RE reducing biology

by

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Dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the Department of Philosophy in the Graduate School of Duke University

2008
ABSTRACT

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Abstract

This dissertation proposes a new working model of reductionism for biology and a new concept of the gene based on the new reduction model. My project aims to help biologists and philosophers understand what reductionism in biology really is, or, should be.

Historical debates about reductionism testify us that the classical reduction model, *i.e.*, Ernest Nagel’s bridge-law model, offers us neither an appropriate ontological reductionism nor a reductive explanation about biological phenomena. Casting doubts on the received view of the layered hierarchical model of ontology, I suggest that many interesting biological properties be construed as second-order functional properties and their first-order realizers. Providing for reduction finely-analyzed biological properties, I offer a new model for reductionism in biology – localized functional reductionism – which evolved from Jaegwon Kim’s view of reductionism presented for the problems of mental causation.

My localized functional reductionism shows that a localized functional property is reduced to its base/structural property. I emphasize that researchers in biology do not deal with abstract general properties but always localized, structure-specific biological properties. A localized functional property and the structure-specific biological
property as its base property are what we are interested in and this is
what makes biological properties appropriate for research and
meaningful for philosophical discussion. The localized functional
reduction model, which is actually a case of token reduction model,
integrates the fine-grained ontological hierarchies of both macro/micro-
levels and higher/lower-orders, and it also synthesizes functional
reductionism and token identity thesis. In my localized functional
reductionism, functional biological properties are not eliminated but
they exist with their own causal powers and true explanatory powers.

I also argue that the gene, construed as a second-order functional
property, must be understood as gene expression network-specific. The
gene, when it is realized on a given occasion, is reduced to, and is
identical with, one of its genomic realizers on the given occasion, that
is, the gene expression network. A new dynamic approach to the
concept of the gene as the gene expression network vindicates
reductionism.
For my husband, Chang-Seong Hong,
and my daughters, Jaehyun and Dahyun
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Introduction: Reduction and Progress in Biology

Ever since the molecular structure of genes was discovered in 1953, almost all the sub-disciplines in biology have undergone radical changes and achieved great progress. Classical genetics and embryology, for instance, welcomed more sophisticated forms of research and improved their own research methods and thereby transformed themselves into molecular genetics and contemporary molecular developmental biology, respectively. Dramatic changes and rapid progress in all fields of biology have led biologists, especially molecular biologists, to believe that they can no longer be satisfied with rough, only approximately acceptable explanations of classical biology in their research. Molecular biology, which uses the concepts and methodologies of chemistry and physics along with functional terms, describes and explains biological phenomena with more clarity and precision than classical, functional biology. The idea and ideal of reductionism lie behind every research project of scientific researchers, whether they like it or not, whether they admit it or not. Should we
then not understand the success of biology in light of reductionist project in scientific practices?

No discussion in philosophy of biology has much value without considering and examining the actual scientific practices which working scientists have been carrying on in biology. The history of biology, which I am going to introduce here brings to light a series of events showing us that researchers and actual research projects have always demanded explanations that illuminate the nature of complex biological phenomena in light of their underlying micro-structural mechanisms. As embryology and classical genetics have advanced and turned into developmental biology and molecular biology, the biological facts that embryology and classical genetics have accounted for only partially produced a more comprehensive explanation at the level of cells, and, later, at the molecular level. As disciplines in biology progress, scientific researchers and their research projects are increasingly more committed to the reductionist research program of molecular biology.

**What Do Researchers Do in Biology?**

The aim of scientific research in biology is to discover the causes of biological phenomena. Biologists choose a model system, such as
mouse, fruit fly, or other animal systems, and seek explanations of why and how certain biological events occur in these model systems under certain conditions.

Consider the history of developmental biology. The biologists in the 19th Century were quite interested in embryonic development. They thought that embryogenesis was a very dynamic process made up of a series of events. This view naturally led them to look for causes of embryonic development. Many embryologists believed that the theory of vitalism explained embryonic development well, as the result of an inbuilt impulse in living organisms. By contrast, others supported the idea of recapitulation and advocated the view that life never emerges spontaneously, rather it always arises from earlier living forms and repeats various evolutionary stages during the embryonic period.

Although they had these two different theoretical views of embryonic development, all embryologists used the same methodology, that is, the morphological examination of embryonic forms at various stages of development.

As scientific technology advanced and Mendel’s laws were rediscovered, embryologists began to question the causal processes that underlie embryogenesis. They realized that it was imperative to shift their attention from comparative and descriptive morphology to the
behavior of chromosomes and cell division. In the late 19th Century August Weismann claimed that what causes embryonic development was inherited determinants, what he called germ plasm. He believed that germ plasm (or germ cells) is inherently different from body cells (or somatic cells) and has sole responsibility for inheritance.¹ Weismann’s ‘dualistic’ approach to heredity was contrasted with Hans Spemann’s experiment. Spemann held the idea that embryonic differentiation was not the result of the function of ‘germ plasm,’ but of epigenetic interactions between cells; in other words, embryonic development is not controlled by predetermined germ cells but orchestrated by the cell-cell interactions. These two mutually incompatible theories competed with each other, and the excitement of the competition was accelerated by reports of research results supporting one or the other. But, the competition was over when Spemann discovered the ‘organizer’: a small lump of cells in the early amphibian embryo with a unique property that organizes the formation of a complete, secondary embryo when transplanted in a host embryo. He found out that a small region of cells at the dorsal lip of the blastopore in a newt embryo caused the emergence of a complete embryo when transplanted in the ventral side.

of the same newt embryo. His experiment showed that cells could have the property of forming and organizing a complete embryo, according to the position of the cells and their interaction with other cells. He coined the term ‘organizer’ for this unique property of cells, and advocated a theory of embryonic induction by the organizer. His contemporaries and the scientific community accepted the notion of organizer, and believed that it answered the first question that the early 19th Century embryologists raised: what causes embryonic development and how it is regulated?

Following the discovery of the organizer, embryology transformed itself from ‘theoretical’ biology to ‘experimental’ biology. By the early 20th Century embryologists were ready to examine the nature of the organizer and its effects using a variety of different approaches and techniques. They designed experiments to find out whether organizers are chemically identifiable and whether they might function like hormones. Major progress in biochemistry and endocrinology enabled embryologists to describe embryonic development in detail and understand it better; however, embryologists failed to find the organizer or equate it with hormones.

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2 Joseph Needham claimed that the organizer was the “morphogenetic” hormone. See Needham, Joseph, 1959, A History of Embryology, Abelard-Schuman, New York.
On the other hand, the electron microscope, radioactive tracers, and many other advances in the technology of cell biology helped biologists examine and manipulate a single cell, rather than a whole organism. In 1962 John B. Gurdon conducted experiments using a single cell, a frog’s egg. He transplanted a nucleus from a gut cell into an enucleated frog’s egg, and showed that the frog’s egg with the nucleus from a gut cell successfully developed into a complete frog – this method would now be called ‘cloning’. Gurdon’s experiment demonstrated that each nucleus in a cell contained a complete set of the genetic information needed to mediate full development of a new frog. With this new discovery, biologists could come to finally explain what Weismann suggested a long time ago – that is, germ plasm (or germ cells) is solely responsible for inheritance. Rapid improvements of experimental methods and advancement in cell biology and molecular biology led biologists to develop a wide variety of interests, and, no doubt, such dynamic changes in biology affected embryology as well. In this period embryology acquired a new name, ‘developmental biology’.

An episode in the history of classical genetics provides an idea of what researchers actually do. Mendel’s laws were rediscovered in 1865

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and became the basis of classical genetics. Mendelian classical genetics was understood through the results of Mendel’s breeding experiments showing that each parent contributes one of the two ‘factors’ (anlagen) that segregate independently into sexual gametes. These gametes recombine in the next generation to form an individual. In 1905 William Bateson discovered that unlike the independent segregation of Mendel’s two ‘factors,’ certain traits appeared more frequently than expected and were passed down to the next generation together. This ‘linkage’ phenomenon – an exception to Mendel’s laws – was later explained by the theory of genetic crossover that Thomas Hunt Morgan proposed. Morgan’s ‘theoretical’ explanation of Bateson’s ‘linkage’ event was that if genes were aligned along the chromosome and if homologous chromosomes exchanged genetic materials in the process of gamete formation, then genes that were close to each other on the chromosome would have a better chance of passing on together to the same gamete than genes that were farther apart. Biologists were able to explain these mechanisms of inheritance with their understanding of the nature of chromosomes whose behavior was the key for describing the transmission of inheritance. The discovery of the molecular structure of the gene in 1953, however, reshaped dramatically the scientific researches in classical genetics and galvanized the formulation of a new
discipline in biology, that is, molecular biology. Biologists of the new era were no longer satisfied with rudimentary knowledge and partial explanations of inheritance and transmission. There was a ‘molecular and structural revolution’ in their approach to genetics. Researchers and their projects, which had been committed to the reductionist view ever since the time of Bateson and Morgan, vindicated the reductionist program of molecular biology with the discovery of the DNA structure.

The foregoing history of biology testifies that all scientists in biology, although their research has different model systems or different methodologies, attempt to describe in detail and explain more precisely multifaceted biological phenomena. Biologists persistently try to find causes of biological phenomena to provide their causal explanations more correctly. As the research in the disciplines of biology progresses and their methods and technology improve, scientists can better describe and explain biological processes at cellular or subcellular levels and, better still, at the level of molecules. More and more complicated biological phenomena are being unraveled as biologists commit their research projects to the reductionist research program of molecular biology.

Reducing biological phenomena is to explain them at the level of molecules, and, ultimately, at the level of chemistry/physics. However,
such reduction does not at all imply the elimination of higher-level
‘functional’ biology or the elimination of biology itself. Neither
molecular biology nor chemistry/physics eliminates biological
‘functional’ descriptions of phenomena. To the contrary, molecular
biology and chemistry/physics play a role in biology in such a way that
they incur no loss to our functional understanding of biological
phenomena.

The goal of my dissertation is to present a clear view of
reductionism and a new model of reduction that help both biologists and
philosophers of biology understand what reductionism in biology
really is, or should be. My view will also help differentiate reductionism
from eliminativism – a claim that the causal power/role and the

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4 Alexander Rosenberg’s coinage of ‘functional’ biology signifies non-molecular biology as the counterpart of ‘structural (molecular)’ biology. But, as he points out, the term ‘functional’ biology is not quite distinctive because molecular biology is functionally analyzed as well. See Rosenberg, Alex, 1997, “Can Physicalist Antireductionism Compute the Embryo?”, p. S360. Rosenberg later elaborates on the concept of ‘functional’ biology as the study of phenomena under their functional ‘kind’ descriptions. He says that what makes a kind functional is that its instances are the products of an evolutionary aetiology. Since natural selection operates at the macromolecular level, some of its kinds will be functional, too. Molecular biology is, on the other hand, the study of certain classes of organic macromolecules. The functional/molecular distinction is a convenient one that reflects widespread beliefs about a real division in the life sciences. See Rosenberg, Alex, 2002, “Philosophy of Molecular Biology” in Encyclopedia of Life Sciences, Macmillan Publisher Ltd, Nature Publishing Group.

5 As biologists read my dissertation, especially the last chapter, they will find that my new definition of the gene shows that their reductive research programs never provide a hint of, let alone a justification for the notorious and controversial claim, genetic determinism. This is one of many reasons why I believe a new clear view of reductionism may help biologists.
explanatory efficacy of higher-level functional biological properties are not real and thus are entirely absorbed into those properties of molecular biology or chemistry/physics. My model of reduction will illustrate that ‘functional’ biological properties are not eliminated but survive with their own and true causal/explanatory powers.

In the first chapter, I discuss a number of problems in the ‘standard’ account of reductionism that philosophers have developed and accepted. Given all those problems of reductionism in philosophy of biology, we need to reveal what has gone wrong with reductionism and the debates on its nature. I will first examine Kenneth Schaffer’s model of reduction in biology as an inter-theoretical relation – the relation of a theory to be reduced to its base reducing theory. I will argue that this model of reduction may capture the idea of scientific change in biology yet fail to explain why and how reductive research programs in science always appear in every branch of scientific research. I argue that reductionism should be a claim about explanations of biological phenomena, not an analysis of the relation between theories.

Next, I will explore the ontological framework of Schaffer’s view of reduction. I will point out the problems of the layered ontology that Ernest Nagel’s model of reduction and Schaffer’s Nagelian model of
reduction hinge on. The layered or hierarchical model of ontology classifies biological properties as higher-level macro-properties and lower-level micro-properties. This ontological scheme restricts our understanding of reduction to a purely mereological form where higher-level macro-biological entities are ‘decomposed’ into lower-level constituents of higher-level entities. Mereological relation is a synchronic relation. There is no temporal gap in between these two different levels of entities and their properties. Instead, higher-level macro-properties exist at the same time as their micro-level constituent properties arise. In other words, the mereological reduction model does not allow for temporal processes. The ‘decomposition’ relation here is understood as synchronic. So, explanations generated by the mereological reduction model are at best explanations of decomposition or explanations of synchronous biological facts. But I seriously doubt that synchronous reductive explanations are the only form of reductive explanations that working scientists in biology are always looking for. I suspect scientists and their research projects must demand not only synchronous explanations but also causal explanations about complex biological phenomena. Causal explanations are diachronic explanations in which the two different types of properties (or events) occur over a period of time, and this temporal gap between the two
types of properties (the cause and effect, to be precise) makes it possible that the causal relation be interfered and easily manipulated. I suggest that the layered ontological model and mereological reductionism are at times useful but may not be the only form of, not to speak of ‘the best’ form of, explanations that should be adopted in philosophy of biology.

I will reject the view that all biological properties are exhaustively characterized as either macro-properties or micro-properties. It seems that many biological properties cannot neatly fit in anywhere on the layered model of ontology. I believe that we need a new ontological scheme to understand and classify biological properties appropriately. This new ontology does not replace or eliminate the mereologically hierarchical model of ontology; instead, it promotes a fine-grained ontological view of biological properties that is compatible with the model of mereologically hierarchical ontology. The model of reduction I will propose is based on a more fine-grained ontology of biological properties and it will help us understand diachronic reductive causal explanations in biology.

To accomplish this goal, I introduce the first- and second-order property distinction that Jaegwon Kim proposes for his reductionism in philosophy of mind. I suggest that we construe most biological properties either as a second-order functional biological properties or as
one of the first-order properties in its realization bases. The first- and second-order property distinction in the new ontological model will help us characterize biological properties more clearly and in a more fine-grained way. The first- and second-order property dichotomy complements the mereologically hierarchical model of higher/lower-level properties, and thereby improves our understanding of biological properties as a whole.

In chapter 2 I will attempt to deal with the two major arguments against reductionism: the ‘gory detail’ argument and the argument from the thesis of multiple realizability. The gory detail argument that Philip Kitcher has proposed targets the claims of the explanatory primacy of molecular biology and of the reducibility of functional biological explanations to the explanations of molecular biology. I will argue that explanations of functional biology, which Kitcher believes are objectively better and completely adequate to explain higher-level biological phenomena, are in fact not reliable explanatorily. This is because changes in lower-level conditions often disrupt the higher-level biological patterns, and they consequently make explanations of higher-level regularities inadequate and unable to cope with anomalous patterns in the higher-level functional biology. Functional biology fails to generate reliable explanations on why and how such anomalies occur
at higher-levels. As the degree of explanatory reliability decreases in functional biology, the primacy of the level of biological explanations must shift from functional biology to molecular biology. I believe that this direction of the shift in explanatory primacy goes hand in hand with the direction of scientific progress.

I will then address the problem of multiple realizability of functional properties, arguably the most recalcitrant problem that undermines the plausibility of reductionism. I will examine and criticize each of the antireductionist arguments of David Hull and Philip Kitcher and claim that their arguments are wrongheaded. Both philosophers erroneously believe that reductionism is either the global model which aims to reduce all higher-level sciences to the lowest level physics all the time, or it is the type reduction model which assumes the ‘lawlikeness’ of the relations of properties in each of two theories and reduces higher-level property types to lower-level property types. I will show that neither the global reduction model nor the type reduction model will work in biology. If both the global and type reduction models are to be out of picture in the debates of reductionism in biology, the antireductionist arguments, which primarily target these two reduction models, should also be out of picture or at least recalibrate their targets.
In chapter 3 I will propose a new model of reduction in biology. My new model hinges on the localized functional reduction that fares well with both diachronic ontological reduction and explanatory reduction. Readers will see that the approach of functional biological properties as second-order functional properties plays a pivotal role in my discussion. I emphasize that researchers in biology do not deal with abstract properties but always localized, structure-specific biological properties. A localized functional property and the structure-specific biological property as its base property are what we are interested in and this is what makes biological properties appropriate and meaningful for our discussion. The localized functional reduction model, which is actually a case of token reduction model, integrates the more sophisticated and fine-grained ontological hierarchy of both macro/micro-levels and first/second-orders, and it also synthesizes functional reductionism and token identity thesis. I will also claim that the model of localized functional reduction satisfies ontological and epistemic requirements of reductionism and thereby protects reductionism from the antireductionist charge of eliminativism.

In the last chapter I aim to reduce the concept of gene with my model of localized functional reduction. We all agree that ‘the gene’ is the most basic and fundamental biological entity. For a reductionist,
however, reducing the gene at the genomic level is crucially important not only to complete her project of reductionism but also to contribute greatly to the success of reductionism in general. The reductionist must face three tasks to reduce the gene: (1) redefine the concept of gene as a second-order functional property, (2) find a genomic (nucleic acid) realizer of the gene to support the claim that a gene is a structure-specific functional property, and (3) locally reduce the functional property gene to its genomic realizers in terms of structure-specific identity. I will suggest that the gene, as a second-order functional property, must be understood as gene expression network-specific. The gene, when it is realized and instantiated on a given occasion, is reduced to, and is identical with, its realizer. This is constituted of a cluster of DNA sequences and their activities. The gene, a functional property, is reduced to one of its genomic realizers on a given occasion, that is, it is identical with the gene expression network on that occasion.
Chapter 1: Problems of Reductionism in Biology

My goal of this first chapter is to illuminate problems in the recent debates about reductionism and propose a new and improved ontological hierarchy of biological properties. I will first examine the model of reduction that Kenneth Schaffer offered for the philosophy of biology and argue that his theoretical and ontological model does not provide satisfactory causal explanations for biological phenomena. Secondly, I will challenge the hierarchy of ontology that has been widely accepted among philosophers of biology. I claim that this ontological model, sometimes called ‘the layered model of ontology’ or ‘the wedding cake model,’ is the very first origin that generates a misleading conception of reductionism in biology. The layered model presents ‘a picture’ that properties are understood in a hierarchical fashion where a macro-property on a higher level is in a mereological relation (i.e., the part-whole relation) with a micro-property at a lower level. This synchronic, mereological consideration has been the pivotal idea of this ontological model that has been providing the framework for
reductionism. However, it does not seem to represent all the varieties of relations that exist among biological properties. I will argue that, although this layered model has been at times useful, it is not the only available form of a comprehensive ontology for framing the debate about reductionism. I believe reductionism has been the victim of an incomplete and unsatisfactory ontology: it requires a more sophisticated and fine-grained ontology for more productive discussion. In place of the layered model of hierarchy, I will introduce into biology the ontological hierarchy that Jaegwon Kim proposes for his metaphysics and philosophy of mind, one that distinguishes between levels and orders. Provided with more finely analyzed and ordered biological properties, we will have the ontological hierarchy that we must adopt for more productive discussion in the reductionism of philosophy of biology.

