis of macromolecules", leading to an increase in the tumor mass during proliferation. These intermediates are known to advance the 'pentose phosphate pathway', which is also crucial for amassing cancer cells. Therefore, glycolysis becomes a crucial pathway for cancer cells due to its ability to provide energy rapidly and facilitate tumor growth. Because of that, scientists can exploit glycolysis as a target pathway to hinder cancer cells' primary energy acquisition pathway.

[84] According to Anya Plutynski (2018: 219), "cancer is typically characterized as a disease of disorderly cell growth and disruption of tissue organization". Its incidence usually increases with age. Plutynski (2018: 222) also emphasizes six „hallmarks" of cancer, based on Hanahan and Weinberg's (2000) famous article, which are the following: "sustained proliferative signaling, evading growth suppressors, resisting cell death, enabling replicative immortality, inducing angiogenesis, and activating invasion and metastasis".

[85] In this instance, I use cancer cells and tumor cells interchangeably. However, they are not the same. Specifically, a tumor relates to abnormal and rapid tissue growth. Cancer relates to diseases caused by the abnormal growth and spread of cells. Having a tumor does not necessarily mean having cancer, as tumors can be either malignant or benign.

To inhibit glycolysis, scientists primarily target its enzymes based on the type of tumors. I present two cases. The first involves Glucose transporter 1 (GLUT1), i.e., a protein that transports glucose into cells. The second one regards Hexokinase (HK), an enzyme which catalyzes the first step of glycolysis, i.e., converts glucose into G6P. Xiao et al. (2018), examined the role of GLUT1 in tumor progression, focusing on prostate cancer. They found that GLUT1 contributes to prostate cancer by promoting proliferation through glycolysis. Their research revealed higher levels of GLUT1 in prostate cancer cells compared to normal prostate cells. To test the impact of inhibiting glycolysis, they conducted a "knockdown"[86] of GLUT1. The results showed that the "knockdown" aided in suppressing glycolysis, subsequently slowing down the growth of cancer cells.

HK is an enzyme that "catalyzes the phosphorylation of glucose to glucose-6-phosphate (G6P), after glucose enters the cell via glucose transporters (GLUT)" (Gill et al. 2016: 89). There are four isoforms of HK, i.e., representing slightly different variations in structure. Among these isoforms, Hexokinase 2 (HK2) is prevalent in the glycolysis occurring in cancer cells. Thus, HK2 is a very important factor in tumor growth rate. According to Patra et al. (2013: 214), "HK2 constitutes an attractive potential selective target for cancer therapy". They examined the role of depleting HK2 in "tumor initiation and maintenance" in types of lung and breast cancers. Their study concluded that HK2 is required for the "oncogenic transformation" of embryonic fibroblasts found in mice, on which the studies were conducted. Moreover, the depletion of HK2 was "sufficient to profoundly reverse oncogenic transformation" (Patra et al. 2013: 214).

Examining examples like GLUT1 and HK2 highlights their important role in tumor growth. Experimental interventions have shown that targeting these factors can prevent cancer cell growth. The development of drugs for this purpose relies on deep understanding of glycolysis, including the knowledge of how the glycolytic pathway operates, along its steps, and the enzymes catalyzing the process.

[86] In molecular biology, a knockdown refers to the temporary and partial reduction of the expression of a specific gene or a protein. In other words, it decreases their expression.

SECTION 3. Interventionist Account, Pathways, and Cancer Treatment

In the preceding section, I emphasized the significance of GLUT1 and HK2 in fueling tumor growth through glycolysis. Inhibition of these components halts cancer cells from obtaining energy via glycolysis, making the development of drugs targeting these structures a key strategy in cancer treatment. To analyze this in a conceptual framework, we can turn to the interventionist account (see Part I, Chapter 2). Particularly, we can explain this phenomenon of cancer treatment by employing Woodward's (2003) causal explanation account. Unlike other accounts of causal explanation (see Part I, Chapter 2), I employ this account due to its connection to scientific practice, and its endorsement by the new mechanists and Ross' pathway concept. In the general framing, Woodwardian explanation means to "show how what is explained depends on other, distinct factors, where dependence in question has to do with some relationship that holds as a matter of empirical fact" (Woodward 2003: 4-5). Woodward terms his account the interventionist account, which revolves around the *manipulability* conception. This conception guides scientists to "think about causal explanation" (Woodward 2003: 9). He bases his account by drawing inspiration from Robert Weinberg's (1985) idea that molecular biology provides explanations through information usable for manipulation or *control* of effects. In particular, emphasizing experimental techniques by which molecular biologists try to intervene and manipulate with biological systems.

Woodward's (2003) account focuses on examining explanation as identifying "factors or conditions such that manipulations or changes in those factors or conditions will produce changes in the outcome being explained" (Woodward 2003: 10). Explanatory information has to be relevant for manipulation and control. Woodward (2003: 10) notes that "the notion of information that is relevant to manipulation thus need to be understood modally or counterfactually". In other words, an explanation should address *what-if-things-had-been-different* question. It should enable us to understand how altering factors in the explanans would impact explanandum in various possible scenarios. This same conceptual framework is at work in the abovementioned cancer treatment research. In other words, scientists are thinking about how the "knockdown" of a specific molecule could alter the entire process. In the context of glycolysis, inhibiting a particular cause (e.g., HK2) prevents the intended outcome (e.g., ATP production) of the process.

As previously discussed, pathways carry causal information in form of sequential steps, that is, employing temporal characteristic. Pathway representations depict these steps by illustrating the subsequent steps of a certain process, such as glycolysis. This information

serves as a foundation for scientists to identify relevant factors that can be manipulated to alter the pathway's outcome. The extraction of this information is facilitated through the pathway explanation process, elucidated in detail in Part II (see Chapter 6). Recall the model mentioned earlier (see Figure 18), comprising the ontic aspect, which is central. Here, scientists aim to capture the pathway inherent in nature, e.g., the glycolytic pathway operating within cells. However, it is crucial to note that the entire explanation process initiates with the strategic aspect. Scientists identify potential causal routes in a network and then expand this network to uncover additional pathways. This is particularly evident in the case of glycolysis, as demonstrated by the historical overview provided earlier. Namely, over several generations, scientists unraveled the pathway, with the network of pathways leading to the current understanding of the pathway being represented in Figure 17. This network-building process reflect scientists' attempts to comprehend the pathway as it exists in nature, i.e., the previously mentioned ontic aspect. The third and final aspect of the process involves the representation of a pathway, namely the epistemic aspect. As previously outlined, pathway representations are specifically made via emphasizing the causal connection between steps of a pathway, namely the temporal feature of it. That representation is then used to *explain* the pathway.

In the case of glycolysis, the progressive elucidation of the pathway through years of experimentation and network construction led to its potential of various applications, including the one in question in this chapter. The information accumulated by scientists regarding the pivotal steps and stages of the pathway, along with its interconnected pathways is crucial for enabling interventions. These interventions involve manipulating specific steps, as exemplified in the case of glycolysis and cancer treatment. In this specific scenario, a targeted enzyme is manipulated, effectively "knocked out", thereby preventing both the investment and pay-off stages of the pathway.

In this sense, the interventionist account emerges as the fundamental conceptual framework of causal explanation in the above case. The targeted structure in this case is a pathway. Through the investigative strategy of elucidating a pathway, by expanding it and subsequently representing it, one can exploit its causes. This involves manipulating these causes to bring about a change in the pathway's outcome.

CONCLUSION. Chapter 7

This chapter examined the case study of inhibiting glycolysis to impede the growth of cancer cells. Initially, I addressed how pathways are represented, emphasizing the distinction from mechanisms. Specifically, I drew comparisons between Ross' (2021), Thagard's (2003), and my perspective on this differentiation. In this sense, I highlighted the divergence from Thagard's stance by asserting that the investigative strategy of pathways is distinct from mechanistic strategy, aligning more closely with Ross. Although my view aligns with Thagard's regarding the ontic aspect, that is, acknowledging shared features between these structures, I emphasized a departure in highlighting the temporal aspect in pathways representations. This distinction anticipated further discussion in the chapter.

Subsequently, I examined the significance of the glycolytic pathway in cancer treatment, providing a historical overview of the discovery of glycolysis. Moreover, I highlighted the process of elucidating the pathway and building related networks to glycolysis. Additionally, I outlined the pivotal role of glycolysis as the primary energy source for the growth of cancer cells. Consequently, specific segments of the glycolytic pathway emerge as potential targets for medical intervention, aiming to inhibit the pathway and hinder the growth of cancer cells. Thus, I singled out glycolysis as a promising pathway for cancer treatment purposes.

Finally, I outlined Woodward's (2003) interventionist account of causal explanation. This account served as the conceptual framework underpinning the inhibition of glycolysis. Thus, it explains the significance of interventions and manipulations on causes to bring about a change in the outcome. Specifically, in the case of glycolysis, to effectively prevent the growth of cancer cells.

Chapter 8: Natural Selection: A Pathway or a Mechanism?

INTRODUCTION. Chapter 8

There are several distinct characterizations of natural selection. It is often characterized as a cause, force, process, mechanism, factor, or principle, with the most frequent ones being named.[87] In this regard, two main philosophical accounts are the following ones: the *force* account, which is a traditional characterization, and the *statistical* account, which has lately gained in its importance by an overall interpretation of Darwinism as a statistical theory.[88] In more detail, the statistical account posits that natural selection is only a certain type of byproduct or consequence at the population-level, resulting from a sequence of lower-level processes involving biological individuals (see Matthen and Ariew 2002; Walsh, Lewens, and Ariew 2002). Standardly, the term 'biological individuals' refers to individual organisms, for instance, Darwin's finches or bacteria. According to the statistical account, in direct opposition to the traditional force account (see Sober 1984), natural selection, along with all other factors influencing evolutionary change, despite its importance from a Darwinian point of view, is not a real force. It does not amount to a certain capacity of "*acting on* populations" (Stephens 2004: 552; italics added), as proponents of the force account, drawing an analogy with Newtonian mechanics, seek to emphasize.

Apart from these two main philosophical accounts, there is an additional approach that aims to examine the concept of natural selection, i.e., the approach by which natural selection is characterized as a mechanism.[89] This perspective has drawn both positive and negative assessments. On the negative side, there are authors such as Skipper and Millstein (2005), Havstad (2011), and Garson (2019).[90] On the positive side, there are authors such as Barros (2008), Illari and Williamson (2010), and DesAutels (2016). I start by examining the seminal

[87] For additional characterizations of natural selection, particularly as framed by Charles Darwin in the first edition of the *Origin*, see Havstad (2011: 513).

[88] For an overview of that shift in the philosophical debate on evolutionary theory, particularly concerning the nature of natural selection, among other factors of evolutionary change, such as, primarily, random genetic drift, but also mutation and migration, see Stephens (2004). However, it is important to note that this work primarily focuses on the force account and does not explore other options beyond the abovementioned dichotomy. The primary focus of this chapter is the examination of the possibility of characterizing natural selection as a causal structure, i.e., a mechanism or a pathway; thus, I do not examine force account in more detail.

[89] In the next section, I explore the question whether the pathway concept aptly characterizes natural selection. For the present discussion, however, I focus on the already established debate surrounding whether the concept of a mechanism accurately captures natural selection.

[90] I would like to add that Perez-Gonzalez and Luque (2019) also take a negative stance. However, I do not examine their perspective as they emphasize the importance of broadening the debate by including other evolutionary causes within the mechanistic framework, beyond natural selection.

paper by Skipper and Millstein (2005), which initiated the ongoing debate on the issue. Basically, all the abovementioned authors either reference or discuss their approaches to the issue by examining the ideas presented by Skipper and Millstein (2005). Thus, I begin with the examination of their paper, utilizing it as a foundation for subsequent assessments.

