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Reproduction and the Reduction of Genetics

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ABSTRACT

In this essay I develop a new, unified perspective on genetics, development, and reproduction. I suggest a heuristic use of theory reduction to address an issue of contemporary theoretical importance: the framing of a theory of developmental units. I claim that the gene concept, properly understood, *is* a concept of developmental unit and suggest that the historiography of genetics should reflect this fact. There is no denying that genetics has been a successful science. If its relation to development could be adequately expressed, genetic theory might also provide clues to a theory of development. Conventionally, the mechanisms of development are expected to be explained in terms of mechanisms of genetics. Development is treated as an epigenetic process and, if theory reduction is possible, a theory of development is expected to reduce to a general theory of genetics. This expected direction of reduction from development to genetics depends on the conventional understanding of genetics and its relations. I argue for a reversal of this expectation by reconceptualizing genetics and development as fields describing aspects of the process of reproduction. The relation of these aspects is not one of simple parts to a whole. They are deeply entwined, as my analysis of reproduction will show. Once certain features of scientific reduction are identified, the new perspective can be used to pursue reductionism heuristically, to use what we know about the theoretical units of genetics to speculate about units of a general theory of development. Thus, reductionism may be scientifically useful even though the conditions for formal reduction are not met.

THE PROCESS OF REPRODUCTION

I urge a change of perspective on genetics and gene concepts. The fundamental entities of biology are processes rather than structures or functions. Genetics is about genetic processes. Development is

about developmental processes. Whether, when, and how entities of a particular structural or functional kind serve such processes are empirical questions. Whether there are genes besides the nucleic acid molecular kind (structure) or besides the replicator kind (function) are empirical questions requiring an ontology of biological processes for answers. Various kinds of objects might serve in genetic units of heredity or development. The functions that are carried by such structures when they serve a process of reproduction are determined by the relation between heredity and development in the process: Functions need not be invariant to changes of structure and structures need not be invariant to changes of function in order to serve reproduction. Thus, necessary and sufficient conditions on structure or function are bound to fail as definitions for units of process.

In a process view of evolution, relations among genetic and developmental *processes* may be distinguished from relations among *objects* serving these processes. In the traditionally interpreted structural hierarchy "biological individuals" serving developmental processes such as cells and organisms are thought to be at a higher level of organization than objects serving genetic processes such as genes. In the process perspective I endorse, developmental processes are more fundamental: Genetic processes contain developmental processes, so development is not *epi*genetic and hierarchical structural order is not equated with processual order.

The prospects for reductionism and mechanistic explanation are altered in the process perspective. We need not reject theory reduction as an alternative philosophy to mechanism (see Brandon 1996, chapter 11) if theories are about processes while mechanisms are function-bearing, structured things that cause behaviors (see Glennan 1996) that we assign to processes.[2] We can use what we know about structures and functions of mechanisms in the special case of genetic processes as heuristic constraints to construct theories of the general case of reproduction processes.

Genetic processes can be treated as special cases of reproduction processes that contain developmental processes. Thus, genetic processes contain development. The direction of reduction is changed from the conventional expectation because the special theory of genetic processes should reduce to a general theory of reproduction that explicitly incorporates development. Reduction of special case

theories to more general theories is a central feature of the traditional view of theory reduction (Nagel 1961; Sarkar 1998). My use of theory reduction is anything but traditional, however. I distinguish between the *image* of a reduction model, meaning the expected direction of reduction between scientific realms and thus which realm is to count as more fundamental, and the *mapping relations* that connect items across realms.[3] Reversal of the biological image of reduction from development to genetics can drive a reductionistic research program to establish new mapping relations by working at the level of a general theory of reproduction without already having in hand an articulate, formal theory of development. This reductionism is a scientific research strategy, not an *a posteriori* philosophical analysis (Wimsatt 1976). To pursue this line of thought, I will first give a general characterization of biological reproduction and then suggest more precisely how genetic processes are special cases of reproduction processes. Once done, the heuristic program and its historiographic implications for the gene concept can be sketched.

What Is Replication?

I do not think that the most general philosophical treatments of evolutionary theory to date have interpreted genetic processes adequately. Dawkins (1976, 1982) and Hull (1980, 1981, 1988), for example, invoke "copying" and "replication" in their definitions of the "replicator," but fail to give satisfactory accounts of these processes. Others invoke properties such as heritability in their accounts of units of selection, but are not clear about the ontological status of such properties and their relationship to the processes that carry them. For example, heritability has been claimed to be a necessary property of a unit of selection (e.g. Lewontin 1970). Heritability is not a sufficient expression for an inheritance *process* because heritability expresses a capacity, not a process.[4]

My purpose in analyzing reproduction as a process is to formulate an alternative to the notion of replicator. The replicator concept appeals to certain examples of replication as definitive. This procedure threatens circularity: A replicator is "an entity that passes on its structure largely intact in successive replications" (Hull 1988, 408). But what is replication? It is tempting to say either that replication is

the process by which replicators pass on their structure or simply refer the reader to a molecular genetics textbook, implying that replication is anything "sufficiently like" nucleic acid replication. The failure to analyze process is widespread in the units of selection literature and reflects the fact that analysts of the structuralist perspective have often been more interested in units that map neatly onto hierarchies of structural organization than in tracing the ontological implications for evolution as a process. Those following the functionalist perspective of G. C. Williams (1966) as well as Dawkins and Hull have returned the emphasis to process, but in a way that has tended to exclude developmental processes as central to the integration of replication and interaction by selection. The exclusion is symptomatic of strategies of theory generalization by abstraction. One cannot appeal to exemplars of replication (e.g., DNA) that have been identified in terms of the structural hierarchy and be confident that their properties are necessary and sufficient for replicators in general without a general analysis of the replication process.

Progeneration: Multiplication with Material Overlap

Reproduction is a process with two aspects: *progeneration* and *development*.[5] Progeneration is the multiplication of entities with *material overlap* of old (parent) and new (offspring) entities. Material overlap means that some of the parts of the offspring were once parts of the parents. If you cut a loaf of bread in half, you progenerate it, making two new things out of one old one; the parts of the new half-loaves were formerly parts of the old loaf. If a cell reproduces by fission, it divides into two new cells, all of whose parts used to be parts of the parent. Reproduction by fission involves progeneration. Progeneration is distinguished from *copying* and *multiplication* by its requirement of material overlap.[6]

The term *progeneration* evokes an impression of self-motion or self-reproduction. Progenerating organisms are agents of reproduction, not merely material or efficient causes. They may be called *reproducers*. Bread loaves must be cut in pieces by an agent with a knife while cells divide *themselves* in reproduction. The contrast between agent and patient should not be taken seriously without a precise

notion of biological agency, but whatever that notion turns out to be, my concern is with reproduction by biological agents.[7]

Progeneration is universal in biological reproduction. We can, of course, imagine that DNA replication might have been fully conservative rather than semiconservative. Furthermore, it is not typical for modern DNA strands to be transmitted to new units of reproduction or to play a role in gene expression without the material overlap of more inclusive entities, the cells. So even if it turned out that DNA replication per se did not satisfy the material overlap condition for biological reproduction, DNA replication would be part of, rather than a kind of, reproduction. What about single stranded RNA viruses? They do not materially overlap their daughter strands except by accidental nucleotide incorporation. Does viral "replication" count as reproduction? No. Viruses replicate only by virtue of the development of their hosts. Viral nucleic acid replication is best understood as a kind of process in terms of virus development, which is part of host development, reproduction and genetics.

The distinction between replication and reproduction and between their associated units comes down to the role of development in the respective processes: The replicator is a concept abstracted from development while the reproducer is a developmental concept. If there is something we would count as a case of biological reproduction that does not involve material overlap, I suggest that is because we have abstracted the concept of reproduction from its developmental aspects and thus mistakenly identified replication with copying rather than with reproduction.

If DNA replication might not have been reproduction, if viral "replication" is not reproduction, then is the material overlap distinction between progeneration and copying mere semantic hairsplitting? If the analysis of reproduction only added precision to the use of words like *replication, copying,* and *reproduction,* perhaps the philological effort would not be worth the scientific gain. However, if the evolutionary dynamics of the flow of matter differ from the dynamics of the flow of information or structural pattern, then there may well be a scientific significance to the distinction.[8] Indeed, why *is* DNA replication semiconservative while viruses can get away without it? If there is an evolutionary cost of material overlap, what evolutionary benefit could counterbalance that cost? One possibility

concerns transmission fidelity. Chemically bonded or bounded matter is a way to "transmit structure largely intact" without having to depend too much on the contingent stability of the environment. In general, information flow is reliable only to the extent that the channel conditions supply stability to messages. If fidelity is low, replication rate must be high if selection is to operate effectively (Wimsatt 1980, 245). Material overlap may buffer information in fidelity-limiting environments.

Besides distinguishing copying from progeneration in terms of material overlap, it is critical to be clear about what types of progenerating entities count as reproducing entities. That clarification is a problem for a general theory of development. Roughly, when we assert that some thing has reproduced, we mean that that thing has produced another thing of a certain type or kind. However, cutting a loaf of bread in half is not reproduction, no matter how much the half loaves resemble the whole "parent" loaf, so the relevant kind is a very special property. To count as biological reproduction, the half loaves would have to be capable of indefinite progeneration. The half loaves can certainly be cut again, but there comes a point when it is impossible to divide the tiny cut pieces into things that are still bread, let alone loaves. Even if matter turns out to be infinitely divisible, a point will undoubtedly be reached when no physical process divides the matter further. The capacity for indefinite possibility of progeneration (i.e., that a lineage of progenerants could be indefinitely long) sounds similar to Dawkins' account of replicators as things with sufficient longevity, fecundity, and fidelity to be the units of selection and the beneficiaries of evolution (Dawkins 1976). However, in process perspective the replicator can be understood to be a very special case of something much more general, the reproducer.[9]

Development: Acquisition of the Capacity to Reproduce

In general, the type of thing that is reproduced by progeneration is the reproducing type. It is not the case that resemblance in just any respect (i.e., having any property in common) is sufficient to turn cases of progeneration into cases of reproduction. Progeneration must result in more things that can reproduce for that progeneration process to count as an aspect of a reproduction process. In other

words, there is exactly one property in which parents and offspring *must* be correlated for their progeneration to count as reproduction: the capacity to reproduce. The production of offspring that do not have the capacity to reproduce is not reproduction, but only progeneration.

