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Reductionism Redux: Computing the Embryo

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Abstract. This paper argues that the consensus physicalist antireductionism in the philosophy of biology cannot accommodate the research strategy or indeed the recent findings of molecular developmental biology. After describing Wolpert's programmatic claims on its behalf, and recent work by Gehring and others to identify the molecular determinants of development, the paper attempts to identify the relationship between evolutionary and developmental biology by reconciling two apparently conflicting accounts of bio-function – Wright's and Nagel's (as elaborated by Cummins). Finally, the paper seeks a way of defending the two central theses of physicalist antireductionism in the light of the research program of molecular developmental biology, by sharply reducing their metaphysical force.

Key words: molecular development biology, physicalist antireductionism, homeo boxes, explanation, causation, ontology

1. The consensus anti-reductionist position in the philosophy of biology

The consensus anti-reductionist position in the philosophy of biology begins with a close study of the relationship of Classical genetics (Mendelism and its successors), to the molecular biology of the nucleic acids, and their immediate protein products. This study reveals that there are in fact no laws of Mendelian genetics to be reduced to laws of molecular biology, and no distinctive laws in molecular biology to reduce laws of Mendelian genetics, that the kind terms of the two theories cannot be linked in general statements of manageable length that would systematically connect the two bodies of theory; and that nevertheless, biologists continue to accord explanatory power to Mendelian genetics, while accepting that Mendelian genes and their properties are "nothing but" nucleic acids and their properties.

The first three of these observations serve to completely undermine the thesis once held in the philosophy of biology that Mendelian genetics smoothly reduces to molecular genetics in accordance with some revision of the post-positivist account of reduction. The last two observations have been joined together as "physicalist antireductionism" – so called because

it attempts to reconcile physicalism – the thesis that biological systems are nothing but physical systems, with antireductionism – the thesis that the complete truth about biological systems cannot be told in terms of physical science alone. In particular, biologists and philosophers who embrace physicalist antireductionism adopt two theses about the autonomy from molecular biology of non-molecular biology ("functional" biology I shall sometimes call it – functional because it identifies biological structures and systems by their causal roles, usually their adaptationally selected effects):

(1) the principle of autonomous reality: The levels, units, kinds identified in functional biology are real and irreducible because they reflect the existence of objective explanatory generalizations that are autonomous from those of molecular biology.

(2) The principle of explanatory primacy: At least sometimes processes at the functional level provide the best explanation for processes at the molecular level.

We can find these two theses of physicalist antireductionism hard at work in thought of philosophers like Philip Kitcher (1984) and Elliot Sober (1993), and biologists as different as Richard Lewontin (1982) and Ernst Mayr (1982).

The argument for these theses proceeds by example. Mendel's laws tell us that genes come in pairs and that only one of each pair is transmitted to each off-spring. Since it is well-established that Mendel's laws are not reducible to molecular ones, in accordance with principle (1), the Mendelian gene is an autonomous kind. Principle (2) is vindicated because Mendelian segregation is most fully explained by considerations from cellular physiology about the movement of chromosomes at meiosis, and not by providing the endless details of what molecule does what in the biochemical transactions that underlie meiosis. The cellular physiology of meiosis is the "right" level at which Mendelian regularities are most fully explained because explanations that begin at more disaggregated levels, like the macromolecular, would miss connections and similarities, would forego significant generalizations, and would introduce unneeded, perhaps even irrelevant details. If meiosis is like other biological processes it does not result from a small set of molecular processes whose members are individually necessary and jointly sufficient for the assortment and segregation of genes. Moreover, many of the molecular processes implicated in meiosis also underlie other very different functional phenomena. So, appealing to the set of processes that doubtless happens to be necessary and sufficient in the circumstances for any one individual case of meiosis may be inappropriate for the next case. Seeking these sets will blind us to what all or most cases of meiosis share in common. It is what they share

in common – that they are all cases of meiosis – which explains Mendelian assortment and segregation. So the argument for (1) and (2) goes.

But if molecular biology does not reduce Classical genetics, how is it related to this theory? Positive accounts of the relationship are not a part of the antireductionist consensus. Pretty clearly, the nucleic acids, and their protein-products provide the structure for assemblages cell physiologists observe. And they provide the processes that implement the behavior of these cellular assemblages. They make the gene 'concrete' by providing the underlying material that preserves and transmits the information Classical genetics deals with. Molecular information about the location and structure of the genetic material also helps the Classical geneticist understand where Mendel's "laws" go wrong, and what exceptions to these rules of thumb are to be expected. Molecular biology substantiated Classical hypotheses about, for example, "mutation", which appear to be *ad hoc* in the absence of independent structural evidence.[1] Molecular biology also enabled the classical geneticist to apply methods and theories that had been shaped with an eye to observable phenotypes – properties of organisms – to molecularly characterized properties of molecular assemblages. Since at the level of molecular traits the Mendelian regularities are much closer to exceptionlessness, Classical genetics came to be vindicated at the level of the macromolecule – where there seems to be one trait for each gene, even as molecular biology was explaining the weaknesses of Mendel at the level of the organism.

Of course another thing molecular biology did *to*, and not *for* Classical genetics, was gravely to undermine its ontology. Molecular genetics reveals that there is no one single kind of thing that in fact does what Classical genetics tells us (classical) genes do.[2] In this respect of course molecular genetics replaces Mendelian classical genetics. The classical theory retains a place in pedagogy for the same reason Newtonian mechanics does. A glance at physics text books shows that Newton's theory is heuristically useful and seriously misleading. Similarly, molecular biology shows why Classical genetics is a useful instrument, even pedagogically indispensable, but is fundamentally flawed. One reason it is difficult for antireductionists to give a positive account of what molecular biology does for Classical genetics is that the conclusion that classical genetics is merely a heuristic device, undercuts the explanatory autonomy antireductionists wish to accord Classical genetics.[3]

The philosophical consensus reflected in (1) and (2) is not substantiated in significant domains of biology. In particular, developmental biology, one of the subdisciplines which molecular biology made its focus in the two decades after its conquest of genetics, seems clearly to repudiate the down-ward direction of explanation countenanced by principle (2) above. Moreover, in developmental biology at least there are no deep explanatory generalizations

we would miss where we to eschew the ontology of functional biology. For there are no explanatory generalizations at higher levels of organizations. There are only descriptive regularities about "almost invariable" sequences at uniform locations like the "imagal disk" reflecting teleological mysteries of development which can only be explained in molecular terms. At most the non-molecular generalizations set out tasks for developmental explanation, and never provide explanations. Thus, principle (1) is without application in this compartment of biology. Even if biology's functional kinds are perfectly genuine, they lack any very satisfying or deep autonomous explanatory role in developmental biology. What is more, the success of molecular developmental biology's program suggests that, in developmental biology at least, physicalist antireductionism is little different from the emergentist dualism between the living and the non-living that embryology cast off even before it was invaded by molecular methods.