Next, I turn to the examination of whether natural selection could be characterized as another causal structure, namely a pathway. In the abovementioned debate, significant criticism has been directed towards characterizing natural selection as a mechanism, particularly because the nature of natural selection, i.e., its features, does not correspond to the mechanistic features that proponents of the new mechanistic account advocate for. In this regard, features of organization, productive continuity, and regulation are particularly criticized.

Furthermore, some authors (see Havstad 2011) argue that, given the abstract nature of natural selection, it cannot be accurately characterized as a mechanism. She claims that: "at the general level, natural selection is an abstract process without the entities/parts and activities/interactions characteristic of a mechanism" (Havstad 2011: 522). To address this critique and other mentioned earlier, I argue for characterizing natural selection as a pathway. I support this argument by applying the three aspects of mechanisms and pathways previously discussed in the thesis. Additionally, I apply Ross' (2021) pathway concept, specifically her characterization of pathways. In this examination, I consider the natural selection schema presented by Skipper and Millstein (2005), which outlines seven stages of natural selection. I argue that stages II, III, and IV exhibit features akin to pathways. I highlight that these stages include features aligning with the aspects of pathways, specifically the strategic pathway.

The structure of this chapter is the following one: in Section 1, I present the debate surrounding the characterization of natural selection as a mechanism. This section places special emphasis on the seminal paper by Skipper and Millstein (2005), proceeding with the subsequent debate on the issue in question. In Section 2, I employ the three aspects of pathways and the pathway concept, both of which I previously examined in Part II (see Chapters 5 and 6). In this section, I argue that natural selection can be partially characterized as a strategic pathway.

SECTION 1. Natural Selection: A Mechanism?

1.1. Skipper and Millstein's (2005) First Point

With the characterization of a mechanism in mind, as examined in Part II (see Chapter 4), a critical question arises: Does natural selection meet the criteria to be considered a mechanism? This question was initially posed by Skipper and Millstein (2005). In this section, I outline two key points from their paper, and these are as follows: (i) Their examination of the proposal regarding whether the new mechanistic notion of mechanism might capture or 'get at' the aspects of natural selection; and (ii) their suggested directions for further work on the conception of mechanism that might capture the aspects of natural selection.[91]

Starting with point (i), Skipper and Millstein (2005) assert that the notion of mechanism, especially as characterized by new mechanists at the time, does not capture the aspects of natural selection. They support this claim by highlighting three specific aspects of mechanisms as found in the work of Glennan (1996; 2002), and Machamer, Darden and Craver (2000) that fall short in characterizing natural selection adequately. These aspects are organization, productive continuity, and regularity. Let me briefly explore each of these aspects.

Firstly, organization. According to Glennan (1996) and Machamer, Darden and Craver (2000), mechanisms are complex systems that are structured and organized, decomposable into their component parts. Recall that Glennan's (1996) illustrative example of a mechanism is a toilet, while Machamer, Darden and Craver's (2000) prime biological examples include protein synthesis and neural depolarization. According to Machamer, Darden and Craver (2000: 3), "entities often must be appropriately located, structured, and oriented, and the activities in which they engage must have a temporal order, rate, and duration". Skipper and Millstein (2005: 338) argue that it is unlikely that natural selection possesses the degree of organization required by either Machamer, Darden and Craver or Glennan. They emphasize that organisms exhibiting variability in their location and orientation lack a stable configuration. Additionally, they consider the variability of organisms, such as finches, stating that despite the controversy surrounding the source of variation in natural populations, variation is deemed a necessary condition for natural selection.

[91] It is important to note that Skipper and Millstein (2005), besides these two points outlined, also analyze the differences between Glennan's and Machamer, Darden and Craver's conceptions of mechanisms. However, I do not address these differences here as they have been, to some extent, discussed in Part II (see Chapter 4).

Secondly, productive continuity. Skipper and Millstein (2005: 339) assert that neither Glennan's nor Machamer, Darden and Craver's conception of productive regularity adequately captures natural selection. In Glennan's framework, the concept of the productive continuity is manifested through his concept of 'interactions' between parts in a mechanism that ensures a robust behavior of a mechanism (see Glennan 1996; 2002). However, Skipper and Millstein (2005: 340) argue that this conception falls short of capturing natural selection because Glennan's account of interaction does not encompass the dynamics or productive continuity between stages in natural selection. Furthermore, Glennan's understanding of interactions does not account for how differences among organisms in a population influence the ways in which that population interacts with its environment. Moreover, Skipper and Millstein argue that Machamer, Darden and Craver's notion of productive continuity also fails to adequately capture natural selection. In particular, Skipper and Millstein contend that the concept of entities and activities proposed by them falls short of addressing adequately this task. As Skipper and Millstein (2005: 341) claim: "It is true that finches are entities and that they engage in activities such as feeding (surviving) and mating (reproducing). The problem is that the activities of surviving, and reproducing do not constitute selection".

Thirdly, regulation. Once more Skipper and Millstein (2005: 342) examine this aspect by addressing both Glennan's and Machamer, Darden and Craver's approaches separately. Glennan perceives regularity as "direct, invariant, change-relating generalizations", and Skipper and Millstein find promise in this approach for capturing the regularity of natural selection. Particularly, they highlight Glennan's assertion that a mechanism is robust, or that the interactions in a mechanism are robust, which aligns with invariant generalizations found in Woodward (see 2000; 2003). Skipper and Millstein point out that a generalization that is invariant supports relevant counterfactual truth claims.[92] They clarify their understanding of natural selection as a probabilistic phenomenon, suggesting that the notion of a stochastic mechanism could adequately capture natural selection. Although they do not explicitly state whether Glennan attempts to capture the stochastic causal relation they have in mind, Skipper and Millstein believe that Woodward (2003) does so. They note that Woodward's understanding of generalizations governing causal relations aligns with how they perceive natural selection. According to them, the key lies in establishing a proper account of probabilistic causal relations.

[92] For the purposes of this section, I do not examine this conception further, particularly because I addressed counterfactual accounts of causal explanation in Part I (see Chapter 2).

Turning to Machamer, Darden and Craver's (2000: 3) approach to regularity, they characterize mechanisms as regular in that "they work always or for the most part in the same way under the same conditions". Skipper and Millstein (2005: 343) argue that natural selection does not exhibit the required regularity according to Machamer, Darden, and Craver, unlike, for example, protein synthesis and DNA replication, which align with their concept of a mechanism. Skipper and Millstein (2005) argue so because, according to them, natural selection is probabilistic, illustrating this point with the famous "finches" example. To sum up, Skipper and Millstein assert that Glennan's (or rather Woodward's) regulatory concept, in contrast to Machamer, Darden and Craver's, is more suitable for accommodating the probabilistic nature inherent in natural selection.

1.2. DesAutels (2016) Critique

Before turning to point (ii), I emphasize DesAutels (2016) criticism of Skipper and Millstein's characterization of Machamer, Darden and Craver's regulatory concept and the nature of natural selection. As a proponent of the positive side, DesAutels argues that natural selection can be characterized as a mechanism.[93] He (2016: 13) asserts that natural selection "can be seen to be regular enough to qualify as an MDC mechanism just fine", MDC referring to an acronym for Machamer, Darden and Craver (2000). However, in order for it to be considered regular enough, DesAutels introduces three crucial distinctions, namely *process vs. product regularity*, *mechanism-internal vs. mechanism-external sources of irregularity*, and *abstract vs. concrete regularity*. I briefly outline these three distinctions. Before that, I also present the regularity critique articulated by Skipper and Millstein (2005), as formed in an argument form by DesAutels (2016: 16):

- P1. MDC requires that mechanisms behave regularly (i.e., they 'work always or for the most part in the same way under the same conditions').
- P2. But natural selection operates probabilistically where this can be couched either in terms of: (i) 'petty influences' which we should expect to differ across evolutionary iterations or (ii) genuine indeterminism operating at the molecular level.

[93] DesAutels (2016: 14) provides *prima facie* reasons for understanding natural selection as a mechanism, roughly summarized as follows: (1) mechanisms are "*set up for something*", and natural selection is a system that is also set up for something, i.e., according to DesAutels, specifically, bringing about adaptation; and (2) mechanisms "support reductionist explanation" (decomposition), and natural selection is also decomposable.

- P3. Given either disjunct of (P2), natural selection cannot meet the regularity requirement set forth by MDC.
- C1. Therefore, natural selection cannot be an MDC mechanism.

Now, after laying out this argument form, DesAutels proceeds to address the regularity critique by Skipper and Millstein, employing the distinctions mentioned above. Let me start with the first distinction, namely the process vs. product regularity distinction.

According to DesAutels, process regularity means consistency in the behavior of the parts of a mechanism, encompassing activities and entities, each time the mechanism operates. On the other hand, product regularity pertains to the consistency in the output of a mechanism each time it operates. Following this distinction, DesAutels argues that Skipper and Millstein's regularity critique is inadequate. He contends that their example[94] demonstrates only that natural selection is product irregular, not process irregular. According to DesAutels, this form of irregularity is not a threat to the Machamer, Darden, and Craver conception of regularity.[95]

Let me now outline the second distinction, namely the mechanism-internal vs. mechanism-external sources of irregularity. DesAutels (2016: 18) explains this distinction by using a toaster example. A mechanism-external source of irregularity occurs when a toaster fails to regularly produce adequate toast because, if one simultaneously uses other appliances in one's kitchen, a fuse is blown, causing the toaster to shut down. Conversely, a mechanism-internal source of regularity occurs when there is a faulty connection in the toaster's wiring. DesAutels argues that Skipper and Millstein rely on mechanism-external sources of irregularity in their critique. However, he asserts that mechanism-external inhibiting factors should not pose a threat to the Machamer, Darden, and Craver regularity concept. DesAutels contends that, for the mechanistic explanatory framework, what matters is some degree of regularity in the function of the mechanism itself.

DesAutels' (2016: 20) third distinction centers around abstract vs. concrete regularity, emphasizing the relationship between mechanism types and tokens. He draws on Andersen's (2012: 417) type-token distinction between mechanisms. Andersen outlines that, on one side, the term 'mechanism' can refer to a single individual chain in the world (a mechanism token), for instance a particular neuron firing on a given occasion, i.e., a mechanism that led to that

[94] Skipper and Millstein (2005) are using finches' example to demonstrate that natural selection is not regular in the mechanistic terms.
[95] For a detail examination of why product irregularity is not threatening to Machamer, Darden, and Craver's regularity concept, see DesAutels (2016: 17).

particular firing of the neuron. On the other side, she states that the term 'mechanism' can also refer to represent a type of a causal chain (a mechanism type), i.e., one that could recur on multiple occasions. For instance, a neuroscience textbook describes the mechanism of neuron firing, but it does so, not to describe a particular instance of a neuron firing, rather a type of a causal chain that possibly occurs on many occasions. Following this distinction by Andersen, DesAutels argues that mechanism types, i.e., general representations of mechanisms, relate to the term 'schema', used by Machamer, Darden, and Craver. Schema is defined as a truncated abstract description of a mechanism type, often depicted with entities in boxes and activities as arrows (see Machamer, Darden, and Craver 2000: 15).[96] Now, following this distinction, DesAutels argues that if natural selection is perceived as a mechanism type with a relatively high degree of abstraction, then it should be understood as behaving "always or for the most part in the same way under the same conditions thereby fulfilling the MDC regularity requirement" (DesAutels 2016: 21).

By introducing and elucidating on the three abovementioned distinctions, DesAutels aims to counter Skipper and Millstein's regularity critique. Moreover, he intends to present a general strategy for considering the mechanism concept in explaining stochastic biological phenomena, particularly such as natural selection. DesAutels suggest that by understanding natural selection as a mechanism type with a relatively high degree of abstraction, one can accommodate its stochastic nature with the Machamer, Darden, and Craver regularity concept.