Typically, the capacity to reproduce must be *acquired* or built-up; things are not born with it. Even cleavage cells in an early embryo that do not take up nutrients or synthesize new RNA must go through some internal rearrangement in order to successfully divide again. At the very least, they must move chromosomes to the metaphase plate and pull them apart in order to divide in a way that yields a reproductively capable offspring cell. Things can be born with the capacity to progenerate, but if they are to be reproductive, they must also be born with the capacity to develop, that is, with the capacity to acquire the capacity to reproduce.

Evolutionary theory requires, minimally, that reproductive capacity be "transmitted" from generation to generation. The means for this have typically been the transfer, in material overlap, of particular *mechanisms* that confer the capacity to develop the capacity to reproduce. It is not essential that an offspring develop or reproduce in the same way as its parents in order that the essential property – the capacity to reproduce – be instantiated in offspring. Some insect species alternate between generations of winged and nonwinged forms, so offspring are rather unlike parents in those structures and functions. Haploid male hemiptera do not resemble their female parent in a number of respects. Moss and fern gametophytes do not resemble their sporophyte parents in many respects that are essential to sporophyte development. Successful academics do not always produce academic offspring (if they have offspring at all), nor do nonacademic parents always succeed in keeping their children out of the ivory tower. These examples dress in processualist clothes the familiar point that heritability of complex, adaptive traits or parts is rarely perfect. The more fundamental point is that heritability is a developmental concept because reproduction must transfer "pieces of development" from parent to offspring, i.e., parts conferring the capacity to develop.[10]

Critiques of heritability as a concept of genetics (e.g., Lewontin 1974a; Sarkar 1998) often point out that heritability is a phenotypic

concept. That critique relies on the partition of structures into phenotype and genotype. From the process perspective, heritability is a developmental concept. However, even from this perspective there is no full partitioning of development and progeneration except by imposing an explicit model of structural organization in which reproduction can be interpreted as operating at a single level of structural organization. The reason for this difference between structuralism and processualism has to do with the recursive relation of progeneration and development in reproduction.

To count as reproduction, a progeneration process must result in entities with the capacity to reproduce. The acquisition of the capacity to reproduce is the process of development.[11] Since development is the acquisition of the capacity to reproduce, we can say that reproduction is progeneration of entities with the capacity to develop the capacity to reproduce. This analysis of reproduction is therefore recursive: Entities with the capacity to develop the capacity to reproduce are entities with the capacity to develop the capacity to develop the capacity to reproduce, and so on. The recursion stops or "bottoms out" when something is progenerated which has the capacity to reproduce and does not need to acquire it through development. This automatic capacity to reproduce may be called *null development*.

Genetics in a Process Perspective

The analysis shows that development is a critical component of reproduction, equal in significance to the progeneration of new individuals, and indicates what is essential to parent-offspring resemblance. Reproduction is neither mere multiplication of individuals nor mere copying. It requires that multiplication involve material overlap and it bounds the specification of development. The analysis can be put to use to reverse the direction of the image expressing the expectation that a theory of development reduces (in principle) to a theory of genetics and to show how, in process perspective, appeal to mechanisms of genetics can explain developmental phenomena. To do so, we first need to reinterpret genetics in a process perspective.[12]

The analysis of reproduction subsumes two special cases: inheritance processes and replication processes. *Inheritance processes* are the subject of classical genetics, in which no special knowledge of the mechanistic basis for particular developmental mechanisms causing reproductive capacity is required. Mendelian genetics is a special case theory of inheritance in which certain developmental mechanics are controlled by experimental design. Mendel expressed his concern for such cases when he characterized his theory as one of the "development of hybrids" (Mendel 1866 [1965], 21). *Replication processes* are the subject of classical molecular genetics, which assumes special knowledge of the Watson-Crick coding mechanisms. Genetics is the field centrally concerned with both of these types of process, so I will call inheritance and replication types of *genetic processes.*

Replication processes can be interpreted as special cases of inheritance processes, which in turn are special cases of reproduction processes. Since reproduction involves both progeneration and development, genetic processes also involve these. Because genetic processes are special kinds of reproduction process, and if theory reduction is possible, theories of genetic processes should reduce to the theory of reproduction processes in the sense that special case theories should be derivable from general theories.

It is important to recognize that, from the conventional standpoint of Weismannism, progeneration and development are logically separable component processes that can be studied in isolation from one another. Progeneration is the physical process described in terms of hereditary character "transmission" in genetic models. Although transmission sounds like a process, its treatment in genetics and evolutionary theory is in terms of an abstraction: the genotype-to-genotype or phenotype-to-phenotype mapping among generations (Lewontin 1974b, 1992). Development is the process represented by the genotype-to-phenotype map within generations. Thus, the traditional Weismannist separation of germ and soma, that provides a cytological basis for Mendel's factor-character distinction and Johannsen's genotype-phenotype distinction, seems untouched conceptually by the analysis of reproduction. But this is not so because progeneration and development are recursively entwined. There is no progeneration without development and no development without progeneration, except in the special case of null-development. Weismannism demands idealizations and abstractions in *both* the scientific domains of genetics and developmental biology, in which developmental aspects of heredity are ignored by geneticists and in which hereditary aspects of development are ignored by developmental biologists. These idealizations and abstractions must be recognized precisely for what they are – heuristics of scientific research – in order to go beyond them to a general, unifying theory of heredity/development that does not limit the science of genetics to classical Mendelian or molecular genetics.[13]

Genetics is distinguished from other fields concerned with reproduction by its particular interest in problems of heredity. Heredity is a *relation* of similarity between parents and offspring that is caused by reproduction processes. Geneticists measure the *degree* of heredity relations in terms of trait correlations among relatives. A trait is any measurable property of parents and offspring. Trait correlation is a measure of the association between trait "values" of the relatives being compared. Traits are classes of trait values. We speak of height as a trait. Being two meters tall is a value of the trait. Heritability, as noted above, is the quantitatively expressed *capacity* to cause relations of heredity that is carried by reproduction processes. The *measures* that are called narrow-sense and broad-sense heritability in the technical literature of quantitative genetics are statistics on populations of individuals that carry the heritability capacity. Because there must be a correlation in the capacity to develop for a process to be a reproduction process, heritability, as noted above, is a developmental concept.

Reproduction processes are the cause of genealogical relations. *Genetic processes*, specifically inheritance and replication processes, are specially structured reproduction processes. The field of genetics as it has developed historically is concerned with reproduction processes in which heredity relations are caused in particular ways. Some genetic problems do not require a detailed knowledge or representation of the mechanisms that cause heredity, but only with measuring the degree of the relation. Darwinian evolution before Mendel could do with a simple inequality: Offspring resemble their parents more (on average) than they resemble other members of the population into which they are born. Classical biometry was con-

cerned to measure empirical correlations among relatives, although the biometricians took varying positions on what could be learned of the "underlying" mechanisms from these empirical values. More recent genetics addresses the chromosomal and molecular mechanisms of heredity and the statistical consequences of their operation among populations of hereditary units.

Despite the variety of historically changing problems, interests, and theoretical needs of genetics, the nature of genetic processes can be characterized simply in terms of reproduction processes. Reproduction processes cause heredity relations as a result of the entwined effects of progeneration and development. *Inheritance processes* are reproduction processes in which there are *evolved* causal mechanisms "for" producing heredity relations in development in particular respects and degrees. Development is the acquisition of the capacity to reproduce. Acquiring a capacity for something usually requires constructing a mechanism – a complex system of component parts – to generate it, so the process of development can be understood as constructing mechanisms for particular hereditary relations in reproductive capacity. *Replication processes* are inheritance processes with a special kind of evolved causal mechanism, namely *coding* mechanisms.[14] Coding mechanisms, like other evolved mechanisms of development, produce heredity in particular respects and degrees; they may also be for the *control* of the degree of heredity relations.

Many molecular geneticists study replication processes in the special sense defined here, although they would characterize their work in structural or functional terms. Other geneticists study nonreplicative inheritance processes such as the so-called epigenetic inheritance systems or EISs (Jablonka and Lamb 1995). Some evolutionary biologists study group reproduction (Wade and Griesemer 1998), although it is not clear to what extent interdemic reproduction processes are genetic (see Griesemer and Wade, 2000). EISs involve inheritance mechanisms that are probably not coding mechanisms (or at least not precise coding mechanisms compared to the nucleic acid system). Interdemic reproduction may or may not include evolved mechanisms of group development, but this way of thinking about group reproduction gives new meaning to Van Valen's aphorism that evolution is the control of development by ecology.

The cytosine methylation system is perhaps the EIS closest to a

genetic coding system known (Jablonka and Lamb 1995, chapter 4). It is dependent on nucleic acid coding in the sense that cytosine methylation codes gene activation states in terms of the degree of methylation of cytosine residues in DNA sequences: More methylation means lower gene activity. The relation is quantitative, but appears not to be exact. Methylation states have been shown to be transmissible in mitosis and in some cases in meiosis for up to a few tens of generations, providing a possible mechanism for *Dauermodifikationen.*

Other EISs include structural inheritance systems such as cortical inheritance in paramecia in which surgical rearrangements of cilia patterns have been shown to be transmitted from parent to offspring through many generations. Heredity of these patterns can be precise and quantitative, but there is no *coded* information in the sense that: (1) the information is not about something other than the cortical pattern itself and, (2) is not specified by a combinatorial set of elements that is small relative to the size of the sequences coded for.[15]

Still less coded (or "digital" or "unlimited" [see Szathmáry 1994]), are metabolic steady state systems of inheritance. When cells reproduce by fission, they divide cytoplasm into two not necessarily equal parts. Cytoplasm is the substance of much of the metabolic machinery of the cell. The division of cytoplasm can preserve structure and hence cause correlations between parent and offspring in that structure. Because these EISs typically depend on chemical diffusion and molecular transport processes to equalize metabolic patterning throughout the cell, they may be of lower fidelity than the nucleic acid coding system. But just because a mechanism is not a coding mechanism does not mean that it is utterly incapable of causing heredity relations. Heredity, like correlation, is a relation that ranges in degree from –1 to +1.