What follows if developmental molecular biology does not substantiate physicalist antireductionism? One possible conclusion to draw is that the relation between molecular biology and Classical genetics is fundamentally different from the molecular biology/developmental biology relation. Perhaps "interfield" relations among subdisciplines in biology are more varied than philosophers have supposed and no interesting general theses in this area are tenable. Alternatively we may decide to re-examine the Classical genetics/molecular biology interface, seeking a way to reconcile it with the relation we uncover in the case of molecular developmental biology. One thing we cannot do is rest complacent with blanket physicalist antireductionism as exemplified in principles (1) and (2).

In the next section I sketch recent work in developmental molecular biology which is sharply at variance with principles (1) and (2). This work suggests that non-molecular generalizations about typical cellular embryology have little explanatory power, even when they have few exceptions. They provide the explananda for this subdiscipline; never the explanans. Section 3 explores the nature of inter-theoretical explanatory relations in this subdiscipline of biology. It sketches a way of dividing the explanatory tasks between evolutionary and developmental biology on the basis of the appropriate range of application for two apparently conflicting approaches to bio-function – those of Nagel (as developed by Cummins) and of Wright. Section 4 offers an argument that vindicates molecular developmental biologists' repudiation of physicalist anti-reductionism, at least for their part of the discipline. It shows that principles (1) and (2) can only be defended against macro-molecular explanations throughout biology by a weakening that deprives them of the ontological strength required to bear the weight of physicalist antireductionism.

2. Diffusible Morphogens: From Dormative Virtue to the Homeobox

In *The Structure of Biological Science* I wrote that

> Nothing is more striking in biology than the apparently goal-directed phenomena of embryology and development . . .
>
> It has long been assumed that descriptions and explanations of goal directed systems were ultimately to be cashed in for nonteleological theoretical explanation at the level of molecular biology. Developmental biologists do not seem at present to be very close to such molecular explanations of cellular development, still less to such explanations of the emergence of whole organs like the chick's wing (Rosenberg, 1984).

A decade later this is a description of developmental biology that has been overtaken by events. One leading developmental biologist broaches the central question of the discipline in terms that suddenly substantiate the most reductionistic of aspirations. Here is how Lewis Wolpert sees the state of play in developmental biology:

> Over the past 20 years, progress in developmental biology has been so dramatic that developmental biologists may be excused for having the view, possibly an illusion, that the basic principles are understood, and that the next 20 years will be devoted to filling in the details. The most significant advances have come from the application of molecular techniques and a greatly improved understanding of cell biology. **So we can begin to ask questions – like whether the egg is computable.**
>
> . . . Will the egg be computable? That is, **given a total description of the fertilized egg – the total DNA sequence and the location of all proteins and RNA – could one predict how the embryo will develop?** This is a formidable task, for it implies that in computing the embryo, it may be necessary to compute the behavior of all the constituent cells. it may however, be feasible if a level of complexity of description of cell behavior can be chosen that is adequate to account for development but that does not require each cell's detailed behavior to be taken into account (Wolpert, 1994).

The significance of Wolpert's tentatively affirmative answer to the question whether the embryo is computable, turns on how we are to understand his use of the term 'computable.' Wolpert must mean something more by 'computable' than the notion of computability of mathematical functions, which makes a given output for a given input a matter of algorithmic, mechanically decidable processes. For so understood the thesis that the embryo is computable from macromolecules alone will not even be controversial among

biologists including anti-reductionist biologists. A mathematical function is computable if a machine can execute it. The system which builds the embryo out of macromolecules is a machine, albeit one cobbled together by natural selection. Accordingly there is a computable function that this machine implements.

To avoid triviality Wolpert's claim must be understood as making restrictions on the form of the algorithm as well as the mapping of molecular input to embryo-output. For an algorithm that adverted to cellular mechanisms not themselves "computable" from the nucleic acids and the proteins that compose the fertilized egg would hardly vindicate the hope to predict the development of the embryo from a description merely of DNA, RNA and proteins.

Thus, emergentists, holists, indeed vitalists, could agree that the embryo is computable, provided they could pack the function that maps molecules into organisms, with their favorite downward causal forces, undisaggregatable functional units, or for that matter vital forces and enteleches.

The requirement that the function rendering the embryo computable not advert indispensably to factors beyond the macromolecule is implied by Wolpert's admission that "it may be necessary to compute the behavior of all the constituent cells" of the embryo. Presumably "Computing" the behavior and properties of cells means predicting this behavior and these properties from a description of the nucleic acids and proteins constituting the fertilized egg. The cell is to be at most a way-station on the path to the developed organism, one algorithmically dependent on macromolecules alone. But, Wolpert's thesis must be understood as claiming that the function which renders the embryo computable will take us from macromolecules to organisms without having to pass through the way-station of a complete description of all the constituent cells, or even any of them. And even if the cellular way-stations are necessary, it may be sufficient to compute only some subset of cellular behavior.

The function Wolpert needs will have to be in a sense "decomposable" as well as computable: complex components of the function – say components describing cellular mechanisms – will have ultimately to be transparently decomposed into simpler components that do not invoke cellular machinery.

There is a further attribution we need to make to Wolpert's hypothesis in order to reflect the significance attached to it by developmental molecular biologists. Its not just that the function is computable, and decomposable, but that it enables us to explain the vast diversity of actual and possible morphologies on the basis of a quite limited stock of elements – maternal proteins, genes, and rules for combining them. Computationalism is overwhelmingly attractive in cognitive science because it enables us to explain the power to

encode and decode an indefinitely large class of signals on the basis of a finite stock of recognizable elements and composition rules. For similar reasons, the computationalist in developmental molecular biology will hold, unless the vast diversity of form is similarly explainable from a tractable base of a relatively small number of regulatory and structural genes (and their protein products) combined by a similarly small number of combination rules, we can surrender all hope of any completeness and generality in the understanding how diversity in development is possible, let alone actual.

It is in this sense then, that the function is decidable, decomposable and relatively simple, that we should understand Wolpert's hypothesis that the embryo is computable.

In order to show what Wolpert's optimism, that the function satisfies these conditions, bodes for contemporary accounts of reductionism in biology, it would be helpful to sketch some of the results that sustain this optimism. In the late sixties Wolpert was already attempting to construct a theory which might guide the search for a purely molecular account of development (Wolpert 1969). The chemical mechanism which Wolpert suggested involved a "diffusible morphogen"[4] – a chemical whose concentration gradient would decline as the distance from its source in the embryo, and would switch on different developmental patterns in different parts of the embryo depending on this concentration and the sensitivity of molecular receptors on cell-surfaces or within them. The theory was advanced as the simplest mechanism to explain certain striking experiments, but there was no independent evidence for the existence of such a substance. At the start, the notion of a "diffusible morphogen" had all the empirical content of Moliere's "Dormative virtue." This is where molecular biology enters the story.