1.3. Skipper and Millstein's (2005) Second Point

I now return to Skipper and Millstein's point (ii). Skipper and Millstein (2005) outlined an important direction for further work regarding this issue; thus, paving the way for the following discussion. Besides presenting three main critiques towards the mechanistic characterization of natural selection, i.e., the problematic aspects of organization, productive continuity, and regularity, they also offer the following important statement:

> To capture natural selection, a main evolutionary mechanism, a conception of stochastic mechanism as a non-unique causal chain is required in which change is produced by virtue of the ways in which

[96] An example of a mechanistic schema is James Watson's (1965) central dogma diagram (DNA → RNA → protein), representing the flow of genetic information from DNA to RNA to protein. This diagram is considered an abstraction of the highest degree, providing an explanation for the fundamental processes occurring in genetic information transfer.

> property differences among members of a population in the context of some environment affect properties of that population. We think the basic resources for characterizing the mechanism of natural selection may be found in the new mechanistic philosophy. And we urge the proponents of the philosophy to explore the directions for further work we suggest. Skipper and Millstein (2005: 344)

In the upcoming section, I particularly address Skipper and Millstein's ideas and directions as I explore the idea whether natural selection could be partially characterized as a pathway. However, prior to that exploration, I examine the already established discussion surrounding the suggested directions for the debate. I begin this examination with Barros' (2008) view.

1.4. Barros' (2008) and Havstad's (2011) Discussion

In the abovementioned quote, Skipper and Millstein emphasized that, to capture the nature of natural selection, a mechanistic account has to accommodate the stochastic nature of natural selection. They propose to consider the idea of examining stochastic mechanisms as a non-unique causal chain. Barros (2008: 318) aligns with this idea, suggesting that natural selection can be characterized as a mechanism explaining adaptation. According to Barros, organisms possess some traits well-suited to their environment, and this phenomenon is termed natural selection (see Barros 2008: 313).

Before presenting his characterization of natural selection as a stochastic mechanism, Barros distinguishes *deterministic* from *stochastic* mechanisms. Deterministic mechanisms allow making predictions with certainty and thus explaining phenomena. Examples of these mechanisms are the heart's pumping, or the protein synthesis described by Machamer, Darden, and Craver (2000). In other words, as long as the mechanism is working properly, the outcome can be determined deterministically, that is, a working heart will pump blood, a working mechanism of protein synthesis will synthesize a protein. In contrast, in defining stochastic mechanisms, Barros follows Glennan's (2002) definition, which involves some degree of randomness or probability that falls short of determinism (see Barros 2008: 310). Barros introduces an example of a stochastic mechanism, the fair-coin flipping machine. Barros further distinguishes stochastic mechanisms. According to him, there are *unbiased* and *biased* stochastic mechanisms. An unbiased stochastic mechanism is a mechanism that operates with outcome probabilities of 50% or less. Examples of that mechanism are, again, the fair-coin

flipping machine or the mechanism that releases neurotransmitters initiating electrical activity in postsynaptic neurons (see Bogen 2005). A biased stochastic mechanism is a mechanism operating with probabilities greater than 50%. Their operations are characterized by terms such as 'are likely to' or 'probably will'. According to Barros (2008: 312), an example of a biased stochastic mechanism is natural selection. In his view, natural selection can be predicted to increase the prevalence of a trait in a population due to its "selective bias in favor of that trait" (Barros 2008: 312). He then characterizes natural selection as a two-level, multistage stochastic mechanism that explains adaptation. Natural selection is two-level "because the phenomenon of adaptation cannot be fully explained using either individual level or populational level mechanisms alone" (Barros 2008: 318).[97] Additionally, natural selection is multistage because it operates over time, rather than on a single occasion. It is a stochastic mechanism, as it operates probabilistically and not deterministically, being biased in favor of certain traits.

Havstad (2011) raised a critique of Barros' (2008) characterization of natural selection, asserting that it is overly general. Her primary concern is that Barros' account lacks specificity, failing to distinguish natural selection from other selective processes. In other words, Havstad argues that Barros' characterization does not adequately identify natural selection but rather encompasses any form of selective processes. Thus, according to Havstad (2011: 519-520), Barros' characterization of natural selection falls short as a precise account of the explanation of the particular phenomenon of adaptation by natural selection.

Havstad identifies the primary challenge in characterizing natural selection as a mechanism to be the extreme variability among specific cases of natural selection in different populations. She contends that accommodating such variability requires a degree of abstraction. Consequently, a generalized account, such as Barros', is deemed insufficient for accurately characterizing natural selection; instead, it tends to characterize any form of selective process.

Havstad elucidates her perspective on natural selection by drawing a distinction between two phenomena, namely, gene expression, and respiration. Gene expression is a well-defined biological phenomenon that is caused by a recognized mechanism in the literature (see Machamer, Darden and Craver 2000). It involves entities such as DNA, RNA, and proteins, along with activities such as transcription and translation. Both entities and activities are specific and well-known. However, Havstad (2011: 519) posits that natural selection is not analogous to gene expression but rather more akin to respiration. She characterizes respiration

[97] For more information on both individual and population level mechanisms, see Barros (2008: 318).

as a phenomenon that occurs through various mechanisms. For instance, mammals employ lungs, fish use grills, and insects use spiracles, each employing distinct mechanisms for respiration. Drawing a parallel to this distinction, Havstad concludes that Barros' account aligns more with describing natural selection as akin to respiration. According to her, Barros' explanation of natural selection tends to address selection in general sense rather than providing a specific account of it.

Following Havstad's negative account on the characterization of natural selection as a mechanism, I would like to briefly present another perspective arguing against characterizing natural selection as a mechanism. Garson (2019: 257-259) contends that natural selection cannot be considered a mechanism because it lacks a distinct function. Although it contributes to the development of functional traits, such as, for example, zebra stripes, natural selection does not possess an inherent function of its own. Unlike other mechanisms, such as protein synthesis, with a specific function, natural selection is not selected for any particular function. Consequently, Garson asserts that natural selection should be viewed not as a mechanism of evolution but as a cause.[98]

1.5. Illari and Williamson's (2010) Account

To conclude on a positive note, I present Illari and Williamson's (2010) characterization of natural selection. Their characterization follows the ideas of Skipper and Millstein's (2005), as well. Recall that Skipper and Millstein posit that natural selection is neither decomposable nor organized. Illari and Williamson seek to refine the understanding of decomposition and organization in this sense. To achieve this, they draw a comparison between the mechanisms of protein synthesis and natural selection, highlighting three core elements of mechanistic explanations. These elements are as follows: *functional individuation, hierarchical nestedness or decomposition,* and *organization*. According to Illari and Williamson, natural selection, akin to protein synthesis, incorporates all three elements in its mechanistic characterization.

Now let me briefly examine these three elements. Functional individuation is highlighted because mechanistic explanation begins with the individuation of the phenomenon to be explained. New mechanistic authors generally agree that all mechanisms are mechanisms *for* some phenomenon or possess a specific functional role (see Machamer, Darden and Craver

[98] I examine this idea of natural selection perceived as a cause in the following section, particularly by partially characterizing natural selection as a pathway.

2000: 6; Glennan 2017: 23). In this sense, Illari and Williamson (2010: 282) stress that functional individuation is a crucial element of mechanistic explanation. They illustrate this with two examples, that is, with the already mentioned protein synthesis and natural selection. In the case of protein synthesis, it serves as the mechanism *for* the production of proteins. Biochemistry textbooks typically define the mechanism of protein synthesis as having the function of decoding the information in DNA to produce proteins.[99] On the other hand, natural selection explains adaptation. According to Illari and Williamson, natural selection inherently produces adaptation, making it a mechanism *for* adaptation. In this sense, natural selection is functionally individuated and qualifies as a mechanism.

The second core element of mechanistic explanation is hierarchical nestedness or decomposition. According to Illari and Williamson (2010: 284), "once the phenomenon is identified, mechanistic explanation characteristically proceeds by decomposing the phenomenon into lower-level components". Decomposition is a common theme among new mechanistic authors. For instance, Machamer, Darden and Craver (2000) highlight entities and activities in mechanisms, while Bechtel and Abrahamsen (2005) outline component parts and component operations (see Part II, Chapter 4). Despite terminological differences, new mechanistic authors concur that decomposition is a crucial feature of mechanisms. The prime example of decomposition is evident in the mechanism of protein synthesis. Using Machamer, Darden and Craver's terminology, entities in the mechanism of protein synthesis include DNA, RNA, and ribosomes, while activities involve replication, transcription, and translation. This detailed breakdown of components allows for the observation of sub-mechanisms within protein synthesis, elucidating how DNA is decoded to produce protein. However, unlike the mechanism of protein synthesis, identifying the entities and activities in the mechanism of natural selection is not as clear.

As I mentioned earlier, Skipper and Millstein (2005) contend that natural selection lacks the necessary parts for decomposition. Conversely, Illari and Williamson argue that the concept of decomposition needs to be reinterpreted to assert that natural selection does indeed involve entities (or component parts) and activities (or component operations). They emphasize the significance of functional decomposition, asserting that mechanism should be decomposed into entities or activities displaying distinct functions. Mechanism of natural selection, as well as

[99] It is important to note that when Illari and Williamson refer to the characterization of 'function', they are specifically considering the causal role account of functions. In their paper (2010: 282-283), they explore various characterizations of functions relevant to mechanisms and ultimately endorse the causal role account, also known as Cummins' account. This choice aligns with Craver's (2001) analysis of the relation between mechanisms and functions, which I discussed in Part I (see Chapter 3).

mechanisms in other domains, besides biochemistry, might not have the parts that are physically similar and functionally relevant in comparison to the mechanism of protein synthesis.

According to the concept of functional decomposition, the mechanism must be broken down into entities and activities that serve different functions. Illari and Williamson (2010: 285) assert that, considering functional decomposition, the mechanism of natural selection aligns with a functional hierarchy, i.e., the hierarchy by which a mechanism decomposes into sub-mechanisms. They propose that natural selection involves entities such as populations, organisms, cells, DNA, chromosomes, and alleles, along with activities such as directional selection, stabilizing selection, disruptive selection, reproduction, and meiosis.

Illari and Williamson contend that protein synthesis and natural selection are analogous, although they acknowledge some differences between them. For instance, in natural selection, activities often play a more crucial role than entities for the explanation of phenomena. The activities in natural selection are more diversified than entities, which are relatively similar (e.g., organisms of the same species). While entities must be present, activities, or rather sub-mechanisms, are more extensively studied due to their greater functional significance. In protein synthesis, on the other hand, entities hold more importance than activities, with a greater emphasis on the microstructural details since structural differences are more functionally vital. Despite these distinctions, Illari and Williamson (2010: 287) conclude that both entities and activities are crucial for both mechanisms.

The third and final core element of mechanistic explanation is organization. Merely identifying entities and activities in mechanisms is insufficient; instead, one has to also comprehend how they collectively produce the phenomenon, i.e., their organization. According to Illari and Williamson, once a phenomenon is identified and relevant entities and activities are discovered, it is crucial to delineate the specific relations these entities and activities have with each other to produce the phenomenon in question. In other words, organization pertains to the coordinated action of entities and activities, working together to bring about the phenomenon.

Illari and Williamson (2010: 290), contrary to Skipper and Millstein, argue that natural selection exhibits organization. However, they acknowledge a distinction between the mechanism of protein synthesis and natural selection. Protein synthesis often operates in a closed system, such as a cell, shielded from external influences. On the other hand, natural selection operates in an open system that is highly sensitive to initial conditions and slight perturbations, occasionally resulting in unexpected outcomes. Despite this difference, Illari and

Williamson assert that, like protein synthesis, natural selection displays spatiotemporal organization. However, it is important to note that the organization observed in natural selection occurs at a certain level of abstraction, which I explore in the next section.