The reproduction of demes by the progeneration and development of propagules (composed of one to many organisms) that colonize new habitat, may, in some ecological contexts, be another sort of steady state inheritance system, albeit at a much higher level of spatial organization than metabolic steady state systems composed of autocatalytic chemical cycles within cells. Group or deme reproduction involves sampling organisms from a parent deme, i.e., deme progeneration, and constructing new offspring demes from it, i.e.,

deme development, resulting in parent deme – offspring deme correlations and measurable group heritabilities, g^2 (McCauley and Wade 1980; Slatkin 1981; Wade and Griesemer 1998; Wade and McCauley 1980).

This kind of sampling process, whether in nature or in the laboratory, satisfies the analysis of reproduction given above. Parent demes progenerate by material overlap of organisms as parts. Offspring flour beetle demes studied in the laboratory must acquire the capacity to reproduce by means of an appropriate balance of organismal processes of development, reproduction, cannibalism, medium-poisoning, and so forth resulting in persistence on an environment (i.e., experimenter) imposed demic generation time.[16] The recursive nature of the analysis of reproduction expresses the fact that group reproduction depends on organism reproduction as part of group development, not merely because groups can only reproduce because organisms reproduce. Group reproduction is more than group subdivision, just as organismal reproduction is more than organismal fission. From the process perspective, controversy over inter-deme selection can be understood to concern whether certain systems, such as the flour beetle laboratory system, model inheritance systems with evolved mechanisms of development for particular heredity relations at the group level, or are only artificially constructed group progeneration systems with little prospect for group level evolution of mechanisms of group development.

Material overlap per se is the material basis for all mechanistic theories of genetics that explain how and in what respects and degrees offspring resemble their parents. The issue is no different in its general aspects whether the structural level is alleles transferred by mitosis to offspring cells or organisms transferred in group reproduction to offspring demes. The mechanisms as well as the population and evolutionary consequences may be quite different, but they are instances of the same kind of process.

Geneticists may complain that this characterization of heredity is too broad to be of any scientific use. Geneticists typically are not interested in low-longevity, unstable, low-fidelity inheritance systems. But lo-fi inheritance systems were probably important before the evolution of more precise mechanisms of inheritance (Dyson

1985; Gánti 1971). They may be involved in major evolutionary transitions to new levels of organization (Maynard Smith and Szathmáry 1995). They may also play a role in evolutionary dynamics now through interaction between epigenetic and replication processes (Jablonka and Lamb 1995). Heredity among reproducers emerging at new levels of organization created in major evolutionary transitions need only be of sufficient fidelity to drive evolution of mechanisms for development that stabilize them to disintegration from below, due to the competing genetic "interests" of their constituents. Thus, evolutionary transition may be characterized as the origin and evolution of new levels of reproduction rather than replication (see also Szathmáry and Maynard Smith 1997).

In general, a reproduction process in which there is a material overlap of mechanisms for development is what is minimally required for an inheritance process. Reproduction transfers from parents to offspring the mechanisms that cause heredity relations to arise in development. A theory of replication processes must interpret these as well as the structure and function of the genetic code abstracted from its material embodiment in physical processes. Differently put, a theory of genetic processes cannot merely be a theory of genetic structure or relations. The transmission of the genetic code itself depends on transfer of a set of aminoacyl-tRNAs and the rest of the translation machinery in cell division, not just the transfer of genes. General theories of genetic transmission cannot take this for granted, or else the origin and evolution of genetic systems cannot be explained.

A HEURISTIC USE OF PHILOSOPHY

On the Scientific Utility of History and Philosophy of Science

The perspective on reproduction formulated in the previous section suggests a scientific research program for constructing an integrated theory of heredity, development, and evolution that accounts for the way heredity and development are entwined in reproduction processes. Populations of genealogically connected, varying reproduction processes are the material basis of evolutionary processes. Exist-

ing accounts of heredity and development each tend to "black box" the other: Development is ignored in transmission genetics and transmission is ignored in the development of the phenotype. The entwinement of these processes, made clear by the analysis of reproduction in the process perspective, might serve as a conceptual scaffold for constructing an integrated theory without these black boxes. However, there is a problem: There are well-developed theories of genetics, but there are no comparably developed theories of development. So how can the integrated perspective help us if there are no corresponding theories to integrate? We must seek a theory of development comparable in scope, detail, and power to existing theories of genetics.[17] The theory we seek should not, however, be constructed in isolation from genetic theories if it is to contribute to a theory of reproduction that can integrate phenomena of heredity, development, and evolution.

Developmental genetics, of course, aims to explain developmental phenomena in terms of the behavior of genetic mechanisms. However, my theoretical project is not simply to theorize developmental genetics. I propose an expanded view of genetics from a process perspective that transgresses the boundaries of function and structure delimited by the tradition of genetic research in the twentieth century. Nevertheless, it would be pointless to ignore tradition and march off in an entirely new direction because the resulting theory would not bear significantly on the large body of empirical work and thus would integrate nothing. We seek a theory of development in the process perspective that is "backward compatible" with the scientific research programs, if not the theories, of today and yesterday.

Historians and philosophers of science have spent the last fifty years studying how scientists construct, test, and replace theories and how scientific methods and products are best classified and interpreted. History may thus provide conceptual and empirical resources for constructing new scientific theories. Philosophy may suggest formal strategies for interpreting these new constructs in terms of familiar ones. In this section, I suggest such a strategy for constructing a theory of development by arguing for a heuristic, scientific use of philosophical analyses of scientific reductionism. In the next section, I suggest that some clues to the units of development may be found in the history of genetics.

Constructing a Theory of Development by Heuristic Application of Reductionism

Reductionism is a complex subject. Its analyses and varieties are too numerous to survey here (Sarkar 1998). Some scientists characterize their research as reductionistic, though it is not always clear what they mean (Brandon 1996, chapter 11; Wimsatt 1976). Philosophers of science have long interpreted relationships among theories, laws, models, fields, properties, and phenomena in terms of formal and substantive accounts of reduction. There is an important contrast between scientists' descriptions of scientific research strategies or heuristics and philosophers' descriptions of formal and informal criteria for what is to count as a reduction. Scientists have sometimes drawn on philosophical work to characterize their activity, for example, when they endorse Popper's view of scientific method as hypothesis-testing and falsification rather than induction and verification as the best description of their work. Similarly, philosophical work on reductionism can be put to use scientifically. I am not merely noting, with philosophers since Nagel (1961), that while formal analyses of reduction do not fully describe scientific norms and practices some broader account of reduction would. I am not aiming to describe scientific practice at all. Rather, I propose a way to do science that makes use of philosophical ideas about the nature of reductionism. In other words, I propose to put philosophical ideas about reduction into scientific practice, as heuristic guides to theory construction.

The basic idea flows from the results of the previous section. I rejected the structural hierarchy as the starting point for interpreting the relation between heredity and development. As has already been noted, in the process perspective, replication processes are a special case of inheritance processes, which are a special case of reproduction processes. Reproduction requires the progeneration (multiplication with material overlap) of the evolutionary minimum mechanisms of development, namely mechanisms conferring the capacity to reproduce. Mechanisms conferring a complete set of species-typical traits provide an evolutionary maximum specification of development. (Anything more would make the origin of new species impossible.) Therefore, general theories of development that can be

integrated with evolutionary theory are bounded by this spectrum of developmental mechanisms. Inheritance requires progeneration of evolved component mechanisms of development. These are mechanisms of development that increase transmission fidelity in particular respects and degrees over what can be achieved at the bare evolutionary minimum. Replication requires progeneration of evolved coding mechanisms of development. So, in process perspective, theories of genetic processes are special cases of theories of reproduction processes. Genetic theories should therefore be reducible to a theory of reproduction in Nagel's formal sense: Special case theories – with terms connectable to terms of a more general theory as well as laws and principles deducible from the general theory – are reduced by the more general theory.

Many philosophers have criticized Nagel's formal criteria for reduction on a variety of grounds. For present purposes, the most important of these criticisms is that his formal criteria of connectability and deducibility only supply analytical tools for interpreting the success or failure of a reductionistic research program *after the fact*. They are philosophers' tools, not scientists' tools. One explanation of this limitation is that Nagel's "syntactic" view of theory structure, in which scientific theories are axiomatizable in first order predicate logic, is not a realistic model of scientific theories in action. In retrospect, it may be possible to express theories this way, but in the heat of scientific theory-building at the cutting edge of empirical work this kind of presentation is rarely possible. Theories are more typically presented in models, not axioms (Lloyd 1988; van Fraassen 1980). Philosophers who have described reductionism as a kind of scientific activity and theorizing as a kind of model-building provide tools that can be put to heuristic, scientific use.[18]

However, to have a general theory of reproduction, we need theories of both "component" processes of progeneration and development.[19] Progeneration has a variety of structural models at different levels of the organizational hierarchy (autocatalysis for molecules, cell division for cells, sexual and asexual modes for organisms, inter- and intrademic models of group reproduction), but these are not complete models of progeneration because we lack a general theory of development, the process that supplies the units that get progenerated in reproduction processes. In contrast, there are explicit structural models of the units of progeneration that cause development.