1. **Problems of Intertheoretical Model of Reduction**

The debate about reductionism in biology started with the discussion of Ernest Nagel’s logical positivist model in the 1970s. According to Nagel, reduction takes place where a theory to be reduced is derived from a base theory that reduces it. To reduce a theory ($T_2$) to
its base theory ($T_1$), the laws of $T_2$ need to be deduced from the laws of $T_1$. To derive $T_2$’s laws from $T_1$’s laws, predicates ($P_s$) of the laws of $T_1$ should connect to predicates ($P_s$) of the laws of $T_2$ via ‘bridge laws.’ For reduction, bridge laws must exist between predicates of $T_1$ and $T_2$ such that $P_1 \leftrightarrow P_2$, that is, $P_1$ and $P_2$ should be coextensive. As we see here, Nagel’s reductionism requires derivability and connectability between predicates, and accordingly these two necessary conditions have been the main topics about which philosophers of biology have argued.

1.1 Schaffner’s Intertheoretical Reduction Model

Kenneth F. Schaffner was one of the first reductionists to introduce Nagel’s model in biology. Schaffner was aware that satisfying the conditions of derivability and connectability might not be as easy as Nagel pictures. Consider classical genetics and molecular genetics. To reduce classical genetics to molecular genetics, Nagelian reduction requires that laws of classical genetics be derivable from laws of molecular genetics, and predicates in classical genetics must connect to those in molecular genetics. However, primitive terms of Mendelian genetics may not be easily coextensive with predicates of molecular genetics. The term ‘gene’ in classical genetics, for example, was
understood as a factor that is responsible for a trait in Mendelian genetics. Yet the same term in molecular genetics is construed as various segments of DNA sequence. Due to the activities of regulatory genes, RNA splicing (i.e., the process of splicing out introns), and many other activities at the genomic level, the gene in molecular genetics fails to be identified with a single segment of DNA sequence. It becomes impossible to identify and designate a single and unique referent of ‘the gene.’ The term ‘gene’ is associated not only with a wide variety of segments of the DNA sequence but also sometimes with segments of RNA, such as, in the case of retrovirus. In other words, the term ‘gene’ in classical genetics connects to indefinitely many predicates of molecular genetics. Given this problem of multiple applicability of the term ‘gene,’ we realize that no uniform connection is possible between two predicates in Mendelian genetics and molecular genetics. If the connection of predicates is not successful, the bridge laws ‘P1 ↔ P2’ are also unavailable. Since the connection of, and bridge laws between, the predicates of two theories are unavailable, the requirement of derivability of a reduced theory from its reducing theory cannot be satisfied. And without bridge laws, Nagel’s model of reduction looks hopeless.
Although Schaffner was aware of the problems in Nagel’s model, he does not dismiss it as something totally inappropriate. To the contrary, he attempts to modify and improve Nagelian reductionism by adding one more condition to the model: the *correction* condition.

Schaffner’s strategy is that the theory to be reduced (T₂) undergoes the process of ‘correction,’ and the ‘corrected’ version of T₂ (i.e., T₂*) is to be reduced to T₁. All principal terms in T₂ are first ‘corrected’ and then the corrected terms come to connect to terms in T₁. Once this connection is secured, the bridge laws between predicates of two theories are possible and available, and the laws of the ‘corrected’ version of T₂* can be derived from the laws of T₁. Thus T₂* reduces to T₁.

The following is Schaffner’s model of reduction:

[SR] An adequate reduction of T₂* to T₁ occurs if and only if:

1. All primitive terms of T₂* are associated with one or more of the terms of T₁,
2. Given fulfillment of condition (1), T₂* should be derivable from T₁,
3. T₂* corrects T₂, i.e., T₂* makes more accurate predictions.
(4) T2 is explained by T1 in that T2 and T2* are strongly analogous, and T1 shows why T2 works.¹

Clearly [SR] includes Nagel’s two conditions in (1) and (2), connectability (or, in other words, the condition of referential identity) and derivability. Consider one of the primary terms, ‘gene,’ in classical Mendelian genetics. According to [SR], the concept of ‘gene’ in Mendelian genetics (T2) can be corrected, not drastically reanalyzed, enriched with new information, and connected with new classes of ‘gene’ (for example, regulatory DNA sequences) in molecular biology (T1). Like the ‘gene,’ all principal terms of classical genetics are corrected and associated with terms in molecular genetics. Then the laws of the corrected classical genetics (T2*) become derivable from the laws of molecular biology (T1), and the corrected classical genetics is reduced to molecular biology by satisfying the two conditions (1) and (2) in [SR]. So far [SR] is not much different from the examination of Nagel’s bridge principle model of reduction.

However, [SR]’s two other conditions (3) and (4) go beyond the requirements of Nagel’s reduction model. At first glance Schaffner seems to simply improve upon Nagel by adding the condition of

'correction' to his two conditions, connectability and derivability. But, in contrast with Nagel’s view of bridge law reduction that focuses on the derivability between two theories, Schaffner’s model attempts to bring to this reduction project something more than a simple logical feature involved in reduction. Take a look again at the two conditions, the condition of correction (3) and the condition of strong analogy (4):

\begin{align*}
[SR] \quad \text{An adequate reduction of } T_2^* \text{ to } T_1 \text{ occurs if and only if:} \\
(3) & \quad T_2^* \text{ corrects } T_2, \ i.e., \ T_2^* \text{ makes more accurate predictions.} \\
(4) & \quad T_2 \text{ is explained by } T_1 \text{ in that } T_2 \text{ and } T_2^* \text{ are strongly analogous, and } T_1 \text{ shows why } T_2 \text{ works.}
\end{align*}

Schaffner was aware that Nagel’s model never explains the nature of the relation between the two theories. He correctly sees the importance of the explanatory aspect of reduction and thereby modifies the model to have it reflect the explanatory relation. The condition (4) in [SR], the condition of strong analogy between T2 and T2*, is his attempt to improve Nagelian reduction along with the condition of correction of T2 to T2*. Prima facie these two conditions in [SR] look quite plausible and seem to effectively defend reductionism in biology.
However, I believe that Schaffner’s introduction of these new conditions also undermines his own accounts of reduction. He must justify the reason for the introduction of the correction and strong analogy conditions by explaining why he needs them for his thesis. To justify the conditions, however, is to explain that there is an independent reason for introducing the conditions to his thesis, that is, a reason other than it’s necessary to make his thesis work. Offering an independent reason is crucially important to make his proposal substantial and productive, not just *ad hoc*, but he offers no other reason. We can reasonably suspect that these problems undermine Schaffner’s ‘improved’ and corrected model of inter-theoretical reductionism. Does this mean that ontological reductionism should be given up once and for all?

An episode in the history of classical genetics will give us an idea of how we can read Schaffner rather differently. As I noted in “Introduction,” Mendelian classical genetics are composed of Mendel’s laws and his breeding experiments. The breeding experiments showed us that each parent supplies one of two ‘factors’ that segregate independently into sexual gametes, and that these gametes recombine in the next generation to form an individual. However, William Bateson discovered an anomaly in this pattern: unlike the independent
segregation of two ‘factors’, certain traits appeared more frequently than expected and were passed down to the next generation together. This ‘linkage’ phenomenon was seen as an exception to Mendel’s laws and led Mendelian classical geneticists to reconsider their theory. Not long after this discovery, Thomas Hunt Morgan proposed the genetic crossover theory and offered an explanation of this ‘linkage’ phenomenon. Morgan’s explanation is that if genes are aligned along the chromosome and if homologous chromosomes exchange genetic materials in the process of gamete formation, then genes that are close to each other on the chromosome would have a better chance of passing on together to the same gamete than genes that are further apart. It is very clear that Morgan’s explanation corrected and improved the original version of Mendel’s genetics, and thereafter Mendel’s genetics became a ‘corrected’ version of classical genetics. This ‘corrected’ classical genetics is the classical genetics that is relevant to the debate of reductionism. Given the discussion of his condition of correction, we can also see that Schaffner’s model of reduction well reflects scientific changes in biology and illuminates how reduction is possible when a theory can be corrected and improved. This point also implies that reduction will not take place if a scientific theory is incapable of, or resists, undergoing changes. Scientific development and changes in
biology are tightly intertwined with reductionism, and scientific change is in part explained by reduction. Reduction must be the mark of scientific development and changes.

Schaffner’s reductionism helps us explain how scientific theories change in biology. In spite of his contribution to the debates of reductionism, however, his model of reduction shares the same problem that Nagel’s has. Nagel’s bridge-law reduction does warrant the coextensiveness of predicates in the two theories, and further shows that a predicate of the theory to be reduced is ‘nothing over and above’ a predicate of the reducing theory. But this ‘nothing but’ locution does not give us an explanation of why these predicates are coextensive. Why, and how, is the ‘gene’ in classical genetics correlated with the ‘gene’ in molecular genetics? There is no explanation of this in Nagel’s model. The correlation between terms in the theories remains something like a brute and fundamental fact, but this is not the kind of explanation it is reasonable to accept. Just like Nagel’s reductionism, Schaffner’s reductionism, even with additional conditions, does not satisfy the requirement of the explanatory role of reduction. The Nagelian model is not explanatory, and Schaffer’s model does not provide reductive explanations either.
As I stated in “Introduction,” the objective of actual scientific research in biology is to explain the biological facts upon which researchers focus. So the value of scientific inquiries in biology must be dependent upon their ability to generate appropriate explanations about biological phenomena. If a model of reduction deserves to be taken seriously, it should be able to provide explanations of why and how reduction occurs in biology. Since the Nagelian model fails to provide explanations and does not work in biology, it is not useful for our debates about reductionism in biology. We need a new reduction model for more productive discussion.

2. Problems of Layered Model

2.1 Metaphysical Implications of the Layered Model

Ever since Paul Oppenheim and Hilary Putnam proposed the layered model of ontological hierarchy, it has served us as a ‘canonical’ view of nature. However problematic it might have been to some philosophers, no other account has replaced it as a standard analysis of

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ontological hierarchy. The layered model helps us view the ontology of reality as a single fixed hierarchical system in which every entity, property, and phenomenon of the world should have a unique designated place in nature. Each entity, property, or phenomenon belongs to its ‘proper’ level in a ladder-like, or wedding cake-like, ontological scheme. According to the layered model of ontology, the ordering of levels among the entities is asymmetrical: some entities (or properties) are classified as ‘higher’ or ‘lower’ than others in the layered ontology. If an object is completely decomposed into entities, the former is called a ‘higher-level’ macro-object whereas the latter the ‘lower-level’ micro-entities. A ‘higher-level’ object at level \( n \) must have a complete decomposition into proper parts all of which are ‘lower-level’ entities at the next lower level, the level \( n-1 \). Now we see that mereology is the key operator in this hierarchical ontological scheme. The higher-level macro-object and its lower-level micro-entities are in the part-whole relation (or mereological relation). Objects (and properties) are categorized in terms of their mereological relations, and each of them is placed at its own ontological level in the hierarchical ontology. The layered model of hierarchy thereby shows that all entities (and properties) are classified in such a neat and comprehensive single fixed system of levels.
Oppenheim and Putnam proposed the six-level hierarchy which comprehensively includes elementary particles through social groups. Elementary particles are assigned level 1 (the lowest level of the hierarchy), atoms level 2, molecules level 3, cells level 4, multicellular living things level 5, and social groups level 6 (the highest level of the hierarchy). Mereological considerations over all entities, properties, and phenomena are responsible for the construction of this six-level hierarchy. Each entity or property at level $i+1$, that is, one level above level $i$, must have a complete decomposition into entities at the next lower level, level $i$. For instance, a eukaryotic cell at level 4 must completely break down into molecules and nothing else. At level 3, every molecule must totally collapse into atoms, as a molecule of cholesterol, for example, reduces to carbon, hydrogen and oxygen. In their turn, atoms at level 2 must disintegrate into elementary particles and nothing else. The descending series of decomposition which Oppenheim and Putnam had in mind with their ontological scheme views social groups as breaking down completely into multiple living things, multiple living things to cells, cells to molecules, molecules to atoms, and atoms to particles.

This hierarchy, of course, is obsolete now, but we can identify several important issues behind Oppenheim and Putnam’s six-level
hierarchy. According to their ontological model, macro-objects at a higher level must completely collapse into lower-level micro-entities leaving no residues at all, and these lower-level micro-entities further break down into more micro-objects at the lowest level. The idea of downward series of complete decomposition implies that each level includes all higher levels, and, further, all higher-level entities are ultimately reduced to the bottom-level entities – social groups are reduced to multiple living things, multiple living things to cells, cells to molecules, molecules to atoms, and atoms to particles. This is global reduction, the kind that Oppenheim and Putnam intended to bring up from the layered model of ontology.

2.2 Problems of the Inter-Level Model in Biology

How would it be possible for each and every entity and property to be classified in a single fixed hierarchy of levels? The layered model is supposed to serve us as the overarching hierarchy of ontology for the whole natural world, but I wonder if there are some properties or phenomena that cannot have their own proper places in this hierarchy. A very neat and clean-cut series such as this layered model makes us suspect that it may look too good to be true and realistically useful.
Also, I believe that this kind of mereological consideration offers a misleading impression that a whole is always different from its parts – in other words, an impression that a whole is always more than its parts and that there is something in the whole that is ‘over and above’ its parts. These two problems – the incomprehensiveness and misleading impression of this layered model – deserve further discussion because this ontological hierarchy itself is the origin of the wrongheaded approach to reductionism in biology.

According to Oppenheim and Putnam’s layered model, there are three options in classifying any two given objects or properties: one object or property could rank (1) at a higher-level than the other, (2) at a lower-level than the other, or (3) both at the same level. As I have discussed above, a higher-level entity/property at one level above level decomposes completely into entities/properties and nothing else at the next lower level, level; (i.e., into ‘lower-level’ entities/properties). This descending breakdown process implies that a higher-level entity/property is reduced to next lower-level entities/property and nothing else. It may also imply that a ‘higher-level’ macro-entity/property can be reductively explained in terms of ‘lower-level’ micro-entities/properties.
Take a diamond, for example. According to the layered model of ontology, this object is decomposed completely into a set of carbon atoms because it is composed of nothing but carbon atoms. So, a piece of diamond must be a higher-level macro-entity while a set of carbon atoms is lower-level micro-entities. This interpretation may imply that a diamond is reduced to a set of carbon atoms, and also that the nature of diamond is reductively explained in terms of being a set of carbon atoms. Now, someone might say that graphite is also composed of nothing but carbon atoms. She might also add that graphite is reduced to a set of carbon atoms and its nature is reductively explained in terms of being a set of carbon atoms. It turns out that the way she explains how graphite is reduced to a set of carbon atoms is exactly the same as the way a diamond is reductively explained in terms of a set of carbon atoms. But, doesn’t a diamond need a different reductive explanation from graphite because it is not the same as graphite in its appearance and properties? Reductive explanations of these two objects need to be differentiated from each other in explaining why and how each of them is reduced to a set of carbon atoms. Can the mereological consideration, which Oppenheim and Putnam proposed in their layered model of ontology, find a way to distinguish a reductive explanation of a diamond from that of graphite? Mereology provides the idea that a
piece of diamond or a piece of graphite is constituted by individual carbon atoms. But such a mereological relation between a piece of diamond (or graphite) and carbon atoms does not itself provide a unique reductionist account of the properties of a diamond or graphite because reductionist accounts must specify how the two objects differ in terms of the bonding and arrangement of these atoms. Unless the mereological relation includes the bonding and arrangement of the atoms, the mereological account ends up lacking any explanation of the unique feature of a diamond or graphite. I believe that any plausible mereological reductionist claim must include accounts of the features of the bonding or arrangement of carbon atoms. It should also be formulated in such a way that a piece of diamond is reduced to so many carbon atoms, and the set of carbon atoms uniquely satisfies specific relations among themselves. The same requirements must be satisfied for the reductive explanation of a piece of graphite. Without the consideration of the relation among parts, the mereological relation cannot be satisfactorily understood, and also a whole cannot be reductively explained in terms of its parts.

An example in biology will show us what a mereological reductionist account should be. An adult Drosophila eye consists of an array of approximately 800 hexagonal ommatidia (or, facets), and each
ommatidium contains eight photoreceptor cells. An adult eye is tied, constitutively, to the compositional details of individual photoreceptor cells. Given this fact, how can the structure of an adult *Drosophila*'s eye be reductively explained? Could there be anyone who incorrectly understands a reductionist claim in such a way that an adult *Drosophila* eye is reduced to *a single* photoreceptor cell? Of course not: it makes no sense to claim that the structure of adult fruit fly’s eye is reduced to a single photoreceptor cell. What is hard to understand is not this kind of incorrect ‘reductionist’ claim but the way antireductionists view reductionism. Consider the comments of David Hull. He attacks reductionism by claiming, “There is little likelihood that all of these phenomena [color coat in mice, feather color in chickens, a certain type of deaf-autism in man and so on] are produced by *a single* molecular mechanism.” 3 This passage is evidence that Hull, along with many other antireductionists, misunderstands what a reductionist claim is about. He erroneously assumes that reductionists try to reduce a property of a macro-level entity to a property of *a single* micro-level entity. Having misunderstood reductionist claims this way, Hull thinks the improbability of reducing all complex phenomena to the

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properties of a single molecular mechanism is a powerful objection. Pace Hall, I must say that his criticism here is just striking a strawman. The reductionist claim that Hull attacks is in fact not a legitimate version of reductionism at all.

2.3 Inter-Level Mereological Reduction

When reductionists claim that an adult Drosophila eye as a macro-entity is reduced to its micro-entities, it should be understood such that the eye is reduced to many (6400) photoreceptor cells and the relations that define how these cells are arrayed. The reductive explanation of an adult Drosophila eye should proceed in a way that an adult eye is reduced to, or identical with, its compositional individual cells of the eye and the relations that define how the individual cells are arrayed.

William Wimsatt also recognizes this type of mereological reduction. He says:

More moderately, it could be taken as a claim that the basic properties and entities of one level are different from the basic properties and entities of another. As such, it is not inconsistent
with reducibility, since the higher-level entities and properties might be identical with configurations and relations of the lower-level entities and properties.\(^4\)

Hence, to generalize this point, when a property of a macro-level entity/property is claimed to be reduced to the properties of micro-level entities/properties, what the reductionist should really claim is that the macro-level entity’s property is reduced to (or explained in terms of) the properties of so many micro-level entities and the relations that connect these micro-level entities. In other words, when a property of a macro-level entity and its micro-level properties are in a mereological relation, \(i.e.,\) when a macro-level property is decomposed into properties of so many micro-level entities, a property of a macro-level entity is reduced to the properties of so many micro-level entities and their relations. This means the reduction of entities at different levels, \(i.e.,\) the inter-level reduction, should be understood in the way that macro-level entities are reduced to micro-level entities and the relations that connect micro-level entities:

[Inter-Level Mereological Reduction]

If an entity or property at level $i+1$ and its constituents at level $i$ are in a mereological relation, and the former is reduced to the latter, then the entity or property at level $i+1$ is reduced to its level-$i$ constituents and the relations among the constituents.