SECTION 2. Natural Selection: A (Strategic) Pathway?

In this section, I explore the possibility of characterizing the natural selection schema proposed by Skipper and Millstein (2005) as a distinct causal structure, specifically a pathway (see Figure 21). I particularly examine this idea since I believe that the natural selection schema in question exhibits a temporal characteristic, aligning with features corresponding to pathways, as extensively discussed in Part II (see Chapter 5). I draw on the three aspects of the pathway concept previously examined, with a particular emphasis on the third aspect, namely, the strategic pathway (SP). However, before delving into the analysis of the natural selection schema, let me first present the schema as outlined by Skipper and Millstein (2005). I employ this schema as a basis to identify specific components that, I argue, could be characterized as SP.

Skipper and Millstein (2005: 329) present a schema of natural selection that significantly influences later debate on the issue, as seen in the previous section. They represent natural selection as a sequence of *temporal* stages, delineating it into seven distinct stages. In Stage I, individuals, such as, for instance, organisms, showcase variation in a particular property within the respective population. In Stage II, these organisms interact with their environments. Throughout Stages III and IV, the nature of the interaction is influenced by the properties these organisms possess. These properties directly impact the survival and reproductive probabilities of individual organisms. Those organisms, whose chances of survival and reproduction are enhanced through interactions with their environments, tend to produce a greater number of offspring with similar properties in the subsequent generations. Conversely, the opposite is true for individual organisms that experience diminished survival and reproductive success. Moving to Stage V, over time, specific properties that enhance survival and reproduction become increasingly prevalent within the population. Transitioning to Stage VI, after numerous generations, the majority of population members exhibit this advantageous property. Finally, in Stage VII, this cumulative process culminates in lineage adaptation.

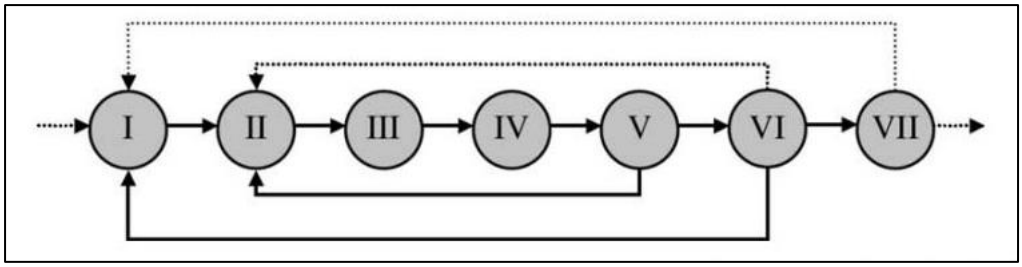

Figure 21: The NS schema (reproduced from Skipper and Millstein 2005: 331). The schema depicts natural selection structured in seven stages. The circles with Roman numerals are stages that are described at the beginning of Section 2.

As an illustration of the schema, Skipper and Millstein (2005) utilize the well-known example of Darwin's finches, a species often referenced in discussions of natural selection:

> There exists a population of finches, *O*s. The finches vary according to their beak length T, the form of a trait that is heritable. And the finches are in a rocky environment E where there is a critical factor, F—variously shaped edible seeds varying in availability. Notice that the critical factor F in conjunction with differences in T is what sets up the selective interaction. Certain beak lengths enable some of the finches to obtain seeds that other finches, lacking the appropriate beak length, are unable to obtain. This constraint on environmental resources that leads to a struggle for existence and a check on the population is the interaction step of the schematic. Given the initial conditions and the causal interaction between the environment and the finches, we expect various downstream effects. That is, we expect differential survival and reproduction, and the other stages described. (Skipper and Millstein 2005: 329)

Before revisiting the abovementioned seven stages and delineating those that could be perceived as SP, I provide a brief summary of the three aspects of the pathway concept examined in Part II (see Chapter 6).

Particularly, because these aspects, partly, correspond to Levy's (2013) three kinds of mechanism and his discussion on natural selection. Here, I provide a brief summary of his view before turning to my perspective. Levy (2013) distinguishes three theses associated with the new mechanistic account, pertaining to causation, explanation, and scientific methodology. These theses are framed as follows: Causal Mechanism (CM), Explanatory Mechanism (EM), and Strategic Mechanism (SM). Levy notes that these three theses "aren't always well-marked in the literature", which, he believes, "hampers several ongoing debates" (Levy 2013: 2). One such debate revolves around whether natural selection can be characterized as a causal structure (e.g., a mechanism). Hence, it is beneficial to differentiate between these theses.

CM offers a perspective on causal relationships, often challenging alternative theories of causation such as the counterfactual account (as proposed by Lewis in 1973) and regularity

approaches (as discussed by Psillos in 2002), among others. In essence, CM posits that causal connections between events exist through the concept of mechanisms. A prominent advocate of this viewpoint is Glennan (1996), who suggests that a mechanism responsible for a particular behavior is a complex system that generates said behavior through the interactions among its various components. Glennan's work addresses Hume's well-known "secret connexion" problem that links cause and effect, proposing a solution grounded in the notion of a mechanism. Consequently, Levy concludes that this thesis primarily pertains to metaphysics.

EM revolves around the notion that to effectively explain a phenomenon, one must provide mechanistic information, encompassing the identification of entities, activities, and their interactions and organization. In other words, mechanisms specify the type of information required for a successful scientific explanation. EM has more proponents than CM, including authors such as Machamer, Darden, and Craver (2000) and Bechtel and Abrahamsen (2005). This thesis primarily pertains to epistemology.

SM revolves around the cognitive and epistemic potential of mechanistic modeling and scientific methodologies. This perspective asserts that mechanistic reasoning represents the optimal approach when analyzing scientific phenomena, providing a highly effective scientific methodology. Described as “a framework for representing and reasoning about complex systems” (Levy 2013: 6-7), this thesis primarily pertains to methodology, earning the label “strategic” due to its emphasis on the importance of mechanistic methods with specific cognitive and epistemic attributes. Prominent proponents of SM, such as Bechtel and Richardson (2010), focus on two mechanistic methods: decomposition and localization. Recall that decomposition involves breaking down a system into its constituent parts, while localization entails identifying distinct causal roles for these parts (see Part II, Chapter 4). These strategies are applicable to examples like clocks, engines, and large enzymatic complexes such as ATPases. However, when components are widely distributed or challenging to isolate, Bechtel and Richardson's approach may have limitations. This suggests that their strategy aligns more with proximal biology, such as molecular and cell biology.

In Part II (see Chapter 4), I proposed a similar distinction among different aspects of mechanism. However, I chose an alternative approach, terming these aspects as ontic, epistemic, and strategic. This choice aimed to align with the already established and ongoing debate within the new mechanistic account, specifically their ontic and epistemic characterizations. Additionally, I introduced the strategic aspect to these characterizations. On one side, inspired by Levy’s (2013) SM, and, on the other side, influenced by investigative strategies present but not spelled out throughout the ongoing debate on the new mechanistic

account. I delineated the ontic aspect as central to new mechanistic authors and corresponding to the mechanism itself, as observed in nature. The epistemic aspect involves considerations related to the representation of mechanisms (e.g., building models). The strategic aspect related to the heuristics of decomposition and localization.

Levy (2013) emphasizes that CM and EM, somewhat akin to ontic and epistemic aspects, are not central to the discussion concerning whether natural selection potentially exhibits mechanistic features. Instead, he directs attention to SM. Levy (2013: 14) outlines this perspective in the following way: "Skipper and Millstein's discussion does not make explicit reference to methods and strategies. But their choice to represent natural selection in a graphic form – a relatively unusual attempt – suggest that such issues are in the back of their minds". Therefore, I do not examine CM and EM in greater detail; rather, I focus on the relationship between SM, that is, the strategic aspect of mechanism, and natural selection. Consequently, I explore the strategic aspect of the pathway concept and its relationship to natural selection.

Levy (2013) employs the following approach to examine the relationship between SM and natural selection: "the applicability of mechanistic methods is chiefly a function of the causal structure of natural selection and kindred populational processes" (Levy 2013: 14). In other words, this approach suggests a comparison between the causal structures of natural selection and the types of systems, which are central to mechanistic methodologies, such as machine-like structures within individual organisms. According to Levy (2013), Skipper and Millstein (2005) adopt this approach to examine whether natural selection involves components, productive continuity, or organization, that is, the basic new mechanistic features.[100] They achieve this by presenting natural selection in a schematic fashion (see Figure 21). In this schema, they delineate distinct stages, portraying natural selection as a sequence of steps. Skipper and Millstein (2005) examine natural selection as if it were a system that can be decomposed into its constituent parts, which are then localized. This is evident from their examination of various features of mechanisms as outlined in the new mechanistic account, namely organization, regularity, and productive continuity. Lastly, I posit that SM influenced

[100] Levy (2013: 15) emphasizes another approach to the issue in question, which explores the possibility of applying mechanistic reasoning to natural selection, despite the potential perception of it not strictly conforming to traditionally endorsed concept of a mechanism (see Matthewson and Calcott 2011). This perspective contends that mechanistic thinking and methods are shaped by the cognitive strengths and limitations of scientists. Portraying a system as though it were a mechanism proves exceptionally valuable in framing and contemplating biological systems. However, I focus on the first approach, as it aligns more closely with the natural selection schema outlined by Skipper and Millstein (2005).

their paper, as well as the following debate, since Skipper and Millstein (2005) explicitly emphasize the following:

> We believe that the schematic representation, as an abstract portrayal of natural selection that exposes its causal essence, offers the best avenue for advancing our comprehension of natural selection as a mechanism in particular, and for understanding the nature and role of mechanisms in science in general. Skipper and Millstein (2005: 331)

Above, I emphasized the need for clarification regarding which of the new mechanistic theses is delineated in the debate in question. Specifically, I posited that mechanisms, namely SM, are a type of causal structure that is considered when assessing the natural selection schema outlined in Skipper and Millstein (2005). However, I advocate for an alternative approach, namely one that considers pathways as causal structures that could characterize the natural selection schema.

Before assessing that argument, I draw attention to a critique aimed at characterizing natural selection as a mechanism, which emphasizes the abstract nature of natural selection. This critique leaves an opportunity to consider another causal structure that could characterize it, namely pathways. The criticism is articulated by Havstad (2011: 520), who contends that "natural selection might be well characterized as an abstract mechanism". Moreover, she argues the following: "at the general level, natural selection is an abstract process without the entities/parts and activities/interactions characteristic of a mechanism" (Havstad 2011: 522).

Recall that the three aspects of the pathway concept outlined in Part II (see Chapter 6), are ontic, epistemic, and strategic aspects. In aligning with Levy's (2013) terminology, I refer to the strategic aspect of pathways as the *strategic pathway* (SP). Now, let me examine whether the natural selection schema (see Figure 21), could be characterized as SP. I argue that it could be, at least partially. Specifically, if one considers Stages II, III, and IV of the schema. On the other side, I argue that Stages I, V, VI, and VII, exhibit SM characteristics.

Both SM and SP revolve around investigative strategies. SM pertains to the causal strategies of decomposition and localization, focusing on breaking down a mechanism into its constituent parts and pinpoint their locations. On the other hand, SP involves the strategy of expanding or mapping out, i.e., an investigative strategy that works as a causal map. Instead of decomposing and localizing, SP involves identifying a set of entities in a given domain. Consequently, the map expands by tracing causal connections. Recall that these causal connections identified within biological pathways hold potential, encompassing information

“pertaining to various causal possibilities rather than a singular, tightly bounded causal process leading to an effect" (Ross 2021: 144). Consequently, as a result: “these causal maps do not resemble car engines or watch mechanisms in the sense that they portray parts, all contingent on a singular primary behavior of some system. They are more akin to a collection of available highways that a vehicle or entity of interest can traverse” (Ross 2021: 144).