Whether these models, for example, of gene-based developmental genetics or of epigenetic inheritance systems, are sufficient for a general theory of development, is unknown. Differently put, the entwinement of the processes of progeneration and development means that a theory of development should supply us with the units of progeneration and a theory of progeneration should supply us with the units of development. Thus, models of either process that ignore the other component of reproduction must be incomplete.

The heuristic application of reductionism aims to take advantage of the asymmetry of theoretical resources in genetics and development to bootstrap one class of general models of development. We do have theories of genetic processes and their units, albeit with the familiar array of idealizations, abstractions, heuristic assumptions, and simplifications in the models that black box development and much else as well. These idealizations blocked traditional interpretations of the relationship between genetics and development as one of reduction because the formal criteria for reduction, term-connectability, and law/principle-derivability could not be strictly met (reviewed in Sarkar 1998, chapter 2). Used heuristically, however, as a means to theory construction rather than philosophically (as an argument that reduction of Mendelian to molecular genetics or of development to genetics failed), the idealizations of genetics at least suggest classes of models for development. A serious reductionistic research program in theoretical developmental biology must explore other classes of models as well if the robustness of the theory to idealizing assumptions of its genetic models[20] is to be tested.

Since genetic theories are special cases of reproduction theory, they must at least implicitly involve development. In other words, genetic units of reproduction are also developmental units, but models in genetics tend to treat the developmental aspects in such a degree of idealization and abstraction that developmental properties are unspecified. One idea of how development has been implicitly assumed in the history of genetics will be suggested in the next section, that genetic units – genes – are modeled as "developmental invariants" – things that play a role in development but are not changed by the process of development.

Indeed, I propose a heuristic use of theory reduction to take advantage of this conception of developmental invariance implicit in

genetic theories. The heuristic is this: We have theories of genetics. We seek a theory of development. Treat genetic theory as a theory of development as well as of heredity. Use the implied units of development in an explicit, albeit limited model for the general case of a reproduction process. Then examine the limitations of the model to account for developmental phenomena outside the range for which it was originally constructed in order to bootstrap richer models of developmental units. In the process, the relationship between the original genetic model of developmental units and the bootstrapped models should be clear enough to relate traditional genetic theory to the general theory of reproduction. In other words, treat genetics as a theory of development in order to discover the limitations of that kind of theory of development in explaining developmental phenomena. This is reductionism in Wimsatt's engineering sense: You perform the reductive analysis in order to explore how the artificially constructed system or model fails to explain the phenomena. Then you systematically study the failure in order to build a better system (Wimsatt 1976; Wimsatt 1987; Wimsatt in press).

The heuristic approach aims to construct models of development using genetic theory we already have, subject to the constraint that the resulting theory of reproduction must reduce the theory of genetics we use to construct it. The reduction constraint is what makes genetic theory, for all its idealizations and abstractions, a useful guide to formulating models of development. Scientific interest is not likely to center on the success of the reduction, which is only a heuristic means to a theoretical end, but rather in the discovery of what is outside the explanatory scope of a theory of development constructed this way. Many biologists working on the evolution of development already have a good sense of what is left out of account (see Buss 1987; Jablonka and Lamb 1995). The theory of reproduction suggests one line of work toward a theory of development: filling the development gap between genetics and reproduction.

Substantive Criteria for the Reduction of Genetics

Following Sarkar (1998, chapter 3), I distinguish between formal and substantive criteria for reductive explanation. Substantive criteria concern the assumptions made in such an explanation, while formal

criteria such as Nagel's concern the logical form of explanation. Sarkar (1998, 43–44) lists three main substantive criteria: fundamentalism, abstract hierarchy, and spatial hierarchy.

In terms of the views expressed in the first section, Sarkar's fundamentalism criterion concerns the image and mapping relations of a reduction. The expected direction of a reduction relation between realms is determined by what counts as more fundamental. Abstract hierarchy applies to the properties bracketed by the evolutionary minimum and maximum specification of development as well as the condition of material overlap in progeneration. Insofar as a given reproductive process involves properties of component processes of progeneration and development, the latter will be at lower levels. The recursive nature of reproduction generates a hierarchy of processes with null development at the lowest level. Spatial hierarchy does not apply to the general theory of reproduction except in the weak sense that some spatial model is called for by any application of the theory because of the universality of material overlap. Material parts are spatial parts. However, the general theory does not specify a particular spatial model or class of models. In that sense, reduction of genetics to reproduction would be a case of "abstract hierarchical reduction" (Sarkar 1998, 44) because the case satisfies the criteria of fundamentalism and abstract hierarchy but not spatial hierarchy. Particular applications of the general theory of reproduction would likely involve some kind of spatial model, for example, of molecular genes, in which case it would be "strong reduction" (Sarkar 1998, 45). Cases in which only fundamentalism is satisfied, for example, the explanation of phenotypes in terms of heritability properties, are cases of "weak reduction" (Sarkar 1998, 44).

A Genetic Model of Developmental Units: Genes as Developmental Invariants

In the process perspective, the reduction image that pictures development as epigenetic is reversed. The arrow of reduction does not point from development as reduced theory toward genetics as reducing theory, but rather the other way, toward development as an aspect of reproduction processes in general, from the special case theories of genetic processes.[21]

The reversed image of reduction seems wrong-headed from the structure perspective because genetics and development are treated there as theories of processes operating on or between different levels of the spatial hierarchy. In the process perspective, genetics and reproduction are at different levels of generality, but since genetic processes always involve developmental processes, they operate at the same structural levels together.

In the functionalist replicator-interactor perspective, as with genotype-phenotype distinctions more broadly, autonomy of genetic processes from development is the basis for the individuation of levels: There are only the two levels of replicators and interactors. Thus, the functionalist perspective inherits the Weismannist constraint from the structuralist perspective. Dawkins-Hull replicators are things that pass on their structure intact, invariant in the developmental processes that construct new vehicles each generation that are exposed to the buffeting of selection. In the functionalist perspective, interactors are characterized in terms of selective interaction, though development is clearly the process that gives rise to interactors. However, development does not figure in the analysis of either replicators or interactors.

Indeed, in structure perspective, development is explained by treating genetic units as *developmental invariants*, things not changed by development but that pass through and control it. As constants in the cycling developmental system, genes are desirable theoretical entities because they have stable properties that can be followed through life cycles and are thus always there to hang explanations on. Genes are not only the only things to which evolutionary benefit can be traced (according to Dawkins' account of the units of selection); they are also the only things that go through development without developing. They epitomize self-reproduction: They are null developers born with the capacity of replication.

In the next section, I suggest that historical challenges to claims about the stability of genes provoke controversy about the nature of the gene because they raise doubt about whether genes can explain characters arising in development. This puts controversy about the concept of a gene on a par with "the species problem" as a perennial conceptual problem in science. Just as with species, scientists debating gene concepts argue strongly for particular conceptions and at the same time wonder whether the concept is even necessary to the science. In any case, the concept of the gene as a developmental invariant suggests a new historiography of genetics may be in order. If developmental invariance is central to the gene concept's history, then it may not be appropriate to interpret the history of relations between genetics and embryology as a divorce. Perhaps genetics never really left embryology after the origination of the gene theory, (cf. Gilbert 1978). Classical geneticists, even Muller in his search for the physical atoms of heredity, really did care about development (Falk 1997).

We can use this property of developmental units of genetics in the process perspective to speculate about theories of development. Genetic theories are theories of development because genetic processes are special cases of reproduction processes, but they are theories expressed in terms of developmental invariants. Are there other developmental invariants besides genes? Can the stability of developmental units be attributed to features of environment rather than internal, structural features? Can the apparatus of theoretical genetics (even if not Mendel's laws) be used to describe other developmental invariants? Are there ways to formulate a theory of development in terms of variable rather than invariant components or quantities?

Asking these questions in a scientifically productive way relies on theory reduction as a heuristic constraint. We seek a theory of development. We know that development is an aspect of reproduction, and we know that genetic processes are a special case of reproduction. Genetic theories describe genetic processes, so we look for the ways genetic theories handle the developmental aspects of "genetic" processes, for example, by treating the units of the theory as developmental invariants. We generalize those ways to a theory of reproduction processes and assess the limitations and failings of developmental theories reached by such generalizations, subject to the heuristic constraint that the formal theory of reproduction must reduce the special case theories of genetics. If we could succeed in constructing such a theory, it might help guide us to mechanisms causing developmental invariance beyond the special cases (genes)

that we already know about. Or it may help show us what mechanisms of development are left out by a theory of developmental invariants.

A PROCESS HISTORIOGRAPHY OF THE GENE CONCEPT?

Genetics tradition treats reduction as a problem ordered by the structural hierarchy, a *strong reduction* in the terminology of the previous section. Reductionist theories and explanations concern entities lower in the hierarchy than those at the level to be explained, while holist, emergentist, and teleological theories and explanations are couched in terms of entities higher in the hierarchy. Geneticists early in the twentieth century generally favored "nuclear monopoly," explaining heredity and development in terms of the controlling role of nuclear substance (Sapp 1987), while embryologists often debated whether the cytoplasm, the cell, or the organism as a whole was the seat of developmental control (Gilbert 1991). Thus, disciplines as well as phenomena were sorted by the structural hierarchy.

As we have seen, the commonly expected direction of reduction linking development and genetics also depends on the structural hierarchy. Although genetics can be treated in a purely phenomenological way, concerned only with parent-offspring resemblance, most theoreticians of heredity beginning with Darwin (gemmules), Mendel (factors), Weismann (biophores, determinants, ids, idants), Galton (stirp), Nägeli (idioplasm), and de Vries (intracellular pangenes) interpreted hereditary phenomena in terms of material, subcellular bodies (Robinson 1979). These entities are constituent parts of cells, just as cells are constituent parts of (multicellular) organisms. Development (at least its classical aspect in embryogenesis) has often been taken to apply only to the determination, differentiation, and growth of cells of multicellular bodies from such constituents. As a cellular or supercellular phenomenon of generation out of components, development necessarily takes place at a higher compositional level than genetics because genes are parts of cells.