(See Figure 1: Levels)

3. **Introducing the Intra-Level model**

Are all biological properties exhaustively characterized as either macro-properties or micro-properties? I doubt that many biological properties neatly fit in anywhere on the layered model of ontology. Can two biological properties, *being a wing*, and *the structure of a wing*, for instance, be classified as a higher-level macro-property and a lower-level micro-property, respectively? It does not seem to be the case that the biological property, *wing*, can be completely decomposed into another property, *the structure of a wing*. They are not in the part-whole relation. These properties and their relation can be explicated by neither mereology nor the idea of higher/lower level distinction. I believe we must hereby reject the view that all biological properties are
exhaustively characterized as either higher-level macro-properties or lower-level micro-properties. Mereology, which is the crucial criterion of the layered model, does not represent the relations of all biological entities, properties, and phenomena.

Darden and Maull also recognized that mereology falls short in explaining the relations among biological properties. They laid out several candidates that can characterize relations among biological properties: the structure-function relation, the cause-effect relation, and the part-whole relation. They correctly cast doubt on the acceptability of the layered model of ontology, but they never proceeded further to a discussion of what went wrong in the layered model.\(^5\) We now have a good reason to reexamine not only the widely-discussed layered model of ontology for its acceptability, but also the nature of the mereological relation.

On the other hand, mereology and mereological reduction upon which the layered model of ontology hinges are understood as synchronic relations. As higher-level macro-biological entities are decomposed and reduced into their lower-level constituents, there is no temporal gap among the occurrences of higher-level macro-properties, their micro-level constituent properties, and the ‘processes’ of

reductions. We may see here that explanations generated by the mereological reduction model are at best explanations of decomposition or explanations of synchronic biological facts. However, I seriously doubt that synchronic reductive explanations should be the only form of reductive explanations that working scientists in biology are always looking for. I think working biologists’ research projects must aim for not only explanations on synchronic relations but also causal explanations about complex biological phenomena. Causal explanations are explanations on diachronic relations in which two different types of properties (or events) are instantiated over a period of time. This temporal gap between the instances of two types of properties (the cause and effect, to be precise) makes it possible that the causal relation be interfered with and easily manipulated. As long as we suspect that mereology does not appropriately classify all biological properties, we cannot be satisfied with either the layered model of ontology or the ‘accepted’ view that this hierarchy should be the overarching ontological framework for reductionism in philosophy of biology.

3.1 Ordering Properties at the Same Level in the Layered Hierarchy
To complement and improve the layered model of hierarchy, I propose a new concept of the ‘orders’ for biological properties. The idea of ‘ordering’ properties stems from the distinction between level and order that Jaegwon Kim employs in his metaphysics and philosophy of mind. He says:

I think we might usefully distinguish between ‘higher-level’ and ‘higher-order,’ or ‘levels’ and ‘orders,’ when speaking of properties in an ordering, using the ‘order’ idiom for first-order, second-order, third-order, ..., properties, and reserving the ‘level’ idiom for tracking the micro-macro hierarchy. That itself is merely a terminological proposal, but there is an important distinction to appreciate. As we saw, the [progression of ‘orders’] does not track the micro-macro ordering: these properties are all properties applying to entities at a single macro-micro level. In contrast, spin, charm, and such are properties of elementary particles, and they have no application to atoms, molecules, and higher objects in the micro-macro hierarchy; transparency and inflammability are properties of aggregates of molecules, and they have no place for atoms or more basic particles. Consciousness and intentionality are properties of biological organisms, or at least
their neural systems, and they have no application to entities that are micro in relation to them.\(^6\)

Kim’s distinction between \textit{level} and \textit{order} suggests that levels represent properties that fit in the mereological hierarchy, while orders catch up properties that cannot neatly fit in anywhere on the layered model of ontology, \textit{i.e.}, on the ‘level’ ontology. Consider again a wing and the structure of a wing. Being a wing and the structure of a wing are properties of biological organism. Can they be classified as a higher-level macro property and a lower-level micro property in organisms, respectively? I do not think that being a wing and wing’s structure are each a higher-level macro-property and a lower-level micro-property of entities on different levels. Rather, they are \textit{different characteristic properties of one and the same entity that of course belongs to the same level} in the layered model of ontology. In other words, they are \textit{intra-level} properties of a level in the layered model. We may further rephrase this view such that they are different \textit{orders of properties} at the same level in the ‘level’ ontology.

The ‘order’ ontology does not replace or eliminate the mereological model of ontological hierarchy. Instead, it promotes a fine-grained

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ontological view of biological properties that is compatible with the model of mereologically hierarchical ontology. If we decide to visualize the ‘level’ ontology as a ‘vertical’ form, then the model of ‘order’ ontology is a ‘horizontal’ form. (See Figure 2: Diagram of ‘Level’ and ‘Order’) With these two ontological hierarchies, the ‘vertical’ level ontology and the ‘horizontal’ order ontology, we can classify biological properties much more appropriately. In the nexus of vertical and horizontal ontological framework, like that of woof and warp, I expect to witness that biological properties will be better characterized and complex biological phenomena will be more precisely understood and explained. I hereby propose a new ontology that includes the ‘order’ hierarchy. The model of reduction I will propose in Chapter 3 will be based on this more fine-grained ontology of biological properties, and it will help us understand diachronic reductive causal explanations in biology.

3.2 Some Biological Properties as Second-Order Properties

I can show my new approach to biological properties with the definition of second-order property. Let D be a domain of first-order properties. We may define the second-order property as follows:
\(F\) is a second-order property over \(D = \text{def.} F\) is the property of having some property \(G\) in \(D\) such that \(S(G)\), where \(S\) is a specification on members of \(D\).

A second-order property is the property of having some property in the domain of its first-order properties specified in a specific way. For instance, the property of being a primary color (for painters) is a second-order property defined over the set of its first-order properties \{red, yellow, blue\}. A color is a primary one if it is one of the three colors. ‘Being a primary color’ is in this way ‘nothing over and above’ being one of the three colors. And the primary colors are specified such that you can mix them to create any other colors but you cannot get any of the three colors by mixing any other colors.\(^7\)

A second-order property is defined over the set of first-order properties. This means that a second-order property is existentially quantified over the set of first-order properties; that is, a second-order property is not the set of first-order properties. The relation between a second-order property and its first-order properties has nothing to do with mereology: it is not the case that a second-order property, say, the

\(^7\) I will show in Chapter 3 that the distinction of second-and first-order is not the determinable and determinate distinction.
property of being a primary color, can be decomposed into one of its first-order properties, say, the first-order property being red, which is at one level below the level of primary color. A second-order property and its first-order properties are different characteristic properties of one entity at the same level in the micro-macro layered model of ontology.

Take for example the biological property of metabolism. Metabolism is a process of chemical reactions that takes place in the cells of a living organism to maintain life, i.e., to grow, reproduce, maintain their structures, and respond to their environments. Metabolism is characterized by two processes, catabolism (so called destructive metabolism) and anabolism (so called constructive metabolism). Catabolism breaks down large and complex molecules in cells, mostly carbohydrates and fats, into more simple substances and releases energy. Taking up this energy, anabolism constructs components of cells including proteins and nucleic acids. In humans and other animals, anabolism enables the muscles to contract and the body to move. In plants, photosynthesis is a form of anabolism. Now we can see that metabolism, catabolism, and anabolism are three different characteristic properties of cells in an organism – these three properties are properties of an entity at one level of the micro-macro hierarchical ontology. Also, notice here that

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8 In plants, photosynthesis is a form of anabolism.
metabolism is ‘nothing over and above’ one of the two processes. The property of having metabolism can thus be understood as a second-order property defined over the set of its first-order properties \{catabolism, anabolism\}. Having metabolism is in this case ‘nothing over and above’ having one of the two properties.

Take a look at the two biological properties, muscle contraction and sliding of actin and myosin filaments. Muscle is composed of bundles of muscle fibers (or, muscle cells), and an individual muscle fiber is made up of myofibrils. Also, myofibrils are composed of two major protein filaments, actin thin filaments and myosin thick filaments. Actin and myosin filaments are organized into sacromere, the unit of muscle contraction, and interact with each other. The coordinated interaction produces contraction and elongation of millions of sacromeres in muscle. (See Figure 3: Diagrammatic Breakdown of a Typical Muscle) As actin filaments bind to the head of the myosin filament, the two filaments crosslink each other and the actin filament slides along the myosin filament. As crosslinking and sliding occur repetitively, the sarcomere shortens, and hence muscle contraction occurs. On the other hand, when the actin-myosin crosslink breaks, the actin filament slides down against the myosin filament. The sacromere consequently gets elongated and muscle becomes relaxed.
Now let us examine the relation between the two biological properties, *muscle contraction* and *sliding of actin and myosin filaments*. These properties are not in mereological relation. Instead, the property of *muscle contraction* may well be defined as a second-order property over the domain of its first-order properties. Its first-order properties are all the mechanical processes, binding myosin’s head with actin, sliding the actin along myosin, releasing the head of myosin from the actin, ....

The property *being a muscle contraction* is a second-order property defined (by being existentially quantified) over indefinitely many first-order properties, \{binding myosin’s head with actin, sliding the actin along myosin, releasing the head of myosin from the actin, ...\}. The properties *being a muscle contraction* and *sliding of actin and myosin filaments* are in a second- and first-order property relation.

### 3.3 Some Biological Properties as Functional Properties

Functional properties are abundant in biology. Many properties of biological systems, such as, heart, eye, wing, sex, etc., are considered functional properties. What makes them functional properties is, roughly put, that they are specified in terms of their causes and effects,
especially the latter.¹⁰ For example, some entities are males as long as they produce the male sex hormone, perform standard male mating behaviors, and so on.

Now let me introduce the definition of a functional property. Functional properties constitute a subset of second-order properties where the members of its first-order properties are specified in terms of their causal/nomic role:

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F \text{ is a functional property over } D = \text{def. } F \text{ is a second-order property over } D \text{ defined in terms of a specification } S \text{ that states causal/nomic relations involving members of } D.
\]

For something to have a second-order functional property \( F \) is for it to have some first-order property or other in a given domain that meets a certain causal specification definitive of \( F \).

¹⁰ ‘Functional property’ here is neutral between the non-historical, causal role account of function and the evolutionary selective effect account of function. I agree that, as Amundson and Lauder (1994) have pointed out, the property being heart, which ranges over both healthy and malformed organs must be construed in terms of the ‘etiological concept’ of function, that is, in term of the evolutionary selective effect (SE) account of function, rather than in terms of non-historical current causal role function alone. The causal role functional analysis of a deformed heart which cannot function as a blood-pump obviously cannot designate its function as blood-pumping. However, provided with the etiological concept of function, the deformed heart can be classified as a heart because even the organism with the malformed heart has a selective history of ancestors which survived because their hearts pumped blood. It is reasonable to embrace the causal role account of function in a history of natural selection.
Consider *being a heart*. Hearts in mammals, birds, and crocodiles contain two circulatory pumps for a total of four heart chambers. Amphibians and most reptiles have a heart that has three chambers in which oxygenated blood from the lungs and de-oxygenated blood from the respiring tissues enter by separate atria and are directed via a spiral valve. In contrast, hearts in many invertebrates, such as bivalves and anthropods, exhibit an open circulatory system where blood flows in vessels and freely in the body cavity. Given these various kinds of physical structures that realize ‘being a heart’ among different animals, we can see that the property of *being a heart* is defined (or existentially quantified) over the domain of its first-order properties \{being a muscular cone-shaped organ which has four chambers in mammals, being two chambers (one atrium and one ventricle) in fish, being three chambers in Amphibians and reptiles, being one chamber in many invertebrates\}. The property *being a heart* is a second-order property. It is also a functional property because it is specified in such a way that something is a heart as long as it performs a certain role in a blood circulatory system. Pumping the blood is a causal/nomic role of the first-order properties over which the second-order property *being a heart* is defined. So, *being a heart* is a second-order *functional* property.
Consider again the biological property *being a muscle contraction*. I discussed above that muscle contraction can be understood as a second-order property defined over the domain of its first-order properties, that is, mechanical interactions of the myosin and the actin filaments {binding myosin’s head with actin, sliding the actin along myosin, releasing the head of myosin from the actin, ...}. Since these first-order properties are specified in terms of their causal/nomic roles (movement of muscle), the property of *being a muscle contraction* is also a second-order functional property.

Consider another example in molecular biology, the property of *being a transcription factor*. Transcription factors are *trans*-regulatory proteins that bind to specific enhancer or promoter regions of DNA sequences to regulate the transcription of genes that are switched on and off by the enhancer or promoter. A specific DNA-binding site of a transcription factor is called a DNA-binding domain or *trans*-activating domain, and this DNA domain enables the transcription factor to interact with other proteins. This interaction causes the formation of the basal transcription complex that in turn binds RNA polymerase and eventually regulates the transcription of the genes. The structure of the DNA-binding domain in a transcription factor is the key for classifying transcription factors. Some transcription factors have a homeodomain
structure or the basic helix-loop-helix (bHLH) motif. Other transcription factors have basic leucine zipper (bZip) or zinc finger motif.\(^\text{10}\) (See Figure 4: Transcription Factors)

The property of being a transcription factor can be understood as a property of proteins whose role is binding to the enhancer or promoter regions of DNA sequence; this regulates the transcription of genes that correspond to the enhancer or promoter. Such proteins contain several kinds of different structural properties: being a homeodomain, being the basic helix-loop-helix motif, being a basic leucine zipper, or being a zinc finger motif. Now, how can we understand the relation between the property being a transcription factor and these structural properties? The relation cannot be understood mereologically: what does the part-whole relation have to do with the relation between being a transcription factor and the structural properties that realize the transcription factor such as being homeodomain, being the basic helix-loop-helix motif, being basic leucine zipper, or being zinc finger motif? Being a transcription factor is specified (or existentially quantified) over the set of its first-order properties |proteins having homeodomain on their DNA-binding sites, proteins having the basic helix-loop-helix motif on their DNA-

\(^{10}\) For the reference of transcription factors, see Scott F. Gilbert, 2006, Developmental Biology, 8\textsuperscript{th} ed., Sinauer Associates Inc.
binding domain, proteins having the basic leucine zipper, proteins having the zinc finger motif, ...}. Also, the property being a transcription factor has a casual/nomic role performed by its first-order properties: binding to the enhancer or promoter regions of the DNA sequence to regulate the transcription of genes that correspond with the enhancer or promoter. The property of transcription factor is a functional property that is a second-order property defined in terms of its causal/nomic relations involving members of its first-order properties. It is a second-order functional property.

In this chapter I have argued that the layered model of ontological hierarchy should not be accepted as the only available form of ontological framework for pursuing debates of reductionism in biology. Although the mereological consideration, which is the origin of the layered model, is sometimes useful, this kind of usefulness does not qualify the layered model as a neat, overarching ontological scheme for biological properties. This is because many biological properties and their relations cannot be exclusively classified as higher-level macro-property or lower-level micro-property. Introducing into biology the new hierarchy of higher- and lower-order properties, I argued that we need to make our ontological framework more fine-grained by integrating the
level and order distinction. This new and more sophisticated ontological scheme is precisely what is required for enlightened discussion of reductionism in biology.
Chapter 2: Antireductionist Arguments

The aim of this chapter is to deal with the two major arguments against reductionism: (1) Philip Kitcher’s ‘gory details’ argument claiming that functional biology has an explanatory primacy over the explanations of molecular biology, and (2) the argument from the multiple realizability of biological properties, perhaps the most recalcitrant problem that reductionists must face. I will discuss the reasons why these two arguments cannot successfully refute reductionism.

1. Arguments for the Explanatory Primacy of Functional Biology

1.1 Kitcher’s ‘Gory Details’ Argument

Philip Kitcher presents an extraordinary antireductionist argument against the explanatory primacy of molecular biology that denies the reducibility of functional biological explanations to the
explanations of molecular biology. He argues that higher-level biological disciplines perfectly and completely explain higher-level biological phenomena. None of the molecular details of the biological processes are explanatorily relevant. According to Kitcher, therefore, reductionism is refuted.

Consider meiosis, the process of two successive cell divisions that generates haploid germ cells in the sexual organs. In meiotic divisions, each chromosome undergoes precise joining and breakage, and the result is the formation of two recombinant chromatids of each chromosome in a cell. (See Figure 5: Stages of Meiosis) In the first meiotic division, homologous pairs of chromosomes, which carry two identical copies of allele, get paired, and the pairs then go through physical crossing-over, i.e. recombination. After recombination, homologous chromosomes separate from each other, and each chromosome goes into a different nucleus. As a consequence, half of the original chromosomes begin to have the second meiotic division. In this second meiotic division, the pair of sister chromatids of each chromosome separate from each other and each sister chromatid moves

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to different daughter cells. These haploid cells are germ cells in the sexual organs.

Given this information, the principal event in meiosis is the cytological process of chromosomal pairing and separation. This chromosomal movement determines how the genes are inherited. Let’s call the process of chromosomal pairing and separation PS-process, as Kitcher does. Kitcher argues that the PS-process fully explains why the genes on the same chromosome are likely to be transmitted together during meiosis, and why the genes at different chromosomes are transmitted independently. The transmission and recombination of genes as macro-level phenomena are completely explained by the PS-process in meiosis at the cytological level. No molecular details or information of the process is needed. He asserts that a cytological explanation that refers to endless ‘gory details’ of molecular information does not deepen our understanding of the gene transmission. Rather, the ‘gory details’ of how the gene transmission occurs at the molecular level would in fact decrease the explanatory power of cytological explanations. What is relevant to explain the gene transmission or gene recombination is not the molecular information of this phenomenon but an account of the PS-process of chromosomes. The PS-process completely explains the distribution of genes. Thus, cytology, a higher-
level biological discipline whose inquiry is about karyotype and chromosomes’s behavior, is the right level of discipline that explains the transmission of genes during meiosis; cytological explanations are objectively better, preferable, and completely adequate for explaining cytological phenomena.²

Kitcher does not deny that molecular biology has greatly contributed to classical genetics. Even so, his acknowledgement of all the successes of molecular biology does not lead him to believe that the explanatory powers of cytology depend on the molecular information – in other words, he does not believe that molecular explanations have explanatory powers. Why not? We can find the reason in his theory of explanatory unification. In “Explanatory Unification and the Causal Structure of the World,” Kitcher writes:

Science advances our understanding of nature by showing us how to derive descriptions of many phenomena, using the same pattern of derivation again and again, and in demonstrating this, it teaches us how to reduce the number of facts we have to accept as ultimate.³

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This passage illustrates the core of Kitcher’s theory of explanatory unification. A scientific explanation consists in providing a unified account of a range of different phenomena. The best explanation of different biological facts generates the largest quantity of knowledge with the smallest set of explanatory patterns, or the smallest number of independent *explanantia*, over and over again.