Now, let me revisit Levy’s (2013) three theses concerning mechanisms and explore the idea whether SM can be applied to the natural selection schema, along with the abovementioned SP. I propose that SP shares similar values with SM by providing a framework for representing and reasoning about complex systems, primarily in terms of methodology, as outlined earlier. SP serves as a framework that highlights a strategic approach, giving significance to methods with cognitive and epistemic features. The key distinction between SM and SP lies in SP's emphasis on the aforementioned cognitive and epistemic features within pathway modeling (e.g., constructing maps and identifying causal connections).

In this sense, I examine whether SP could more accurately accommodate the previously presented natural selection schema. I argue that it can, as it underscores the representational potential of causal connections relevant to diverse explanatory outcomes, aligning well with the concept of natural selection. Consequently, in the following discussion, I emphasize that the natural selection schema exhibits SP in Stages II, III, and IV. Simultaneously, the natural selection schema demonstrates SM in the remaining stages, namely, Stages I, V, VI, and VII.

Let me first address the SP characterization of the natural selection schema. Recall that in Stage II of the schema in question, organisms within a population exhibit variation in certain properties, while engaging with their environments. In this stage, it is crucial to note that these properties influence the organisms’ prospects for survival and reproduction in a *variety of ways*. If we consider these properties to be heritable, organisms with improved chances of survival and reproduction due to their interaction with the environment tend to produce more offspring, passing on these advantageous properties. Conversely, organisms with reduced chances of survival and reproduction due to their interaction with the environment tend to produce fewer offspring (see Skipper and Millstein 2005: 329).

To illustrate this, recall the case study of Darwin’s finches. In line with the natural selection schema (see Figure 21), Stage I depicts a population of finches. In Stage II, these finches showcase variation in their beak length, representing an inheritable fitness trait. The finches inhabit a rocky environment with differently shaped edible seeds, each with varying availability. Based on their beak lengths, some finches can access seeds that others cannot. This scarcity of resources in their environment leads to the well-known ‘struggle for existence’.

According to Skipper and Millstein (2005: 329): “given the initial conditions and the causal interaction between the environment and the finches, we anticipate various downstream effects”. In other words, different rates of survival and reproduction are expected, as detailed in the subsequent stages of the natural selection schema.

I argue that Stage II in the schema is better accommodated as an SP. This is primarily because, as discussed earlier in the context of Ross’ characterization of pathways, SP identifies a set of entities and traces their causal connections. These connections possess investigative potential, encompassing a range of causal possibilities rather than a singular causal process leading to an outcome. In this sense, natural selection, especially in Stage II, is characterized similarly. Specifically, there is a population of individual organisms, such as finches, and several potential causal possibilities exist, i.e., various potential causal processes leading to a *downstream* effect. Different finches vary not only in their properties, but also in their interaction with their environments, resulting in various probabilities of survival and reproduction. Therefore, to use Ross’ analogy, SP operates more like the selection of available highways that an entity travels along, rather than a mechanism, such as a car engine or a watch, which aligns more closely with the way in which SM operates.

Moreover, I contend that Stages III and IV in the natural selection schema are also characterized as an SP. This is particularly evident because these stages represent an extension of Stage II. Throughout these stages, one can observe a process in which several causal possibilities exist, and the interaction in place is influenced by the properties that organisms possess.

Furthermore, I advocate for SP because it addresses the concern raised by Havstad (2011), as previously emphasized. Specifically, the notion that natural selection is an abstract process. Ross (2021) highlights that one of the features of pathways, which distinguishes them from mechanisms, is their abstract nature. In other words, pathways represent causal sequences that abstract from a significant number of details. This is particularly true for the natural selection schema in question, where natural selection encompasses seven distinct stages. One can draw parallels between the schema (see Figure 21) and the glycolytic pathway (see Part II, Figure 17). Both schemas are depicted in stages, aligning with the SP characterization by sharing the aforementioned pathway features.

Let me now consider the remaining stages of the natural selection schema, namely, Stages I, V, VI, and VII. I argue that these stages can be characterized as SM. I believe that these stages exhibit investigative strategies more specifically tied to mechanisms, namely, decomposition and localization. For instance, localization can be employed by identifying

causal roles for the system's component parts. It seems to me that in these stages, there are populations with stable properties, and we can identify those properties and organisms, which have entities and activities that are organized in such a way as to produce an outcome, in contrast to the various causal possibilities found in SP, as in Stages II, III, and IV of the natural selection schema.

Recall that in Stage I, there are certain initial conditions, i.e., there is a population (e.g., of finches) with determined phenotypic properties (e.g., the beak length). In this stage, we can decompose the population into different subgroups of organisms, and those subgroups can be further decomposed into entities performing specific activities (e.g., the beak used for eating). In Stages I, V, VI, and VII, we can pinpoint the prevalent properties in the population at that point (e.g., finches with the fitness-enhancing beak length given the availability of a certain type of seeds). Furthermore, these stages exhibit more of a spatial characteristic discussed earlier in Part II (see Chapter 6), which is more prominent in mechanisms. Particularly, since these stages emphasize populations with their specific properties, these populations exhibit their advantageous properties, which, in the final stage of the schema, leads to lineage adaptation. On the other side, as stated earlier, Stages II, III, and IV exhibit more of a temporal characteristic, which is more prominent in pathways. Specifically, the natural selection schema outlines that these stages are process-like, namely through organisms' interaction with their respective environments.

Although the entire natural selection schema, as I highlighted at the beginning of this section, exhibits a temporal characteristic due to its representations in sequential steps, implying a pathway characteristic, I also emphasize that certain stages display mechanistic characteristics. One might criticize this view by referring to my earlier argumentation in Part II (see Chapter 6), where I outlined that mechanisms exhibit both spatial and temporal characteristics. However, I also argued for a distinction between the investigative strategies found in mechanisms and pathways. In this chapter, examining the natural selection schema, I concluded that these investigative strategies are evident in the schema offered by Skipper and Millstein (2005), and followed in the further debate on the issue in question. Thus, there is room for both SP and SM, as both aspects of these causal strategies contain temporal characteristics. However, as I argued, Stages II, III, and IV, have a specific feature of creating causal connections and routes similar to the investigative strategies displayed by the pathway concept.

CONCLUSION. Chapter 8

To sum up, I addressed the ongoing discussion surrounding the characterization of natural selection, with a particular focus on its representation as a causal structure. I examined the debate initiated by Skipper and Millstein (2005) presenting both positive and negative accounts addressing the issue in question. Proponents of the negative side include authors such as Skipper and Millstein (2005), Havstad (2011), and Garson (2019), while on the positive side, proponents include authors such as Barros (2008), Illari and Williamson (2010), and DesAutels (2016).

I began by examining the seminal paper authored by Skipper and Millstein (2005), providing a detailed analysis of the natural selection schema consisting of seven distinct stages discussed in their work. In that sense, I argued that this schema can be characterized not only as mechanistic but also as exhibiting pathway characteristics, depending on the various stages identified in the schema. Specifically, I advocated that Stages II, III, and IV exhibit SP characteristics, while the remaining stages exhibit SM characteristics (as found in Levy's (2013) depiction of three theses of new mechanism).

The stages characterized as exhibiting SP features involve population of individual organisms, such as, for instance, finches, with several potential causal possibilities or various potential causal processes leading to a downstream effect. These potential causal possibilities align with the investigative strategy of mapping or expanding out, which is a key aspect of the pathway concept characterized by Ross (2021). On the other hand, I argued that Stages I, V, VI, and VII correspond to the investigative strategies of decomposition and localization, particularly because of the properties prevalent in the population of, for instance, finches (e.g., the fitness-enhancing beak length), which can be pinpointed, that is, localized.

Moreover, I reiterated the significance of spatial/temporal aspects in both pathways and mechanisms (see Part II, Chapter 6). I argued that SP stages exhibit more of a temporal aspect, aligned with their respective investigative strategy, while SM stages exhibit more of a spatial aspect, along with the decomposition and localization investigative strategies.

CONCLUSION. Part III

In this part of the thesis, I focused on applying the previously examined concepts, such as pathways and mechanisms, to scientific practice. In this sense, I emphasized two case studies, namely glycolysis and the characterization of natural selection.

In Chapter 7, I examined the case study of inhibiting glycolysis to prevent the growth of cancer cells. Firstly, I revisited the three aspects of pathway concept discussed in Part II (see Chapter 6) and particularly focused on the epistemic aspect. Specifically, I addressed how pathways are represented, emphasizing their distinction from mechanisms. I drew comparisons between Ross' (2021), Thagard's (2003), and my perspective on this distinction. In that sense, I highlighted the divergence from Thagard's perspective by arguing that the investigative strategy of pathways is distinct from mechanistic, thus aligning with Ross. On the other hand, my perspective aligned with Thagard's regarding the ontic aspect, acknowledging shared features between these structures. Finally, I emphasized a distinction in the epistemic aspect, where I pointed out the temporal characteristic in pathways' representations, which was further explored in the subsequent chapter.

Moreover, I examined the significance of the glycolytic pathway in cancer treatment. I highlighted the strategic aspect related to glycolysis by presenting its historical background, which includes the creation of various networks, thus pinpointing to the investigative strategy of pathways. Furthermore, I outlined strategies for inhibiting glycolysis, which is a primary energy source for the growth of cancer cells. I presented two of these strategies, namely targeting HK2 and GLUT1. Consequently, I asserted that Woodward's (2003) interventionist account of causal explanation may be utilized as the conceptual framework, which underpins the glycolysis case in question. Thus, explaining the significance of interventions and manipulations on causes to bring about a change in the outcome.

In Chapter 8, I addressed the ongoing debate on whether natural selection can be characterized as a causal structure. I presented the debate initiated by Skipper and Millstein (2005). Subsequently, I outlined both positive and negative accounts addressing the issue. In particular, I provided a detailed examination of Skipper and Millstein's natural selection schema, consisting of seven distinct stages. I argued that some of these stages exhibit strategic pathway characteristics, while others mechanistic. Here, I again revisited the three aspects of pathways and mechanisms, focusing on the strategic aspect. In that sense, I concluded that Stages II, III, and IV of the natural selection schema align with the investigative strategy of mapping or expanding out, prominent in the pathway concept, while Stages I, V, VI, and VII

align with the mechanistic investigative strategies of decomposition and localization. Moreover, I revisited the spatial and temporal aspects of both pathways and mechanisms and argued that stages corresponding to the pathway concept exhibit more of a temporal characteristic, while other stages exhibit more of a spatial characteristic, pertaining to mechanisms.

CONCLUSION

The thesis focused on the causal explanation in biology, particularly molecular biology, closely examining terms like explanation and causation. The thesis argued that these concepts are intertwined and ubiquitously employed in everyday discourse. Although other forms of explanation exist, such as those arguing for an explanation in the form of an argument (e.g. covering law theory; unificationist account) or non-causal explanations (e.g., mathematical), explanations in the life sciences usually take the form of causal explanations. The explanans in these explanations can be various. The thesis engaged with the ones tied to the new mechanistic account, the predominant framework in the discourse surrounding molecular biology. This explanans usually adopt the form of causal structures, such as mechanisms, pathways, or cascades, aimed at explaining biological phenomena. The thesis attempted to examine these structures, exploring their specific similarities and differences, and arguing for mechanisms as the privileged causal structure for explanation. Additionally, the thesis outlined Woodward's (2003) interventionist causal explanation account, commonly employed by new mechanistic authors and the one deemed suitable for scientific practices. In this sense, the thesis argued that this account can serve as a conceptual framework in case studies, such as those examined in Part III. Specifically, it explored the case of inhibiting glycolysis to prevent the growth of cancer cells, and the case of characterizing natural selection in terms of causal structures.