Wolpert's model system is the chick limb, but the story is more simply and dramatically told for the fruit fly, **Drosophila melanogaster**.

The fruit fly embryo begins as a single fertilized egg and within 24 hours emerges as a larva, which then passes through three molts, until it pupates, and emerges nine days later as an adult. The head, thorax, wings, legs, abdomen all develop from segments which are already clearly differentiated in the first few hours after fertilization. Developmental biologists have long been able to trace out these steps, and even to work back to the earliest events in the egg after fertilization, including the striking multiplication-division of nuclei within the single cell, after which each nucleus is enclosed in a cell of its own. By observation of some cells, their individual developmental fates can be mapped. Little in the generalizations describing this process well known to Drosophila biologists is more than description of fairly regular sequences. Little seems explanatory. Indeed, the sequences traditional developmental biology reports express the deep mystery of teleology at its richest.

The developmental molecular biologists task is to "discharge" this teleology (in the way the cognitive psychologist is expected to discharge intentional homunculi). For example, for the developmental molecular biologist it is no explanation of the rapid synchronous division of the nuclei before cellurization to point out that the genetic information they contain **is needed to** direct subsequent differentiation throughout the embryo. This is evolutionary or ultimate biology. Developmental molecular biology is not satisfied with ultimate – adaptational explanations; it seeks proximal ones.

In 1995 the Nobel Prize in Medicine honored the molecular explanation of the process whereby the fruit fly embryo becomes the larva.[5] It is now known that the development of the embryo of the fruit fly is the result of a cascade of Wolpert's diffusible morphogens, and over the last decade or so they have been identified, and the genes that express them characterized. Development of the egg into an embryo and eventually a fly requires initial differentiation between the back (dorsal) and front (ventral) surfaces, and the front (anterior) and back (posterior) of the animal. Both differentiations are the result of a chemical gradient in the concentration of a protein that regulates gene expression by binding to sites of the DNA that turn on and turns off genes which produce still other proteins. Let's consider how anterior/posterior differentiation proceeds.

Prior to fertilization, so-called nurse cell's surrounding the oocyte express the messenger RNA (mRNA) for the protein product of the Bicoid gene, which binds to a molecule on the oocyte's anterior end. Once fertilization occurs, the mRNA is translated into a protein which diffuses from higher concentration in the oocyte close to the nurse cells to lower concentration in the other end. This protein is a long range "signal", which turns on one gene in high concentrations, turns it off at lower concentrations and whose absence turns on still a third gene. The bicoid gene expresses a protein which turns on and/or turns off some twenty five segmentation genes, of which there are three known groups: gap genes, whose products produce the basic (para)segments of the embryo; the so called pair-rule genes – a brace of genes whose differential expression divides (para)segments into further segments, and segment-polarity genes, which orient segments. Most of the segmentation genes are known to code for further regulatory proteins, chemicals that switch on and off still other genes. And these genes are themselves all switched on by different concentrations of the product of the bicoid gene. In turn the gap genes produce proteins that spread out as diffusible morphogens, constituting positional signals switching on and off genes in nearby cells and controlling their development. The next level of spatial differentiation is controlled by the pair rule genes. These two appear to code for proteins that diffuse to transcription sites in the genomes of neighboring cells, switching on other

genes, and so on until the production of regulatory proteins gives way to that of structural proteins in amounts that result in the different cells, tissues, and organs of the fly. As yet much less is known about these structural proteins, but it is already clear that the difference between organisms is largely a result of differences in regulatory gene products and not structural gene products.[6]

The most striking discoveries in developmental molecular biology are those which have identified the so-called Homeotic selector genes, the next level in the cascade of morphogen-producing genes after the segmentation genes. There is in fact fairly startling evidence that these genes produce the most complex of organs in a straight line manner similar to the fixing of the fates of segments of the fruit fly embryo. Walter Gehring has reported experiments in which a previously identified homeotic gene, **eyeless**, when activated in somatic cells all over the body of adult **Drosophila**, results in the growth of complete eyes These eyes including cornea, pseudocone, cone cells, primary, secondary, and tertiary pigment cells, are functional at least to the extent that their photoreceptor cells respond to light (Gehring 1995). Gehring's team has induced eyes in the wings, antennae, halteres, and in all six legs, and they were able to do so in 100% of the flies treated under conditions in which the eyeless promoter gene functions.

Eyeless appears to be a "master-control" gene whose activation by itself is necessary and sufficient (Gehring 1995, p. 1791) to trigger a cascade of genes harbored in all the cells, but normally silent in all but those which give rise to eyes. Presumably, a protein coded by eyeless binds to some set of genes, switching them on and producing a cascade of proteins that ectopically builds an eye on the fly's back, or under its wing, or on its haltere, or even on the end of one of its antenna. And this set of genes is of course to be found in every nucleus in the fruit fly's body. Gehring estimates that the number of genes required for eye-morphogenesis is 2500 (out of approximately 17,000 genes in the Drosophila genome), and that all are under direct or indirect control of **Eyeless**. Moreover, **Eyeless** appears to directly control later stages of eye-morphogenesis. Apparently, the same master-control gene functions repeatedly to switch on later genes, crucial to eye-development, suggesting that evolution has employed the same developmental switch several times in selecting for eye-developing mechanisms.

What is more, **Sey**, the mouse gene homologous to the fruit fly's **Eyeless** gene will produce the same result when inserted into fruit fly somatic cells and switched on. And there is evidence that in the mouse, **Sey** is a master-control gene as well. Proteins encoded by the homologous genes in the two species share 94% sequence identity in the paired domains. Gehring's laboratory has identified counterparts to the fruit fly's **Eyeless** gene, which are implicated in

eye-development across the whole range of species from planaria to squid to humans. **Eyeless** and its homologues may be present in all metazoa.

These results suggest that one of the most complex of organs is built by the switching on of a relatively small number of the same genes, across a wide variety of species, and that the great differences between, say mammalian eyes, and insect eyes, are the result of a relatively small number of regulatory differences in the sequence and quantities in which the same gene products are produced by genes all relatively close together on the chromosome, and that these genes build the eye without the intervention of specialized cellular structures beyond those required for any developmental process. Identifying the other genes in the cascade that produces the entire eye should in principle be a piece of normal science, which will enable the developmental geneticist to "compute" the eye from nucleic acids and proteins alone. For if switching on **Eyeless** can create the eye, surely its creation is "computable" at least in principle. Moreover, as Gehring concludes, "The observation that mammals and insects, which have evolved separately for more than 500 million years, share the same master control gene for eye morphogenesis indicates that the genetic control mechanisms of development are much more universal that anticipated" (Gehring 1995, p. 1792). The more universal, the simpler as well, for a very complex package of instructions to build the eye is open to mutation, and recombination that must reduce its universality as evolution proceeds.