Kitcher evidently thinks that cytology explains gene transmission and recombination by using the PS-process repeatedly, generating our knowledge of this biological phenomenon. According to his theory of explanatory unification, a cytological explanation is the best explanation because it increases our understanding of the biological event with the smallest number of explanatory patterns, the PS-process. In contrast, he argues that molecular details could provide an expanded ‘set of explanation’ which “collectively provides the best systemization of our belief”\(^4\) but such ‘explanatory extension’ would *unnecessarily* enlarge our explanatory store of patterns. Molecular ‘gory’ details and explanations in molecular biology easily direct the explanatory unification astray. Hence, professes Kitcher, gorily detailed molecular information and explanations of molecular biology are irrelevant to generating a unified account of the gene transmission and

recombination: they do not contribute to producing the best explanation of gene transmission. He concludes that the cytological level constitutes an “autonomous level of biological explanation” and explanations of cytology have explanatory primacy over those in molecular biology.

However, Kitcher’s thesis does not aptly reflect the power and strength of molecular biology. We can see this by first examining the way Kitcher understands the nature of molecular biology. He treats molecular biology as a ‘structural’ biology that has only a limited ‘job description,’ say, identifying genes and finding the loci of the genes. His ‘molecular biology’ is a discipline that at most provides only detailed microstructural information on macromolecular biological phenomena, and such information is too ‘gory’ to be relevant in explaining those biological facts. If this should be unfortunately what Kitcher has in mind, it would naturally be difficult for him to see that molecular biology discovers mechanisms of gene expression or molecular pathways/processes that tell us rich stories of organisms’ development at the molecular level.

Consider sexual dimorphism in humans and mice. Usually a female has two X chromosomes (XX) and a male one X and one Y (XY).

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However, sometimes both male and female phenotypes can be found in one individual. An embryo with an XY karyotype, which thus should turn the embryo into a male, has a female phenotype. And an embryo with an XX karyotype has a male phenotype. Can cytology help us understand why sexual dimorphism occurs? More to the point, can the PS-process, the pairing and separation of the X and Y chromosomes, explain this biological phenomenon? I do not think that the chromosomal behavior that the cytological account provides can explain sexual dimorphism. Contra Kitcher, biologists have found that molecular ‘gory’ details are very important for explaining sexual dimorphism. Molecular biologists have reported that a certain gene plays the role of the master gene and sets off the process of sexual determination that converts an embryo into a male. The gene is SRY (Sex-determining region Y) located on the short arm of Y chromosome. Without this gene, an embryo retains femaleness as the ‘default’ program. So, despite the presence of the Y chromosome and the normal behavior of chromosomes, an embryo turns out to have a female phenotype when the embryo has a destructive mutation in the SRY gene. This molecular detail and its corresponding ‘molecular explanation’ help us explain adequately and successfully why sexual dimorphism occurs. This example clearly shows that Kitcher’s view of
‘molecular explanations’ fails to recognize the real explanatory power of molecular biology.

Also, scientific researchers testify that complex biological phenomena in embryonic development, for instance, are better explained in terms of ‘gory’ molecular details. The early development of embryonic *Drosophila*, for example, is nicely illustrated by well-coordinated cascade ways of expressing various genes: maternal-effect genes, gap genes, terminal genes, pair-rule genes, segment polarity genes, homeotic genes, and many others. Due to our knowledge of these genes’ activities and their interactions, the complex biological processes of early embryogenesis are more accurately explained than ever before. It must be accepted that explanations of molecular biology tell us a rich story about early embryonic development more adequately and comprehensively than ever.

I believe that Kitcher’s characterization of molecular biology erroneously generates an extreme and exaggerated chasm between functional biology and molecular biology. Consider his papers, “1953 and all that. A tale of two sciences” and “Hegemony of Molecular Biology”. He declares, “Molecular studies cannot cannibalize the rest of
Kitcher’s choice of the word, “cannibalization” is prejudicial: it insinuates not only a presumed battle for hegemony between two biological disciplines; it also expresses his anxiety about a unification scenario, that is, unification in ‘the reductive sense’ where classical genetics is completely reduced to molecular genetics. He also chooses the word ‘unification’ for his antireductionist thesis. Interestingly, this concept is just as essential for Kitcher’s own theory of explanation as it is for reductionism. Elliot Sober once commented on this, saying “it is ironic that ‘unification’ is now a buzz word for antireductionists.”

At any rate, keeping in mind Kitcher’s worries, let us proceed with a discussion of his argument on explanatory irrelevance. He claims that explanations of cytology have explanatory primacy over explanations in molecular biology. This is because explanations about meiosis at the level of molecules expand the ‘set of explanations’ which collectively provide the best systemization of our belief about meiosis to an unnecessary extent. Molecular ‘gory’ details can enhance our knowledge of the world as they explain more and more biological phenomena. As molecular details (the explanans) apply widely to other biological phenomena (the explananda), explanatory extension is

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obtained. However, Kitcher contends, these molecular details themselves cannot be unified with other basic beliefs and become a member of the set of basic explanatory patterns that explain a wide variety of phenomena over and over again. In fact the molecular details threaten to obscure the unification of the biological facts. Kitcher thus maintains that molecular biological explanations are irrelevant to explaining meiosis: they play no role in generating the unified account of this process. What is worse, the failure to be members of ‘the set of explanations’ about meiosis entails the striking conclusion that explanations of molecular biology are not merely irrelevant, they are non-explanatory.

Kitcher might respond here that cytological explanations are more unified than molecular explanations about meiosis. This encourages us to read him as making a claim about explanatory pluralism. A cytological explanation or a molecular explanation needs to be regarded as one of many alternative explanations, and in the case of meiosis cytology explains it better than molecular biology. If this pluralist picture of unification is what Kitcher actually has in mind, then a molecular explanation must be less explanatory incomplete here but not non-explanatory. However, if he had really advocated explanatory

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8 I am going to discuss this point in depth shortly.
pluralism, he would have bestowed some explanatory power on molecular biology and would have said that two kinds of explanations are both adequate and acceptable relative to given cases. Unfortunately, Kitcher unfairly denies the explanatory power and the explanatory relevance, especially, the causal relevance of molecular biology. He neither grants explanatory relevance to the molecular account nor appreciates the explanatory power of molecular biology. So, it seems impossible, in the first place, to expect him to endorse molecular biology in explaining higher-level biological phenomena. His rejection of the explanatory power of molecular biology is supposed to support his desire “I hope that I have said enough to make plausible the view that molecular studies cannot cannibalize the rest of biology.”9 He wishes that the hegemony of non-molecular biology should remain in classical genetics and functional biology, and that functional biology successfully resists being cannibalized by molecular biology.

1.2 The Problem of Explanatory Reliability

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9 Kitcher, 1984, ibid., p. 373.
Kitcher argues that cytological explanations appealing to the PS-process are the best explanations and that cytology has explanatory primacy over molecular biology in explaining meiosis and gene transmission. *If his account is right*, it must be the case that cytology, a higher-level biological discipline, explains completely why the gene transmission occurs, and how it happens and when it will happen. Cytology is the right-level of explanation, and these are the explanations we can and must rely on and refer to when we explain the gene transmission. Also, explanations of cytology should always give us correct and trustworthy stories with consistent powers of prediction. They are supposed to be consistently confirmed and we can expect them to perform the same job in the future with the same explanatory power. This means that cytology is supposed to provide complete, robust and projectible explanations of gene transmission all the time. Thus, if Kitcher’s argument is correct, cytology must explain, with robustness, reliability and projectibility, why and how some anomalies occur in meiosis, for example, polyploidy or aneuploid. He needs to hold that the PS-process (i.e., the activities of chromosome’s pairing and separation) must completely explain the occurrence of these anomalous phenomena. He must also show that the PS-process is a robust and irreducible property so it is never disrupted by any changes, say,
molecular changes. He also has to insist that a high-level explanation that appeals to the PS-process is the right-level explanation. Cytology provides reliable and projectible explanations of these anomalous phenomena regardless of any changes in ‘gory’ conditions at the molecular level.

However, recent scientific reports prove otherwise. Consider polyploidy or aneuploid as an anomaly in meiosis. If cytological explanations that invoke the PS-process are the best explanation, as Kitcher argues, they should explain completely why these anomalies occur by using the PS-process over and over again. Recent research shows that the PS-process is not a robust and irreducible property, so cytological explanations involving it cannot be robust or reliable in explaining these biological phenomena. Take a look at some ‘gory’ scientific data. Researchers have found Separase and Securin are key players in controlling the process of chromosomal paring and separation (PS-process).\(^\text{10}\) These meiosis-specific proteins, interacting with each other, regulate proper activities of the PS-process. First, Securin checks the cleaving activity of Separase and is degraded by Cdc20-dependent proteolysis when all chromosomes are attached to the

spindle. Due to Securine’s inactivity, Separase becomes free from Securin’s control and regains its own cleaving activity. Separase cleaves the meiotic-specific cohesion subunit Rec8 and the kinetochore protein SLK19. The dissociation causes homologous chromosomes to be separated. Given the role of Separase, we can easily predict that the mutation or deletion of the Separase gene will generate disruptions in the cleavage of the meiotic-specific subunits and consequently prevent homologous chromosomes from getting separated. Mutation or deletion of Separase, a meiosis-specific protein, produces disturbances in the PS-process. This disorder (to be precise, the failure of chromosomal segregation) results in polyploidy or aneuploid in the mutant Separase embryos.

Now, is the PS-process so robust that it can generate reliable and projectible cytological explanations of why polyploidy or aneuploid occurs and how they will happen? It is clear from the scientists’ report above that the higher-level PS-process is easily disrupted by changes in lower-level, ‘gory-detailed’ molecular conditions. So, the PS-process, which is regarded in Kitcher’s antireductionist thesis as an irreducible and robust higher-level phenomenon, turns out to be easily disturbed by lower-level molecular changes. As a result, the higher-level cytological explanations that involve the PS-process cannot explain why
and how PS-processes are interrupted, and why and how the abnormal higher-level phenomena, polyploidy and aneuploid, occur. Cytology is unable to offer higher-degree robust and projectible explanations about these higher-level biological phenomena, so we must conclude that cytological explanations lose explanatory power and can no longer remain complete and reliable. Contra Kitcher, it turns out that explanations drawn from ‘gory-detailed’ molecular biology tell us more causally complete stories about why and how the higher-level phenomena, polyploidy and aneuploid, occur. The higher-level phenomena are explained more thoroughly and more accurately at the level of molecules. Explanations at the molecular level have much stronger explanatory power and can make more precise predictions about the phenomenon.

1.3 The Problem of Explanatory Pluralism

Not only antireductionists but also some reductionists argue that it is wrong to think that only one type of explanation must be objectively superior among several different kinds of explanations, each of which is
already deemed as a correct explanation at each level.\footnote{Steel, 2004, “Can a reductionist be a pluralist?” Biology and Philosophy 19:55-73.} This explanatory pluralism presents the view that the multilevel accounts of a given entity/phenomenon are what science provides: explanations for the same *explanandum* always come in different levels of detail. An ideally complete explanation of a singular occurrence of an event must include the macro-story, the micro-story, and an account of how these two stories are connected. There is no objective *preference* among different levels of explanations even if some of them are more appropriate for given points of interest. The view of explanatory pluralists has an appearance of plausibility. In spite of its popularity and influences, however, I do not think that explanatory pluralism can ease the tension between reductionists and antireductionists; neither do I think that it can help us better understand the nature of biological phenomena.

Let’s first unpack the pluralist argument. According to the pluralistic view, different and multilevel explanations are equally acceptable as long as they explain a single world of the same reality. This means that different types of explanations, say, one in functional biology and one in molecular biology can both be equally acceptable alternatives. The gist of this point is that the biological realm is so
complex that we need many equally acceptable and compatible explanations to fully represent all the biological complexity. If one claims that the complexity of biological processes justifies explanatory pluralism, her view may involve two ideas. First, biological phenomena are so complicated that it is impossible to approach them with a single perspective. We need many perspectives and, accordingly, many different explanations. Second, a perspective is ‘subjective’ and relative to the subject’s interests. So, an explanation derived from a perspective may not give us any ‘objective’ understanding of the given phenomenon. Evaluating an explanation as adequate or correct seems to depend upon the subject’s viewpoint and interests. No objective preference is found among varied explanations. Every explanation is independent yet compatible with any other explanation and they are all equally acceptable. Thus, given these two ideas, we can see that multiple perspectives generate many independent yet equally acceptable explanations. Let me sum up this point as follows:

(EP)  Explanatory pluralism is composed of two ideas:

(1) Multiple perspectives are required for examining the complex biological processes.
(2) A variety of perspectives generate multiple explanations. These explanations are independent but compatible with each other, and they are equally acceptable as explanations for the same phenomenon.

Elliott Sober also advocates explanatory pluralism in responding to Hilary Putnam’s ‘square peg-round hole’ argument. In a way reminiscent of Kitcher, Putnam argues that we do not need to know the ‘gory’ molecular details to explain adequately why a particular spare peg goes through one hole but not the other in a board. The complete and correct explanation is provided by considerations from geometry, that is, with the macro-properties of the peg and the holes. Putnam denies that the micro-properties of molecules (or atoms or particles) in the peg and board explain this fact. He argues that the micro-description is long and complicated, and it brings in a welter of irrelevant details. To explain why the peg goes through one hole but not the other, as long as the peg and board have the macro-properties it does not matter what micro-properties the molecules have. Putnam concludes that to explain the event a geometrical explanation is objectively better than a physical

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explanation. In response to Putnam’s argument of the explanatory relevance of higher-level properties, Sober asks:

What makes a more general (more invariant) explanation objectively better than one that is less? Putnam’s answer is that “one of the things we do in science is to look for laws. Explanation is superior not just subjectively, but methodologically, in terms of facilitating the aims of scientific inquiry, if it brings out relevant laws.” (1975:301) My reply is that the goal of finding “relevant” laws cuts both ways. Macro-generalizations may be laws, but there also may be laws that relate micro-realizations to each other, and laws that relate micro- to macro-as well. ... There is no objective rule concerning which is better.¹³

Sober argues that higher-level sciences can describe patterns invisible at lower levels and might offer more ‘general’ explanations, but physics provides ‘deeper’ ones. Physics might not be able to describe all the

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patterns, but it can nevertheless explain any singular occurrence that a higher-level science can explain. Sober says:

Explanations come with different levels of detail.... There is no objective rule concerning which is better. The claim that the preference for breadth over depth is a matter of taste... In claiming that it is a matter of taste whether we prefer the macro- or the micro-explanation, I am claiming that there is no objective reason to prefer the unified over the disunified explanation.\textsuperscript{14}

Sober’s point is that it is wrong to claim that a broader higher-level explanation is objectively better and more adequate than a gory-detailed lower-level explanation. He rejects Putnam’s argument (and Kitcher’s) by saying “Monolithic theories about simplicity and parsimony – which claim that these considerations are never evidential or that they are never merely pragmatic – should be replaced by a more pluralistic approach.”\textsuperscript{15} There is no objective rule to offer for preferring higher-level explanations to lower-level ones. It is only a matter of our subjective interests. I will incorporate Sober’s explanatory pluralist

\textsuperscript{15} Malcolm Forster and Elliott Sober, 1994, “How to Tell When Simpler, More Unified, or Less Ad Hoc Theories Will Provide More Accurate Predictions” \textit{The British Journal of the Philosophy of Science} 45 (1) 1-35. This quote is from p. 27.
attitude into the formulation of explanatory pluralism (EP) introduced above:

(EP') Explanatory pluralism is composed of three ideas:

(1) Multiple perspectives are required for examining the complex biological processes.

(2) A variety of perspectives generate multiple explanations. These explanations are independent but compatible with each other, and they are equally acceptable as explanations for the same phenomenon.

(3) The preference for one explanation over the other is a matter of our interest.

The account of explanatory pluralism appears to be convincing. However, I do not think that explanatory pluralism helps us better understand the nature of biological phenomena. As we see in EP’ above, explanatory pluralists try to justify their view by appeal to the fact that biological phenomena are complicated and complex. The complexity of phenomena would be preserved when and only when it is examined with multiple perspectives and accordingly explained by
multiple explanations. A single explanation can never come close to explaining a multifaceted biological process; it can never be successful in explaining why the biological event occurs. This means that a single explanation cannot be a sufficient and complete causal explanation of the event. Many different explanations need to jointly contribute to representing a complex phenomenon fully – to save the phenomenon. This point may imply the view that a single explanation is, at best, a partial explanation. But in that case, how could many ‘partial’ explanations that are all independent from one another each be equally acceptable?

To dodge this problem, explanatory pluralists may claim that each of these multiple explanations is not a partial explanation but a full-fledged version of a complete explanation which refers to a sufficient cause of the given biological phenomenon. This move may also sound plausible, but explanatory pluralism cannot avoid difficulties even with this maneuver. Explanatory pluralism promotes the idea that multilevel explanations are compatible and equally acceptable as long as each of them satisfactorily explains a single world of the same reality. Suppose two independent but compatible causal explanations at different levels are equally acceptable for the same phenomena. This view implies that the same event is explained by two
different causal explanations. In other words, two causal explanations of the same event refer to two different causes. This conclusion further suggests that either one of these two causes would have produced the very event just the same even without the occurrence of the other cause. If we generalize this problem situation further, we will see here that explanatory pluralism in fact promotes a view that a single event has multiple disjunctive causes. This view implies that any single biological phenomenon is always caused by multiple causes each of which is a sufficient cause of the phenomenon. In this interpretation of explanatory pluralism, any biological phenomenon is causally over-determined and is ‘over-explained’ by numerous independent, equally acceptable causal explanations. Is this kind of view acceptable?

Consider the causes of Parkinson’s disease. There are several causes suggested so far, and accordingly, several different explanations of the malady are available. Some say that Parkinson’s disease is caused by the progressive impairment or deterioration of neurons in a certain area of the brain known as the substantia nigra. Others say that Parkinson’s disease is the result of the lack of the chemical Dopamine in the brain. Dopamine serves as a chemical messenger, or neurotransmitter, passing messages within the brain and from the brain to the muscles. A lack of dopamine results in abnormal nerve
functioning, causing a loss in the ability to control body movements. So now we have two explanations for the same phenomenon:

1. Parkinson’s disease is caused by the progressive impairment or deterioration of neurons in the *substantia nigra* of the brain.
2. Parkinson’s disease is caused by the lack of Dopamine in the brain.

The two explanations are at different levels – (1) is a functional biological explanation and (2) is a molecular biological explanation. Both explanations appear to be correct. According to explanatory pluralists, we need to understand that (1) and (2) are independent of each other yet mutually consistent and equally acceptable. It is only a matter of taste to prefer one to the other.