The rigidity of the structural hierarchy guarantees a direction of reduction. Images of reduction based on structure cannot easily be reversed because parts and wholes are not likely to trade assigned

places in the hierarchy. It is extremely unlikely that genes will ever turn out to be wholes of which cells are parts or that organisms are really parts of cells. Yet that is the kind of reversal that would be required to keep the structural hierarchy as the proper framework for reduction and also try to reduce genetics to reproduction. There are, of course, genes that at times are not parts of cells such as genes inside viral phage particles, genes spilled out of lysed cells, and artificial genes manufactured in vitro. If Dawkins (1982) is right about the extended phenotype, there can be genes that control phenotypes of bodies in which they do not reside, placing such genes "outside" the cells and cell complexes bearing "their" phenotypic expressions. But these cases do not come close to what would be required to recognize that cells are parts of genes. The radical change of perspective required to make *that* reversal would involve a dramatic change in much more than the gene concept.[22]

Weismannism

An important source of the image that development reduces to genetics is the view that genes are the causes of development as well as the causes of heredity. This is a version of Weismannism.[23] Weismann (1892) espoused the view that germ-plasm is continuous while somato-plasm is discontinuous. The nuclear plasma of cells destined to become germ cells passes untouched through cell lines to form a line of hereditary continuity from zygote to germ cell in development. The parts of the plasma of other cell line nuclei, destined to become the differentiated cells of the body, were thought to be parceled out to the cells of different types such that the qualitative division explains observed cellular differentiation in embryogeny. Related views of heredity, though not of development, are implicit in Mendel and Johannsen (Lewontin 1992). This similarity was noted by Johannsen's contemporaries early in the history of genetics. For example, E. G. Conklin, in his 1914 Harris Lectures, wrote: "This contrast between the germ and the body, between the undeveloped and the developed organism, is fundamental in all modern studies of heredity. It was especially emphasized by Weismann in his germ-plasm theory and recently it has been made prominent by Johannsen under the terms 'genotype' and 'phenotype'" (Conklin 1919, 126–127).

Although T. H. Morgan rejected Weismann's theory of development (understood by him as the Roux-Weismann mosaic theory of qualitative division), Morgan endorsed the view of heredity common to Weismannism and his own factorial theory of heredity, which "rests on the assumption that the germ plasm contains a host of elements, that are independent of each other in the sense that one allelomorph may be substituted for another one *without alteration of either,* and that these allelomorphs will now perpetuate themselves *unchanged* although in company of different factors" (Morgan et al. 1915, 277; *emphasis added*).

Some prominent claims attributed to Weismann should more accurately be attributed to those of his interpreters who inspired the formation of genetics as a distinct discipline, notably E. B. Wilson (Griesemer and Wimsatt 1989). Wilson interpreted Weismann's view as one of "nuclear monopoly" on biological causation (Sapp 1987, 1991). The germ-plasm is the cause both of new germ-plasm and of the soma in development (Wilson 1896, figure 4). In modern guise, genes are double causal agents while characters are epiphenomenal, acausal entities. The noncausal role of the body in modern molecular terms is best shown in Maynard Smith's "rosetta stone" diagram comparing Wilson's interpretation of Weismann to Crick's central dogma of molecular genetics. DNA gives rise to new DNA and to protein, just as the germ gives rise to new germ and to the soma (Maynard Smith 1975, figure 8).

An alternative to strong reduction to lower levels of the spatial hierarchy is abstract hierarchical reduction of special to general cases of reproduction processes. Generalization, like spatial composition, is a hierarchy-forming relation. This is a common sense of classical theory reduction in the physical sciences, as in the case of the reduction of Kepler's laws of planetary motion to Newton's laws of mechanics. If general cases are described by fundamental laws of nature and special cases by phenomenological laws, rules, regularities, or singular observations or events, then reduction may be interpreted as the theoretical enterprise of explanation by fundamental laws.

The purpose of this section is to urge a new historiography of the gene concept, in process perspective, in which the character of the gene as a developmental unit is as central to the gene concept as is the gene's role as a unit of hereditary transmission among generations. As suggested above, from the point of view of genetics, the gene is a unit of reproduction that is invariant in development, stable in heredity in the short term and variable in evolution over the long run (through mutation and other processes of genetic change). The main goal in urging a new historiography is scientific: to gain heuristic purchase on a general theory of development by viewing theories of genetics as theories of development based on invariant units. For that reason, the historical observations below are offered only to illustrate my conviction that historical defenses of the gene as a developmental invariant, as an entity stable in development that explains hereditary transmission and somehow explains development, will be found wherever there is controversy about the nature of the gene.

The proposal that genes are developmental invariants implies a more radical view of the history of genetics than Scott Gilbert's argument that genetics originated in embryology and then superseded it, at least in "theological" rhetoric (Gilbert 1978). Instead, I suggest that genetics never left its ancestral home in embryology. Geneticists of all periods from Mendel onward have expressed direct interest and concern with development as well as hereditary transmission, with the gene-character relation as well as gene-gene and character-character correlation among generations. What has happened historically is that interpretation of heredity has shifted from a developmental aspect of parent-offspring relations in reproduction to genes as autonomous master molecules controlling development (Keller 1995; Sapp 1987). Geneticists have always been intensely interested in development. The historical change of rhetoric masks the developmental nature of the gene.

Mendelian Factors

In a sense, classical transmission genetics beginning with Mendel is the first modern theory of development because it identified the first developmental invariant, the Mendelian factor. Invariant entities are useful theoretical units because they are units one can calculate with and that one can use as tools of experimental intervention. They are conceptually and empirically "solid," "tame," and "domesticated." Entities that change while one is using them are difficult to work

with, hard to manage and keep track of, hard to defend. Mendel described factor segregation in terms of passage unchanged through the hybrid organisms he created and controlled in his experimental garden.

Mendel's paper on hybridization in peas illustrates the notion of a gene (factor) as a developmental invariant (Mendel 1866). The law of segregation expresses an invariance principle. Mendelian factors in hybridization experiments segregate. They can be brought together in hybrids and separated again without any effect on their capacity to act subsequently as factors: "among the progeny of the hybrids constant forms appear, and that this occurs, too, in respect of all combinations of the associated characters" (Mendel 1866, 20). Dominant and recessive factors are both recovered in subsequent generations, meaning in developmental contexts similar to those of the parental generation. Segregation shows that putting a factor into the developmental environment of a hybrid, that differs from that of the true-breeding lineage from which it was extracted, does not alter factors. Segregating factors show themselves to be stable over multigenerational inheritance involving processes of syngametic combination in zygotes and assortment in reproduction that cause developmental contexts to fluctuate. Of course, Mendel characterized these processes and contexts crudely; he considered whether pollen and egg cells were of "like" or "unlike" character and described their combinatorial possibilities without attempting to explain how the factors get from parent cells to offspring cells or how they play a role in the development of offspring traits.

While recessive traits are not expressed in the hybrid, recessive factors carry the capacity for recessive trait expression even in developmental contexts where the recessive trait is not actually expressed. This is revealed when traits equivalent to those of the pure-bred parentals appear in the second generation bred from the hybrids (grandoffspring). Although systems may be such that a gene "is expressed" in one developmental context and not in another, this does not mean that the gene fails to carry a capacity into different developmental contexts and is therefore not developmentally invariant. However, some theories of dominance and recessiveness, such as Bateson's presence-absence theory, challenge the stability of the recessive capacity. That theory interprets recessive traits as due to the

absence of a gene and dominant traits to the presence of a gene. If true, this theory would imply that the capacity for expression of recessive traits is carried, not by a particular material factor, but by the genome, nucleus, cell, or organism lacking dominant factors. Morgan, for one, attacked Bateson's theory on precisely these grounds (Morgan 1926).

Factors are not altered by passage through different kinds of hybrids. Trait expression in Mendel's diploids depends on factor pairs (or more complex collections), not individual factors, so changes in expression with change of developmental context do not constitute changes in the gene. The gene is identified and characterized solely in terms of its invariant qualities. If it also has developmentally variable qualities, these will be judged accidental with respect to its status as a gene. That judgment is made explicit by the use of phrases like "non-Mendelian" to label "genes" that, because of their variability, are by definition aberrant. The label does not signal a belief that non-Mendelian factors must be relatively rare to be counted aberrant. Instead it signals the belief that what it means to be a Mendelian gene includes the invariance specified by Mendel's laws.

It is clear from Mendel's text that his theory was developmental as well as hereditary in nature. In the section "The Reproductive Cells of the Hybrids," Mendel offers a hypothesis to explain his results, that "So far as experience goes, we find it in every case confirmed that constant progeny can only be formed when the egg cells and the fertilizing pollen are of like character, so that both are provided with the material for creating quite similar individuals, as in the case with the normal fertilization of pure species. We must therefore regard it as certain that exactly similar factors must be at work also in the production of the constant forms in the hybrid plants" (Mendel 1866, 20). He goes on to offer an assumption that clearly expresses the joint developmental-hereditary nature of the theory: "In point of fact it is possible to demonstrate theoretically that this hypothesis would fully suffice to account for the *development of the hybrids in the separate generations*, if we might at the same time assume that the various kinds of egg and pollen cells were formed in the hybrids on the average in equal numbers" (Mendel 1866, 21, *emphasis added*).

Subsequent interpreters of Mendel's achievement characterized

this stability as the hallmark of his theory. In *Heredity and Environment,* for example, Conklin characterizes stability in terms of the "purity" of the germ cells of hybrid organisms despite their origin from hybrid parent cells (Conklin 1919, 88). The principle of segregation, which describes the maintenance of purity through crosses and the development of hybrid offspring, is "the most important part of Mendel's law" (Conklin 1919). Offspring ratios not predicted by simple Mendelian assumptions threaten the concept of the factor as stable in development, unless they can be interpreted in an expanded Mendelian sense, for example, by multiple factors, partial dominance, or maternal effects (Conklin 1919, 107–112).