Once **Eyeless** is switched on by regulatory proteins, nothing else beyond the constituent macromolecules is needed, apparently, to "compute" the eye. But, comes the reply, surely, the molecular developmental biologist cannot simply build an eye, still less an animal **in vitro**, by combining the right macromolecules in the right proportions in the right sequence, in the right intervals. Surely the cellular milieu in which these reactions take place is causally indispensable and so the claim to compute the embryo is exaggerated at best.

Molecular developmental biologists may admit that building the eye **in vitro** is beyond the present dreams of the discipline, but in the long run the cellular milieu cannot serve as a "black box", through which macromolecules are transformed into embryo structures. That is the whole point of the "computability" claim. Just as cell-cell signaling is ultimately to be cashed in for a chain of molecular interactions that extend from one stretch of nucleic acids to another across several lipid bi-layers (the cell membranes), all other cellular structures implicated in the machinery of differentiation, will eventually have to be disaggregated into their molecular constituents, if development is fully to be explained.

Here, in developmental molecular biology, there is no room for downward explanation, in which some regularity at the level of cell physiology plays a role in illuminating the molecular processes that subserve development. And this is for two reasons. The first is that such generalizations as obtain at the level of cell physiology are either wholly descriptive histological reports or functional regularities which are developmental biology's job to explain, but never provide its explanations. This is a point to which I return below. The second reason is that cellular structures only come into existence through the molecular processes that precede them. There is in developmental molecular biology therefore no scope for claims about the indispensable role of cellular structures in these molecular processes. The future cannot cause the past.

It is true that maternal cellular structure, as well as that of the oocyte and the sperm-cells, plays a role in our explanation of how the molecular process build the embryo. One might accept that developmental molecular biology takes as given maternal cellular structures, and perhaps even assumes the cellular structure of the egg. It then aims to explain everything else that happens in embryological processes without adverting further to ineliminable cellular physiology. After all, the switching on and off of regulatory and structural genes is not by itself causally sufficient for cellular differentiation, any more than striking a match is causally sufficient for its lighting. Much else is required, and much of this – it will be held – can only be described at least for the moment in terms that advert to cellular structures, ones which have an explanatory role in regulatory and structural molecular pathways. Below I explore the significance of the fact that developmental molecular biologists do not seem to be interested in providing all the conditions – molecular or cellular – that would be causally sufficient for the development of the embryo.

But the idea of according cellular structure a permanent and ineliminable autonomous explanatory role along with molecular processes does not represent the program of molecular developmental biology. Developmental molecular biologists may delimit their area of interest to what happens after oocyte formation. However, it would be a double standard for them to suppose that "computability" might be vindicated for the embryo but not for its mother. Unraveling the molecular details of development has to start somewhere, and the natural starting place is the stage composed of the fertilized egg, and the cellular structure that supports fertilization. On this stage the first players to appear are maternal RNAs and proteins. But because the stage, the reproductive apparatus of the mother is also the result of a developmental process, molecular developmental biology's commitment to "computability" means that this reproductive apparatus is ultimately to be given a molecular account. and indeed, much of the attention of molecular developmental biology is devoted to uncovering the molecular mechanisms of the differentiation of

reproductive cells at the very beginning of embryological development. For these cells are among the most distinctly different and specialized from the very beginning of development.

It remains to be seen of course, whether the program of computing the embryo can be carried out. And there are certainly developmental biologists who have expressed doubts about its ultimate success. However, some of this dissent may rest on a terminological presupposition that divides computationalists from non-computationalists in developmental molecular biology. T.J. Horder has long argued that "a number of diverse but well-known categories of data call into question the adequacy of a purely genetic view of form." He concludes:

> The available evidence (incomplete and selective though it may be) is incompatible with any simple conception of a one-to-one relationship between morphology and the normal genetic programming of morphology whereby each morphological locus derives its unique features through independent control by appropriate specific genes. The complexity of the evidence is such that there is no obvious way to deduce how or in what degree genes alone or in combination define and limit morphology. Since detection of genetic factors depends on morphological outcomes, the situation is, in the absence of independent information about intervening mechanisms, essentially circular (Horder, 1989, p. 324).

One matter worth immediately addressing is the threat of circularity under which molecular developmental biology may lie. Taking it too seriously would undermine the search for all underlying unobservable processes which can initially only be individuated in terms of the phenomena the factors are invoked to explain. It is only when no means are forthcoming independent of the phenomena they are invoked to explain, that inferences to explanatory factors should be suspect as circular. This is the Dormative virtue problem that Voltaire so effectively identified. It might have been a serious criticism of Wolpert's diffusible morphogens: had nothing ever been shown for them – no molecules identified, no genes to express them, no assay to measure their diffusion, then the initial circularity would have turned out to be vicious. This conspicuously was not the case.

More important, Horder's doubts may in fact belie a fundamental agreement with advocates of computability. For the non-genetic factors Horder wishes to invoke as integrally involved along with the genes in determining form are apparently themselves molecular, or potentially so.

Horder writes:

> A simplistic dichotomy between "genetic" and "environmental" factors neglects the internal environment of the organism. This is not normally amenable to manipulation. . . . The possibility therefore remains open that

> "internal" nongenetic factors may represent an unsuspectedly large and systematic contribution to the determination of form (p. 323).
>
> . . . The more we discover in molecular terms about genetic systems . . . , the clearer it becomes why "nongenetic" considerations cannot be ignored. In each cell, differentiation depends on the selective expression of large numbers of structural genes. It is the pattern of selection of expression in different cells that defines an organism as well as the character of the structural genes themselves. The selection process can only be mediated across dispersed genetic elements by selective molecules within a cells repertoire other than DNA. . . .
>
> The fundamental facts of cell biology make it inevitable that many factors remote from DNA are necessary for, and set limits upon, the ways in which DNA can influence morphology (Horder 1989, pp. 324, 325).

There is nothing in these passages with which a computationalist about the embryo should disagree. To begin with, all recognize that genes by themselves code for no phenotype without an environment to interact with, and to select from among their gene-products. As the debate about genic selection revealed, the environment of the gene begins with the molecular milieu of the nucleus. It is equally clear that the computability thesis is not committed to the notion that nucleic acids are enough to determine the embryo. It invokes other macromolecules, at the start maternal proteins and RNA, whose products kick off development. Moreover the diffusible morphogen is just the sort of intercellular messenger Horder requires to select developmental fates for cells that produce morphogenic patters.

So, where does the dispute among developmental biologists lie? It lies, I think, in divergent hunches about the degree of complexity of the "function" that takes molecular inputs into embryological outputs, and the similarity of the "function" across widely divergent species. Wolpert and other reductionists hold that the absolute number of genes – regulatory and structural – that determine morphology is relatively small, and that the differences among organism are due to relatively simple differences in the order in which regulatory genes are switched on and off.