The two explanations (1) and (2) suggest that the occurrence of Parkinson’s disease is determined by *either* the impairment of neurons in the *substantia nigra* of the brain, *or* the lack of Dopamine in the brain. Each of the two causes equally, and independently, produces the same phenomenon. So, in this case an event is determined by two sufficient yet disjunctive causes. In other words, Parkinson’s disease should occur due to one of the two causes even if the other cause does
not take place. So, the disease should be in principle detectable even when either the impairment of neurons or the lack of Dopamine in the brain does not take place. However, research data have indicated Parkinson’s disease does not occur if one of the two ‘independent’ causes is absent. Scientists discovered that Dopamine is produced in the cells of the *substantia nigra* in the brain, and the damage in this area results in a lack of Dopamine. So, Parkinson’s disease does not occur unless the two causes are both present. This discovery teaches us that the two causes are not really independent. The occurrence of Parkinson’s disease is not equally determined by each of the two causes. Contrary of the pluralist view, it does not look like this biological phenomenon has more than one sufficient cause, which means Parkinson’s disease is not causally over-determined. Since the two different causes are not independent of each other, explanations (1) and (2) above that appeal to the two related causes are not independent, either. I believe that many other claims of causal over-determination in biology are spurious as well. The same line of argument can be applied to those cases.¹⁶

¹⁶ On the other hand, if there really were cases of causal over-determination in biological phenomena, I believe, the effects of these independent causes may not be of the same kind of effects but different sorts of more than one kind of effects, which will make the claim of causal over-determination a nonstarter. Unfortunately, I cannot here discuss this issue more.
I also argue that two different explanations at different levels cannot be equally acceptable. If explanations are equally acceptable, they should be equally reliable and robust in the sense that we can always rely on and refer to them when we explain a biological phenomenon. However, as I discussed in 1.2 above, a higher-level explanation, for example a cytological explanation, is not robust or reliable. A higher-level explanation is easily disrupted when the lower-level conditions change. Recall the higher-level property PS-process that we also discussed in 1.2, and the other examples polyploidy and aneuploid. Changes in molecules at the lower level interfere with the PS-process. Cytological explanations that invoke the PS-process cannot explain why and how the abnormal higher-level phenomena, polyploidy and aneuploid, occur. It seems that Kitcher also notices this kind of fragility in higher-level causes and explanations. He says: “There can be interference with normal cytological processes so that segregation of nonhomologous chromosomes need not be independent.”

Higher-level explanations are not robust but in fact vulnerable to breakdowns due to interference from below. Given this fragility, how can we then believe that both higher-level and lower-level explanations are equally acceptable? Although explanatory pluralism has an apparent

plausibility, after a closer examination, we come to realize that it is hard to find an acceptable reason for promoting the pluralist view.

Now, recall the explanatory pluralism (EP)

**(EP)** Explanatory pluralism is composed of the following three elements:

1. Multiple perspectives are required for examining the complex biological processes.
2. A variety of perspectives generate multiple explanations. These explanations are independent but compatible with each other, and they are equally acceptable as explanations for the same phenomenon.
3. The preference for one explanation over the other is a matter of our interest.

As I have discussed above, (2) does not look convincing anymore. There do not seem to be multiple explanations that are all compatible and equally acceptable for the same biological phenomenon. This leaves explanatory pluralists with (1) and (3). The trouble is now that these two ideas cannot play a justificatory role for the explanatory pluralism. What (1) and (3) jointly lead us to is not explanatory pluralism but
perspectivalism. Each one’s perspective is subjective, so there is no need to quarrel about whose is superior or correct. There is no objective preference. It is just a matter of taste. Different perspectives leave us with little to explain and discuss. Various perspectives themselves do not bring us any further or deeper causal explanations of biological phenomena. Explanatory pluralists believe that their pluralist account provides us with a rich presentation of the world, but as I see it, it actually suffers from explanatory poverty.

2. The Multiple Realizability of Biological Properties

David Hull was the first to cast doubt on the applicability of the logical positivist reductionism to biology. He held that the logical positivist model of reduction is only a ‘rational’ construction whose analysis does not tell us much about the true situation in genetics. He showed that the ‘corrected’ Mendelian genetics in Schaffner’s model of reduction cannot be deduced from molecular genetics. A term/predicate of Mendelian genetics, say, ‘gene’, which is supposed to be connected to ‘molecular gene’ in molecular genetics, is associated with various kinds of terms/predicates in molecular genetics. Also, in molecular genetics the term/predicate ‘gene’ is correlated with many
terms/predicates in Mendelian genetics. A one-to-one connection is not available between predicates of the two theories; instead it is a many-to-many relation. The true situation in genetics tells us that the one-to-one relation is impossible; this implies that it is impossible to establish bridge laws between the two theories. It is then out of the question to derive a higher-level theory from its lower-level theory. The inter-theoretical reduction from Mendelian genetics to molecular genetics is thus unattainable. These considerations completely undermine the Nagelian inter-theoretical reduction.

Philip Kitcher agreed with Hull’s argument of ‘many-to-many relations’ and developed it into an example of the classic multiple realization argument. The multiple realizability thesis was originally introduced in Hilary Putnam’s article “Psychological Predicates.”\footnote{Putnam, 1967, “The Nature of Mental States” in Rosenthal, David, 1991, The Nature of Mind, Oxford University Press.} It is the claim that the physical realization bases of a mental property are ‘wildly heterogeneous’ across varied species. \textit{Pain}, for instance, is a functional mental property and it has extremely heterogeneous realizers. Due to this phenomenon of the multiple realizability of mental property, the possibility of identifying a mental property \textit{type} with any physical property \textit{type} is completely blocked. Accordingly, the
type reductionism in philosophy of mind was quite decisively undermined. Ever since, the multiple realizability argument has been the most recalcitrant problem that reductionism needs to meet.

As Kitcher sees it, the reductionist position on genetics requires us to accept three fundamental theses:

R1: Classical genetics contains general laws about the transmission of genes which can serve as the conclusions of reductive derivations.

R2: The distinctive vocabulary (predicates) of classical genetics can be linked to the vocabulary of molecular biology by bridge principles.

R3: A derivation of general principles about the transmission of genes from principles of molecular biology would explain why the laws of gene transmission hold.\(^\text{19}\)

Kitcher argues that reduction fails because all three of these fundamental theses are false: (1) there are no laws in classical genetics, (2) there are no bridge principles that connect predicates in classical genetics to predicates in molecular genetics, and (3) non-molecular

biological explanations are completely adequate and autonomous from molecular biological explanation.

Kitcher assumes that the Nagelian model of reduction is the only model of reduction, and he argues that the reduction of one theory to another theory is effected by using bridge laws to derive the laws of the former from those of the latter. Kitcher first tried to tackle the bridge principle:

BP: \( x \) is a gene if and only if \( Mx \) (\( M = \) a predicate from molecular genetics.)

As Hull did in his argument for ‘many-to-many relations,’ Kitcher also shows that the predicate of classical genetics, ‘is a gene,’ cannot be coextensive with \( M \) because \( M \) is an unsystematic, heterogeneous set of disjunctive predicates. Molecular biologists found that the presence of ‘regulatory’ gene sequences - ‘structural’ gene, introns and exons, and many others - hinder the predicate of classical genetics, ‘is a gene,’ from connecting to a single predicate of molecular genetics, say, ‘is a DNA sequence.’ The predicate, ‘is a gene’ is linked to various disjunctive predicates of segments of DNA (or, a segment of RNA in the case of retrovirus) in molecular genetics. \( Mx \) in the sentence BP becomes an
indefinitely long, disjunctive sentence. So it is impossible to construct any sentence of the form BP. Bridge principles are unattainable, and so is reduction.

Kitcher also contends that there are no laws of classical genetics and thus there is nothing to reduce. Consider Mendel’s second ‘law’ – the probabilities of a gamete receiving any of the possible genetic combinations are equal. Kitcher points out that this ‘law’ does not hold in general. There are exceptions to this ‘law’ such as the linkage phenomenon I discussed in Introduction. He thus goes on to argue, plausibly, that various possible restrictions or emendations of Mendel’s second law will not make it completely precise and exceptionless. So there are no laws in classical genetics; and there are no bridge principles that connect predicates in classical genetics and molecular genetics. Reduction is impossible.

Let me first examine the concept of reduction as Hull and Kitcher understand it. They construe it as *type reduction* or *global reduction* and criticize it. Type reduction assumes ‘laws’ governing the relations of properties in each of two theories and reduces higher-level property types to lower-level property types. Global reductionism, on the other hand, aims to reduce all higher-level sciences to the lowest level physics. Rosenberg interprets Hull and other antireductionists (and
many reductionists as well) as understanding reduction to be type reduction, which is about the relation between ‘natural kinds’ or property types of two theories. As is well known, however, it is impossible to establish the one-to-one connection between higher-level property types and lower-level property types. Hull’s argument of ‘many-to-many relations’ is deemed quite plausible. Type reductionism is refuted, and the logical positivist model (i.e. Nagelian reduction) is out of the picture. Even so, I do not think that the reductionism that we talk about and debate over in philosophy of biology is the kind that Hull and Kitcher understood and criticized. Sober also challenges the antireductionist argument of multiple realizability, which primarily targets the global model:

The third premise in the multiple realizability argument also has come in for criticism. Perhaps pain is multiply realizable, but human pain may not be. And if human pain is multiply realizable, then some even more circumscribed type of pain will not be. What gets reduced is not pain in general, but specific physical types of pain (Nagel 1965). The multiple realizability

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argument is said to err when it assumes that reductionism requires *global* reduction; local reduction is all that reductionism demands. To this objection, a defender of the multiple realizability argument might reply that there are many questions about reduction, not just one. If human pain gets reduced to a neurophysiological state, but pain in general does not, then reductionism is a correct claim about the former, but not about the latter. If psychology provides explanations in which pain – and not just *human* pain – is an *explanans*, then reductionism fails as a claim about *all* of psychology.\(^{21}\)

According to the foregoing passage, the multiple realizability argument undermines global reduction. Nonetheless, suggests Sober, global reduction is not something that reductionism must always demand. Agreeing with Nagel, he emphasizes that what gets reduced is not *properties in general* but *specific physical types of properties*. He takes into account the consideration of empirical *regularities* that are preserved *locally*, say, species- or structure-specifically. A local, structure-specific property is realized only in one property type among

the multiply disjunctive and heterogenous realizers. The local, structure-specific property is reduced to its base, so reduction is possible. The model of reduction here is a local type reduction. Local type reduction is what Sober offers to vindicate reductionism from the antireductionist argument of multiple realizability.

I subscribe to the first half of Sober’s point. In actual biological research we do not deal with abstract properties; instead, the targets of biological research are always localized, structure-specific biological properties. For instance, when scientists research a heart, they do not handle an abstract, general property of being a heart; rather they choose a certain model system and approach a heart as a localized, structure-specific heart, say, a human heart, or a frog heart, etc. The localized functional properties are what we are interested in, and they are what scientists and scientific practices are focused on. I propose that a new focus of localized functional properties will make our discussion of reductionism more realistic and meaningful.

Although I agree with Sober’s approach to biological properties, I do not think that his view of local type reduction is about the reductionism that we discuss in philosophy of biology. Here’s why. If we take biology seriously, we will want to understand reductionism in light of evolution. We all recognize that natural selection operates
comprehensively in the biological realm. As Alex Rosenberg has emphasized, all sub-fields of biology and all biological phenomena are to be construed in the course of evolution. Since they are conditioned by the operation of natural selection on local circumstances – every biological phenomenon is a local phenomenon produced by an endless ‘arms race’ of natural selection working on local conditions – anything biological undergoes endless persistence of structural and functional variation.\(^{22}\) The same function (or effect) is realized in wildly heterogeneous disjunctive structural traits. The multiple realizability of function appears in the course of natural selection. The feature of multiple realizability is the mark of the actual operation of natural selection in our biological realm. So, the nature of our biological world teaches us not to expect neat, non-disjunctive natural kinds anywhere. Our biological space gives us no clean-cut or lasting concept of ‘natural kind’ or ‘type’. No nomological regularities about biological functional kinds are secured during the long course of evolution. There are no trans-spatiotemporally true laws, in any subfield of biology.\(^{23}\) This

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\(^{23}\) The argument of the absence of law plays a triple role in Rosenberg’s thesis. Casting doubt on all the reductionism debates conducted so far, he disputes the validity of post-positivist reductionism, and at the same time he renders useless all
point is consistent with Dennett’s view of evolution as a ‘universal acid’ eating through everything we believed and all the ways we look at the world.24 Once we see through the ‘operating system’ of our biological world, that is, natural selection, we cannot but realize that any kind of type reduction model leads us nowhere in biology. What we see is not a kind or type, but a particular biological phenomenon in a local condition. The particular biological events are something that are explanatorily and causally relevant to our inquiry of biology. Rosenberg proposes token reductionism as a new model of reduction to deal with particular biological phenomena.25 I believe that this model satisfies the objective of reductionism in biology. I will discuss token reductionism in more details in the following chapters.

I have argued that Kitcher’s ‘gory detail’ argument is not a successful refutation of the explanatory primacy of molecular biology. Cytology or other higher-level functional biology can offer only limited

antireductionist criticisms aiming at the issue of laws in the post-positivist reductionism.
and rough-and-ready explanations. Cytological explanations can never achieve higher degree of explanatory power unless the functional level refers to molecular details and molecular explanations. Explanations of molecular biology provide more complete and more accurate explanations about why and how these higher-level biological phenomena occur. Molecular biology always has explanatory primacy over functional biology. I also claimed that the global or type reductionism that Hull and Kitcher criticize cannot qualify as a working model of reduction in philosophy of biology. Global or type reduction is out of the picture in the debate of reductionism, and, therefore, so is the antireductionist argument which primarily targets these two models.
Chapter 3: Localized Functional Reduction in Biology

The goal of this chapter is to propose a new model of reduction in biology. This model evolves from my second-order functional property approach to biological properties and is applied to the problems of reduction in biology. Utilizing the distinctions of macro/micro-levels and first/second-orders I offered in the previous chapters, we are now provided with more fine-grained biological properties. Unfortunately, there still is a great deal of misunderstanding about the nature of reductionism, and one of the reasons for this is the failure to comprehend the nature of second-order functional biological properties. To rectify this situation, I will suggest that we construe second-order functional properties as localized functional properties. A simple example will explain my point quickly.

The property of being a heart is construed as a functional property: we all know that a heart is a blood pump regardless of what constitutes its material bases: in other words, something is a heart as long as it satisfies the job description - the function - of a heart.
However, when we research the nature of a heart, we do not look for an *abstract*, general property of *being a heart* that must be common among all the species that have hearts; rather, we always research a specific heart with a specific structure, say, a human heart, or a frog heart. I will argue that, although we may not like to accept the existence of general biological properties as abstract metaphysical entities, we can show that structure-specific biological properties do indeed exist. In fact, real-life researchers in biology do not deal with *abstract* metaphysical properties but always with *localized, structure-specific biological properties*. A localized functional property and the structure-specific biological property are what we are interested in and these are what make biological properties ripe for debates of reductionism. Also, this new focus on *localized functional properties* will make our discussion of functional biological properties realistic and meaningful. I will conclude that the localized functional reduction model, which is actually a version of a token reduction model, integrates the more sophisticated and fine-grained ontological hierarchy of both macro/micro-levels and first/second-orders. Furthermore, my localized functional reduction model synthesizes functional reductionism and token identity theory. I will show that the model of localized functional reduction satisfies ontological and epistemic requirements of
reductionism and thereby protects reductionism from the antireductionist criticism of eliminativism.

1. The Second-Order Approach and Functional Properties in Biology

Jaegwon Kim originally proposed functional reductionism for inquiry in the philosophy of mind. It crucially hinges on the second-order functional property approach to mental properties. I claim, as I did in Chapter 1, that functional properties in biology - being a heart, having a muscle contraction, and being a transcription factor - should also be understood as second-order functional properties. Consider, again, the biological property having a (process of) muscle contraction. Muscle contraction can be understood as a second-order property defined over the domain of its first-order properties, that is, mechanical interactions of the myosin and the actin filaments: {binding myosin’s head with actin, sliding the actin along myosin, releasing the head of myosin from the actin, ...}. Since these first-order properties are specified in terms of their causal/nomic roles (i.e., movement of muscle), the property of having a muscle contraction is a second-order functional property. Let me explain this point in more detail.
Recall the definition of a second-order property. Let D be a set of (first-order) base properties.

\[ F \text{ is a second-order property over } D = \text{def. } F \text{ is the property of having some property } G \text{ in } D \text{ such that } S(G), \text{ where } S \text{ is a specification on members of } D. \]

Second-order functional properties are a subset of second-order properties where the members of their first-order properties are specified in terms of their causal/nomic roles:

\[ F \text{ is a functional property over } D = \text{def. } F \text{ is a second-order property over } D \text{ defined in terms of a specification } S \text{ that states causal/nomic relations involving members of } D. \]

So, for something to have a second-order functional property \( F \) is for it to have some first-order property or other in a given domain that meets a certain causal specification definitive of \( F \).

Consider (the process of) meiosis. I argued in Chapter 2 that meiosis is a second-order functional property. Meiosis is the set of two successive cell divisions which separates homologous chromosome
pairs and leads a diploid cell (46 chromosomes, or 2n) to become haploid gametes (23 chromosomes, or n). In the process of meiosis, the number of chromosomes is reduced to exactly one-half. Meiosis, the two successive meiotic cell divisions, is composed of a number of distinct stages. (See Figure 5: Stages of Meiosis) At the stage of meiotic prophase, homologous chromosomes get paired, and the paired chromosomes attach to the meiotic spindle and line up on the middle of the meiotic spindle at metaphase I. At anaphase I the pairs of homologous chromosomes separate and move to opposite poles, and the first cell division takes place. Each chromosome then recondenses and becomes two sister chromatids. These chromatids align themselves on a new pair of spindles at metaphase II, and move to opposite poles at anaphase II. At telophase II, each sister chromatid reaches opposite poles and nuclei begin to reform. The second cell division usually occurs at this time, and at the end of this second meiotic division four daughter cells, each with a single copy of each chromosome, are created.

With this cytological information, we can define meiosis as the second-order property existentially quantified over the set of its first-order base properties: homologous chromosomes’ pairing, their aligning, their separation, sister chromatids’ aligning, their separation, and so
These various first-order properties all have the same single causal role: reducing the number of chromosomes to half and eventually generating haploid gametes. We may understand meiosis as a second-order functional property over the set of first-order base properties which are specified in terms of the causal task of bringing about haploid gametes. This example shows us that (the process of) meiosis, construed as a second-order functional property, is in each of its occurrences ‘nothing over and above’ one of its first-order base properties, \textit{i.e.} one of its stages. There is nothing more to a specific case of meiosis than one of its stages.

2. \textbf{Second-Order Functional Properties and the Multiple Realizability Thesis}

What is the relation between a second-order functional property and its first-order properties (realizers)? Meiosis is a functional biological property defined over the set of its first-order base properties. Meiosis is specified in such a way that something, some phenomenon, or some event is meiosis insofar as it takes up the causal/nomic role of meiosis, that is, if it is caused by the successive events of chromosomai pairing and separation. Given the definition of a functional property, it
is clear that the function of meiosis can in principle be realized in many base physical properties.