Castle's argument in the 1910s that natural selection might alter Mendelian factors and that he had experimental evidence in support of this interpretation was dealt with severely by the Morgan school, particularly by Muller. The case is discussed by Provine (1971, chapter 4; 1986, chapter 2) and need not be repeated here. The lesson, though, is the same: If selection alters the gene, then the gene is not invariable in the developmental process described by Mendelism. Such challenges must be met if genetics is to be built on Mendelian units.

In *The Theory of the Gene,* Morgan wrote that "Mendel's theory of heredity postulates that the gene is stable. It assumes that the gene that each parent contributes to the hybrid remains intact in its new environment in the hybrid" (Morgan 1926, 292). Morgan offered a series of examples to show the variety of evidence that the central Mendelian assumption is true, despite the apparent challenge to the stability of the gene implicit in unexpected ratios.

Despite much diversity of behavior in development, the stability of genes is never allowed to remain at issue for long. Even mutation gets interpreted as a stable change in gene state. Whenever the stability of an alleged genetic unit becomes doubtful, its status as a gene does also. Genes are the epitome of Salmon's notion of a causal process: Genes can transmit "marks" as the result of causal interactions (Salmon 1984). Salmon (1994) argues that invariance is central to the general concept of a causal process (see also van Fraassen, 1989; Woodward 1993). Here, invariance with respect to a developmental frame of reference is central: It is what allows us to interpret a reproduction process as genetic, as having the capacity to transmit

particular kinds of developmental marks, meaning mutations, along replication processes.

An additional basis for thinking invariance is central to the gene concept lies in the use of phrases like *a gene for X.* This expression is applied carefully in classical genetics, in situations where hybridization experiments have been done to reveal in what respect the gene is a differential, in other words a cause that distinguishes between organisms with and without the gene. This differential quality of the gene concept has been emphasized by many geneticists throughout the history of the field (see Schwartz, this volume). Without the possibility of hybridization and its requirement of distinguishable phenotypes, genes that are developmentally invariant as specified by Mendelism cannot be detected. This is because, as the classical geneticists understood, characters are determined by many or all the genes and each gene may have many effects. The experimental geneticist relies on the developmental invariance of the gene to discover its presence through hybridization, so invariance is embedded in the instrumental notion of Mendelian factors as well as in its conceptual analysis. For Mendel, the relevant contexts were the zygotic environments from which the factor is extractable by further crossing. Extractability is the experimental sign of invariance.

A process historiography of the gene concept would show that challenges to the stability or invariance of the gene cut deeply because they challenge not only concepts, but the epistemological basis of experimental utility of the gene concept. A careful history of controversies over gene concepts should show that each time apparent variability in the gene itself was discovered, geneticists faced a familiar array of options. They could defend the gene concept, showing how challenging cases, for example, of "non-Mendelian" ratios, could be given Mendelian explanations, thereby vindicating purity of the germ and stability of the factor. Or, they could narrow their gene concept to that part or property of the gene as previously conceptualized that remains developmentally invariant in the new phenomena (essentially refining general descriptions of genetic phenomena). If pseudoallelism or fine structure mapping reveals that genes can no longer be thought of as units of structure, function, mutation, *and* recombination, then perhaps genes can be interpreted more narrowly as units of structure that must team up to constitute

units of function. If the operon theory forces a divorce between units of structure and function, then perhaps genes can be kept on as units of structure whose functions are explained in terms of cell-level regulation. In other words, challenges to gene concepts throughout history might be understood in terms of such a list of options if we interpret the gene as something that is supposed to be developmentally invariant, in other words, something defined with respect to the developmental process. Finally, geneticists could alter the meaning of *development* in such a way that their original gene concept is of an invariant in the modified sense of development.

The history of repeated defense against challenges of instability may lend, ironically, a sense of progress. If narrowing the gene concept to ever smaller sequences preserves the developmental invariance of the concept, it is a march in step with the progress of mechanism toward lower and lower levels of organization. But what stops progress from going too far, to the level of single nucleotides? Geneticists' sense that single nucleotides are chemical rather than biological objects that create a counter-pressure to make the gene concept more inclusive. Otherwise, explanatory power would be handed over, along with the gene concept, to chemists. On the other hand, expanding the gene concept leads to holism – the genome as unit of heredity – which geneticists have typically, though not invariably (remember Goldschmidt), regarded as unproductive because of their heuristic bias in favor of strong reduction and mechanism leading down the biological hierarchy.

The important point is that successful defense of the Mendelian gene concept does not thereby *explain* development. The Mendelian gene is an entity that is invariant in the frames of reference defined by Mendelian experimental construction of those developmental processes. Mendelian methods supply no account of how genes are involved in the development of the organism's traits. Mendel's concept does not address the "paradox of development."

The Paradox of Development

The fundamental problem of development is to explain how heterogeneity comes from homogeneity. It is a hard problem because the sources, nature, and mechanisms of change must all be understood

and articulated. It poses a dilemma for biologists who have committed to a particular approach or point of view if they do not see how their chosen field, theory, and methods can solve the problem directly. Either the failing is internal to their point of view, requiring internal changes in theory or methods, or the failing is external in the sense that a solution requires theories or methods from outside the chosen approach or point of view. Either way, the committed scientist who fails must give up something: either explanatory power by sticking to the point of view but failing to solve the problem, or explanatory control by admitting fundamental units from other fields or points of view.[24] Few scientists are willing to accept either horn of the dilemma and instead try to solve the problem head on by articulating, modifying, or otherwise advancing their paradigm – activities Kuhn called "normal science."

Genetics faces the fundamental problem of development because, as I have argued, genetics is about development, even if practitioners have tried to insulate their field from embryology and developmental biology. The paradox of development poses a dilemma for genetics (Gilbert 1994; Sapp 1991). How can cell differentiation, the basis of form in multicellular organisms and hence of variation in evolution, be explained by genes, since the genes of all somatic cells are identical? The dilemma was familiar to Morgan when he wrote in *Embryology and Genetics* that "At first sight it may seem paradoxical that a guinea pig that can develop areas of black hair should have white areas of hair if, as is the case, the cells of both areas carry all the genes" (Morgan 1934, 134). The paradox is the geneticist's way of stating the fundamental problem of development. The genetical version became urgent after the defeat of the Roux-Weismann mosaic conception of development, that did explain cellular differentiation out of genetical homogeneity.

The geneticists' paradox is a consequence of assumptions about the gene as an invariant. Genes must be stable in heredity in order to explain the basic phenomenon of heredity by Mendelian means: Offspring tend to resemble their parents. Deviations from this tendency are explained in terms of changing collections of genes rather than alterations of genes themselves (except for rare mutations). The basic tendency to heredity is explained as a result of the hereditary stability of the genetic units. But the experiments which originally

showed this hereditary stability, Mendelian hybridization experiments, also required genes to be developmental invariants: Genes could not change as a function of developmental context and be detected by Mendelian means. However, if somatic cells are genetically equivalent and also cause development as dictated by Weismannism then they must change in order to cause differentiation. Commitment to Mendelian methods *and also* to genetic explanations of development generates the paradox: Genes must be developmentally invariant because otherwise they could not be shown to be hereditarily stable, but they must be developmentally variable if they are to be the cause of differentiation in development. The paradox of development is a paradox of genetics.[25]

Homogeneity of genes among cells of a developing organism is an aspect of developmental invariance: The genes don't change in quantity or quality as they are passed from cell to cell in the dividing organism. But what must geneticists give up in order to address the paradox? Should they stick to their classical genes and Mendelian methods and give up the power to explain development? Should they modify their gene concept in the hope that some other notion of what makes a gene a stable unit in hereditary transmission will also explain the gene's role in expression and differentiation? Or should they refine the description of the developmental invariance property of the gene in such a way as to cede explanatory control but not give up the classical gene or its explanatory power?

Morgan explored all three of these strategies for explaining development by genes. He outlined them in the introduction to *Embryology and Genetics* (9). First, all genes might act all the time in the same way. That would leave differentiation completely unexplained. Still, the dilemma would be solved by putting one's head in the sand, treating differentiation as a black box to be described phenomenologically for the purpose of character analysis. Second, it may be that "different batteries of genes come into action as development proceeds" (Morgan 1934, 10). This solution, that Morgan interprets along the lines of the Roux-Weismann hypothesis, is contradicted, he claims, by results of experiments compressing embryos to alter the sequence of cleavage planes. Third, Morgan speculates that differences of protoplasm might influence the "growth of the chromatin" and affect "the substances manufactured by the genes,"

so that "The initial differences in the protoplasmic regions may be supposed to affect the activity of the genes. The genes will then in turn affect the protoplasm, which will start a new series of reciprocal reactions" (Morgan 1934). Since the initial protoplasmic differences will trace to the action of maternal genes, we have a genetic solution to the problem of development. This last explanatory strategy is most interesting. In effect, it narrows the nature of the developmental invariance of the gene to its structural properties, allowing its functioning to depend in complex and changing ways on its interactions with protoplasm. As a result, control of the explanation of development is partially ceded to the students of protoplasm – cytologists, embryologists, and colloidal chemists – but the nature of the gene as an invariant in development, though narrowed, is preserved. Genes remain the locus of explanatory power because it is their role in interaction that is explanatory.

Operons

Weismann's speculative hypotheses of genomic spatial hierarchy and mosaic development were being rejected at the same time that Weismannism's causal structure was spreading as part of the central theoretical perspective of the emerging field of genetics. The genetic equivalence of all the cells of the body, including germ-cells, only exacerbated the problem of the paradox of development for geneticists who accepted the developmental invariance of the gene resting on Weismannism's theory and Mendel's methods. Morgan's speculations in the 1930s, trying to hold on to the classical Mendelian gene concept *and* the goal of a genetic explanation of development, far outran both theory and experimental data.