As noted in section 2 "computability" in developmental molecular biology turns out to be a variant of "computationalism" in cognitive psychology: The thesis that a capacity to produce an indefinite variety of forms from a finite stock of units – the genes – is only explicable if the stock of units can be combined in accordance with a syntax – rules about switching on and off given in the case of development by natural selection – to produce the variety we know is possible.

Those who dissent from "computationalism" in fact have no stake in the denial that there is some sort of function from molecules to embryos, and

physicalists among them will even grant that the function be in principle decomposable in the sense advanced in section 2. The dispute is whether the function is at the same time simple enough actually to be formulated and powerful enough to explain the morphological diversity nature manifests. Consider for example, Gehring's claim that "only" 2500 genes may be involved in the most complicated of systems, the eye. If these 2500 genes interact in accordance with 2 to the 2500 power different "rules" for the construction of gene-products, then the function from molecule to eye will not be computable for any practical or even explanatory purpose.[7] Whether the computable function is like this will clearly be an empirical dispute about contingent matters of fact. But it is one with implications for the philosophy of biology and on which philosophical arguments may bear.

3. Reducing functions without reduction-functions

Reductionism in developmental molecular biology has some modest morals for debates in the philosophy of biology, and at least one quite unmodest lesson. The principle explanatory task of molecular developmental biology is to discharge the teleology of functional developmental biology. This is not news of course. For the better part of a century now, reductionists have held that cashing in teleological for nonteleological processes is an important part of the agenda of physicalist science. What is news however, is that the program has begun to be successfully articulated by molecular developmental biology. The promissory notes are being honored in macromolecular **specie**. But for our purposes what is significant is that because it aims to cash in physicalism's promissory notes, developmental molecular biology will not countenance down-ward causation in the spirit of principle (2). Nor does it advert to explanatory generalizations that make autonomous the kind terms of functional biology, as principle (1) proclaims.

Starting with a generalization like

(f) the function of the fifth segment in the **Drosophila** is to produce the wing.

molecular developmental genetics provides a nonfunctional account that explains claims like (f). These explanation in developmental molecular biology nicely substantiate the Cummins/Nagel theory of functional explanation (Nagel, 1961, Cummins, 1975) I describe it as the Cummins-Nagel theory, because unbeknownst to almost every one, including Cummins (who thought he was refuting Nagel), his account of function works because it relies on Nagel's directively- organized system approach, as I shall illustrate by application to an example.

Explanations in molecular developmental biology proceed by showing how functional processes are implemented by macromolecular ones that operate biochemically. It cashes in functional attributions for non-functional attributions in accordance with Cummins' schema (Cummins 1975 in Sober 1994, p. 64).

(3) **x** functions as a **F** in **s** (or the function of **x** in **s** is to **F**) relative to an analytical account **A** of **s**'s capacities to **G** just in case **x** is capable of Fing in **s** and **A** accounts for **s**'s capacity to **G** by appealing to the capacity of **x** to **F** in s.

Instantiated in terms of the function of the product of the bicoid gene, (3) reads

(3′) The bicoid gene-product functions as a morphogen in **Drosophila melanogaster** embryo (or the function of bicoid gene-product in **Drosophila** embryo is to act as a morphogen) relative to **A** – an analytical account of the **Drosophila'**s embryo's capacities to segment, just in case the bicoid gene-product is capable of being a morphogen in the **Drosophila** embryo and **A** accounts for the **Drosophila**'s embryo's capacity to segment by appealing to the capacity of bicoid gene-product to act as a morphogen in Drosophila embryo.

The pay-off of this account is to be found in how it "discharges" the teleological attribution to the bicoid gene-product by analyzing capacities into biochemical regularities at the macromolecular level. And this is where Nagel's directively organized system approach enters, as I will show shortly.

Following Cummins, we explain functions by capacities, which are already well understood: capacities are sets of dispositions. The capacity of the **Drosophila** embryo to segment is explained by appeal to a number of other capacities of components of the **Drosophila** embryo – the regulatory genes and their products "such that programmed manifestation of the components dispositions results in or amounts to a manifestation of the embryo's capacity" (Cummins 1975, in Sober 1994, p. 63). Basic capacities or dispositions – like the disposition to bind preferentially to a certain DNA sequence – are explained by "instantiation": showing how the disposition is realized in the things which have it.

But wait, what is the role of the expression "programmed manifestation" in this analysis? By programmed manifestation, Cummins tell us, he means "organized in a way that could be specified in a program or a flow chart" Elsewhere the appeal to the notion of a program has either trivialized accounts of

teleology or function (see for example, Mayr 1982, p. 48) or wrongly required us to count non-functional processes as functional ones. When "program" is understood to itself describe a process or a state requiring intentional intervention or judgement, appeals to it are question-begging. When programs are treated as mechanically decidable procedures, a variety of purely physical processes appear to qualify as teleological. For example, changes in the values of pressure, temperature and volume in a gas can be expressed as a program with the function of maintaining their relation to the gas-constant **r**. But such a program is clearly no basis for identifying a gas as a functional or teleological system.

Nagel dealt with both these potential counterexamples by requiring that a goal directed system consist in a set of subsystems, whose non-teleological behavior implements the teleology of the whole system, just as Cummins does. However, Nagel gives an account of the nature of the implementation which avoids the counterexamples. A directively organized system is one composed of subsystems which are themselves (ultimately) not directively organized but interact in such a way that a state of the whole system is maintained – either the state of tracking some goal through environmental variations, or the goal state itself – by the temporally asymmetric feed forward and/or feed back effects of changes in the values of causal variables of each of the subsystems on one another. This temporal asymmetry requirement precludes counterexamples like those which turn ideal gases into teleological systems.[8] The flow chart of the program under which the subsystems interact is mechanically decidable, and engenders no regress to further teleology.

One issue on which molecular developmental biology's discharge of functions should cast light is the vexed relationship between developmental and evolutionary biology. In particular, it is for evolutionary biology to answer the question (to the extent it is answerable) of how and why individual non-teleological processes came to be packaged together in such a way as to constitute jointly directively organized systems, and how these directively organized systems should themselves be packaged into larger such systems. This individuation of packages and their components via their "bio-function" is underwritten by the theory of natural selection in the ways Larry Wright uncovered in his teleological or etiological analysis of functional attributions (Wright 1976). It is for evolutionary biology to individuate the entities and processes which developmental biology explains; it does so by an implicit or explicit appeal to their adaptational etiologies – their bio-functions. It is for developmental biology to explain how they accomplish these bio-functions by appeal to their capacities. Thus developmental and evolutionary subdisciplines are distinguished in part by the differing notions of function which they employ, and related by the fact that developmental biology's functional

attributions are dependant on evolutionary biology's functional attributions. If the latter are wrong, then the former are ungrounded. But etiological individuation simply sets the problems for developmental biology, it is no part of their ultimate solution.