Consider another example, being a transcription factor. As I noted in Chapter 1, a transcription factor is a trans-regulatory protein that binds to a specific enhancer or promoter region of DNA sequences to regulate the transcription of genes that are switched on and off by the enhancer or promoter. A specific DNA-binding site of a transcription factor is called a DNA-binding domain or trans-activating domain, and this DNA domain enables the transcription factor to interact with other proteins. This interaction causes the formation of the basal transcription complex that in turn binds RNA polymerase and eventually regulates the transcription of the genes. The structure of the DNA-binding domain in a transcription factor is the key for classifying transcription factors. Some transcription factors have a homeodomain structure or the basic helix-loop-helix (bHLH) motif. Other transcription factors have a basic leucine zipper (bZip) or a zinc finger motif. (See Figure 4: Transcription Factors)

The property of being a transcription factor can be understood as a property of proteins whose role is binding to the enhancer or promoter regions of DNA sequence; this regulates the transcription of genes that correspond to the enhancer or promoter. Such proteins contain several
kinds of different structural properties: being a homeodomain, being the basic helix-loop-helix motif, being a basic leucine zipper, or being a zinc finger motif. Now, we should understand the property being a transcription factor as a second-order functional property that is defined over the set of its first-order properties specified in terms of their causal role; in this case, binding to enhancer or promoter regions of DNA sequence in order to regulate the transcription of genes that are under the control of the enhancer or promoter. Here, we can also see that being a transcription factor as a second-order functional property is realized in one of its various first-order base properties, {being a homeodomain, being the basic helix-loop-helix motif, being a basic leucine zipper, or being a zinc finger motif.}

‘Realization’ is the best way to understand and explain the relation between second-order functional properties and their first-order properties (realizers). A biological property, construed as a second-order functional property, cannot be instantiated without being realized in one or another of its first-order base properties. However, a first-order base property does not have to rely on any other higher-order property for its instantiation. The realization model specifies an asymmetric dependence relation between second- and its first-order properties.
3. **The Determinate-Determinable Relation (DDR)**

The realization relation between a second-order functional property and its first-order properties should be clearly differentiated from the determinate-determinable relation. To see the subtle but important difference, I will reformulate the traditional model of determinate-determinable relation between properties (DDR, hereafter) and show why it is different from the first/second-order relation. First, I will show that DDR is a necessary relation, and that determinates are related to determinables in an asymmetric necessitation relation. But DDR is also a conceptual relation, so a determinable can be derived via conceptual analysis of one of its determinates. In contrast, the relation between a second-order property and its first-order base is neither metaphysically necessary nor conceptually derivable. The first-order properties of a given second-order property are empirically discovered in nature, so their relation is contingent and non-conceptual. Second, I will prove that, unlike the second-order functional property approach, DDR is inconsistent with the multiple realizability thesis of functionalism. However, as I discussed in Chapter 2, the multiple realizability of biological phenomena is precisely what indicates that natural selection has been operating in our biological realm. Due to the
endless and persistent activities of natural selection, our biological world has provided widely disjunctive and ‘wildly heterogeneous’ base properties for any biological function. In this respect, if DDR is not compatible with the multiple realizability thesis that we cannot give up, we must conclude that DDR is useless for reductionism in biology.

3.1 Definition of the Determinate-Determinable Relation

W.E. Johnson (1921) introduced the term ‘determinable’ in his *Logic*:

I propose to call such terms as color and shape determinables in relation to such terms as red and circular which will be called determinates.¹

What he means is that some properties stand to others as determinate to determinable: being red is a determinate of the determinable color, and being circular is a determinate of the determinable shape. It can also be that crimson is a determinate of the determinable red, being square is a determinable of the determinable being rectangular, guzzling

is a determinate of the determinable *drinking*, and so on. So what exactly is this determinate-determinable relation?

Stephen Yablo refines Johnson’s DDR and introduces his own definition:

(D) \( P \) determines \( Q \) only if:

(i) necessarily, for all \( x \), if \( x \) has \( P \) then \( x \) has \( Q \);

(ii) possibly, for some \( x \), \( x \) has \( Q \) but lacks \( P \).\(^3\)

Yablo’s definition, (D), shows that he prefers to understand DDR as a necessitation relation: \( P \) necessitates \( Q \) just in case *necessarily* if anything has \( P \) it has \( Q \). The way Johnson understands DDR is not so different from Yablo’s (D). So we must ask of them: What is it that explains or grounds the necessitation relation between \( P \) and \( Q \)? Yablo’s definition of DDR says only that there is a certain necessary *relation* between \( P \) and \( Q \); but does not explain the nature of the necessary relation. (D) is silent in explaining *why* \( P \) determines \( Q \); all it says is that DDR is the relation of an asymmetrical necessitation. However, we would all agree that this kind of metaphysical

necessitation relation cannot be introduced into biology. Our biological world simply cannot accommodate ‘necessitation’ per se. The occurrences of biological phenomena are all contingent, and they never show any sign of necessity. Moreover, the relation between biological properties/events is crucially dependent upon local conditions. Due to the endless and persistent operation of the process of natural selection, any ‘necessary’ relation between properties or even ‘lawlikeness’ is not in the cards for the biological realm.

The claim of ‘brute’ metaphysical necessitation relation cannot serve as a model of reductionism in biology. Reductionism should be able to provide an explanation of why and how a certain biological event determines another event. But a mere affirmation of the existence of asymmetric necessitation relation between the two biological events does not say much at all. For example, let $P$ be a property in molecular biology, say, the process of releasing histamine, and $Q$ a property in functional biology, say, an abnormal immune system reaction (caused by a harmless substance), including a runny nose. I formulate this case applying (D) as follows:

The process of releasing histamine determines a runny nose only if (i) necessarily, for all humans, if a human has histamine
release, then she has a runny nose, and (ii) possibly, there are some people who have a runny nose but lack the process of releasing histamine.

Does this appear to be a plausible and acceptable explanation of anything? What does the ‘determination’ tell us? —Not much. It could say only that ‘the process of releasing histamine’ is necessarily related to ‘having a runny nose’. But it does not provide any explanations of why these two properties are related and how these two different events occur in a related fashion. Unless (D) satisfies this basic requirement of explanatory accounts, we cannot adopt DDR in biology to represent and explain the relations of biological properties.

John Searle has attempted to explicate the determinate-determinable relation in terms of predicate entailment.4 ‘Red’ entails ‘colored’ because it is impossible for something to be red and not colored. Guzzling and sipping both conceptually imply drinking because it is impossible for someone to guzzle or sip but not be drinking. Also, crimson, scarlet, and pink each conceptually entails red. However, if DDR is properly understood as conceptual, that is, if the relation

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between a determinate and its determinable is something analytic, the DDR model cannot be used to represent the relations among biological properties. This is simply because the relation among biological properties is not something analytic. We cannot get the property, ‘being a heart’, for instance, by conceptually analyzing different physical and molecular features of organs that pump blood. The concept or meaning of a certain structure of an organ does not logically entail its having the function of a heart, or its being a heart. It is instead a matter of empirical discovery that different structures of organs happen to have the function of a heart, namely, pumping blood. Also, a conceptual entailment relation in biology cannot play an explanatory role where all the important relations are contingent. The conceptual entailment aspect of DDR shares the same problems with the necessary relation aspect of DDR.

Sara Schwartz introduces DDR to define the relation between a trait and a change in a trait. She understands DDR as a necessary or a conceptual relation just as Johnson, Yablo, and Searle did, and she believes that DDR can play a role in explaining biological properties. In her essay, “The Differential Concept of the Gene,” she writes:
The differential concept of the gene states that there is a relation between a change in a gene (an allele) and a change in a trait. In order to distinguish between a change in a trait and a trait, I will name the first an alternative appearance of a trait (AAT). The distinction between a trait and an AAT [an alternative appearance of a trait] is analogous to W.E. Johnson’s now classical distinction between determinable and determinate properties. ... Johnson drew attention to three features in which determinables relate to determinates that are of interest here. 1) If a particular has a determinate property (AAT), it then follows that the particular has the determinable property (a trait). *If a pea is round, it necessarily has a shape.* 2) If a particular falls under a determinable (a trait), it follows that it has one of the corresponding determinate properties (AATs), although the specific determinate property is not entailed. Thus, if a pea has a shape, it *necessarily* has some particular shape, *i.e.*, round, wrinkled, etc. 3) One particular cannot at the same time represent more than one of the determinates (AATs) which fall under a common determinable (a trait).\

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The relation between a trait and what Schwartz calls an AAT (an Alternative Appearance of a Trait or changes in a trait) is, according to her, DDR. Like Johnson and Yablo, she understands this relation as a necessary one. She also seems to construe DDR as a conceptual relation, since she writes “If a pea is round, it necessarily has a shape.” We can have a concept of shape by analyzing the concept of a determinate, that is, a particular shape (round, wrinkled, etc.). This should mean that we can have the determinable biological properties by analyzing the concepts of determinate biological properties. If DDR represented the relation of first- and second-order properties and if functional biological properties were the determinables of the first-order base properties, then we should be able to discover and identify the functional properties by logically analyzing the concepts of the first-order base properties. But the conceptual analyses of the first-order properties can never give us the concepts of functional properties. The relation between a functional property and its base properties (realizers) can only be discovered empirically. Think about a simple example, *being a heart*. This property construed as a second-order functional one, is realized in a disjunction of first-order physical properties, that is, {being four chambers in mammals, being two chambers in fish, being

three chambers in amphibians and reptiles, being one chamber in many invertebrates). Can we understand the relation between being a heart and its realizers by conceptual analysis? —No. We cannot infer being a heart from any logical analysis of the concepts of the physical properties of a specific organ. Therefore, the relation between functional biological properties and their first-order base properties is not conceptual. If we accept the multiple realizability of functional biological properties as something non-conceptual, and if DDR is understood as expressing conceptual relations, then we cannot use the DDR model to illuminate the nature of biological properties. I will further discuss the problems of the DDR model in the following section.

3.2 The Incompatibility of DDR with the Multiple Realizability Thesis

Consider the property of having H$_2$O molecules. The properties of being steam, of being liquid water, and of being ice can be said to be determinate properties of the determinable, being composed of H$_2$O molecules. There exists an asymmetric necessitation relation between each of these three properties and the property of being composed of H$_2$O molecules. It satisfies Johnson’s and Yablo’s definitions of DDR. Therefore, although the three determinates each have different causal
powers, they are all determinates of the same determinable property: being composed of H₂O molecules. How can this be so? Because the three determinates have in common the property of being composed of H₂O molecules. This point is essential to understand the nature of DDR. If there is nothing in common among determinates, how could they fall under the same determinable? Things that are scarlet or crimson or pink have something in common: they are all red. Yellow, red, or blue things all have the property of being a primary color. Guzzling and sipping have the property of drinking in common. This is what is required of the properties standing in the determinate-determinable relation. Arthur Prior explicitly addresses this point.

Determinates under the same determinable have the common relational property... of characterizing whatever they do characterize in a certain respect. Redness, blueness, etc., all characterize objects, as we say, ‘in respect of their color’; triangularity, squareness, etc., ‘in respect of their shape.’ And this is surely quite fundamental to the notion of being a determinate under a determinable.\(^6\)

W. E. Johnson apparently believes that co-determinates at the same-level do not have anything in common except their incompatibility.\textsuperscript{7} For instance, red and blue do not share the same properties and they are incompatible. However, to make sense of Johnson’s view, it has to be interpreted in such a way that if blueness characterizes a thing, then redness cannot characterize it, and that blueness and redness are incompatible in this respect. Without this interpretation, his view seems completely implausible: it is very hard to believe that blueness and redness have absolutely nothing in common. David Armstrong also mentions this problem: “Johnson’s view that co-determinates at the same level have nothing in common except their incompatibility is phenomenologically implausible. The class of shapes or the class of triangles, the class of colours or the class of reds, appear to have much more in common than that.”\textsuperscript{8}

Scarlet, crimson, and pink have redness in common; red, yellow, and blue have the property of being a primary color in common.

Otherwise, they cannot make the co-determinates of the same determinable. However, the multiple realizability thesis obviously argues for exactly the opposite claim: \textit{There is no significant, non-}

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functional property common to all the realization bases of the functional property of being a heart. Since the continuous selection process in nature is blind to specific structures of organs, there is no common physical condition or criteria with which to group all the varied realizers of functional biological properties. DDR requires a significant common property in the determination bases, but the multiple realizability thesis denies the existence of such a common property in the realization bases. We must conclude that DDR is not compatible with the multiple realizability claim of functionalists, and thus DDR does not specify the relation between functional properties and their base properties. Therefore, any attempts to use the DDR model to illuminate the relations among biological properties will be at best misleading and in any case will eventually fail.

4. Functional Reduction

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9 One might argue that there exists among all the realizers at least the common property of instantiating the same functional property. But this is not the functionalists’ point when they affirm the multiple realizability of functional properties. They claim that there is no significantly common physical (and non-functional) property among the varied base properties, and this is exactly the objection with which they defeated the classical type-identity theory in philosophy of mind once and for all.
I employ Kim’s second-order functional property approach to tackle the issues of reduction. For the philosophy of biology, I modify his model as follows\(^\text{10}\):

\begin{itemize}
  \item [1\textsuperscript{st} Step] Construe or reconstrue a biological property as a second-order functional property in the following fashion:

  Having a biological property \( F = \text{def.} \) Having some property or other such that the property performs a causal task \( C \).

  \item [2\textsuperscript{nd} Step] Find \( F \)'s realizers, that is, the base properties that perform the causal task \( C \) of the functional property.

  \item [3\textsuperscript{rd} Step] On a given occasion, having a second-order functional property \( F \) is \textit{nothing over and above} having a base property or other where the base property is the realizer of \( F \).
\end{itemize}

As we see above, the observation of property instance identity (or token identity) completes the process of functional reduction. The following

\(^{10}\) For Kim’s detailed arguments, see \textit{Mind in a Physical World}, MIT Press, 1998, Chapter 4.
steps will also show us how we can develop a scientific theory to reduce a property $F$.

*Step 1.* Functionalize $F$ by providing a functional definition or functional characterization of the following form:

For $x$ to have $F$ (or to be an $F$) = \text{def.} for $x$ to have some property $G$ such that $C(G)$, where $C(G)$ states a causal specification that $G$ must meet.

*Step 2.* Identify – discover – the $G$, or $Gs$, that meet the causal specification $C$ in the individual, or a population of individuals, of interest in a given research program. Such $Gs$ are called the “realizers” of $F$ in these individuals.

*Step 3.* Develop a theory that explains how the realizer $G$ performs the causal task specified by $C$ in the individuals in question.

Steps 2 and 3 are obviously matters of empirical scientific research. Yet the question whether $F$ can be defined functionally is a conceptual issue. Even so, how to formulate or characterize a functional definition
for a given $F$ is likely dependent on empirical findings and theoretical needs. What is important for functionalizability is that once $F$ has been functionalized (or shown to be functional) we know that $F$ is functionally reducible. Given that $F$ is a functional property, if anything has $F$, then it follows that it has one or another of the realizers of $F$. An instance of $F$ is reducible and $F$ is reductively explainable in terms of its realizer.

Let us examine how the functional reduction works in biology. Take for example the property of *being a transcription factor*. The first step in functional reduction is to construe *being a transcription factor* as a second-order functional property. Having a transcription factor is defined as having some property or other that performs the causal task of *being a transcription factor*: binding to the enhancer or promoter regions of a DNA sequence to regulate the transcription of genes that control the enhancer or promoter. The next step is to find out, empirically, those varied properties that have the causal role of *being a transcription factor*, i.e. regulating genes’ expression. Scientific researchers have discovered that proteins with DNA-binding domains of several different structures (homeodomain, basic helix-loop-helix motif, basic leucine zipper, or zinc finger motif) perform the causal task of a transcription factor. These different properties are the realizers of the second-order functional property, *being a transcription factor*. Following
Kim, the last step of a functional reduction model is to see that having a transcription factor is on a given occasion nothing over and above having one of its first-order properties (realizers). Developing a theory that explains how the realizers perform the causal task of being a transcription factor among the individuals in question will complete the task of functional reduction.

5. **Disjunctive Properties and Local Reduction**

It is impossible to seek a straightforward and simple reduction model with which we can reduce a biological functional property to a specific physiochemical property. It has been widely accepted that, due to the multiple realizability of a functional property, a second-order functional property is realized in varied physical bases, and the heterogeneity of realizers makes such a simple reduction model as global-reductionism or type-reductionism a nonstarter. To examine this problem in more depth, I now suggest that we first look at the original thesis of the multiple realizability which Hilary Putnam proposed in 1967.
Putnam presented the multiple realizability argument in a paper called “Psychological Predicates.” He changed this title to “The Nature of Mental States” when he republished it in a collection of his essays. I believe this simple change of the title might have led us to forget the original focus of the multiple realizability argument. The multiple realizability that Putnam first proposed was about the one-to-many relation between a predicate and its ‘wildly heterogeneous’ referents, not about the relation between a property type and its varied realizers. I do not know why Putnam changed the title of his paper, but philosophical discussion of the multiple realizability thesis has ever since focused on the nature of properties, not on the nature of predicates and their multiple applicability.

Functional properties have been often regarded as disjunctive properties such that if a functional property $P$ is realized in $P_1$ or $P_2$ or $P_3$ then $P$ is in fact identical with a disjunctive property $P_1 \lor P_2 \lor P_3$ [$P = P_1 \lor P_2 \lor P_3$]. However, I do not think this is the correct way to understand the point of multiple realizability, especially when Putnam himself makes it clear that ‘disjunctive properties’ should not be taken seriously. I contend that multiple realizability should be understood in

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terms of the multiple applicability of a functional predicate, and not always with a property locution that tempts us to erroneously postulate the existence of disjunctive properties. Let me further elaborate on this point.

Whenever a property is multiply realized, we should be able to find something in common among all the things in which the property is instantiated. Everything that instantiates the property *being blue*, for example, shares something in common, that is, *being blue*. Now, suppose there is a property \( P \) in such a way that \( P \) is a disjunctive property \{being red or being round\}. Can we here find out that everything that instantiates this ‘disjunctive property’ \{the property red or the property round\} shares anything in common? Obviously not: it is impossible to find anything in common between all red things and all round things. This is why we cannot accept ‘disjunctive properties’ as properties.

Historically, many philosophers have argued against the notion of a disjunctive property. David Armstrong, for one, says that a disjunction of predicates such as ‘red’ and ‘hard’ does not itself have a disjunctive property. There is no property of *being red or hard*, although each disjunct of the meaningful *predicate* ‘red or hard’ does
correspond to the properties red and hard, respectively.\textsuperscript{12} Alex Rosenberg also points out:

\textit{[W]e need to distinguish predicates in languages from properties in objects... the only predicate we employ that is true of every [P.sub.i] is a disjunctive one, but it does not follow that the property picked out by the disjunctive predicate is a disjunctive property.}\textsuperscript{13}

To sort out and clarify all the problems involved in ‘disjunctive properties,’ I suggest we distinguish \textit{sentential disjunction} from property disjunction. When a property $\Phi$ is a ‘seemingly’ disjunctive property $[\Phi_1 \lor \Phi_2]$, the claim $[\Phi = \Phi_1 \lor \Phi_2]$ should not be understood as:

\begin{align*}
\text{Property } \Phi &= \text{ disjunctive property } [\Phi_1 \lor \Phi_2]
\end{align*}

We should instead interpret the ‘or’ or ‘$\lor$’ in $[\Phi = \Phi_1 \lor \Phi_2]$ as a \textit{sentential disjunction}. ‘Property $\Phi = \text{ disjunctive property } [\Phi_1 \lor \Phi_2]$’ is to be

\begin{footnotesize}
\end{footnotesize}
understood as saying that something has \( \Phi \) amounts to the fact that it has \( \Phi_1 \) or it has \( \Phi_2 \). Also,

\[
\text{Having Property } \Phi = [\text{having } \Phi_1] \lor [\text{having } \Phi_2]
\]

This can also be understood as follows:

\[
\{\text{Having Property } \Phi = [\text{having } \Phi_1]\} \lor \{\text{Having Property } \Phi = [\text{having } \Phi_2]\}
\]

This sentential interpretation of ‘disjunctive properties’ vindicates the ontological status of functional properties. Due to the heterogeneity of the realizers of a functional property, it could be argued that a functional property is in fact a disjunctive property and as such cannot be regarded as a well-behaved, single nomic kind that has a single causal power. Since a functional property is not a single nomic kind, however, there is no guarantee that it is a causally efficacious, legitimate property in its own right. But with the sentential interpretation of a functional property, we now have a better chance to

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save its legitimacy as an ontological entity. I will discuss this point in detail in the next section.