Support for Morgan's third explanatory strategy came only in the 1960s after genetics began to go molecular, when it was recognized that cellular difference could be interpreted in terms of differences in states of molecular gene activity rather than differences of gene presence/absence, structure, or arrangement (position effect, inversion). The operon theory of Jacob, Monod, and others provided an interpretation of molecular feedback mechanisms by which differences in cellular and external environments could cause different states of gene activation. As Sapp put it, "The operon scheme solved

the problem of cellular differentiation without invoking environmentally directed gene mutations and without invoking a distinction between somatic heredity or development (cytoplasm) and sexual heredity (nucleus)" (Sapp 1991, 246). The operon model avoids ceding explanatory power to other fields, theories, or methods in the sense that the gene remains the central explanatory unit. However, while the operon theory provides a genetical solution to the paradox of development, it sustains the essential tension of the paradox insofar as genes remain the cause of development. In the operon model, explanatory control is ceded to developmental biology or (horrors!) nutritional biochemistry by making gene activation states the central concern rather than gene structure. This opens the door to other cell constituents as possible units of explanation for development. Moreover, activation states are not properties of classical genes, so the status of genes as developmental invariants, and thus the nature of the gene concept, is muddied.

The Jacob-Monod operon theory of bacterial gene regulation is on the opposite side of the molecular biology revolution from Mendel. Part of the triumph of molecular genetics is due to narrowing of the neoclassical gene concept, identifying genes with nucleotide sequences. This move eliminated much uncertainty about the nature of genes due to their complex role in development. The Watson-Crick molecular gene narrows the sense in which a gene is a developmental invariant to that of a structural invariant – the nucleotide sequence does not change in development even if its activation state does. However, narrowing the gene concept in this way does not avoid ceding explanatory control. Even though genes code for proteins, proteins could not be invoked to explain differentiation and thus, by transitivity of causes, used as an interpretation of how genes cause development. The reason is that sequence invariance does not explain how different cells come to have different constellations of proteins that cause differentiation.

The operon concept of the gene illustrates options for resolving challenges to the notion of the gene as a developmental invariant. As discussed above, neoclassical replies to challenges to the classical Mendelian gene showed that non-Mendelian genes – genes that exhibited apparent developmental variability – have Mendelian explanations. In contrast, molecular solutions to challenges to the power

of classical genes to explain development involve either modification of the genetic properties that are developmentally invariant or a changed interpretation of developmental invariance that grounds the gene concept. The option of modifying the gene concept was not open to geneticists of the Morgan era for social-institutional reasons. Genetics would have withered with that decision just as embryology appears to have done as it pursued one unit concept after another (organizers, morphogenetic fields, and so forth). Histories of the period between the chromosome theory and the operon should supply further evidence of genes as invariants (e.g., Dietrich, this volume).

The operon theory interprets genes as collections of nucleotide sequences having different functions.[26] Some sequences – promoters and operators – are regulatory, providing binding sites for molecules that regulate gene expression. Other sequences – structural genes – code for amino acid sequences of polypeptides. RNA polymerases bind promoters to begin transcription of downstream structural DNA sequences into RNA. After transcription, RNA molecules can be processed, transported out of the nucleus, and translated on ribosomes into amino acid sequences. Transcription can be blocked if a regulator molecule binds the operator, preventing RNA polymerase from moving down the line to the structural gene. Transcription can be (re)started if effector molecules bind the regulators to prevent them from blocking transcription. The ordered collection of regulatory and structural sequences, regulatory molecules, and effectors (that can be molecules imported from the extracellular environment), form an environmental response system altering, not the nucleotide sequences of the genes, but the state of activation of the collective. Jacob and Monod called this collective a "macromolecular society" (Sapp 1991, 245).

The operon concept can address the paradox of development genetically by expanding the gene concept to include activation states as well as sequence as part of the gene concept in order to explain development and resolve the paradox of development. But precisely by ascribing the feedback that explains developmental diversification to the state of activation and not to the sequence, the operon theory of gene regulation distinguishes the sequence as a developmental invariant from activation state as a developmental variable.

The question is, what kind of a solution to the paradox is the decision to include activation state as part of the gene concept and what sort of scientific commitments does that decision require? If the operon concept helps solve the paradox of development by expanding the gene concept to include activation state, it comes at the cost of rejecting the idea that genes are developmental invariants, or else it is on the road to genetic holism: The gene is not a molecule but rather the whole macromolecular society, that is the new locus of developmental invariance. As a result, operon-based genetics ceases to be genetics in the classical sense, a serious break with tradition.

Alternatively, if the operon concept of a gene, like the classical Watson-Crick molecular gene, equates gene with nucleotide sequence, then the problem of development and its explanation by genetics is abandoned. Instead, a new interfield theory (Darden 1991; Darden and Maull 1977) of "developmental genetics" is called for that is neither development nor genetics in the classical sense and whose domain is all that lies "between the base pairs of the genotype and the limb or eye of the phenotype" (Gilbert 1991, 136).

This story illustrates how the image of reduction is tied to the core concept of a gene as a developmental invariant by the paradox of development. Genetics cannot explain development without being developmental, but in being so, it ceases to look very "genetic." As genetics incorporated ever more sophisticated interpretations of the macromolecular regulatory environment of the genes, holding out hope for a genetic solution to the paradox of development, the fundamental idea that the genes are stable in development crumbled. Genes are the locus of developmental explanation because they are fixed points in the developmental flux. The attempt to resolve the paradox of development genetically, through the operon model of gene regulation, either narrowed the concept of the gene to that of nucleotide sequence or threatened to expand it so much that the new view ceases to be genetics from a structuralist perspective.

Developmental genetics counts as distinct from genetics if genetics adopts a narrow gene concept that is developmentally invariant. Developmental genetics counts as part of genetics if the gene concept is expanded to include the activation states that provide an account of genetic control with environmental feedback. But then the gene is no longer a developmental invariant, hence developmental

genetics is not genetics in the classical sense. Thus, the paradox of development continues to be a dilemma for genetics in the molecular age. This is to be expected if what is essential to a gene concept is the idea of developmental invariance.

Developmental Genetics

The fundamental assumption of developmental genetics and its application to evolution is succinctly stated by Raff and Kaufman: "Our premise is that developmental processes are under genetic control and that evolution should be envisaged as resulting from changes in the genes regulating ontogeny" (Raff and Kaufman 1983, 2). This premise supports the traditional image of reduction. One expects development to reduce to genetics because genetics is the field whose theories describe the causal mechanisms by which genes regulate ontogeny and support causal explanations of evolutionary changes in form when coupled to the laws of evolutionary genetics.

Gilbert (1991, 1994, 38–39) traces the origins of developmental genetics to work by Gluecksohn-Schoenheimer and Waddington in the late 1930s, quoting the former's reflection of 1938 that embryology and genetics are each incomplete without the other (Gilbert 1994, 38). But for Gilbert, the significance of developmental genetics is that it puts to rest the criticism of genetics by embryologists, that mutations and Mendelizing traits in general affect only superficial adult characters and not fundamental Bauplans constructed in development. Developmental genetics claims to study the control of major Bauplans in development by genes (or gene complexes). These are significant advances into the study of the developmental role of gene action, to be sure, but they do not serve to alter the expectation that *development* reduces to genetics. Instead, developmental genetics provides richer detail than the austere formal mapping of genotype to phenotype in neoclassical genetics, which skirts the whole problem of development. Developmental genetics provides a new theoretical level in its interfield theories tracing gene action located and localized spatially and temporally in the developing organism.

From the process point of view, developmental genetics does not address the important conceptual issues because it inherits the structuralist tradition of reductionism in which development is expected

to reduce to genetics. In the process perspective, developmental genetics is not the genetics of "developmental" traits, but rather is an idealized model of development, one in which the progenerative role of environmental variation is simplified for the sake of emphasizing the role of the developmental resources called molecular genes. It is dubious that an adequate theory of development can be formulated that takes only these resources as its central units, since molecular biology is daily challenging the developmental invariance of nucleotide sequences right down to the level of single nucleotides (see e.g., Fogle this volume; Moss 1992; Neumann-Held 1998). A more adequate theory of development will probably not be possible without a more radical change of perspective, such as the one urged at the outset of this paper.