For the moment we have only the sketchiest idea of how for example the Eukaryotic cell might have evolved from the packaging together of the mitochondria and bacteria. It may be possible to recover the sequence of events which packaged the various RNAs, and DNA into the directively organized system which produces proteins. But we will be able to do this only if there are but a small number of possible routes from the existence of individual nucleic acid bases to macromolecules.

By providing an evolutionary etiology that explains the persistence and multiplication of these packages of non-teleological subsystems, evolutionary theory finds its way into every compartment of developmental biology, even the most fundamental level.[9] But molecular developmental biology takes these packages as given, and uncovers the molecular details of how they operate. These details both highlight evolutionary lineages in the homologies they reveal, and more importantly, enable the evolutionary molecular biologist to provide an account of how selection actually brings molecular subsystems into directively organized wholes. Above the level of the macromolecule, functionally characterized structures are supervenient on a large disjunction of alternative actual and possible mechanisms. Since each of these different mechanisms is the result of a different causal process, there is no unique path of adaptations that resulted in a functionally characterized cell, tissue, organ, structure or behavior, nor probably a managable disjunction of them. And what these path-ways all do share in common – their selectively similar effects – is just what is to be explained! At most evolutionary biologists can identify design solutions and tell "just so" stories about how they might have emerged. At the level of the macromolecule the chemical and physical constraints may be narrow enough so that a manageably small number of causal routes from mere matter in motion to a macromolecular subsystem can sometimes be uncovered, by for example laboratory experiment.

This relatively clean division of responsibilities between developmental and evolutionary biology makes it particularly clear why selection operates so overwhelmingly at the earliest stages of development of the fertilized egg. Packages that will not work together are wiped out early, and adaptations at any level above the macromolecule have no chance of even appearing unless they are the result of macromolecular combinations selected for their own relatively immediate adaptive advantage.

Another of the modest lessons of molecular developmental biology is the light it sheds on the unit of reduction. Reductionism-for-and-against is

traditionally viewed as a debate about statements, theories, laws, models, or other linguistic items that reflect generalizations. But reductionism in developmental biology is not a thesis about laws, or surrogates for them like practices, models, or other conceptual items. It is about specimens, or perhaps even about particular organisms. Having traced the developmental pathway from maternal messenger RNA all the way to the adult fly in one batch of fertilized eggs, the developmental molecular biologist is satisfied to find homologies and similarities in the development of other organisms, and is even willing to accommodate different pathways in the same or similar organisms with slightly different genomes or different environmental milieu. The vindication of molecular biology is more a matter of proving an existence proof for one or a small number of purely molecular pathways to a biological system. It is not a matter of tracing all or most or even many of the different pathways that eventuate in the same outcome.

Why is an existence proof that at most establishes just one complete story from molecule to animal for one sample of embryos sufficient in the developmental molecular biologists' program to sustain the "computability" thesis? Part of the answer must be that developmental molecular biologists understand that the diversity of organisms that evolution fosters is so great that empirical generalizations about development which combine strength and simplicity are unlikely. Developmental molecular biologists seem to recognize that theirs is not a science that aims at laws, but the application of other nomothetic sciences to tracing out singular causal chains. If there are generalizations in this discipline, they are either of the sort developmental biology aims to explain or else they are about laboratory techniques, methodologies, assays, tricks that will turn genes on or off, produce mutations, move genetic elements from one place to another.

The existence proofs molecular developmental biology seeks for its computable functions establish a general possibility on the basis of a small number of actualities: showing how a complex biological system does emerge from a purely chemical process in one case, establishes the possibility that it can so emerge in many others. At this stage it is more important to go on to another model system – a more complicated one – to establish yet another general possibility. Going back and tracing out more actual routes is an exercise left to later in the research program.

The apparently attainable object of the subdiscipline is not a function that will map every molecular input to an embryological output. One mapping at most establishes the existence of a class of functions. But why should uncovering one causal chain from maternal RNA and regulatory proteins all the way to the developed embryo suffice to establish the existence of the general function? And why are developmental molecular biologists satisfied with

establishing merely the existence of the function, not its full specification? These are important questions. But the very fact that they present themselves suggests that there is something quite different going on in molecular developmental biology than what philosophers of biology have supposed happens when a more fundamental theory reduces, "extends", or otherwise unifies a less fundamental one.

Reductionism and its surrogates are viewed as driven by an imperative to unify. As Kitcher writes, "the unification of our account of the world is a cognitive desideratum for us, a desideratum that we place ahead of finding the literal truth on the many occasions when we idealize the phenomena. The causal structure of the world, the division of things into kinds, the objective dependencies among phenomena are all generated from our efforts at organization."[10] But the reduction molecular developmental biology aims at places literal truth ahead of idealized models that unify, and indeed substitutes for unified accounts of apparantly homogeneous phenomena disaggregation into distinct pathways that give the causal structure of the world. In this department of biology "the objective dependencies" are not the result of our efforts at organization, they are the results of our search for the literal truth.

Why this is so is an important question. But there is a prior one that awaits the defender of reductionism in developmental molecular biology. If physicalist antireductionism is correct, there may be some developmental processes for which the function Wolpert seeks does not even exist. In that case, molecular developmental biology will have to curtail its reductionist pretensions. For all Wolpert knows, there may turn out to be obstacles to "computability" in the form of non-molecular embryological processes that honor principle (1) and (2).

4. Developmental molecular biology's exemption from physicalist antireductionism

Whether the embryo is computable, whether there is a function mapping every biological outcome to a unique set of molecular inputs, is a contingent matter. This makes reductionism in developmental molecular biology an empirical theory, and not an a priori doctrine. And if physicalist antireductionism holds sway elsewhere in biology, who is to say it will not obstruct the reductionistic aspirations of developmental molecular biology.

What developmental molecular biology needs is an exemption from the writ of physicalist antireductionism. It needs an argument against principle (1) – the levels, units, kinds identified in functional biology are autonomous and irreducible, just because they figure in explanatory generalizations we

would miss if we did not adopt the language of these functional kinds; contra principle (2) it needs to show that in development at least processes at the biological level never provide the best explanation for processes at the molecular level.

These two principles entail a commitment to "downward causation", to the thesis that functional states of biological systems cause molecular processes, and do not do so simply owing to their molecular composition and properties. Now, this is a thesis that some antireductionists willingly embrace. But downward causation is a commitment intolerable to developmental molecular biology, or at least to Wolpert's program. If molecular developmental biology's commitment to physicalism is inimical to the very possibility of downward causation, then it will be exempt from the writ of physicalist antireductionism.