To appreciate the value of sentential interpretation, think once more about ‘being a heart.’ The property of being a heart is a second-order functional property, as I argued in Chapter 1. Pumping blood is a causal/nomic role of the second-order property of being a heart, and being a heart is realized in varied properties of various structures, such as a muscular cone-shaped organ which has four chambers in mammals, three chambers in amphibians and reptiles, two chambers (one atrium and one ventricle) in fish, and one chamber in many invertebrates. The relation between the second-order functional property being a heart and its diverse realizers can be formulated as follows:

\[
\text{Being/Having a Heart} = \text{[Having four heart chambers]} \lor \text{[Having three chambers]} \lor \text{[Having two chambers]} \lor \text{[Having one chamber]}
\]

This formulation, again, can be construed as follows:

\[
\{\text{Having a Heart} = \text{Having four heart chambers}\} \lor \{\text{Having a Heart} = \text{Having three chambers}\} \lor \{\text{Having a Heart} = \text{Having two chambers}\} \lor \{\text{Having a Heart} = \text{Having one chamber}\}
\]
\{Having a Heart = Having two chambers\} or
\{Having a Heart = Having one chamber\}

From the sentential interpretation of multiple realizers, we can see here that each disjunct sentence shows that the functional second-order property, \textit{being a heart}, is identified with its first-order realizer as long as the first-order realizer exhibits the function of a heart, that is, implementing a blood circulatory system.

Let me unpack this idea a little more. Each disjunct sentence implies that \textit{being a heart}, which is seemingly an abstract biological property, is a \textit{localized, structure-specific biological property} when it is instantiated in a given structure. The localization of general biological properties is exactly what happens in the real-life field of biology: working scientists never deal with a heart as an abstract entity in their research. Rather they always choose a research model system such as a rat, mouse, fruit fly, or worm and engage their research on a heart in one of these model systems. We can make this point clearer as follows:

\{Having a Heart = Having four heart chambers in mammals, birds, and crocodiles\} or
{**Having a Heart** = Having three chambers *in amphibians and most reptiles*} or

{**Having a Heart** = Having two chambers *in fish*} or

{**Having a Heart** = Having one chamber *in many invertebrates*}

Each disjunctive sentence shows that the property *being a heart* is localized in a specific structure, say, a human heart, or a frog heart, etc. Scientists and scientific practices have always focused on the properties of a ‘structure-specific heart’ as the locus of a localized functional property. It is also what makes our discussion of functional biological properties relevant and meaningful. Thus, the biological property, *being a heart*, is realized and localized in its first-order property of a given structure on a given occasion; the instance of the biological property *being a heart* is, on this occasion, identical with the instance of its first-order property (realizer) in this structure. Hence, *being a heart* is ‘functionally reduced’ to its realizer on this occasion.

*Cell polarity* is known as a biological property of cells that has the causal role of establishing the anterior-posterior axis (A-P axis) that generates asymmetrical cell divisions at the early stages of embryonic development. Recently, molecular biologists have found that *cell polarity* is shown when some cell-fate-determinant proteins are localized
asymmetrically in a cell. Early blastomeres of *Caenorhabditis elegans* zygotes exhibit cell polarity when PAR and CDC42 proteins are asymmetrically localized in cells.\(^{15}\) *Drosophila’s* epithelia show cell polarity when Bazooka (Bas) protein and Atypical Protein Kinase C (aPKC) are asymmetrically distributed.\(^{16}\) To interpret these reports of the molecular biologists, I suggest we understand the biological property, *cell polarity*, as a functional property defined over the domain of the first-order causal/structural properties, that is, \{asymmetric localization of PAR protein and CDC42 protein in the early blastomere of *C. elegans*, asymmetric localization of Bas protein and aPKC protein in the epithelia of *Drosophila*\}. These various structural properties perform the causal role of *cell polarity*, causing A-P axis in cells and subsequently generating asymmetrical cell divisions in the early stages of embryonic development. Asymmetric localization of many different proteins realize *cell polarity* in different cells, so we can say that *cell polarity* is a functional property that is realized in its many different physical bases:


Cell polarity = [Asymmetric localization of PAR proteins and CDC42 protein in the early blastomere of C. elegans] v [Asymmetric localization of Bas protein and aPKC protein in epithelia of Drosophila]

This disjunctive character of cell polarity can be interpreted as a kind of sentential disjunction:

Having cell polarity = [Having asymmetric localization of PAR proteins and CDC42 protein in the early blastomere of C. elegans] or [Having asymmetric localization of Bas protein and aPKC protein in epithelia of Drosophila]

This is again understood as follows:

{Having cell polarity = [Having asymmetric localization of PAR proteins and CDC42 protein in the early blastomere of C. elegans]} or {Having cell polarity = [Having asymmetric localization of Bas protein and aPKC protein in epithelia of Drosophila]}

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As we see here, *having cell polarity*, which is a second-order functional property, is realized in its different first-order properties. Each disjunct shows that *having cell polarity* is localized in a specific property/structure, say, the early blastomere of *C. elegans* or epithelia of *Drosophila*. This localized functional property, *having cell polarity*, is identified with and reduced to its realizer on a given occasion.

6. **Localized Functional Reduction is Not Elimination**

A functional property $F$ is not really a single nomic property because if $F$’s realization bases are wildly heterogeneous, as functionalists claim, $F$’s causal powers would also be as wildly heterogeneous as its realization bases are. This observation has raised many issues about the nature of functional properties. To discuss these problems more productively, let us first recall Kim’s suggested process of functional reduction\(^{17}\):

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\(^{17}\) For Kim’s detailed arguments, see *Mind in a Physical World*, MIT Press, 1998, Chapter 4.
[1st Step] Construe or reconstrue a biological property as a second-order functional property in the following fashion:

Having a biological property $F = \text{def.}$ Having some property or other such that the property performs a causal task $C$.

[2nd Step] Find $F$’s realizers, that is, the base properties that perform the causal task $C$ of the functional property.

[3rd Step] On a given occasion, having a second-order functional property $F$ is *nothing over and above* having a base property or other where the base property is the realizer of $F$.

As we see at the first step, if we construe $F$ as a functional property defined over a given set of first-order properties, it would be hard to accept $F$ as a property in its own right. This is because a mere logical operation (existential quantification) over a given set of first-order properties does not create a new property in the world. It might be less misleading if we talk about a second-order *predicate* of a property in the set of first-order properties, or a second-order *concept*, rather than a second-order property. All things considered, it may be that there
exists no such general (vis-à-vis local structure-specific) biological property that is realized in such varied physical bases across all the different structures. It may just be a concept or a predicate, which does not by themselves have causal powers. It is properties (or instantiations of properties, to be precise) that have causal powers. If what we have considered as functional biological properties are in fact just functional predicates/concepts with no distinct causal powers, then we will arrive at an unacceptable conclusion: at best an epiphenomenalism of functional biological properties beyond the instantiation of first-order properties, or worse, an eliminativism of the functional biological properties. Very few philosophers would tolerate such a conclusion. We need to find a way to avoid this problem.

I should emphasize again that we need to understand a disjunctive property not as a property per se but as a set of disjunctive sentences. Only with this new understanding can we clearly see that each disjunct guarantees the notion of a localized functional property, one that is more fine-grained and structure-specific. If we adopt the sentential interpretation of disjunctive properties, we will see that the multiple realizability of a functional property should be understood as the realizability of a functional property in one of its many local conditions, that is, in one of its local base/structural properties. The
localization of a functional property turns the general functional property, which could be regarded as merely a second-order functional concept/predicate, into a real, explanatorily relevant localized structure-specific functional property. Further, having a localized functional biological property is, as Kim’s third step of functional reduction shows, nothing over and above having its local realizer.

A localized functional property becomes identical with its base/structural property, and the identity relation between a localized functional property and its base/structural property lets them share the same causal powers. But we need to note that this identity is not a matter of ‘necessity’. Unlike the classic examples of necessary identity - ‘Mark Twain is Samuel Clemens,’ ‘Hesperus is Phosphorus,’ ‘Water is H$_2$O,’ - the identity between a localized functional property and a base/structural property, and their realization relation, is strictly contingent. Likewise, the identity between the causal powers of a localized functional property and those of its base/structural property is contingent. In our biological world, if $x$ has a functional biological property $F$, $x$ has a realizer $G$ of the functional biological property $F$ in virtue of the constant operation of natural section that binds functional properties to base/structural properties. This means that a localized functional property shares the same causal role as a base/structural
property at this local condition at this time, but not necessarily at any other condition or at any other times. The identity between the causal powers of a localized functional property and those of its base/structural property is only contingent. Nonetheless, it survives as a legitimate property with its own causal powers.

So why is a localized functional property $F$ reduced to, but not eliminated by, its base/structural property $G$? Because to have a localized $F$ is to have a causal role and $G$ has this causal role at this local condition at this time. The localized $F$ and $G$ share exactly the same causal role/powers in the given local conditions. Since the local functional property does have causal powers, it is not eliminated; it is only reduced to its local realizer by sharing its causal powers. Thus local functional reduction is not a kind of eliminativism.

7. **Localized Functional Reduction in Biology**

Here is my model of localized functional reduction:

**[Localized Functional Reduction]**

If a functional property has a realizer property in a structure of
type S, then the localized structure-specific functional property is reduced to, and identical with, the realizer property.

This localized functional reduction is a version of intra-level reduction:

[Localized Functional Reduction]

The higher-order functional properties at the level i are reduced to their first-order base/structural properties at the same level i.

This model shows us that a localized functional property is reduced to its base/structural property. In biology, a localized structure-specific functional property is understood and explained in terms of its first-order base/structural properties. This is also an intra-level reduction model: the localized functional biological property is reduced to, and is reductively explained in terms of, its base/structural properties at the same level. The localized functional biological property and its base/structural property are different characteristic properties of one and the same entity which of course belongs to the same level in the layered model of ontology. This model differs from mereological reduction, but they complement each other and provide a richer story for reductionism in biology (See Figure 6: Reducing Biology).
Reductionists working in biology must consider the localized functional reduction model.
Chapter 4: Reducing Genes

This last chapter of my dissertation aims to reduce the concept of gene using my model of localized functional reduction. Many of us may agree that the gene is the most fundamental biological property. Hence, reducing the gene at the genomic level is crucially important for a reductionist not only to show the validity of her reduction model but also as a significant contribution to the success of reductionism generally. In this final chapter I will first show that the concept of gene has been undergoing conceptual changes. Its referents are unmanageably diverse, wildly heterogeneous, and seemingly unrelated to each other: reducing the gene at the genomic level may thus appear to be an impossible job. I will then discuss the reasons why locutions containing gene properties are still legitimate and useful even though none of the properties with which the gene is identified can ensure a unified concept. With these preliminary discussions, I will work on three tasks to reduce the gene: first, I will argue that the concept of gene be understood as a second-order functional property; second, I will
find genomic realizers of the gene and support the claim that a gene is a structural-specific functional property; third, I will reduce the functional property being a gene to its genomic realizers via structure-specific identity. I will claim that the gene should be understood as the ‘gene expression network’-specific functional property and when it is realized on a given occasion, is reduced to, and identical with, its realizer that is constituted of DNA sequences and their interactions. My proposal provides a new, dynamic approach to the concept of gene and vindicates reductionism.

1. Mendelian Gene

Let’s first examine how the concept of gene has evolved in the history of biology. It appeared for the first time in the work of Gregor Mendel in 1865. Mendel explained that each parent contributes one of two “factors” and these factors segregate independently into sexual gametes. He thought of this “factor” as the unit of inheritance that transmits heritable characteristics from parents to their offspring.\(^1\) In the 1920s, Thomas Hunt Morgan refined the concept of gene. He hypothesized that if the gene is the unit of inheritance transmitting

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\(^1\) In 1909 Wilhelm Johannsen coined the term ‘genes’ for Mendel’s hereditary ‘factors’.
heritable characteristics, and, consequently, if it directs the
development of unit characters, then the gene is not only the unit
characters but also the unit of genetic transmission, genetic
recombination, gene mutation and gene function. In 1922, Hermann J.
Muller further extended the concept: the gene is something that has the
property of self-replication, the property of mutation, and the property
of manufacturing products other than itself that provide equipment for
the developing organism. Up until that time, all biologists approached
the gene type as a ‘theoretical’ entity which had a pivotal causal factor
in the development of organisms, but its referent was yet to be known.

In 1941, Beadle and Tatum sought a way to replace the
theoretical entity with a real one. They advanced the hypothesis that
genes are chromosomal segments and the function of each gene is to
produce its corresponding enzyme. They intended to establish the gene
as a concrete entity by counting its product, an enzyme. If a gene is a
chromosome, then it is something that encodes its corresponding
enzyme (and later, polypeptide). People understood the gene as a
causal factor that has several different properties – properties of
transmission, recombination, self-replication, mutation of heritable
characteristics, and manufacture of products for an organism’s
development. These properties are the gene’s causal roles in
development, or *causal specifications* that something must meet to qualify as the gene.

[The Mendelian Gene]

For an organism to have genes is for it to have the properties of transmission, recombination, self-replication, mutation of heritable characteristics, and manufacture of products for its development.

2. **The Fuzzy Molecular Gene**

The advent of molecular biology shifted our inquiry from searching for its properties to finding the referent of “the gene” itself. With their understanding of the gene’s properties (or causal roles), working scientists endeavored to prove that the ‘theoretical’ Mendelian gene exists. In 1944 Oswald Avery successfully purified and demonstrated that it was the nucleic acid – not protein – that transfers the infectious characteristics of the strain of bacteria to another one. The gene exists as the nucleic acid. In 1953 James Watson, Francis Crick, Linus Pauling, and many of their contemporaries jointly or independently contributed their expertises to unveil the structure of
deoxyribonucleic acid (DNA). They revealed the simple yet elegant double helix of DNA, and hinted scientists the mechanism of genetic transmission right out of the DNA structure. The two strands separate from one another, and then each strand functions as a template for the assembly of a new strand through complementary base pairing. This explanation of genetic transmission suggested by Watson and Crick was satisfactory in that DNA has the property of self-replication, one of the three properties of genes which Muller theoretically suggested three decades ago. Crick proposed the idea that genes as sequences of deoxyribonucleic acid carry the genetic information for the assembly of a particular protein. His idea was based on the hypothesis of co-linearity between one gene and one protein. He also suggested that a genetic message transfers from deoxyribonucleic acid to ribonucleic acid and finally to protein.\(^2\) The genetic message encoded in DNA is transcribed into a molecule of RNA, and then the same message encoded in RNA (a messenger RNA, to be precise) is translated into a sequence of amino acids in a protein molecule. This idea advocated a simple linear relationship between DNA sequences and protein molecules. Due to the simplicity, the gene looks to have a *one-to-one* relationship.

\(^2\) In 1953, Crick did not know that the sequence of ribonucleic acid also carries genetic information in certain viruses, i.e. retroviruses.
relation to the DNA sequence. The identification of DNA with the gene itself implies that DNA molecules have a gene’s causal roles in the most straightforward way.

[The 1953 Gene]
For an organism to have a DNA sequence is for it to have the properties of {transmission, recombination, self-replication, mutation of heritable characteristics, and the manufacture of products for its development}.

In the early 1960s, François Jacob and Jacques Monod constructed a regulatory model of gene activation, the operon model, and showed that there are two different types of genes: ‘structural genes’ which are responsible for carrying the genetic information, and ‘regulatory genes’ which play a regulatory role in the expression of structural genes. The discovery of these two different types of genes immediately undermined the one-to-one relation between the gene type and the DNA sequence. It had to be decided whether the gene type refers only to the structural genes or to the structural genes plus regulatory genes as well.
In the 1970s, advances in DNA technology enabled working scientists to make many exciting discoveries. Findings of repeated genes, movable genes, nested genes, and polyprotein genes all challenged the one-to-one relation between the gene and the DNA sequence. The genes of rRNA are repeated in several tandem copies, thus contesting the idea that the units of transmission and transcription may not always be coextensive. Some genes’ have the ability to move in the chromosome from one location to another, refuting the idea that the locus of a gene is always fixed in the chromosome. The nested gene which resides within an intron of another gene testifies that genes are not located in a linear order on the chromosome. Polyprotein genes prove that each gene does not always and necessarily encode for a single polypeptide. Further, the discoveries of an open-reading-frame (ORF) and ‘upstream’ cis-regulatory elements provoked the question of how to delimit a gene: how far upstream or downstream from the structural DNA sequence should we trace to identify a gene? Since genes cannot be expressed without sequences that immediately or remotely regulate and encode all of the relevant transcription factors, and since these regulatory sequences are located in the genome, it becomes difficult to determine and measure every gene.
Where are we now? We find ourselves leaving behind the heyday of simplicity in genetics. Simple identification between the gene type and the DNA sequence was impressively uncomplicated and straightforward, but none of the DNA sequences showed a complete set of properties or causal specifications that it is believed the gene has: it cannot be true that a segment of DNA has all the causal roles that genes do. Identifying the gene type with particular DNA sequences and then reducing the gene to those sequences is a troubling conjecture.

3. Illusions of the Gene

Evelyn Fox Keller has proclaimed the demise of the gene concept. She writes:

Even though the message has yet to reach the popular press, to an increasingly large number of workers at the forefront of contemporary research, it seems evident that the primacy of the gene as the core explanatory concept of biological structure and function is more a feature of the twentieth century than it will be the twenty-first. What will take its place? Indeed, we might ask,
will biology ever again be able to offer an explanatory framework of comparable simplicity and allure?³

She argues that our conception of the gene as the ultimate explanatory factor of biological structure and function is obsolete. In addition to eliminating the gene concept, she tries to abolish many distinctions in biology - between genetic and epigenetic, and nucleus and cytoplasm, for example - and she also wants to jettison the association between genetic and active, and epigenetic and passive.⁴ She is skeptical about the concept of gene and intends to prove its illusory nature to the scientific community. Her ‘radical view’ will make sense only if we decide to let the gene succumb to the tremendous number of discoveries of anomalous phenomena which tend to decrease the usefulness of the concept. However, I believe, as many people still do,⁵ that an obituary for the gene is rather premature. No matter how vague and fuzzy its definition is, this does not prevent working scientists from

successfully communicating with one another about complex molecular phenomena. Difficulties in defining the gene do not entail the incommensurability of locutions using the concept, so there is no need to worry about the possibility that the gene might face the same fate as phlogiston. Like the concept of *species*, which involves a multitude of definitions, “it may not be important to know what the precise meaning of ‘gene’ is,” and the fuzzy concept would not have any problem playing its crucial role in biology for the future as well. So why should we now stop defining the gene?