NOTES

1. Part of this work was pursued while the author was a fellow of the Wissenschaftskolleg zu Berlin (1992–1993) and of the Collegium Budapest (1994–1995) and was facilitated by USDA Cooperative Agreement PNW 95–0768. I thank the Rektors and Fellows of both institutes, and the respective Biology Group Conveners, Peter Hammerstein and Eörs Szathmáry. This work would not have been done without the interest and support of Leo Buss, Eva Jablonka, and Eörs Szathmáry. I owe many thanks to the editors, especially Rafi Falk for detailed criticism and advice, and to an anonymous reviewer for valuable suggestions on how to organize this material. I am grateful to the other workshop participants, to audiences at Chicago, Duke, Northwestern, and San Diego, and especially to my former colleague Michael Dietrich for helpful discussion of the manuscript and clarification of the argument. I thank Connie, Ellen, and Kate for their support.
2. This suggests a pragmatic view of theories that adds to the semantic view the notion that our assignments of objects to processes must pick out from the network of causation those pathways of interest and concern to us. Perspectives play the role of guides to commitment to scientific action in the pragmatic view of theories parallel to the role of models as guides to acceptance and belief in the semantic view.
3. I adopt Sarkar's (1998) terminology of factors, rules and realms rather than the traditional causes, laws, and domains to avoid confusion of my use of terms like *image* and *mapping* with terms from logic and mathematics having different meanings.
4. In forthcoming work I criticize the philosophical literature on replicators. Work in preparation will show how to interpret heritabilities and selection differentials as quantities for causal capacities.
5. Two authors who express similar philosophical views are Brandon (1990, chapter 3) and Burian (1992, 11). Burian equates intergenerational transmission with "replication plus development." Brandon considers the role of development in relation to that of replicators in evolution. The relation between these views and mine are too complex to enter into here. Thanks to Michael Wedin for suggesting the term *progeneration.*
6. Compare progeneration to Maynard Smith's multiplication condition for something to be a unit of evolution (Maynard Smith 1988). He includes the notion of "same kind" in his definition of multiplication, but provides little elaboration and does not acknowledge the general requirement of material overlap. Copies need not materially overlap their originals to count as copies, but progenerants must materially overlap their parents if they are to count as progenerated. A photocopy of a painting does not materially overlap the painting that was laid on the copier glass, but it is nevertheless a copy. A copy of a painting resembles the original, though it may not be made of paint on canvas, let alone be made out of the very paint and canvas of the original. But a progenerant of a painting might be a torn half of the original painting. In virtue of material overlap, it will resemble the painting in some respects, but certainly not in all the same respects that copies typically do. Thus, some cases of so-called "cultural" evolution will turn out to be "biological" in the sense that they satisfy the material overlap condition for biological reproduction, which in turn is a necessary condition for evolution. Other cases will only satisfy weaker copying criteria. This differentiation of cases of cultural evolution disagrees with Dawkins' (1976) memetic theory as well as with some developmental systems theories (Griffiths and Gray 1994), that treat all cases of cultural evolution on a par due to their lack of distinction of replication and reproduction (Griesemer in press). Genetic reductionist views also tend to treat all cases of cultural evolution as reducible to biology in the same way. In my view, some cases of cultural evolution may be genuine cases of biological evolution because they are genuine biological reproduction processes, whereas others may only be analogous to reproduction processes and therefore not reducible in a theory of biological reproduction.
7. Things get interesting as we push back to the origin of life. Do autocatalytic chemical cycles count as biological agents? Must we invoke an idea of chemical agency to explain biological agency? Is a loaf of bread an agent if it "invites" a hungry knife-wielder to cut it, as Dawkins suggests when he characterizes a sheet of paper as an "active" replicator (meme) if the words written on it cause someone to put it into the photocopier and make more copies? Actors and agents must be dis-

tinguished if we are to understand the biological autonomy of reproducers (Eilhu Gerson, personal communication).

8. This is an argument I first made in Griesemer (in press).
9. The reproducer concept generalizes the replicator in parallel to the theory of reproduction processes generalizing genetic processes. In Griesemer (in press), I argued that the reproducer concept redresses grievances from the developmental systems perspective against reductionism (e.g., Gray 1992; Griffiths and Gray 1994; Oyama 1985). It also argues against the divorce of genetics and development reinforced by Dawkins (see Sterelny, Smith, and Dickison 1996).
10. Thanks to Walter Fontana for the wonderful phrase *pieces of development*.
11. In work in preparation, I argue that this is the minimal specification of development needed for a general theory of evolution, in which the only relevant essential properties of offspring are whether they can reproduce and the extent to which they vary in that capacity. That is enough for so-called "replicator dynamics" to apply to reproducers. The maximal specification of development for evolutionary theory is the acquisition of species-typical traits. All traits with evolutionary biofunction can be thought of as ways and means to the minimal developmental end.
12. I say *a* process perspective because mine is not the only one. Developmental systems theorists also offer a process perspective. See Griesemer (in press).
13. In short, Weismann was no Weismannian (Griesemer 1994; Griesemer and Wimsatt 1989).
14. Following Glennan (1996, 52), mechanisms are things – physical objects or processes – which have parts that interact in such a way as to cause a specifiable behavior: "A mechanism underlying a behavior is a complex system which produces that behavior by the interaction of a number of parts according to direct causal laws." One might also interpret mechanisms for heredity in terms of Cartwright's notion of "nomological machines" (Cartwright 1997). Saying that a mechanism is "for" heredity means that it is efficient at producing heredity (with respect to some trait) compared to processes that do not have the mechanism. Most such mechanisms will be efficient in virtue of being evolutionary adaptations. In forthcoming work, I analyze the concept of coding in terms of the process perspective suggested here. Discussion of coding is beyond the scope of this paper.
15. Coding, in other words, may consist in excess combinatorial information (Gánti 1971 [1986]). The number of nucleotide (amino acid) kinds is much smaller than the number of sequences of nucleotides (polypeptides) coded for. There are four basic kinds of nuclotides and a vast number of possible sequences of chromosome length. Heredity is "limited" when the number of possible states specifiable by the system is comparable to the number of actual units realizing states of such a system, whereas heredity is "unlimited" when the number of possible states vastly exceeds the number of actual states realized by any physical system (Szathmáry and Maynard Smith 1993).
16. See Wade and Griesemer (1998) for an empirical study of propagule pool reproduction as a kind of group-level genetics and Griesemer and Wade (2000) for an interpretation of group genetics with Punnett Squares.
17. Different views of theory structure may lead to different scientific research programs than the one proposed in this section, which assumes that theories are collections of models and their robust consequences.
18. Wimsatt 1976, cf. Sarkar 1998. For a catalogue of reductionistic research heuristics, see Wimsatt (1980). The papers in Wimsatt (in press) articulate his philosophy of theorizing as model-building.
19. 'Component' is in scare quotes because I have not specified an ontology of processes. Whether and how progeneration and development can be genuinely interpreted as parts of reproduction processes must be worked out. This is an important conceptual problem for the heuristic application of reductionism proposed here because it bears on whether substantive reduction criteria are met by the theory of reproduction presented in the first section.
20. That is, models of development framed from the point of view of traditional genetics. A certain amount of violence to traditional language is unavoidable. In the process perspective, genetics is much broader than its traditional meaning because it applies to all the developmental aspects of the special classes of reproduction processes here called inheritance and replication.
21. It should be clear that the reversal is not simply one of turning the arrow around. There is also a change of basis in the switch from the structure or spatial hierarchy perspective, in which genetic units are lower in level than phenotypic units, to the abstract hierarchy of the process perspective in which genetic processes are special case but higher *level* processes of reproduction as compared to the general case but lower level processes of progeneration and development.
22. Faced with the choice of holism or part-whole reversal, developmental systems theorists (Gray 1992; Griffiths and Gray 1994; Oyama 1985) chose a form of holism by identifying whole developmental systems as units of replication (cf. Sterelny, Smith, and Dickison 1996). I argue instead that genetic systems (replicators) reduce to developmental systems, i.e. to reproducers.
23. For recent literature describing and analyzing Weismannism, see Churchill (1985, 1987); Gilbert (1991); Griesemer (1994) ; Griesemer and Wimsatt (1989); Maienschein (1987); Maynard Smith (1989); Mayr (1985); Sapp (1991).
24. Obviously, this is not an exhaustive list of options. Scientists can change

their basic commitments, lines of work or even careers in order to pursue a problem. The point is that the problem compels choice.

25. I do not have space to characterize how this paradox is generated and averted in embryology, but see Sapp (1991) for further discussion.
26. See the excellent review of the operon theory in the context of a discussion of the embryological origins of the gene theory by Gilbert (1994, chapter 2).

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The Concept of the Gene in Development and Evolution

Historical and Epistemological Perspectives

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CAMBRIDGE
UNIVERSITY PRESS

PUBLISHED BY THE PRESS SYNDICATE OF THE UNIVERSITY OF CAMBRIDGE
The Pitt Building, Trumpington Street, Cambridge, United Kingdom

CAMBRIDGE UNIVERSITY PRESS
The Edinburgh Building, Cambridge CB2 2RU, UK http://www.cup.cam.ac.uk
40 West 20th Street, New York, NY 10011-4211, USA http://www.cup.org
10 Stamford Road, Oakleigh, Melbourne 3166, Australia
Ruiz de Alarcón 13, 28014 Madrid, Spain

First published 2000

Printed in the United States of America

Typeface Palatino 10/13 pt. *System* MagnaType™ [AG]

A catalog record for this book is available from the British Library.

Library of Congress Cataloging in Publication Data

The concept of the gene in development and evolution: historical and epistemological perspectives / edited by Peter J. Beurton, Raphael Falk, Hans-Jörg Rheinberger.
p. cm. – (Cambridge studies in philosophy and biology)
Includes bibliographical references and index.
ISBN 0-521-77187-0
1. Genes. 2. Genetics – Philosophy. 3. Developmental genetics. 4. Evolutionary genetics. I. Series. II. Beurton, Peter, J. III. Falk, Raphael. IV. Rheinberger, Hans-Jörg.
QH447 .C66 2000
572.8'38 21 – dc21 99-042106

ISBN 0 521 77187 0 hardback

Contents

Introduction

Everybody knows about genes. One can read about them in the press. Often we are told that genes are selfish and help themselves rather than the bodies they are housed in. Genes play their role in the nature/nurture debate and in health care. Also, there is an urgent need to conserve the biodiversity around us for future generations that has, of course, to do with genes. There is a big science industry of genome sequencing that is an inventory-taking of all of man's (and other organisms') DNA. Darwin searched a lifetime in vain for the hereditary units, and indeed, genes are indispensable in modern Darwinian evolutionary theory. Early this century, genes were inferred from the Mendelian behavior of traits. The year 1953 marked a breakthrough when Watson and Crick disclosed the double-stranded helical structure of DNA. This suggested an elegant explanation of how genes could replicate themselves from one generation to the next but also serve the purpose of building an individual organism in each generation. Henceforth, the gene came to be viewed as a piece of DNA that coded for a protein or, more generally, a functional or structural product. Genes were seen as inviolable messages passed down the generations (save for occasional mutations) and as the ultimate causal factors lying behind development. Once, these findings were considered evidence for one of the most successful research strategies in the life sciences during the first half of the twentieth century.

Molecular biological discoveries over the last fifty years have made this story vastly more complicated in the details. Somewhat detached from the gene as a public icon, but also unknown to many biologists, these new findings have caused a watershed during the