It is relatively easy to see why principles (1) and (2) require downward causation. Principle (1) is grounded on the notion that to be real, and not just artificial, a kind or category's instances must have distinct causal powers, ones reported in causal laws we would miss if we did not commit ourselves to their reality. But the causal powers of biological kinds must be distinct from the causal powers of assemblages of molecular properties. Otherwise, the distinctness of biological kinds from molecular ones would be threatened. For their causal powers would not be distinct from those of molecular kinds.

Principle (2) tells us that the instantiation of these non-molecular real properties of biological systems sometimes provides the best, most complete explanation of the instantiation of molecular properties. Now, since these explanations are objectively the best ones, and not just the most heuristically tractable for creatures of our cognitive and computational powers, their explanatory excellence presumably reflects the accuracy of their reports of causal processes. Consequently, in these cases the direction of best explanation will follow the direction of causation: i.e. if explanation is downward, then this must be owing to the downward direction of causation from the level of the functional to the level of the molecular.

Keep in mind that the downward causation required here is not one that can be cashed in for more fundamental "upward causation" from molecular kinds to biological ones. It cannot be the case that biological processes cause molecular ones just in virtue of the biological processes **being** themselves molecular. For this would turn downward causation into a mere way-station for more upward or sideways causation.

Now, physicalism holds that all biological properties are realized by combinations – sometimes vastly complex combinations – of molecular properties: Whenever an organism or system instantiates a biological property *F*, it has some molecular property *M* such that *M* realizes *F* in systems or organ-

isms that have *F*. Of course, the agenda of developmental molecular biology is explicitly to uncover how molecular properties realize biological ones. The notion that functionally described processes are composed of nothing but macromolecular processes is a core commitment all physicalist biologists share.

To see the problem downward causation makes for physicalism, suppose that a given functional property, say segmentation by a parasegment in **Drosophila**, has a macromolecular effect, say blocking the diffusion of a morphogen. If segmentation supervenes on some complex conjunctive/disjunctive macromolecular property, *Mx*, then every instance of segmentation, is realized by some instance of the molecular property, *Mx*. Downward causation from the biological to the molecular requires, for example that sometimes cellular action blocks chemical diffusion of the morphogenic molecule. But of course, segmentation is implemented molecularly by the parasegment, because the parasegment instantiates the (very complex conjunctive/disjunctive) macromolecular property *M*. If the complex macromolecular property *M* is how the parasegment implements segmentation, then the question arises why it is that the parasegment blocks the diffusion of the molecule by segmenting, and not the macromolecules that implement the segmenting which do the molecular blocking job? Surely this is not a case of overdetermination by independent molecular and cellular processes. We need to block the claim that the macromolecular property *M* does the job, just because we attributed the cause of morphogen-blocking to cell-segmentation – this is its distinctive causal power. We cannot also attribute this blocking power to segmentation's molecular implementation without depriving segmentation of the distinctive causal role which guarantees its autonomy, indispensiblity and irreducibility. But this means, contra physicalism, that the segmentation has distinct non-physical causal powers uncovered in functional biology.

Physicalism can be reconciled with downward causation, but only at a cost that is probably too heavy for developmental molecular biology and certainly prohibitive for most physicalists. The problem is to combine two claims: the mereological – whole/part, "nothing but" dependence of concrete biological systems on their constituent macro-molecular implementations must be combined with the claim that the biological systems have **distinctive** causal powers – powers with macromolecular effects different from the effects of the macromolecular assemblages that implement them. Remember, without distinctive causal properties, there is no basis to accord functional states, entities and processes reality autonomous from molecular phenomena. Principles (1) and (2) derive autonomy from causal/explanatory role.

The physicalist antireductionist needs to block the shift of causal powers from the biological down to its macromoleular implementation. This requires two controversial "moves" in the philosophy of science.

First we need to embrace the view that causation is a relation among states, events and processes that is conceptually dependent on explanation. That is, the notion of explanation is more basic than the notion of causation, and the latter can only be understood on the basis of an understanding of the former. This is hard to swallow because it makes a fact apparently about the world – causation, depend on a fact about explanations we request and provide. But it is a view that at least some physicalist antireductionists embrace (see Kitcher 1993, p. 172).

To the thesis that explanation is conceptually prior to causation we must add another controversial claim about explanation: that it is heavily "pragmatic": Explanation is pragmatic roughly when it is viewed not as a relation just between propositions, but as a relation between questions, and answers offered in a context of an interlocutor's beliefs. These beliefs reflect presuppositions of interlocutors which together determine the correctness or goodness or explanatory worth of various answers.

Here is how pragmatism about explanation combined with its conceptual priority to causation can save physicalist antireductionism. Suppose that some biological system *a*, has some functional property *F* and *a*'s having the functional property *F* is implemented by *a* having some complex molecular property *M*.

So *a* has *F* because *a* is *M*, and the way *F* is realized in nature is through the molecular mechanism *M*.

Now, how can

a has *F*

explain

a has *G* (where *G* is some effect of being an *F*)

without

a has *M*

(also) explaining why

a has *G*?

After all, *a*'s being an *F* is "nothing but" *a*'s being an *M*.

The only way this can happen is when "explains" is a non-extensional relation not just between matters of fact, but between them and cognitive agents with varying beliefs. For someone who doesn't know that *a*'s *F*ing is realized by *a*'s *M*ing, the substitution of *M* for *F* in the explanation won't

work. If we don't know about *M*, or understand how it does *F*, then *a*'s having *M* doesn't explain to us what *a*'s having *F* explains to us.

This means that when explanation is treated as "subjective", "pragmatic", its direction can move downward from the functional to the molecular. And if causation is just explanation (or depends on it), it too can move "downward" from the biological to the molecular, following the direction of explanation.

For cognitive agents like us many of the complex macromolecular properties on which the biological ones supervene may be too complex to uncover, to state, to employ in real time explanation and/or prediction. Under these circumstances, the biological property may provide the best, or the only explanation (for us) of the phenomenon in question. On this view down-ward explanation of the sort principle (2) envisions may be possible: it may turn out that sometimes the instantiation of biological properties autonomously and indispensably explains macromolecular processes, without the functional property's instantiation being explainable (to us) by appeal to the instantiation of macromolecular properties that we are intelligent enough to uncover and use in real time.

Putting the pieces together we can now construct an account of downward causation compatible with physicalism: explanation is sometimes downward because there are contexts in which correct molecular answers to explanation-seeking questions are non-explanatory (to us). Causation will be downward in these cases if it follows the direction of explanation.

However, this reconciliation of physicalism with downward causation will not suffice for the antireductionist component of physicalist antireductionism. For antireductionism requires we interpret principles (1) and (2) on either a non-pragmatic notion of explanation, or on a conception of causation as prior to explanation.