The thesis had three aims, each corresponding to one of the three parts. Part I, divided into three chapters, aimed to systematically present and structure the debate. It placed a particular focus on elucidating the background concepts related to the new mechanistic account. These concepts included explanation, causation, mechanism, biological function, and law of nature. Each concept is revisited, to some degree, in other parts of the thesis, particularly when addressing causal-explanatory structures, i.e., mechanisms, pathways, and cascades, and their application to scientific practice. Among these background concepts, Part I particularly emphasized Woodward's interventionist account of causal explanation, deeming it suitable conceptual framework for examining causal-explanatory structures in biology, especially within molecular biology. The choice of this account is motivated, on one side, for being commonly endorsed by new mechanistic authors, and on the other side, for its significance in the context of scientific practice. The interventionist account highlights the importance of intervention and manipulation in explaining a phenomenon, assessing whether manipulating causes can alter the effect, ultimately changing the outcome of a process.

Part II, divided into three chapters, aimed to advocate for a consensual characterization of the new mechanistic account, attempting to establish a unified perspective among various new mechanistic authors. This part also highlighted distinct causal-explanatory structures beyond mechanisms, encompassing pathways and cascades. Moreover, for each structure, the thesis delineated the three aspects of the explanation process, namely ontic, epistemic, and strategic aspects. This part particularly emphasized the difference in the strategic aspect, where, unlike mechanisms, pathways and cascades do not rely on the heuristics of decomposition and localization but instead employ the strategy of building networks, that is, mapping or expanding out their processes. The difference was also evident in the epistemic aspect, where pathways and cascades use more abstract representations and incorporate process-like, i.e., temporal characteristics. On the other hand, mechanisms encompass both system and process-like characteristics, with the system-like referring to spatial characteristic. Despite these differences, all three structures share similar features in the ontic aspect, being encompassed by the features of productive continuity and organization, originally associated with mechanisms. Consequently, Part II advocated for mechanisms as the explanatory privileged causal structure, specifically due to them incorporating both spatial and temporal characteristics, corresponding to the system and process-like aspects.

Part III, divided into two chapters, aimed to apply the previously examined causal-explanatory structures and their three aspects of explanation to scientific practice. The case studies in question were: (a) the inhibition of glycolysis to prevent the growth of cancer cells, and (b) the characterization of natural selection in terms of causal-explanatory structures. Considering case study (a), Part III outlined two potential targets, specifically Glucose transporter 1 and Hexokinase 2, as suitable targets for intervention, leading to the inhibition of the glycolytic pathway. Moreover, it asserted that the conceptual framework of the interventionist account best accommodates this case study. Particularly, because it emphasizes the role of intervention and manipulation of causes to alter outcomes, which is evident in this example. Furthermore, regarding the same case study, the thesis advocated for a distinct epistemic approach to observing pathways, differing from other views such as Ross' (2021) and Thagard's (2003). The thesis argued that pathway representations involve a strong temporal characteristic, which is crucial in the context of the case study in question. Regarding case study (b), the thesis examined whether the natural selection schema proposed by Skipper and Millstein (2005) exhibits mechanistic characteristics. It proposed a novel perspective, characterizing the natural selection schema as partially a pathway and partially as a mechanism.

Specifically, because some parts of the schema exhibit more of a temporal characteristic, while others display spatial characteristics.

To sum up, the thesis contributed to the debate on the causal explanation in molecular biology in three ways, as follows: (i) by structuring the debate, emphasizing the interventionist account of causal explanation, the causal role of functions, and the mechanistic approach to laws of nature as key conceptual frameworks in the debate; (ii) by advocating a distinction between ontic, epistemic, and strategic aspects of mechanisms, pathways, and cascades, asserting that mechanisms hold the explanatory privileged status among these structures; and (iii) by applying the conceptual framework of the interventionist account of causal explanation, encompassing the examined structures, to two significant case studies from scientific practice, namely cases in molecular oncology and the characterization of natural selection.

REFERENCES

Akram, M. (2013). Mini-Review on Glycolysis and Cancer. *Journal of Cancer Education* 28, 454-457.

Allen, E. G. (2005). Mechanism, Vitalism, and Organicism in Late Nineteenth and Twentieth-Century Biology: The Importance of Historical Concept. *Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Sciences* 36(2), 261-283. https://doi.org/10.1016/j.shpsc.2005.03.003.

Andersen, H. (2011). Mechanisms, Laws, and Regularities. *Philosophy of Science* 78(2), 325-331.

Andersen, H. (2012). The Case for Regularity in Mechanistic Causal Explanation. *Synthese* 189(3), 415-435.

Andersen, H. (2014). A Field Guide to Mechanism: Part I. *Philosophy Compass* 9(4), 274-283. https://doi.org/10.1111/phc3.12119.

Anić, Z. (2022). *Mechanisms and Mechanistic Reasoning in Medicine* (PhD thesis). Sveučilište u Rijeci, Filozofski fakultet, https://urn.nsk.hr/urn:nbn:hr:186:253993.

Ayala, J. F. (1970). Teleological Explanations in Evolutionary Biology. *Philosophy of Science* 37, 1-15.

Baetu, T. (2019). *Mechanisms in Molecular Biology*. Cambridge University Press

Balorda, V. (2023). Reductionism Debate in Molecular Biology: Max Delbrück's Complementarity Approach. *Studia Historiae Scientiarum* 22, 587-610. https://doi.org/10.4467/2543702XSHS.23.016.17707.

Barros, D. B. (2008). Natural Selection as a Mechanism. *Philosophy of Science* 75(3), 306-322.

Bartel, P. D. (2004). MicroRNAs: Genomics, Biogenesis, Mechanism, and Function. *Cell* 116(2), 281-297.

Bechtel, W. (2011). Mechanisms and Biological Explanation. *Philosophy of Science* 78, 533-557.

Bechtel, W. (2015). Can Mechanistic Explanation Be Reconciled with Scale-Free Constitution and Dynamics? *Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Science,* 53, 84-93.

Bechtel, W. (2019). Analyzing Network Models to Make Discoveries about Biological Mechanisms. *British Journal for the Philosophy of Science*, 70, 459-484.

Bechtel, W., and Abrahamsen, A. (2005). Explanation: A Mechanist Alternative. *Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Science* 36 (2), 421–41. https://doi.org/10.1016/j.shpsc.2005.03.010.

Bechtel, W., and Abrahamsen, A. (2013). Thinking Dynamically about Biological Mechanisms: Networks of Coupled Oscillators. *Foundations of Science*, 18, 707-723.

Bechtel, W., and Richardson, R. (1993). *Discovering Complexity: Decomposition and Localization as Strategies in Scientific Research.* Princeton University Press.

Bechtel, W., and Richardson, R. (2010). *Discovering Complexity: Decomposition and Localization as Strategies in Scientific Research.* Cambridge: MIT press.

Berryman, S. (2009). *The Mechanical Hypothesis in Ancient Greek Natural Philosophy*. Cambridge: Cambridge University Press.

Bigelow, J., and Pargetter, R. (1987). Functions. *Journal of Philosophy*, 84(4), 181-196.

Blečić, M. (2022). The Notion of 'Information' in Genetics: A Pragmatic Model. *Journal of Biological Education*, 1-12.

Bogen, J. (2005). Regularities and Causality: Generalizations and Casual Explanations. *Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Sciences.* 36(2): 397-420.

Bogen, J., and Woodward, J. (1988). Saving the Phenomena. *Philosophical Review*, 97(3), 303-352.

Bois-Reymond, E. D. (1884). *Untersuchungen über Thierische Eelektricität.* Berlin: G. Reimer.

Boniolo, G., and Campaner, R. (2018). Molecular Pathways and the Contextual Explanation of Molecular Functions. *Biology & Philosophy*, 33(3-4), 24.

Boorse, C. (2002). A Rebuttal on Functions. In: Ariew, A., Cummins, R. C., and Perlman, M. (eds.), *Functions: New Essays in the Philosophy of Psychology and Biology*. Oxford University Press.

Brandon, R. (1985). Grene On Mechanism and Reductionism: More Than Just a Side Issue. *PSA: Proceedings of the Biennial Meeting of the Philosophy of Science Association 1984*, 345-353.

Brigandt, I. (2013). Systems Biology and the Integration of Mechanistic Explanation and Mathematical Explanation. *Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Sciences,* 44(4), 477-492.

Brigandt, I., and Love, A. (2022). Reductionism in Biology, *The Stanford Encyclopedia of Philosophy* (Summer 2022 Edition). https://plato.stanford.edu/archives/sum2022/entries/reduction-biology.

Briggs, R. W. (2016). How Do Sunflowers Follow the Sun – and to What End? *Science* 353(6299), 541-542. https://doi.org/10.1126/science.aah4439.

Bromberger, S. (1966). Why Questions. In: Colodny, R. (ed.), *Mind and Cosmos: Essays in Contemporary Science and Philosophy*. Pittsburgh: University of Pittsburg Press.

Brzović, Z., and Šustar, P. (2020). Postgenomics Function Monism. *Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Sciences* 80, 101243. https://doi.org/10.1016/j.shpsc.2019.101243.

Brzović, Z., Balorda, V., and Šustar, P. (2021). Explanatory Hierarchy of Causal Structures in Molecular Biology. *European Journal for Philosophy of Science,* 11 (2): 1-21. https://doi.org/10.1007/s13194-021-00380-7.

Buller, J. D. (1998). Etiological Theories of Function: A Geographical Survey. *Biology and Philosophy*, 13, 505-527.

Caro, T., Izzo, A., Reiner Jr., R. C., Walker, H., and Stankowich, T. (2014). The Function of Zebra Stripes. *Nature Communications*, 5, 3535.

Carroll, W. J. (2020). Laws of Nature. *The Stanford Encyclopedia of Philosophy* (Winter 2020 Edition), https://plato.stanford.edu/archives/win2020/entries/laws-of-nature/.

Chaudhry, R., and Varacallo, M. (2022). Biochemistry, Glycolysis. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; (2023) January. Available from: https://www.ncbi.nlm.nih.gov/books/NBK482303/

Craver, F. C. (2001). Role Functions, Mechanisms, and Hierarchy. *Philosophy of Science*, 68(1), 53-74.

Craver, F. C. (2005). Beyond Reduction: Mechanism, Multifield Integration, and the Unity of Neuroscience. *Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Sciences*, 36, 373-397.

Craver, F. C. (2007). Constitutive Explanatory Relevance. *Journal of Philosophical Research*, 32, 3-20.

Craver, F. C. (2009). Mechanisms and Natural Kinds. *Philosophical Psychology*, 22(5), 575-594.

Craver, F. C. (2013). Functions and Mechanisms: A Perspectivalist View. In: Huneman, P. (ed.), 2013, *Functions: Selection and Mechanisms*, 133-158.

Craver, F. C., and Darden, L. (2013). *In Search of Mechanisms: Discoveries Across the Life Sciences*. Chicago and London: The University of Chicago Press.

Craver, F. C., and Kaplan, D. M. (2020). Are More Details Better? On the Norms of Completeness for Mechanistic Explanations. *The British Journal for the Philosophy of Science*, 71(1), 287-319.

Craver, F. C, and Tabery, J. (2019). Mechanisms in Science. The Stanford Encyclopedia of Philsoophy (Summer 2019 Edition). https://plato.stanford.edu/entries/science-mechanisms/.

Crick, F. (1970). Central Dogma of Molecular Biology. *Nature* 227, 561-563.

Cummins, E. R. (1975). Functional Analysis. *Journal of Philosophy*, 72, 741-764.

Cummins, E. R. (1983). Analysis and Subsumption in the behaviorism of Hull. *Philosophy of Science* 50(March), 96-111.

Cummins, E. R. (2000). Reply to Millikan. *Philosophy and Phenomenological Research*, 60(1), 113-127.