4. The Gene as a Process

As molecular biology and its methodology advance and improve, the way molecular biologists think of the gene becomes dramatically different from the way non-molecular biologists do. Molecular biologists identify it with DNA sequences, while non-molecular biologists, including physiologists, embryologists, and evolutionists, insist on the view that the gene is something more than just a stretch of DNA molecules, something more than the entity coding for a single polypeptide chain. Holding firmly to Dobzhansky’s dictum, “Nothing in

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biology makes sense except in the light of evolution,”⁷ non-molecular biologists believe that the gene must not be an exception to the pervasive activities of natural selection. Traditionally, the gene has been treated as a ‘black box’, where the input is a pair of parental factors, and the output, via some simple rules of dominance, is the phenotype of the offspring. Utilizing the 1953 gene concept, above, molecular biologists attempt to open the ‘genetic black box’ by identifying it with DNA sequences. They think that the concept of the black box is nothing but DNA molecules which they believe contain necessary and sufficient information on the relevant phenotype. In contrast, non-molecular biologists contend that, although DNA sequences contain genetic information that is required for the development of the organism, such information is not sufficient to produce the relevant phenotype. According to them, to understand the gene and its function we have to go beyond molecular structures; the gene and its function have to be understood in light of the development of other structures and the evolution of several hereditary vehicles.

Paul Griffiths and Eva Nueman-Held argue that the gene is not to be identified with DNA sequences alone but with the whole range of

resources expressing a particular product, that is, the molecular process. They understand that the gene “denotes the recurring process that leads to the temporally and spatially regulated expression of a particular polypeptide product.”

According to Griffiths and Neumann-Held, what is inside the ‘genetic black box’ is not DNA sequences but complex molecular processes. They propose that the whole range of molecular developmental processes is something that is sufficient to bring about the development of organisms. Setting aside their original intention, regarding molecular process, I think we should notice their contributions to the inquiry on the concept of gene by focusing on the complexity of the development details. Doing so will lead us to shift our attention from the limited concept of molecular gene to an inclusive view of developmental process.

Griffiths and Neumann-Held explain their notion of molecular process in the course of an argument for ‘causal democracy’. They offer causal democracy to refute the view that genes have a special role in the development of organisms: “The role of the genes is no more unique than the role of many other factors, such as the role of the

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9 I will discuss their intention in the following paragraph.
 maternal cytoplasm.\textsuperscript{11} Genes need to be regarded as just one of many informational sources for organisms’ development. Griffiths and Neumann-Held cast off the privilege of genes and naively want to bestow on every actual (and possible) causal factor an equal standing in the molecular processes.

I am not sure how much they want to stretch the notions of causal factor and ‘molecular process,’ but they need to include a great number of things that constitute molecular processes: stretches of DNA sequences, their products, biosynthetic pathways, regulatory mechanisms, many factors of the organism’s environment, and so on. Isn’t this excessively inclusive? If it is, then what is it that they call the molecular process, anyway? To hold their ‘causal democracy’ thesis, removing the privileged role of genes from the gene concept and accommodating the complexity of developmental intricacies, they need a great number of actual and possible causal factors of a molecular process. But this turns their notion of molecular process itself into an empty one: it is so profligate and extended that all its explanatory adequacy and predictive power has been eviscerated. Furthermore, it becomes exceedingly difficult to individuate molecular processes. If they are not then demarcated, it is impossible to know whether

\textsuperscript{11} Griffiths and Gray, 1994, \textit{ibid.}, p. 277.
molecular processes have the properties of a gene, properties of transmission, recombination, self-replication, and mutation of heritable characteristics.\textsuperscript{12} I believe Griffiths’ and Neumann-Held’s approach is fatally damaging both to their own claim and to our understanding of genes. The price they have to pay is too high to keep their idea of causal democracy, even though they recognize correctly that an inclusive view of the developmental process must avoid the limited concept of molecular gene.

5. Reducing Genes

5.1 The Gene as a Second-Order Functional Property

I argue that we construe the gene as a second-order functional property. Like muscle contraction, meiosis, and transcription factors, which I discussed in previous chapters, the gene should be understood as a second-order functional property. Let us recall the definitions of a second-order property and a second-order \textit{functional} property:

\textsuperscript{12} Rosenberg claims “Griffiths and Neumann-Held’s proposal is another version of sheer eliminativism about genes. Though it retains the word, it gives up the kind that the word \textit{gene} is used to pick out.” See Rosenberg, 2006, \textit{Darwinian Reductionism Or, How to Stop Worrying And Love Molecular Biology}. p.124.
For something to have a second-order property $F$ (or to be an $F$) is, by definition, for it to have some property $G$ such that $S(G)$, where $S(G)$ states a specification that $G$ must meet.

Functional properties are a subset of second-order properties when they are specified by the causal relations they are embedded in:

For something to have a second-order functional property $F$ (or to be an $F$) is, by definition, for it to have one of its first-order properties specified in terms of its causal/nomic role.

With the definition of a second-order functional property in hand, let us try to define the gene:

To have (or to be) the gene is to have certain causal/nomic roles, such as transmission, recombination, self-replication, mutation of heritable characteristics, and the manufacture of products for development of an organism.

Recent scientific findings show that we should think of the gene with many different bases, for example, various DNA sequences, regulatory
DNA sequences, and their interactions, and these various bases perform the causal tasks that we believe the gene has. All this justifies construing the gene as a second-order functional property:

For something to be a gene is for it to have some first-order base property which is specified in terms of its causal/nomic role.

Or,

For something to be a gene is for it to have a variety of first-order base properties that perform the causal tasks that we believe the gene should have.

5.2 Structures of Genes and the Gene Token

Once the gene is regarded as a second-order functional property, the next step in my reduction model is to discover realizers of the gene. The realizers of the gene are, by definition, first-order base properties that perform the causal tasks of the gene. As we saw above, the realizers are ‘wildly heterogeneous’ and unmanageably disjunctive. A gene can be either the structural gene which is responsible for carrying the genetic message, or the structural gene plus the regulatory sequences which controls the expression of structural genes.
Alternatively, a gene can be a certain cluster of DNA molecules at one time but a different cluster of DNA molecules at another time. In a more dramatic expression of diversity, a gene can also be either a part of the genome or a whole genome. A variety of different DNA or RNA sequences are the realizers of the gene, and such diverse structures and properties make unattainable a general molecular definition of the gene. Due to the heterogeneity of realizers, it is impossible to individuate a gene as a single count noun “gene” and characterize the gene as a natural kind. The gene cannot be regarded as a natural kind or a type because of this heterogeneity of its realizers. Instead, we must think of a gene as ‘a gene token,’ meaning something that is a spatio-temporally distributed particular object. Rosenberg argued that a gene is a local phenomenon produced by natural selection working on local conditions. Since natural selection is blind to physically different structures for their own survival and replication as long as they have identical or even just similar effects, it is no surprise that even at the level of nucleic acids, many different structures would be selected to perform the (same) function of the gene. Owing to the persistence of variation, multiple realizability becomes visible as the mark of natural selection working in the local environment, and thus no neat, non-

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disjunctive natural kinds are found anywhere else in the biological realm. So the gene needs to be understood as gene tokens.

5.3 Localization and the Gene Expression Network

As Dobzhansky teaches us, genes, along with everything else biological, should be understood in light of development and evolution. We need to see that the gene is not a single DNA molecule or an isolated entity which carries a genetic message to its receiver. The gene is a dynamic entity that not only allows transmitting genetic messages but also is itself subjected to a vigorous process of evolution. If we adopt this dynamic view of the gene, we can easily see that some antireductionist arguments are wrong. For example, antireductionists argue that it is impossible to reduce a complex process to a single macromolecule, and they quickly conclude that reductionism has limits. But reductionism is not such a naïve theory that unintelligently tries to reduce or explain a complex phenomenon into or in terms of a single DNA molecule. The attempt to do so will be neither possible nor worth the effort. If we accept the dynamic view of the gene, we can immediately recognize that the arguments for its privileged role have been so far focused on one single DNA sequence and its phenotypic
outcome. This picture is fundamentally wrong and misleading; once we see how a gene operates in *dynamic gene networks*, a single DNA molecule can never be thought of as a lone player. Instead, we need to realize that the genetic player which has a causal role is not a single DNA molecule but the *gene expression network* which is composed of many molecules, their interactions and activities in a cluster of genes. Here, a DNA molecule plays its role as a part of the network. The restrictive view of the gene as identical to DNA molecules is nothing but an obstacle to our understanding of biological phenomena as a whole.\textsuperscript{14} Unfortunately, this kind of narrow approach has been prevalent in biological research and often serves as a justificatory factor for genetic determinism. This restrictive view of the gene and its consequent genetic determinism are both mistaken. Holland and Galas are also aware of this problem. They point out that the immune system has been studied for more than 100 years yet we still have only a partial understanding of its properties because up until recently immunologists have been able to study this complex system with only one gene or one protein at a time.\textsuperscript{15} I suggest, as Hood and Galas realize that we consider the scope of the gene as going beyond a single DNA molecule;

\begin{itemize}
\item \textsuperscript{14} This might appear to be like the antireductionist approach Griffiths and others take. However, the gene expression network exploits Griffiths’ notion of process to reduce the gene and consequently vindicate reductionism.
\item \textsuperscript{15} Hood and Galas, 2003, “The digital code of DNA” *Nature* 421:444-448.
\end{itemize}
the gene must be understood as a dynamic gene expression network composed of many molecules and their interactions and activities in a cluster of genes.

Take for example homeotic genes. Homeotic genes are clustered homeobox (TATTA box)-containing genes present in all metazoans, and they have an evolutionarily conserved function of conferring positional identity along the axis of the main body. The function of homeotic genes is considered crucial in the early development of metazoans, especially during the period of segmental formation. Because of the presence of the homeobox in a whole range of genes controlling development in metazoans, a great number of different genes began to be identified as belonging to this family. In the fruit fly *Drosophila*, the homeotic genes *bithorax complex* control the individual identity of segments from the posterior thorax and the abdomen. The homeotic genes *Antennapedia complex*, in contrast, regulate the segmental formation of the anterior part of thorax. Many other homeotic genes, including *engrailed, deformed, fushi tarazu*, and *Sex combs reduced*, also play key regulatory roles in the development of segmentation in *Drosophila*. Homeotic genes are multiple genes and perform different roles in a wide range of development. This can be illustrated as follows:

The homeotic gene is realized in heterogeneously diverse genes, and each gene has a causal role that is responsible for the early development of organisms such as the formation of segmental patterns. Many of the homeotic genes’ products function as transcription factors by recognizing sequence-specific binding sites, i.e., the homeodomain, the region of the protein coded by the homeobox, and by regulating the activities of other genes. It is possible that diverse functions with different homeotic genes are acquired through internal changes, their activity as transcription factors on downstream targets, and through interactions between the genes and other proteins. This means that a huge array of different control factors regulates the activity of homeotic genes, and complex control elements modulate their function and roles. When we examine homeotic genes, we find that their expressions are strongly and precisely regulated by a highly conserved gene network composed of many DNA sequences, many proteins, and interactions between sequence-specific proteins and DNA sequences.
Consider *Drosophila*’s homeotic gene *Sex combs reduced* (Scr). *Scr* encodes a sequence-specific transcription factor that controls, in concert with other gene products, the segmental identity of the labial and prothoracic segments in the embryo and the adult. During embryogenesis, the product of *Scr* accumulates in a discrete spatio-temporal pattern that includes the labial and prothoracic ectoderm, the subesophageal ganglion of the ventral nerve cord, and the visceral mesoderm of the anterior and posterior midgut. Gindhart-Jr., *et al.*, show that a regulatory network containing *Scr* sequences and other homeotic segmentation genes, including *fushi tarazu*, determine the activity of *Scr* during embryogenesis.\(^\text{16}\) This implies that the function and activity of *Scr* cannot be explained without considering the gene expression network since *Scr* plays a role in the genetic regulatory network and is controlled by it.

The activity of a gene depends upon the gene expression network where it is embedded. In their “Developmental Gene Network Analysis” Roger Revilla-i-Domingo and Eric H. Davidson testify that the network approach has already begun in the scientific community, especially in the field of developmental biology. They believe that the information

processing functions executed by the cis-elements regulating the expression of the participating genes control the developmental process. According to Revilla-i-Domingo and Davidson, the project of developmental biology is to unravel the developmental gene network to provide insights into how the programs for development work and how they evolve.\textsuperscript{17} Schroeder, \textit{et al.}, also report that the \textit{Drosophila} homeotic gene \textit{odd skipped} is regulated by a segmentation gene network consisting of maternal and zygotic genes. They suggest that since a gene’s activity is context-dependent, or network-dependent, it is possible to switch the status of a gene’s activity from a repressive function to an activating function by manipulating the gene network.\textsuperscript{18} The gene network approach can also be found in a study of sea urchin embryos. Eric H. Davidson, \textit{et al.}, report:

\begin{quote}
Development of the body plan is controlled by large networks of regulatory genes. A gene regulatory network controls the specification of endoderm and mesoderm in the sea urchin embryo. ... The network contains over 40 genes at present, and each node can be directly verified at the DNA sequence level by
\end{quote}


cis-regulatory analysis. Its architecture reveals specific and general aspects of development, such as how given cells generate their ordained fates in the embryo and why the process moves inexorably forward in developmental time.¹⁹

These researchers try to explain the development of sea urchins with the concept of a gene (regulatory) network. They think that gene networks secure dynamic processes of genetic digital information to produce phenotypic outcomes and ultimately control the development of this creature.

Let us go back to my reduction model and see how it fares with this new view of the gene. I have so far argued that we need to construe the concept of gene as a second-order functional property. We discovered that a gene is realized in multiple genomic realizers. I have also stressed that a gene is not just a DNA molecule or sequence but must be considered in its gene expression network which is composed of a set of many DNA molecules whose state of activity is connected by sequence-specific cis- and trans- regulatory interactions. The gene expression network does not solely rely on the gene sequence. Instead,

it is determined by DNA molecules, their interactions, and their products. The characteristics of the predicted gene relationships depend on the underlying networks, and networks concern biological processes rather than molecular functions. In a gene expression network each realizer and its activity are to be understood in the context of that network. Absent consideration of the gene expression network, there can be no satisfactory explanation of the activity and function of each realizer of homeotic genes, for example. In other words, the function of each realizer of homeotic genes is dependent on its gene expression network. Therefore, as I have claimed above, if we construe homeotic genes as second-order functional properties, we can see here that they are realized in, or are reduced/identical to, specific gene expression networks that are their first-order base properties. A gene *is* in fact the gene expression network where it is realized on a given occasion: the gene is reduced to, or is identical with, its realizer that is constituted of DNA sequences, their products, their interactions, and others. The gene token is on this occasion identical with its gene expression network token.20

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20 The gene expression network is ‘open textured’ enough to add new information as it comes in.
This new concept of the gene as a gene expression network is compatible with, and thus helps us see more clearly that what is conserved in the course of evolution is not the gene that we have thought of so far, but is in fact the gene expression network. Accordingly, this new approach promotes the view that the carrier unit of inheritance is not really a gene: it is a gene expression network of genes. Scott Gilbert, a developmental biologist, also expresses a similar view:

The inheritance is not of a gene but of a regulated network of genes and the binding regions for their product. The interesting questions of evolutionary biology will involve how these pathways were modified to bring about the formation of new cell types and new body plans during the development of life on earth.\(^{21}\)

The notion of gene expression network is a key to understanding how genotype determines phenotype. In this new approach, ‘genotype’ does not have to mean only a set of DNA molecules; it means a set of

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gene expression networks. The flow of a genetic message is stored and secured in the gene expression network, and cells’ response to external/environmental conditions is controlled by it. Through coordinating and regulating the expression of gene products, the network performs specific functions. Gene expression networks must be the most fundamental source of developmental programming and they are what provide us with the best explanation for organisms’ development. Also, this notion must be accepted as a crucial factor in explaining the course of evolution. Hood and Gallas observe that the networks can change significantly in short periods of evolutionary time, such as during the Cambrian explosion of metazoan organisms when the huge diversity of body plans emerged over perhaps 10–30 million years. Evidently, the activity of the gene networks is responsible for the body plans. Hood and Gallas also say that remarkable changes in the gene networks caused the development of the human brain about 6 million years ago during its divergence from the common ancestor humans share with chimpanzees.\(^\text{22}\) I conclude that the notion of gene network explains not only the development of organisms but also evolutionary processes.

I have claimed that the gene, as a second-order functional property, must be understood as gene expression network-specific: the gene, when it is realized on a given occasion, is reduced to, and is identical with, its realizer that is constituted of a cluster of DNA sequences and their activities. As a functional property, the gene is reduced to one of its genomic realizers on a given occasion, that is, in the gene expression network. My proposal provides a new dynamic approach to the concept of the gene by exploiting the antireductionist idea of molecular process. Reducing the gene is possible in my model of localized functional reduction. Reductionism is vindicated.
Figure 2: Diagram of ‘Level’ and ‘Order’

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<th>Second-order Properties</th>
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<tr>
<td>Micro-properties at level $i-1$</td>
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Figure 3: Diagrammatic Breakdown of a Typical Muscle

Figure 3 shows how actin thin filaments and myosin thick filaments are arranged to form the myofilaments of a sarcomere.

http://www.med.unibs.it/~marchesi/muscle.html
**Figure 4: Transcription Factors**

**A. The homeodomain transcription factors**

The homeodomain structure has a helix-turn-helix that is made up with 60 amino acids. The third helix extended from the homeodomain binds to the major groove of the DNA, and amino acids in the amino-terminal portion of the homeodomain also contact the bases in the minor groove of DNA.

**B. The basic helix-loop-helix transcription factors**

The basic helix-loop-helix (bHLH) is a domain of basic amino acids (typically 10 to 13 residues) that precedes the first α-helix binds to the promoter or enhancer site of DNA sequence.
**C. The bZip transcription factors**

The basic leucine zipper (bZip) transcription proteins are dimmers. Each subunit of the dimmers has a basic DNA-binding domain that is followed closely by a helix containing several leucine residues. These leucines in a helix interact with other leucine residues on the other subunit, and hence the interaction forms a ‘leucine zipper’ between the two subunits. It results in dimmers. The leucine zipper domain interacts with the promoter to stimulate or repress transcription of certain genes.

**D. The zinc finger transcription factors**

The zinc finger transcription factor proteins have two or more ‘DNA-binding fingers,’ that is, the looping out of the ‘zinc fingers’ in that cysteine and histidine amino acids hold a zinc atom.

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Figure 5: Stages of Meiosis

The Visual Dictionary http://www.infovisual.info/
**Step 1: Localized Functional Reduction**
(Reduction of Intra-level Properties)

*Horizontal process*

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**Step 2: Mereological Reduction**
(Reduction of Inter-level Properties)

*Vertical Process*

- Level i: Structural Properties
- Level i-1: Structural Properties and Relations
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