Principle (1) holds that there are generalizations which are explanatory "objectively", not merely relative to some interlocutor's beliefs. Only such non-pragmatic "objective" explanatory power will underwrite ontological inferences to existence of functional kinds as irreducible. Principle (2) invokes a notion of "best explanation", either context free or relative to some privileged context, say, that which obtains among interlocutors with the most complete and accurate beliefs about causal processes. And this notion of "best explanation" will be circular if its presuppositions about these causal processes are explanation-dependent. For purposes of a debate between reductionists and antireductionists about principles (1) and (2) a pragmatic approach to explanation and the assimilation of causation to explanation are not merely unavailing. They are likely to undermine both principles (1) and (2) by cutting the connection between explanatory power and metaphysical commitment to what really exists independent of our interests. If biological properties are

real it is because they are, like electrons, indispensable to the most well established theory under some context-free standard, or because there is good non-explanatory, observational evidence for their existence, not because they satisfy the explanatory itch of cognitive agents of our sort.

Let us accept for purposes of argument that explanation is heavily pragmatic. What no physicalist can accept is that explanation so viewed is conceptually prior to causation – i.e. that causation can be defined in whole or in part by appeal to explanation. That way lies Kantian idealism at best and social constructivism at worst. By itself however, the (pragmatically) explanatory indispensiblity of biological kinds will not justify their biological reality or their autonomy from the macromolecular. And if best explanations are ones which report objective causal sequences most completely, then in the end functional biology may turn out to be a way-station in the direction of a thoroughly reduced and perhaps even computational molecular biology.

5. Conclusion

That physicalist antireductionism is the common wisdom of modern philosophy of biology might surprise the non-cognoscenti. If I am right, it should not be surprising to the cognoscenti that at least one component of modern biology does not support this consensus position. And this component – molecular developmental biology – bids fair to be at least as fruitful a source of insights into the nature of biological processes over the next few decades as molecular genetics has been in the past few. Furthermore, the commitment to physicalism it shares with the rest of biology and on which molecular developmental biology relies most directly, undermines the common wisdom of physicalist antireductionism, unless interpreted as a thesis about biologists – their interests and limits – as opposed a thesis about the ontology and metaphysics of the biological realm.

Notes

[1] As Kitcher notes, "Classical genetics makes certain presuppositions – that genes replicate, that some mutations are viable, which apparently seem impossible, given premises that classical geneticists accepted. Molecular genetics should how these presuppositions could in fact be true, consistent with the premises classical geneticists accepted." See Kitcher (1984).

[2] As Kitcher says, molecular biology provides "a specification of the entities that belong to the extensions of predicates in the language of the earlier theory [Classical genetics], with the result that the ways in which the referents of these predicates are fixed are altered in accordance with new specifications." See Kitcher (1984) p. 364. Here Kitcher is assimilating the way in which regularities about molecular phenotypes successfully instantiate Classical generalizations about phenotypes which are disconfirmed regularly at the level of gross morphological

features. By substantiating these Classical generalizations at the level of biochemistry, while falsifying them at the level of the cell and above, molecular biology drastically shifts causal roles away from the classical gene and towards so many different molecules as to extirpate the entire gene concept.

[3] For a broader discussion of the relations between classical and molecular genetics, and their implications for intertheoretical relations generally, see Rosenberg, 1994, Shaffner, 1993, and Waters, 1990.

[4] The idea originated with Morgan, 5 (1897): 582.

[5] This work was carried on by Edward Lewis of Cal Tech, Eric Wieschaus of Princeton, and Christiane Nuesslein-Volaed of the Max Planck Institute.

[6] A highly accessable account of the last decade and more's discoveries in the embryology of the fruitfly are recounted in Lawrence (1992).

[7] Here I am indebted here to Peter H. Schwartz.

[8] Note that changes in the values of pressure, temperature and volume of a gas fix one another's values instantaneously.

[9] This should be no surprise, cf. the difference between DNA and RNA. The former differs chemically from the later only because of the differences in the functions they perform. DNA stores information with high fidelity, RNA transmits information with low cost. This explains why RNA was selected for containing uracil and DNA selected for continaing thymine. See Rosenberg, 1984, chapter five.

[10] Kitcher (1993), p. 172.

References

Cummins, R.: 1975, 'Functional Analysis', *Journal of Philosophy* **72**, 741–765, reprinted in E. Sober (ed.), *Conceptual Issues in Evolutionary Biology*, second edition, MIT Press, Cambridge, 1993.

Gehring, W., Halder, G., Callaerts, P.: 1995, 'Induction of Ectopic Eyes by Targeted Expression of the **Eyeless** Gene in Drosophila', *Science 267*, 1788–1792.

Horder, T.J.: 1989, 'Syllabus for an Embryological Synthesis', in D.B. Wake and G. Roth (eds), *Complex Organizational Functions: Integration and Evolution in Vertibrates*, John Wiley, New York.

Kitcher, P.: 1984, '1953 and all that: A Tale of Two Sciences', *Philosophical Review* **93**, 335–373.

Kitcher, P., Sterelny, K., Waters, K.: 1990, 'The Illusory Richness of Sober's Monism', *Journal of Philosophy* **87**, 158–161.

Kitcher, P.: 1993, *The Advancement of Science*, Oxford University Press, Oxford.

Larence, P.: 1992, *The Making of a Fly: The Genetics of Animal Design*, Blackwell Scientific Publishers.

Lewontin, R. and Sober, E.: 1982, 'Artifact, Cause and Genic Selection', *Philosophy of Science* **47**, 157–180.

Mayr, E.: 1982, *The Growth of Biological Thought*, Belnap Press, MIT Press, Cambridge, 1994.

Nagel, E,: 1977, 'Teleology Revisited', *Journal of Philosophy* **74**, 261–301.

Nagel, E.: 1961, *The Structure of Science*, Harcourt Brace, New York, reprinted Hackett, Indianapolis, 1986.

Rosenberg, A.: 1984, *The Structure of Biological Science*, Cambridge University Press, Cambridge.

Rosenberg, A.: 1994, *Instrumental Biology or the Disunity of Science*, University of Chicago Press, Chicago.

Schaffner, K.: 1993, *Discovery and Explanation in Biology and Medicine*, University of Chicago Press, Chicago.
Sober, E.: 1993, *The Philosophy of Biology*, Westview, Boulder.
Morgan, T.H.: 1897, *Wilhelm Roux Archives* **5**, 582.
Waters, K.: 1990: 'Why the Antireductionist Consensus Wont Survive: The Case of Classical Mendelain Genetics', *PSA 1990*, The philosophy of Science Association, East Lansing.
Wolpert, L.: 1969, 'Positional Information and the Spatial Pattern of Cellular Formation', *Journal of Theoretical Biology* **25**, 1–47.
Wolpert, L.: 1994, 'Do we understand development?', *Science* **266**, 571–572.
Wright, L.: 1976, *Teleological Explanation*, University of California Press, Berkeley.