Cummins, E. R. (2002). Neo-Teleology. In: A. Ariew, R. Cummins, and M. Perlman (eds.), 2002, *Functions: New Essays in the Philosophy of Psychology and Biology*, Oxford University Press.

Darden, L. (1991). *Theory Change in Science: Strategies from Mendelian Genetics*. New York: Oxford University Press.

Darden, L. (2005). Relations Among Fields: Mendelian, Cytological and Molecular Mechanisms. *Stud. Hist. Phil. Biol. & Biomed. Sci.*, 36, 349-371.

Darden, L. (2006). *Reasoning in Biological Discoveries: Essays on Mechanisms, Interfield Relations, and Anomaly Resolution*. Cambridge University Press.

Davies, S. P. (2001). *Norms of Nature: Naturalism and the Nature of Functions.* Cambridge: MIT Press.

Deaton, A. (2010). Understanding the Mechanisms of Economic Development. *Journal of Economic Perspectives*, 24(3), 3-16.

DesAutels, L. (2016). Natural Selection and Mechanistic Regularity. *Studies in History and Philosophy of Biological and Biomedical Sciences*, 57, 13-23.

Descartes, R. (1664). *Le Monde.* In: S. Gaukroger (ed.). 1998. *The World and Other Writings.* New York: Cambridge University Press.

Dowe, P. (2000). *Physical Causation.* Cambridge: Cambridge University Press.

Dupre', J. (2012). *Processes of Life: Essays in the Philosophy of Biology*. Oxford University Press.

Feyerabend, P. (1975). *Against Method: Outline of an Anarchistic Theory of Knowledge*. New Left Books.

Finkelstein, G. (2013). *Emil du Bois-Reymond: Neuroscience, Self, and Society in Nineteenth-Century Germany.* The MIT Press.

Fodor, A. J. (1974). Special Sciences, or Disunity of Science as a Working Hypothesis. *Synthese*, 28(2), 97-115.

Franklin-Hall, L. (2016). New Mechanistic Explanation and the Need for Explanatory Constraints. In: K. Aizawa, and C. Gillet (eds.), *Scientific Composition and Metaphysical Ground*, Palgrave, 41-74.

Fredrick, K., and Ibba, M. (2009). Protein Synthesis: Errors Rectified in Retrospect. *Nature*, 457(7226), 157-158. https://doi.org/10.1038/457157a.

Friedman, M. (1974). Explanation and Scientific Understanding. *Journal of Philosophy*, 71(1), 5-19.

Gale, A. J. (2011). Current Understanding of Hemostasis. *Toxicol. Pathol.* 39 (1), 273-280. . https://doi.org/10.1177/0192623310389474.

Ganapathy-Kanniappan, S, and Geschwind, H. J.-F. (2013). Tumor Glycolysis as a Target for Cancer Therapy: Progress and Prospects. *Molecular Cancer*, 12, 152.

Garson, J. (2013). The Functional Sense of Mechanism. *Philosophy of Science*, 80(3), 317-333.

Garson, J. (2017). A Generalized Selected Effects Theory of Function. *Philosophy of Science*, 84(3), 523-543.

Garson, J. (2019). *What Biological Functions Are and Why They Matter*. Cambridge University Press.

Gasking, D. (1955). Causation and Recipes. *Mind*, 64(256), 479-487.

Gill, S. K., Fernandes, P., O' Donovan, T. R., McKenna, S. L., Doddakula, K. K., Power, D. G., Soden, D. M., Forde, P. F. (2016). Glycolysis Inhibition as a Cancer Treatment and its Role in an Anti-Tumour Immune Response. *Biochimica et Biophysica Acta*, 1866, 87-105.

Glennan, S. (1996). Mechanisms and the Nature of Causation. *Erkenntnis*, 44, 49-71.

Glennan, S. (2002). Rethinking Mechanistic Explanation. *Philosophy of Science*, 69 (S3), S342–53. https://doi.org/10.1086/341857.

Glennan, S. (2010). Ephemeral Mechanisms and Historical Explanation. *Erkenntnis*, 72(2), 251-266.

Glennan, S. (2011). Singular and General Causal Relations: A Mechanist Perspective. In: Phyllis Illari, Federica Russo and Jon Williamson (eds.), *Causality in the Sciences*, Oxford University Press, 749.

Glennan, S. (2017). *The New Mechanical Philosophy*. Oxford: Oxford University Press.

Godfrey-Smith, P. (2009). Abstraction, Idealizations, and Evolutionary Biology. In: A. Barberousse, M. Morange, and T. Pradeu (eds.), *Mapping the Future of Biology: Evolving Concepts and Theories*. Berlin: Springer.

Godfrey-Smith, P. (2014). *Philosophy of Biology*. Princeton: Princeton University Press.

Goodman, N. (1947). The Problem of Counterfactual Conditionals. *Journal of Philosophy*, 44(5), 113-128.

Goodman, N. (1955). *Fact, Fiction, and Forecast.* Cambridge: Harvard University Press.

Grüning, N.-M., and Ralser, M. (2021). Glycolysis: How a 300yr Long Research Journey that Started with the Desire to Improve Alcoholic Beverages Kept Revolutionizing Biochemistry. *Current Opinion in Systems Biology*, 28: 100380.

Havstad, J. (2011). Discussion: Problems for Natural Selection as a Mechanism. *Philosophy of Science*, 78 (3), 512-523.

Hempel, G. C. (1965). *Aspects of Scientific Explanation and other Essays in the Philosophy of Science*. New York: The Free Press.

Hempel, G. C., and Oppenheim, P. (1948). Studies in the Logic of Explanation. *Philosophy of Science*, 15(2), 135-175.

Hess, B., and Boiteux, A. (1968). Mechanism of Glycolytic Oscillation in Yeast. I. Aerobic and Anaerobic Growth Conditions for Obtaining Glycolytic Oscillation. *Hoppe-Seyler's Zeitschrift Physiologische Chemie*, 349(11), 1567-1574.

Hitchcock, R. C. (1995). Salmon on Explanatory Relevance. *Philosophy of Science*, 62, 304-320.

Hsu, P., and Sabatini, D. (2008). Cancer Cell Metabolism: Warburg and Beyond. *Cell*, 134(5), 703-707.

Hull, D. (1976). Informal Aspects of Theory Reduction. in R.S. Cohen and A. Michalos (eds.), *Proceedings of the 1974 Meeting of the Philosophy of Science Association*, Dordrecht: D. Reidel, 653–670. https://doi.org/10.1007/978-94-010-1449-6.

Illari, P., and Williamson, J. (2010). Function and Organization: Comparing the Mechanisms of Protein Synthesis and Natural Selection. *Studies in History and Philosophy of Biological and Biomedical Sciences*, 41, 279-291.

Illari, P., and Williamson, J. (2012). What Is a Mechanism? Thinking about Mechanisms Across the Sciences. *European Journal for Philosophy of Science*, 2(1), 119-135.

Illari, P., and Russo, F. (2014). *Causality: Philosophical Theory Meets Scientific Practice.* Oxford: Oxford University Press.

Ioannidis, S., and Psillos, S. (2017). In Defense of Methodological Mechanism: The Case of Apoptosis. Axiomathes, 27 (6): 601-619.

Jansson, L., and Saatsi, J. (2019). Explanatory Abstraction. *British Journal for the Philosophy of Science*, 70(3), 817-844.

Jones, M. (2005). Idealization and Abstraction: A Framework. In: M. R. Jones, and N. Cartwright (eds.), *Idealization XII: Correcting the Model. Idealization and Abstraction in the Sciences* (86, 173-217). Amsterdam: Rodopi.

Kauffman, A. S. (1970). Articulation of Parts Explanation in Biology and the Rational Search for Them. *PSA: Proceedings of the Biennial Meeting of the Philosophy of Science Association*, 257-272.

Kitcher, P. (1981). Explanatory Unification. *Philosophy of Science*, 48(4), 507-531.

Kitcher, P. (1984). 1953 and All That: A Tale of Two Sciences. *Philosophical Review*, 93: 335–373. https://doi.org/10.2307/2184541.

Kitcher, P. (1985). Two Approaches to Explanation. *Journal of Philosophy*, 82(11), 632.

Kitcher, P. (1989). Explanatory Unification and the Causal Structure of the World. In: P. Kitcher and W. Salmon (eds.), *Scientific Explanation.* Minneapolis: University of Minnesota.

Kripke, S. (1963). Semantical Considerations on Modal Logic. *Acta Philosophica Fennica*, 16, 83-94.

Kuhn, T. (1970). *The Structure of Scientific Revolutions*. Chicago: University of Chicago Press.

Leuridan, B. (2010). Can Mechanisms Really Replace Laws of Nature? *Philosophy of Science*, 77(3), 317-340.

Levy, A. (2013). Three Kinds of New Mechanism. *Biology and Philosophy*, 28 (1), 99-114. https://doi.org/10.1007/s10539-012-9337-z.

Levy, A. (2018). Idealization and Abstraction: Refining the Distinction. *Synthese* 198, 5855-5872.

Levy, A., and Bechtel, W. (2013). Abstraction and the Organization of Mechanisms. *Philosophy of Science*, 80(2), 241-261.

Lewis, D. (1970). General Semantics. *Synthese*, 22(1-2), 18-67.

Lewis, D. (1973a). *Counterfactuals*. Wiley-Blackwell.

Lewis, D. (1973b). Causation. *Journal of Philosophy*, 70, 556-567.

Lewis, D. (1986). Causal Explanation. In: David Lewis (ed.), *Philosophical Papers Vol. II*, Oxford University Press, 214-240.

Liu, B., Han, Y., and Qian, S.-B. (2013). Cotranslational Response to Proteotoxic Stress by Elongation Pausing of Ribosomes. *Molecular Cell*, 49(3), 453-463. https://doi.org/10.1016/j.molcel.2012.12.001.

Machamer, P., Darden, L., and Craver, C. F. (2000). Thinking about Mechanisms. *Philosophy of Science*, 67 (1), 1–25. https://doi.org/10.1086/392759.

Matthen, M., and Ariew, A. (2002). Two Ways of Thinking about Fitness and Natural Selection. *The Journal of Philosophy*, 99 (2): 55-83.

Matthewson, J., and Calcott, B. (2011). Mechanistic Models of Population-level Phenomena. *Biology and Philosophy*, 26 (5): 737-756.

Menzies, P, and Price, H. (1993). Causation as a Secondary Quality. *British Journal for the Philosophy of Scienc,e* 44(2), 187-203.

Menzies, P., and Beebee, H. (2020). Counterfactual Theories of Causation. *The Stanford Encyclopedia of Philosophy* (Winter 2020 Edition). https://plato.stanford.edu/archives/win2020/entries/causation-counterfactual/

Millikan, G. R. (1984). *Language, Thought, and Other Biological Categories.* Cambridge: MIT Press.

Millikan, G. R. (1989). In Defense of Proper Functions. *Philosophy of Science,* 56(2), 288-302.

Mitchell, D. S. (2000). Dimensions of Scientific Law. *Philosophy of Science,* 67(2), 242-265.

Moss, L. (2012). Is the Philosophy of Mechanism Philosophy Enough? *Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Sciences,* 43(1), 164-172. https://doi.org/10.1016/j.shpsc.2011.05.015.

Mossio, M., Saborido, C., and Moreno, A. (2009). An Organizational Account of Biological Functions. *British Journal for the Philosophy of Science,* 60(4), 813-841.

Nagel, E. (1961). *The Structure of Science: Problems in the Logic of Scientific Explanation*. New York: Harcourt, Brace and World.

Nathan, J. M. (2020). Causation vs. Causal Explanation: Which is More Fundamental? *Foundations of Science,* 28(1), 441-454.

Neander, K. (1983). *Abnormal Psychobiology*. Dissertation, La Trobe.

Neander, K. (1991). Functions as Selected Effects: The Conceptual Analyst's Defense. *Philosophy of Science* 58, 168-184.

Nicholson, J. D. (2012). The Concept of Mechanism in Biology. *Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Sciences*, 43(1), 152-163. https://doi.org/10.1016/j.shpsc.2011.05.014.

Nicholson, J. D., and Dupre, J. (eds.) (2018). *Everything Flows: Towards a Processual Philosophy of Biology*. Oxford University Press.

Okasha, S. (2002). *Philosophy of Science: A Very Short Introduction.* New York: Oxford University Press.

Okasha, S. (2019). *Philosophy of Biology: A Very Short Introduction*. Oxford: Oxford University Press.

Patra, C. K, Wang, Q., Bhaskar, P. T., Miller, L. Wang, Z., Wheaton, W., Chandel, N., Laakso, M., Muller, W., J., Allen, E. L., Jha, A. K., Smolen, G. A., Clasquin, M. F., Robey, B., and Hay, N. (2013). Hexokinase 2 is Required for Tumor Initiation and Maintenance and its Systemic Deletion is Therapeutic in Mouse Models of Cancer. *Cancer Cell*, 24(2), 213-228.

Perez-Gonzalez, S., and Luque, V. (2019). Evolutionary Causes as Mechanisms. *History and Philosophy of the Life Sciences*, 41: 13.

Plutynski, A. (2018). *Explaining Cancer: Finding Order in Disorder.* Oxford University Press.

Popa, T. (2017). Mechanisms. In: S. Glennan and P. Illari (eds.), *The Routledge Handbook of Mechanisms and Mechanical Philosophy.* London: Routledge.

Povich, M., and Craver, C. F. (2017). Mechanistic Levels, Reduction, and Emergence. In: S. Glennan and P. Illari (eds.), *The Routledge Handbook of Mechanisms and Mechanical Philosophy*. (2017), 185-97.

Prior, W. E. (1985). *Dispositions*. Atlantic Highlands. N. J.: Humanities Press.

Popper, K. (1959) (2002). *The Logic of Scientific Discovery*. Translation by the author of *Logik der Forschung* (1935), London: Hutchinson. Republished 2002, London and New York: Routledge Classics.

Potochnik, A. (2017). Idealization and the Aims of Science. The University of Chicago Press.

Psillos, S. (2002). *Causation and Explanation*. New York: Routledge.

Railton, P. (1978). A Deductive-Nomological Model of Probabilistic Explanation. *Philosophy of Science*, 45(2), 206-226.

Railton, P. (1980). Explaining Explanation: A Realistic Account of Scientific Explanation and Understanding. Dissertation, Princeton University.

Reichenbach, H. (1951). *The Rise of Scientific Philosophy.* University of California Press.

Reichenbach, H. (1958). *The Philosophy of Space and Time.* New York: Dover.

Rescher, N. (1996). Process Metaphysics. An Introduction to Process Philosophy. *Transaction of the Charles S. Peirce Society*, 32(4), 689-697.

Robins, S. K., and Craver, C. F. (2009). Biological Clocks: Explaining with Models of Mechanisms. In: J. Bickle (ed.), *The Oxford Handbook of Philosophy of Neuroscience*. Oxford University Press, 41-67.

Rosch, E. (1978). Principles of Categorization. In: Eleanor Rosch and Barbara Lloyd (eds.), *Cognition and Categorization*. Lawrence Elbaum Associates.

Ross, L. N. (2018). Causal Selection and the Pathway Concept. *Philosophy of Science,* 85(4), 551-572.

Ross, L. N. (2021). Causal Concepts in Biology: How Pathways Differ from Mechanisms and Why It Matters. *The British Journal for the Philosophy of Science*, 72(1), 131-158. https://doi.org/10.1093/bjps/axy078.

Ross, L. N. (forthcoming). Cascade Versus Mechanism: The Diversity of Causal Structure in Science. *The British Journal for the Philosophy of Science.*

Roux, S. (2017). From the Mechanical Philosophy to Early Modern Mechanisms. In: S. Glennan and P. Illari (eds.), *The Routledge Handbook of Mechanisms and Mechanical Philosophy.* London: Routledge.

Ruse, M. (2005). Darwinism and Mechanism: Metaphor in Science. *Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Sciences,* 36(2), 285-302.

Russell, B. (1948). *Human Knowledge.* New York: Simon and Schuster.

Salmon, C. W. (1984). *Scientific Explanation and the Causal Structure of the World.* Princeton: Princeton University Press.

Salmon, C. W. (1989). *Four Decades of Scientific Explanation.* Minneapolis: University of Minnesota Press.

Salmon, C. W. (1994). Causality without Counterfactuals. *Philosophy of Science*, 61(2), 297-312.

Salmon, C. W. (1998). *Causality and Explanation.* New York: Oxford University Press.

Sarkar, S. (1992). Models of Reduction and Categories of Reductionism. In: S. Sarkar (ed.). 2005. *Molecular Models of Life*. Cambridge: The MIT Press.

Schaffner, F. K. (1993). *Discovery and Explanation in Biology and Medicine*. Chicago: University of Chicago Press.

Schaffner, F. K. (2016). *Behaving: What's Genetic, What's Not, and Why Should We Care?* Oxford University Press.

Schickore, J. (2022). Scientific Discovery. *The Stanford Encyclopedia of Philosophy* (Winter 2022 Edition). https://plato.stanford.edu/archives/win2022/entries/scientific-discovery.

Sheredos, B., Burnston, D., Abrahamsen, A., and Bechtel, W. (2013). Why Do Biologists Use So Many Diagrams? *Philosophy of Science*, 80(5), 931-944.

Skipper, R., and Millstein, R. L. (2005). Thinking about Evolutionary Mechanisms: Natural Selection. *Studies in History and Philosophy of Science Part C Studies in History and Philosophy of Biological and Biomedical Sciences*, 36(2).

Smith, S. A., Travers, R. J., and Morrissey, J. H. (2015). How it All Starts: Initiation of the Clotting Cascade. *Crit. Rev. Biochem. Mol. Biol.*, 50(4), 326-336. https://doi.org/10.3109/10409238.2015.1050550.

Sober, E. (1984). *The Nature of Selection: Evolutionary Theory in Philosophical Focus*. Chicago: University of Chicago Press.

Sober, E. (1987). Explanation and Causation. *British Journal for the Philosophy of Science*, 38(2), 243-257.

Stegmann, U. E. (2005). Genetic Information as Instructional Content. *Philosophy of Science*, 72(3), 425-443.

Stephens, C. (2004). Selection, Drift and the 'Forces' of Evolution. *Philosophy of* Science, 550-570.

Stryer, L., Berg, J., and Tymoczko, J. (2012). *Biochemistry*. W.H. Freeman.

Šustar, P. (2007a). Neo-Functional Analysis: Phylogenetical Restrictions on Causal Role Functions. *Philosophy of Science*, 74(5), 601-615.

Šustar, P. (2007b). Crick's Notion of Genetic Information and the 'Central Dogma' of Molecular Biology. *Britihs Journal for the Philosophy of Science*, 58(1), 13-24.

Šustar, P., and Brzović, Z. (2014). The Function Debate: Between "Cheap Tricks" and Evolutionary Neutrality. *Synthese* 191(12), 2653-2671.

Tahmasebi, S., Sonenberg, N., Hershey, W. B., and Mathews, M. B. (2019). Protein Synthesis and Translational Control: A Historical Perspective. *Cold Spring Harbor Perspectives in Biology*, 11(9): a035584, https://doi.org/10.1101/cshperspect.a035584.

Taillefer, Marc, and Richard Sparling. (2016). Glycolysis as the Central Core of Fermentation. *Adv Biochem Eng Biotechnol.* 156: 55-77.

Thagard, P. (1988). *Computational Philosophy of Science*. MIT Press.

Thagard, P. (2003). Pathways to Biomedical Discovery. *Philosophy of Science*, 1-20.

Tilly, C. (2001). Mechanisms in Political Processes. *Annual Review of Political Science*, 4(1), 21-41. https://doi.org/10.1146/annurev.polisci.4.1.21.

van Riel, R., and Van Gulick, R. (2023). Scientific Reduction. *The Stanford Encyclopedia of Philosophy* (Winter 2023 Edition), https://plato.stanford.edu/archives/win2023/entries/scientific-reduction.

von Wright, H. G. (1971). *Explanation and Understanding.* Ithaca: Cornell University Press.

Walsh, D., Lewens, T., and Ariew, A. (2002). The Trials of Life: Natural Selection and Random Drift. *Philosophy of Science* 69: 452-73.

Wei T, Sui, H., Su, Y., Chen, W., Liu, Y., He, Z., Ji, Q., and Xu, C. (2020). Research Advances in Molecular Mechanisms Underlying the Pathogenesis of Cystic Fibrosis: From Tehcnical Improvement to Clinical Applications (Review). *Medicine Reports*. 4992-5002. https://doi.org/10.3892/mmr.2020.11607.

Williams, C. G. (1966). *Adaptation and Natural Selection: A Critique of Some Current Evolutionary Thought.* Princeton: Princeton University Press.

Wimsatt, C. W. (1972a). Complexity and Organization. *PSA: Proceedings of the Biennial Meeting of the Philosophy of Science Association*, 67-86.

Wimsatt, C. W. (1972b). Teleology and the Logical Structure of Function Statements. *Studies in History and Philosophy of Science*, 3, 1-80.

Wimsatt, C. W. (1976). Reductive Explanation: A Functional Account, in R.S. Cohen and A. Michalos (eds.), *Proceedings of the 1974 Meeting of the Philosophy of Science Association*, Dordrecht: D. Reidel, 671–710.

Wittgenstein, L. (1953). *Philosophical Investigations.* New York: Wiley-Blackwell.

Woodward, J. (2003). *Making Things Happen: A Theory of Causal Explanation*. Oxford: Oxford University Press.

Woodward, J. (2006). Sensitive and Insensitive Causation. *The Philosophical Review*, 115(1), 1-50.

Woodward, J. (2008). Explanation. In: M. Curd and S. Psillos, *Routledge Companion to Philosophy of Science*, London: Routledge. 199-209.

Woodward, J. (2010). Causation in Biology: Stability, Specificity, and the Choice of Levels of Explanation. *Biology and Philosophy*, 25(3), 287-318.

Woodward, J. (2016). Causation in Science. In: P. Humphreys, A. Chakravartty, M. Morrison and A. Woody, *Routledge Companion to Philosophy of Science*. Oxford: Oxford University Press.

Woodward, J. (2018). Some Varieties of Non-Causal. In: A. Reutlinger and J. Saatsi (eds.), *Explanation Beyond Causation: Philosophical Perspectives on Non-Causal Explanations*, Oxford University Press.

Wright, L. (1973). Functions. *Philosophical Review* 82(2), 139-168.

Ylikoski, P., and Kuorikoski, J. (2010). Dissecting Explanatory Power. *Philosophical Studies: An International Journal for Philosophy in the Analytic Tradition*, 148 (2): 201-219.

Xiao, H., Wang, J., Jan, W., Cui, Y., Chen, Z., Gao, X., Wen, X., Chen, J. (2018). GLUT1 Regulates Cell Glycolysis and Proliferation in Prostate Cancer. *The Prostate*, 78 (2), 86-94.

Xu, G., Ko, P. H., and Du, R. (2011). A Study on the Precision of Mechanical Watch Movement with Tourbillon. *Vibration* 330(16), 4019-4028.

Zhou, D., Duan, Z., Li, Z., Ge, F., Wei, R., and Kong, L. (2022). The Significance of Glycolysis in Tumor Progression and its Relationship with the Tumor Microenvironment. *Front. Pharmacol*., 13.

LIST OF FIGURES